Andrea M. Trescot
Editor

Peripheral Nerve Entrapments

Clinical Diagnosis and Management

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This book is dedicated to my family, friends, and students, without whom I would never have had the strength to finish this work, and to my patients, who keep me motivated to find answers for their pain.

Andrea M. Trescot

Foreword

The contribution of Dr. Andrea M. Trescot and the multiple authors in the entrapment neuropathy field is very significant. These are topics that often are not covered at conferences, as the time limitations and program limitations exclude what appear to be minor areas. Ultimately, patients suffering from entrapment neuropathies are treated with inappropriate, alternative, excessive technological implants and/or heavy medications such as opioids and others. All these occur because the explanation of the problem, if you do not know it, does not exist.

There are multiple very significant and successful treatment recommendations covered in this book, including, for example, occipital neuralgia. Releasing the compression in the suboccipital compartment by the suboccipital decompression technique can very successfully treat migraine headache-type problems. Previously published and presently released results show that injecting the multiple nerves involved in the production of headache usually gives 2 weeks pain relief whereas the suboccipital decompression gives 24 weeks pain relief. Studies include a multitude of accurate and inaccurate diagnoses, but certain patients can experience years without recurrence of the occipital pain after a single suboccipital decompression from the inferior oblique muscle between C1 and C2, which is a considerable distance away from the superficial injections. In other words, if you know the mechanism of causation, then you are more likely to get long-term success.

The explanation for lower quadrant abdominal hypersensitivity is the entrapment of the iliohypogastric and ilioinguinal nerves going through three muscle layers (the external oblique, the internal oblique, and the transversus abdominis), before becoming cutaneous below the umbilicus. This condition often occurs in the second trimester of pregnancy, especially in muscular young mothers, where the growth of the baby stretches the abdominal musculature. This pain raises significant differential diagnostic problems, such as a misdiagnosis of acute appendicitis where surgical intervention may be hazardous and unnecessary at the same time. The treatment is simple, injecting along the iliac crest, popping through the external oblique aponeurosis and there is usually immediate pain relief; however, it may not last long enough. One can repeat the procedure or, alternatively, choose a longer-lasting solution such as cryoneurolysis in the same location, locating the nerve with the nerve stimulator at the tip of the cryo probe and freezing the nerve. The pain relief can be from 2 days to 1,000 days. Here, you see the diagnosis being made and appropriate treatment being carried out by short- and long-lasting therapeutic methods such as cryoneurolysis.

Another form of entrapment neuropathy that is often missed is saphenous nerve entrapment. The physician has to remember that the saphenous nerve comes off the femoral nerve and not only carries a sensory nerve to the inside ankle (medial malleolus) and the knee, but also it has significant sympathetic fibers. A saphenous block in Hunter's Canal, 4 in. above the knee, with a safe blunted Stealth™ needle, can relieve knee pain. This often helps regain mobility of knee joints, reduce swelling and discoloration of the foot and lower leg following long duration splinting and immobilization of ankle and knee joint. Here, the entrapment neuropathy comes from the extended disuse of the knee or ankle joint following immobilization after surgery or fractures. The resolution of the swelling comes from the lysis of the autonomic sympathetic fibers.

After upper extremity shoulder surgery and fractures, because of immobilization, one may see entrapment of the brachial plexus between the anterior and middle scalene muscles. Simple interscalene injection, however, can be performed with an occluded tip needle so that intraneural or intra-arterial injection should be less likely; even ultrasound guidance does not assure safety if open-ended sharp needles are used. There have been a considerable number of brachiopathies and cord injuries resulting in significant medical legal costs from the use of sharp needle injections in vascular regions where nerve and arteries travel together at the same injection site.

Using needles of too small a gauge can also be dangerous. Pneumothorax is one of the most common medical legal consequences of injections around the lungs; with small gauge needles, the needle can penetrating the lung multiple times, since small gauge needles have a mind of their own and do not go where the doctors wish they would go. The medical legal cost for a pneumothorax can be from zero to multiple six figures. In medicine, the principle must always be: *primum non nocere*, first do no harm.

Nerve entrapments can be caused by scarring anywhere along the path of the nerve, and the "lysis of adhesions" concept extends to scar tissues, where tendons may be limited by scarring and bleeding. Scarring also causes severe neuropathy in the spinal canal. Solving one source of neuropathic pain does not necessarily solve all pains and the doctor needs to remain vigilant by not just asking the question, "How is your pain?" but also examining the patient. It is surprising how many times the patient does not know where the pain is coming from.

I believe Dr. Trescot's recognition for the need to collect the subject matter in this book and subsequent assemblage of its contents is truly remarkable. It has been my pleasure to know and respect the work of Dr. Trescot; my reverence for her work originates in her realization of the need to expand our horizon and treat the patients appropriately rather than excessively. I feel the examples that I listed served to emphasize that not only does one need to recognize problems, but also solve problems with long-term results in mind. Alternatively, the short-term pain pill that may appear to work for hours will become a long-term issue by leading to addiction, chronic pain, and loss of work. All of this starts with the premise, "If you don't know it, it doesn't exist," which is not in the best interest of the patient or doctor. In conclusion, Dr. Trescot deserves all the accolades for exercising a tremendous effort to bring such valuable and extensive information to all of us.

Dallas, TX, USA Gabor Racz, MD, FIPP

Preface

Peripheral nerve entrapments are a commonly overlooked cause of painful conditions, resulting in pain literally from the head to the toe. Even the astute clinician may not be aware of these syndromes, and entrapment of these often small nerves can lead to debilitating pain, mimicking "migraines," cardiac disease, intra-abdominal pathology, "endometriosis," complex regional pain syndrome (CRPS), and "plantar fasciitis." Knowledge of these entrapments can prevent expensive ineffective testing and treatment and can ideally avoid unnecessary pain and suffering.

This book is a culmination of many years of my personal clinical observations as well as collaboration between many providers. Over the years, when I would lecture on peripheral nerve entrapments, I would be met with blank stares, or worse, derision. However, this lack of knowledge is slowly changing. Fifteen years ago, when I would ask the audience to raise their hand if they had ever even heard of the cluneal nerve, perhaps two or three hands would go up. Now, with the same question, sometimes a majority of the room will raise their hands.

There is suddenly a plethora of articles in the literature regarding peripheral nerve entrapment diagnosis and treatment, and the emergence of ultrasound-guided injections in pain medicine has confirmed some of the mechanisms, while at the same time elucidated new mechanisms of entrapment. One of the hardest parts of writing this book has been the decision to stop adding new information to the chapters, since every time that I would find a new reference, my developmental editor (Connie Walsh) would have to reformat the chapter.

This book has been designed to be a guide as well as a reference. We chose pain pattern images that will hopefully trigger the clinician to think about peripheral nerve entrapment as a cause of their patient's pain, while at the same time providing the scholarly anatomic descriptions of the nerve. We hope that this book will help you diagnose as well as treat your patients, using physical exam, differential diagnosis, medications, injections (landmark-guided, fluoroscopic-guided, and ultrasound-guided), neurolytics, neuromodulation, and surgery. Videos showing the physical exam and landmark-guided injections are included for most of the described nerves. We have also created an Index of Symptoms, so that a patient who is complaining of an "ice pick in my eye" should lead you to consider the greater occipital nerve as a possible etiology.

I hope that you will find this book useful to help the patient who is asking "who will stop the pain?"

Anchorage, AK, USA — Andrea M. Trescot, MD, ABIPP, FIPP

Acknowledgements

A book of this size and scope is never created in isolation. Many thanks (and perhaps blame) go to Dr. Peter Staats, who 3 years ago sent *Springer Publishing* to talk to me about doing this book. Thanks also go to Connie Walsh (my developmental editor) and especially Joanna Perry (my original supervising editor), who talked me out of my panic half way through this project. When Joanna left Springer to pursue an MBA, she handed me over to Becky Amos, who shepherded this book to its final form.

I would like to thank my dear friend and collaborator, Dr. Helen (Ellie) Karl, who saw efficacy of these treatments and has become a "true believer." She has been the engine and organizer of this monumental project, and this book would have faltered and failed without her help. She checked and challenged every statement and image, so that this book would be as accurate as possible.

To my section editors, I give special thanks for their flexibility and trust, as the format and even the selection of nerves changed over the course of the development of this book. Each chapter author is a friend (or a friend of a friend), a fellow pain provider, and an advocate for recognition of these clinical syndromes. Many are mentees who were taught by me and who are now becoming the experts. Although each nerve chapter has an author's name attached, we have used a collaborative approach, with contributions by multiple authors, most unnamed, along the lines of Wikipedia. A few, however, deserve special recognition.

Drs. Dan Krashin and Natalia Murinova, an extraordinary husband/wife team (he is a psychiatrist and interventional pain physician, and she is a well-respected neurologist and migraine specialist), not only wrote their own chapters, but also wrote and rewrote many of the other chapters, some of which have their names on them and others that do not. In the same way, Dr. Michael Brown (interventional physiatrist) and Dr. Beth Pearce (podiatrist) provided a special expertise regarding pathologies of the lower extremities.

Dr. Thais Vanetti of Brazil and Dr. Tiffany Zhang of Seattle both did a wonderful job of editing many of my "problem" chapters, all the more amazing because English is their second language. Dr. Terri Dallas Prunskis jumped in to help me finish chapters as the final deadline approached and painted her family and staff to provide many of the nerve pattern pictures. Dr. Eric Wilson from South Africa helped to create the index of symptoms. My sister Leigh Trescot Tobias spent hours helping me to reformat the book when I had the "bright idea" to change the entire format after the book was three-quarters of the way done.

Dr. Agnes Stogicza, my "daughter" and (according to my husband) "mini-me," spent hours helping me by dissecting cadavers as well as creating US images of nerves as we traced nerves to their site of entrapment. Many of these US techniques have never been described in the literature, so expect a flurry of articles to follow the publishing of this book. She arranged for access to fresh cadavers in Hungary, as well as a wonderful anatomist, Gabor Balsa, who patiently and skillfully helped us to isolate nerves. We ultrasounded each other to trace nerves, and Agi always asked the tough questions of "why?" and "how?" and "what about this?" Her enthusiasm for pain treatment and new knowledge has kept me motivated.

Drs. Thiago Nouer Frederico and Fernando Mauad, from Brazil, and Drs. Michael Brown and Brian Shilpe, from the United States, generously provided many ultrasound pictures of the nerves. Thiago patiently traced nerves for me with ultrasound, showing potential entrapment sites not yet well described. Dr. Michael ("Micha") Sommer from the Netherlands reviewed nearly every image and provided valuable insight regarding the ultrasound and non-ultrasound images as well reviewing the "readability" of the language. Dr. Christ Declerck from Belgium also shared a variety of intriguing images. David Spinner, DO, contributed his ultrasound images of the supraorbital, infraorbital, and mental nerves, while Drs. Gladstone McDowell and Porter McRoberts donated several peripheral stimulator images. Holly Long, editor of the journal *Pain Physician*, graciously provided pictures and permissions from the American Society of Interventional Pain Physicians (ASIPP) textbooks and articles.

Accuracy is critical in a book such as this, and I had help from Dr. J. David Prologo (an interventional radiologist) who reviewed the MR images that I created and from Dr. Micha Sommer who reviewed the US images. Dr. Rubina Ahmad also helped to review images.

Over a dinner conversation during a conference in Poland, Ben Zylicz, MD, provided insight into the use of peripheral nerve injections in the treatment of cancer pain and agreed to put these thoughts down in a chapter. Heather Tick, MD, provided a balanced, integrated view of the non-interventional approach to these entrapments.

Edit Debreczeni, daughter of my friend Dr. Edit Racz of Hungary, volunteered on short notice to be my model for the videos of examination and injection that accompanies this book. Tamara Brothers, PA-C, Dr. Thais Vanetti, Dr. Joshua Balch, and my sister Caroline Kirkland also served as willing models. And my brother, David Trescot, volunteered his skills as a photographer and videographer/video editor to create almost every image in this book and to edit the raw video into the educational clips available here.

I would like to give special thanks to my children, Nicole and Joseph Gear, who were indispensible assistants, serving as models as well as copy editors. They allowed me to draw on and poke and scan their bodies to create just the right image, and then corrected my grammar and syntax. Nicole, especially, spent multiple sessions posing for images, and the images of her (I hope) have helped to unify the book; she also spend countless hours reviewing every word of the book to make sure that the information made sense. And this book would never have been possible without my loving, supportive, and long-suffering husband, Harold Gear, who even offered to let me inject him, just so I could get a better image for the book.

A final thanks goes to the support and encouragement over the years from students and colleagues, who would come up to me after my lecture to ask – "Where can I find a book with all this information?" Hopefully, this book will now answer that question.

Andrea M. Trescot, MD
St. Augustine Beach, Florida, USA

The plastinated human specimens shown in this book were produced by Prof. Dr. Hong-Jin Sui, Director of Dalian Medical University, China and Dalian Hoffen Bio-Technique Co,. Ltd, China, and are used here with their permission. The author wishes to thank Dr. Sui and Dalian Hoffen Bio-Technique Co., Ltd., for permission to use the photographs of these specimens.

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Part I

Peripheral Nerve Entrapments: General Principles

Andrea M. Trescot and Daniel Krashin

Introduction

An entrapment neuropathy is defined as a pressure-induced injury to a peripheral nerve in a segment of its course due to anatomic structures or pathologic processes [1]. Entrapment neuropathies for many conditions have been known for years. For instance, Paget [2] described entrapment of the ulnar nerve at the elbow in 1864, Learmonth [3] described carpal tunnel syndrome in 1933, tarsal tunnel syndrome [4, 5] was described in 1962, and radial nerve entrapment at the elbow in 1972 [6]. However, these are still often misunderstood, and there are many other poorly recognized or misrecognized peripheral nerve entrapments associated with clinical pain syndromes. Even for the astute clinician, these conditions may be difficult to diagnose without a high clinical index of suspicion. Knowledge of the syndromes and recognition of the patterns and symptoms will help the clinician to make the right diagnosis.

Kopell and Thompson [7] stated that peripheral nerve entrapment occurs at anatomic sites where the nerve changes direction to enter a fibrous or osseofibrous tunnel, or where the nerve passes over a fibrous or muscular band, and that entrapment occurs at these sites because mechanically induced irritation is most likely to occur at these locations. Trauma, such as surgery or constriction, and peripheral swelling, such as seen perimenstrually or with dietary indiscretions, can induce or perpetuate these entrapments, causing direct injury to the nerve or compromising its blood flow. Peripheral nerve injections (peripheral nerve blocks) are interventional pain management techniques used to treat patients with nerve entrapments presenting as a variety of painful conditions. By delivering local anesthetic and deposteroid directly onto the injured nerve, these injections can provide diagnostic as well as therapeutic benefit for patients suffering from pain anywhere from the head to the toes. Recognition of these conditions will lead to quicker diagnosis and treatment as well as decrease the inappropriate use of expensive (and for these conditions, useless) imaging and painful surgeries [8].

Peripheral nerve entrapments can cause a variety of painful conditions as diverse as headache, backache, "sciatica," "endometriosis," and foot pain. In addition, painful conditions with well-described pathology such as chronic regional pain syndrome (CRPS) or postherpetic neuralgia (PHN) may have a component of nerve entrapment, either as the initiating event (CRPS) or as a consequence of the pathology (PHN).

Nerve entrapments may occur in varying degrees, leading to a variety of clinical presentations. Somatic neuropathic pain originating from these nerves can have multiple etiologies. Nerve injury [2] has been reported from:

- Stretching
- Blunt trauma
- Compression with hypoxia
- Fibrosis with entrapment
- Suture ligature

The pain will often have a burning, shooting, or lancinating quality. Although initially the pain may be intermittent, the pain will usually become constant and more intense with time. If postsurgical, the pain can occur immediately after surgery, or it may start weeks to years after the surgery, as the scar cicatrix gradually tightens around the nerve. Pain is usually aggravated by activity, menstruation (due to perineural edema, hormone-induced increased neurotransmitters, and dorsal horn transmission cell sensitivity), or activity. Clinical diagnosis is dependent on a high index of suspicion and physical exam, but peripheral nerve blocks that provide complete relief, albeit temporary, are the *sine qua non* for establishing this diagnosis [9].

The injectate consists of a long-lasting local anesthetic (usually bupivicaine) and a depo-steroid. Because entrapment of the nerve is usually the underlying pathology, care must be used to avoid further entrapment with large volumes of injectate. Methylprednisolone (Depomedrol®) may be the steroid of choice, because of its high lipophilic nature (to enter the myelin sheath) and its high concentration (80 mg/cc). Total dose of steroid would normally be limited to 80 mg methylprednisolone (or equivalent), with no more than 40 mg at any one site (less if the skin is thin or the injection superficial, because of the risk of steroid-induced skin atrophy).

In this book, we hope to introduce the clinician to the myriad of pain conditions caused by peripheral nerve entrapment that may be diagnosed and treated with peripheral nerve injections. This book is divided into sections: the first is an overview of the history taking, physical exam, and diagnostic injection techniques. This is followed by sections on headaches, face and neck pain, chest wall pain, upper extremity pain, abdominal pain, low back pain, pelvic pain, and lower extremity pain. Each nerve has its own chapter, and each chapter is designed to stand alone, describing the clinical presentation, the anatomy and entrapment, the physical exam, the injection technique (blind, fluoroscopic, or ultrasound) and then the treatment modalities, such as neurolysis or surgery. The chapter concludes with a review of the literature and references. We have added an index of symptoms to aid the clinician in narrowing down the search for a specific nerve.

There have been many books written on regional anesthesia, and many pain practitioners come from this arena, but we want to emphasize that these entrapments cause pain syndromes, and, unlike regional anesthesia, require low-volume precise injections for diagnosis and treatment.

References

1. Toussaint CP, Perry 3rd EC, Pisansky MT, Anderson DE. What's new in the diagnosis and treatment of peripheral nerve entrapment neuropathies. Neurol Clin. 2010;28(4):979–1004.
2. Paget J. Clinical lecture on some cases of local paralysis. Med Times Gazette, London. 1864;1:331.
3. Learmonth JR. The principle of decompression in the treatment of certain diseases of peripheral nerves. Surg Clin North Am. 1933;13:905–13.
4. Keck C. The tarsal-tunnel syndrome. J Bone Joint Surg Am. 1962;44A:180–2.
5. Lam SJS. A tarsal-tunnel syndrome. Lancet. 1962;2:1354–5.
6. Roles NC, Maudsley RH. Radial tunnel syndrome. J Bone Joint Surg Am. 1972;4B:784–90.
7. Kopell HP, Thompson WA. Peripheral entrapment neuropathies. Baltimore: Williams and Wilkins; 1976.
8. Bora Jr FW, Osterman AL. Compression neuropathy. Clin Orthop Relat Res. 1982;163:20–32.
9. Kline DG, Hudson AR. Nerve injuries: operative results for major nerve injuries, entrapments, and tumors. Philadelphia: W.B. Saunders; 1995.

Epidemiology and Pathophysiology

1

Andrea M. Trescot, Daniel Krashin, and Helen W. Karl

Epidemiology

More than 80 million people in the United States suffer annually from serious pain, leading to disability, suffering, drug addiction, depression, and suicide [1].

Although diagnostic tools such as MRI and endoscopic techniques have become more sophisticated, there are a significant number of patients who have been told that "it is all in your head," "you just want pain medication," "there is nothing on the MRI," or "the surgery looks perfect, so I don't know why you are hurting." Pain management clinics have been viewed as the place of last resort – "if all else fails, send them to pain management." As such, the patient arrives on our doorstep, traumatized by ineffective surgeries, hyperalgesic because of high-dose opioids, depressed because of the multiple failed treatments, and hostile because once more they have to tell their story, only to watch the physician shrug and say "I don't know."

Pain is a problem throughout the entire medical field. In fact, the most common reason that patients present in the emergency department (ED) is pain. Todd et al. [2] noted that the median pain scores of patients on arrival in the ED were 8/10 on a number rating scale (NRS), and only half of them had a decrease in their pain by 2 or more points while there. Upon discharge from the ED, 45 % of patients reported an NRS of 4–7/10, and 29 % still had a pain score of 8–10/10.

A recent examination of the UK General Practice Research Database (which contains 1.8 million patient years of data), looking for new peripheral nerve entrapments, reported that they are relatively common [3] (Table 1.1). In another recent survey [4], 30 of 998 patients (3 %) referred to a gastroenterologist were diagnosed as having *chronic abdominal wall pain* (CAWP), presumably caused by myofascial spasm or peripheral nerve entrapment (see *ACNES syndrome* – Chap. 42).

Post-traumatic neuropathy is nerve pain that has been triggered after an injury or as a consequence of medical interventions such as surgery, injections, or radiotherapy. A recent evaluation of more than 2,500 soldiers returning from Iraq and Afghanistan revealed that 44 % had chronic pain, with 48.3 % of them having been in pain for more than a year. More than half (55.6 %) of the soldiers with chronic pain described it as constant; it was moderate to severe in 51.2 % [5].

Acute postoperative pain can develop into *chronic postsurgical pain* (*CPSP*), which affects daily life in 10–50 % of patients after surgery; this pain can be severe in 2–10 % of them [6]. A prospective study of approximately 5,000 surgical patients identified acute neuropathic pain in 1–3 %; 56 % of these had persistent chronic pain 1 year later [7]. The incidence varies widely with the kind of surgical procedure [8]. Simanski et al. confirmed these findings: more than 500 of 911 (57 %) patients had pain scores of greater than 3/10 for a mean of 29 months after orthopedic, abdominal, and/or vascular surgery. Almost 15 % of these patients reported severe pain [9].

Pain after inguinal hernia repair has been reported to range from 0 % to 60 %. A 2007 systematic review of pain after mesh hernia repair showed that 11 % of patients had persistent groin pain. More than a quarter of them had severe pain, and more than a third had limitations of daily activities [10]. Postherniorrhaphy pain is likely due to *ilioinguinal* (Chap. 40) and/or *genitofemoral* (Chap. 41) nerve injury. Similar

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Table 1.1 Incidence of peripheral nerve entrapments in UK primary care in 2000 [3]

	Men	Women
Carpal tunnel syndrome	87.8[a]	192.8
Morton's metatarsalgia	50.2	87.5
Ulnar neuropathy	25.2	18.9
Meralgia paresthetica	10.7	13.2
Radial neuropathy	2.97	1.42

[a]Per 100,000 European standard population

outcomes were reported in 690 consecutive patients surveyed 2 years after a Pfannenstiel incision for cesarean delivery or abdominal hysterectomy. One third still had incisional pain, and nearly 10 % described pain that interfered with their lives. Over half of the patients with moderate to severe pain (17 of 32) were found to have peripheral nerve entrapments [11].

Saphenous vein harvesting is performed in up to 27 % of patients undergoing *coronary artery bypass grafting* (CABG). In a survey of more than 1,000 CABG patients, 130 had chronic chest pain, 100 had leg pain, and 194 reported both. Although leg pain after vein harvest is usually described as mild, in about a third of the patients with pain, the prevalence of moderate to severe pain at a mean of 28 months after CABG was about 40 % [12].

Hicks and Simpson state that about 10–15 % of patients with *cancer-related pain* could benefit from peripheral nerve blocks (see Palliative Care – Chap. 12) [13].

We propose that many of these pain conditions are the result of peripheral nerve entrapments.

Pathophysiology

An *entrapment neuropathy* is defined as a pressure-induced segmental injury to a peripheral nerve due to an anatomic structure or pathologic process [14–16]. The defining criteria of an entrapment, according to Kashuk [17], include altered transmission as a result of mechanical irritation from impingement of an anatomic neighbor. Most of the nerve entrapments discussed in this book occur at areas where the nerve travels through a canal, channel, or tunnel. The nerve has its own blood flow (*vasa nervorum*) as well as accompanying vascular structures. Compression at these sites, whether intrinsic or extrinsic, can cause damage to the neurovascular structures running in the common course.

Multiple approaches have been used to try to categorize these entrapment phenomena, and the naming is therefore not consistent. The name of the condition can come from:

- The compressed nerve (e.g., lateral femoral cutaneous neuralgia)
- The classic Greek or Latin name (e.g., meralgia paresthetica)
- The anatomic area affected (e.g., metatarsalgia)
- The anatomic tunnel (e.g., carpal tunnel syndrome, tarsal tunnel syndrome)
- The motion that causes the compression (e.g., hyperabduction syndrome)
- The names of the describing authors (e.g., Kiloh-Nevin's syndrome)

Some people are susceptible to a particular entrapment neuropathy from congenital narrowing of a tunnel or thickening of an overlying fascial structure, while others with a systemic disorder such as diabetes mellitus (DM) show entrapment signs and symptoms much more frequently than nondiabetics [18–20].

Nerve injuries can result in clinical symptoms extending from mild discomfort to numbness, paralysis, or incapacitating pain. These changes parallel the histologic changes that occur in the implicated nerve (Fig. 1.1) [21]. In order to provide a common language for clinicians and researchers, some generally accepted classifications have evolved [16, 22, 23] (Table 1.2). Most peripheral nerve entrapments result in Grade 1 or Grade 2 injury. The most common mechanism of nerve injury is mechanical, but thermal or chemical injury may also occur (Table 1.3).

Mechanical injury may involve compressing, overstretching, or partially or totally cutting a nerve. Compression usually occurs at a site of direction change in a relatively noncompliant corridor, such as a fibro-osseous tunnel or fascial opening. Inflammation or edema of adjacent structures (e.g., tendons) can reduce the size of the passageway. Graded experimental compression results in profound short- and long-term effects on in vivo blood flow. Mild compression (20–30 mmHg) decreases venous flow; moderate compression decreases capillary and arterial flow; and pressures of 60–80 mmHg cause frank ischemia [24]. These pressures correspond to those measured clinically in the tarsal tunnel [25], carpal tunnel [26], and cubital tunnel [27]. Axonal transport is blocked by pressures of 50 mmHg [28], and nerve impulse conduction is blocked after less than an hour of compression of 70 mmHg in a peripheral nerve [29].

Other animal models of compression neuropathy surround a large nerve with a Silastic tube and evaluate histology and nerve conduction [21, 30]. Histologically, prolonged compression leads to neural edema, which, in the absence of relief, can progress to epineurial fibrosis and scarring, further thickening the nerve and worsening the entrapment. Damage to the myelin sheath and axonal disruption are end stages of chronic compression, resulting in irreversible damage [21] (Fig. 1.2).

Upton and McComas observed that 81/115 (70 %) patients with carpal or cubital tunnel syndrome also had electrophysiological evidence of a nerve injury in the neck [31]. They named the phenomenon the "*double crush*

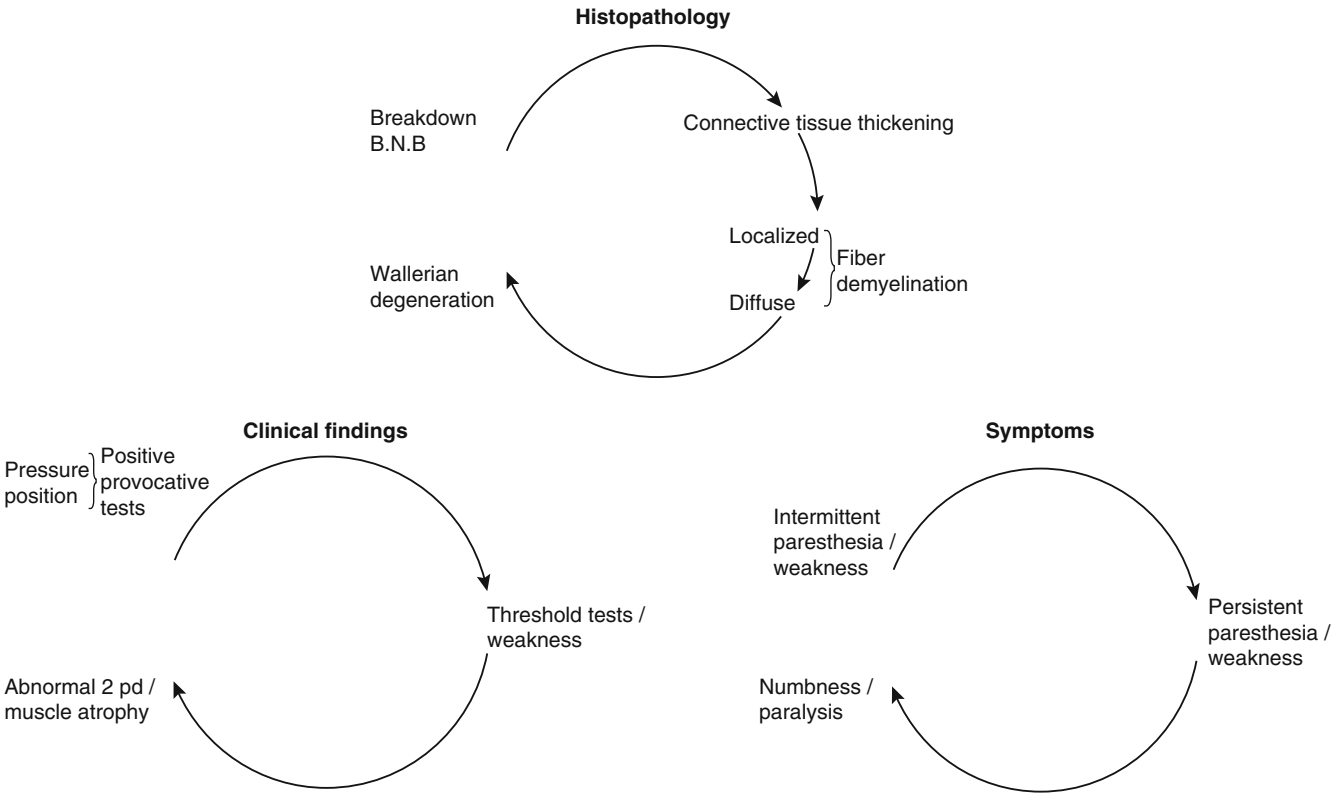


Fig. 1.1 The relationship between nerve histopathology, the patient's symptoms, and the clinical findings (From Mackinnon [21]. Reprinted with permission from Thieme Publishers). *BNB* blood nerve barrier

Table 1.2 Seddon and Sunderland classifications of nerve injury [16]

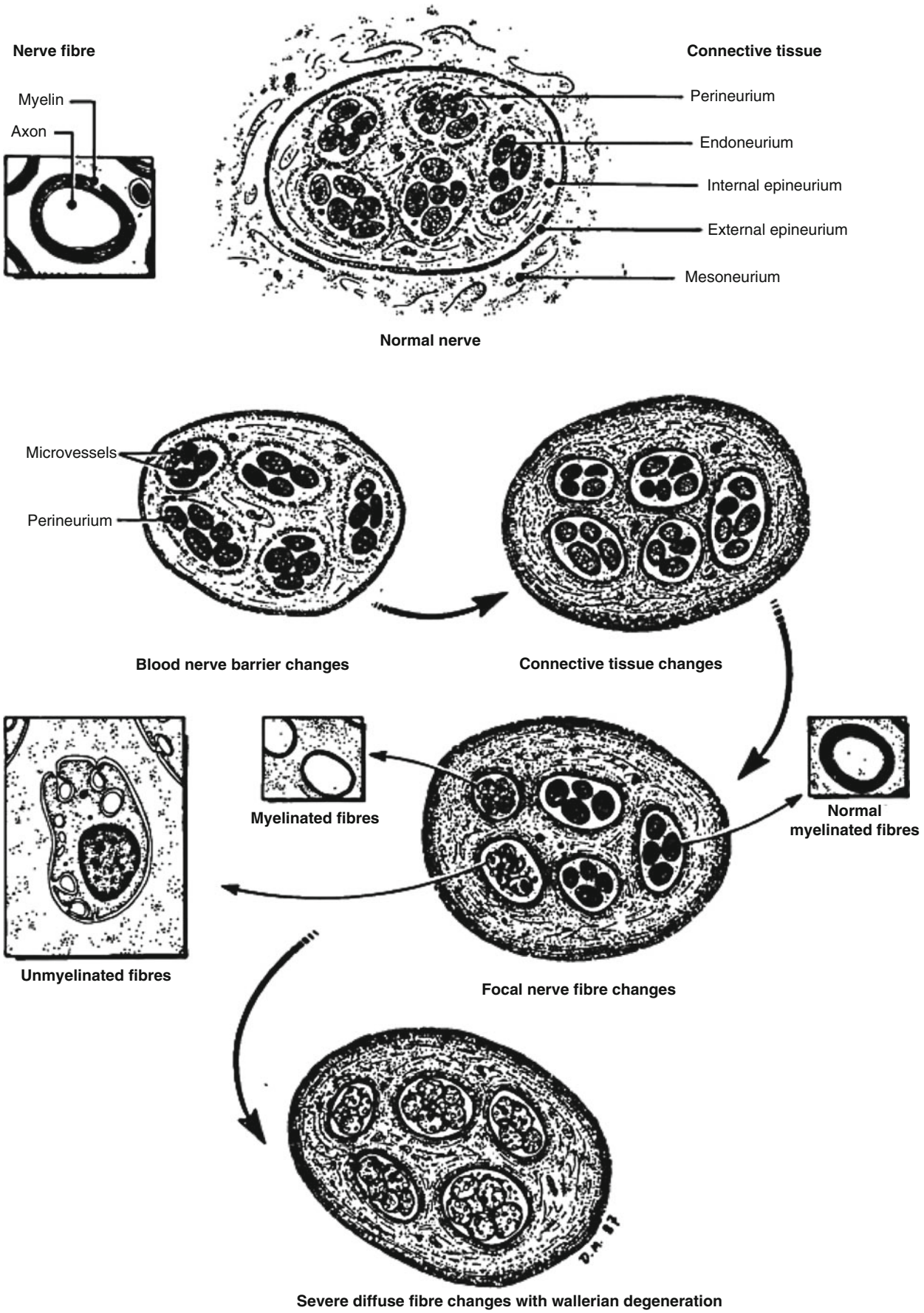
Seddon	Sunderland	Injury
Neurapraxia	Grade I	Focal segmental demyelination
Axonotmesis	Grade II	Axon damaged with intact endoneurium
Axonotmesis	Grade III	Axon and endoneurium damaged with intact perineurium
Axonotmesis	Grade IV	Axon, endoneurium, and perineurium damaged with intact epineurium
Neurotmesis	Grade V	Complete nerve transection
	Grade VI (MacKinnon and Dellon)	Mixed levels of injury along the nerve

From Menorca et al. [16]. Reprinted with permission from Elsevier Limited

Table 1.3 Causes of nerve injury

Mechanical	
Compression and/or stretch	Intraoperative retraction, suture material, scar [8]
	Joint hypermobility [42]
	Expanding tumor or cyst
	Dental work
	Infection
	Edema [60]
	Congenital anomalies (e.g., muscle bands)
	Blunt trauma with or without fracture
Systemic susceptibility	Diabetes mellitus [18, 20]
	Chemotherapy [61]
	Thyroid disease [62]
Chemical	Dental care [52]
	Infection
	Leaking intervertebral disk
Thermal	Total joint replacement [50]

syndrome" (DCS), where the presence of a more proximal lesion renders the distal nerve trunk particularly vulnerable to compression, with a degree of pain and dysfunction greater than that expected from either entrapment alone. They postulated that this was due to the effect of compression on anterograde axoplasmic flow, as later confirmed by other investigators [28, 32]. However, other mechanisms, and indeed whether this phenomenon actually exists, are also under discussion [33]. Lundborg's observation that patients with symptoms of ulnar compression in the wrist subsequently developed compressive symptoms of the same nerve at the elbow led to the concept of "reverse DCS," thought to be due to disturbed


Nerve fibre
Myelin
Axon
Connective tissue
Perineurium
Endoneurium
Internal epineurium
External epineurium
Mesoneurium
Normal nerve
Microvessels
Perineurium
Blood nerve barrier changes
Connective tissue changes
Myelinated fibres
Normal myelinated fibres
Unmyelinated fibres
Focal nerve fibre changes
Severe diffuse fibre changes with wallerian degeneration

retrograde axoplasmic flow [34]. DCS has been observed at several common locations, clinically [21, 35, 36], upon electrodiagnostic investigation [37] (Table 1.4), and experimentally [32]. The surgical outcome of carpal tunnel release is poorer in those patients, suggesting that both entrapments likely require treatment for optimal results [21].

The role of overstretching a nerve is sometimes overlooked, but it is especially important with respect to nerves that cross over joints, where changes in position are known to change the amount of stretch [38, 39]. *Stretch injury* may have a significant role in the pain after joint injuries, suggesting that pain from degenerative joint disease (DJD) may not be purely due to intra-articular pathology [40], as evidenced by the pain relief seen with injection and denervation of the infrapatellar saphenous nerve [41] (see Chap. 58). Also, patients with joint hypermobility from Ehlers-Danlos syndrome have a much higher incidence of ulnar nerve subluxation and potential entrapment at the elbow than patients without Ehlers-Danlos [42] (see Chap. 37). Animal models that investigate the underlying pathophysiologic mechanisms support these clinical observations [38, 43]. High degrees of stretch result in decreased blood flow, but electrophysiologic changes are measurable at levels well below those that cause ischemia [38]; as little as 6 % stretch of a nerve can cause permanent injury [44].

Diabetes mellitus predisposes patients to not only entrapment neuropathies but also to inflammatory ones [18, 45], thereby acting like the first "crush." This often results in the classic stocking and glove symptom patterns in diabetic patients [20, 46]. Entrapment susceptibility in diabetes is thought to be the result of three factors: increased sorbitol concentration leading to neural swelling, abnormal axoplasmic transport, and the presence of intraneural collagen-glucose complexes that make the nerve less compliant [20].

Dellon has advocated early surgical decompression of nerves to prevent and treat diabetic tissue loss. In the foot, combined neurolysis of the common peroneal nerve at the knee (Chap. 67), the superficial peroneal nerve above the ankle (Chap. 68), and the posterior tibial nerve at the tarsal tunnel (Chap. 73) has improved symptoms in the "stocking" distribution. A recent multicenter prospective study of tibial nerve neurolysis alone in 628 diabetic patients with well-documented tibial nerve entrapment documented improved foot ulceration [47], and a randomized clinical trial in 42 patients showed that a four-site decompression significantly improved foot pain [48]. In the upper extremity, combined neurolysis of the median nerve at the wrist (Chap. 37) and the ulnar nerve at the elbow (Chap. 38), with or without release of the radial sensory nerve in the forearm (Chap. 36), improved symptoms in the "glove" distribution [49].

Other causes of nerve compression and entrapment include hematomas, especially with the increased use of prophylactic anticoagulation postoperatively [44]. These patients will present with swelling and increasing pain postoperatively, and nerve testing is not helpful in this acute setting. Hematoma-induced entrapment requires prompt decompression to avoid permanent compromise. In addition, there can be intraoperative nerve injury.

Thermal injury and *chemical injury* are less common, but may occur after total joint replacement (because of the exothermic reaction of the methyl methacrylate cement) [50] or with leaking intervertebral disks (which contain a "soup" of inflammatory cytokines) [51]. Dental materials

Table 1.4 Double crush syndromes

Median (Chap. 37) [21, 36]	CTS and cervical radiculopathy
	CTS and TOS (Chap. 33)
	CTS and median nerve compression at the elbow (pronator syndrome)
Ulnar (Chap. 38) [21, 36]	CuTS and cervical radiculopathy
	CuTS and TOS
	CuTS and ulnar nerve compression at the wrist (Guyon's canal syndrome)
Radial (Chap. 35) [21, 36]	RTS and cervical radiculopathy
Deep peroneal (Chap. 69) [37]	ATTS and LBP
Posterior tibial (Chap. 72) [37]	TTS and LBP

CTS carpal tunnel syndrome, median nerve entrapment at the wrist; *TOS* thoracic outlet syndrome; *CuTS* cubital tunnel syndrome, ulnar nerve entrapment at the elbow; *RTS* radial tunnel syndrome, radial nerve entrapment near the elbow; *ATTS* anterior tarsal tunnel syndrome, peroneal nerve entrapment; *TTS* tarsal tunnel syndrome, tibial nerve entrapment; *LBP* low back pain

Fig. 1.2 Histopathology of chronic nerve compression. The initial changes occur at the level of the blood-nerve barrier. These changes are followed by connective tissue changes and then focal nerve fiber changes. The large myelinated fibers undergo segmental demyelination. The small, unmyelinated fibers demonstrate evidence of degeneration and regeneration with the presence of a new population of very small unmyelinated fibers. With progression of the compression, diffuse Wallerian degeneration is noted (From Mackinnon [21]. Reprinted with permission from Thieme Publishers)

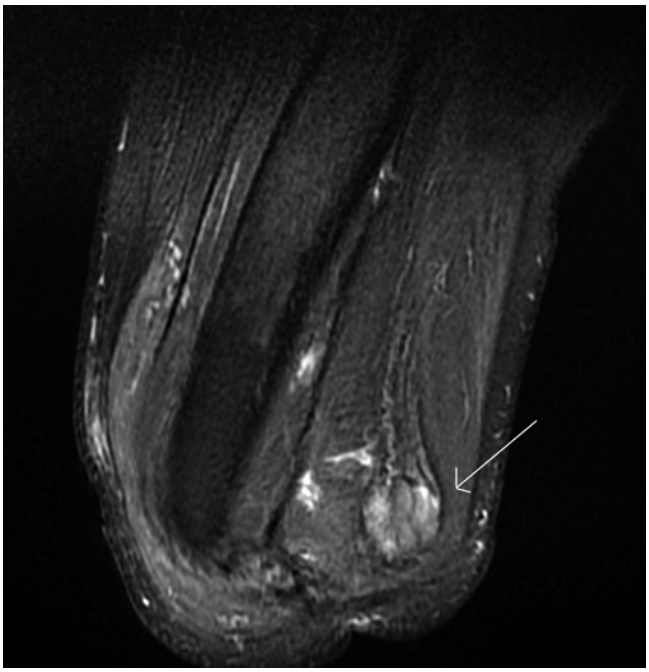


Fig. 1.3 MRI image of a large sciatic nerve neuroma after a leg amputation – *white arrow* (Image courtesy of Andrea Trescot, MD)

(such as those used for a root canal) can also create chemical injury. Chemical damage to a nerve can occur locally from toxic materials such as paraformaldehyde for endodontic filling. Dental irrigation chemicals like sodium hypochlorite can also be culprits. Additionally, chemical injury can be caused internally by the body's own local inflammatory markers and cytokines, including those released by cells fighting infection [52] or from a damaged intervertebral disk [51].

When nerves are injured and try to regenerate, the proximal segment may curl up on itself, causing an ectopic-firing "knot" of nerve fibers called a *neuroma* (Fig. 1.3). Neuromas are mostly composed of sprouting axons, with a significant degree of sympathetic innervation, and can cause pain spontaneously or with stimulation. They can mimic entrapments or they can be caused by the entrapments or the treatment for that entrapment (such as surgery or neurolytics).

Results of Peripheral Nerve Entrapment

Peripheral nerve entrapment can lead or contribute to a wide variety of disorders (Table 1.5). In addition, painful conditions with well-described pathology such as *complex regional pain syndrome* (CRPS) (as described below) or *postherpetic neuralgia* (PHN) likely have a component of nerve entrapment, either as the initiating event (CRPS) or as a consequence of the pathology (PHN).

Table 1.5 Conditions that may be caused by entrapment of a peripheral nerve

Headaches, including "migraines"	"Endometriosis"
Atypical face pain	Postherpetic neuralgia
Chest wall pain	CRPS (previously known as RSD)
Carpal tunnel syndrome	Low back syndrome
Abdominal wall pain	"Sciatica"
Pelvic pain	Foot pain

Recent histological [53] and animal [54] data show that some form of initial nerve trauma is "an important trigger for the cascade of events leading to CRPS" [55]. The distinction between the pathogenesis of CRPS-I and that of CRPS-II is a matter of degree and *not* mechanism [56].

Therefore, patients presenting with CRPS symptoms should be carefully questioned as to where the pain started and where it is most intense. If this is in the distribution of a peripheral nerve, targeting that nerve for diagnostic block and cryoneuroablation (see Chap. 8) can have beneficial effects on the overall CRPS clinical picture. Identification of entrapment neuropathy as the initiating event to CRPS could provide a method for definitive treatment. Interestingly, CRPS has been identified 4–6 months before the diagnosis of a malignancy, perhaps from peripheral nerve entrapment [57].

Recovery with a resolution of the symptoms of nerve injury depends on the inciting event, the severity of the nerve injury, and the patient's ability to heal; it is often difficult to predict. Factors that influence healing include the severity, duration, and location of the injury, the integrity of any involved muscle, the patient's age, underlying genetics, and other diseases (e.g., diabetes) influencing the health of the nerves. Since the blood flow and nutrients for the nerve come from its origin in the spinal column [58], distal nerves tend to be at more risk for injury, due to limited "resources"; however, because of the length of regeneration needed, proximal peripheral nerve injuries take longer than more distal ones to resolve. Spontaneous recovery is often incomplete and may require up to 2 years or longer [59].

Conclusion

Chronic pain, occurring after surgery, after injury, and occasionally spontaneously, may be caused and perpetuated by peripheral nerve entrapments. The recognition that "every chronic pain was once acute" [8] suggests that accurate diagnosis coupled with early treatment may decrease the number of acute pain "failures" and perhaps decrease the risk of chronic pain. Knowing the clinical features and treatment of these peripheral nerve disorders can provide relief for patients who have often suffered for many years.

References

1. Rasor J, Harris G. Using opioids for patients with moderate to severe pain. J Am Osteopath Assoc. 2007;107(9 Suppl 5):Es4–10.
2. Todd KH, Ducharme J, Choiniere M, Crandall CS, Fosnocht DE, Homel P, et al. Pain in the emergency department: results of the pain and emergency medicine initiative (PEMI) multicenter study. J Pain Off J Am Pain Soc. 2007;8(6):460–6.
3. Latinovic R, Gulliford MC, Hughes RA. Incidence of common compressive neuropathies in primary care. J Neurol Neurosurg Psychiatry. 2006;77(2):263–5.
4. Adibi P, Toghiani A. Chronic abdominal wall pain: prevalence in out-patients. J Pak Med Assoc. 2012;62(3 Suppl 2):S17–20.
5. Toblin RL, Quartana PJ, Riviere LA, Walper KC, Hoge CW. Chronic pain and opioid use in US soldiers after combat deployment. JAMA Intern Med. 2014;174(8):1400–1.
6. Kehlet H, Jensen TS, Woolf CJ. Persistent postsurgical pain: risk factors and prevention. Lancet. 2006;367(9522):1618–25.
7. Hayes C, Browne S, Lantry G, Burstal R. Neuropathic pain in the acute pain service: a prospective survey. Acute Pain. 2002;4:45–8.
8. Katz J, Seltzer Z. Transition from acute to chronic postsurgical pain: risk factors and protective factors. Expert Rev Neurother. 2009;9(5):723–44.
9. Simanski CJ, Althaus A, Hoederath S, Kreutz KW, Hoederath P, Lefering R, et al. Incidence of chronic postsurgical pain (CPSP) after general surgery. Pain Med. 2014;15(7):1222–9.
10. Nienhuijs S, Staal E, Strobbe L, Rosman C, Groenewoud H, Bleichrodt R. Chronic pain after mesh repair of inguinal hernia: a systematic review. Am J Surg. 2007;194(3):394–400.
11. Loos MJ, Scheltinga MR, Mulders LG, Roumen RM. The Pfannenstiel incision as a source of chronic pain. Obstet Gynecol. 2008;111(4):839–46.
12. Bruce J, Drury N, Poobalan AS, Jeffrey RR, Smith WC, Chambers WA. The prevalence of chronic chest and leg pain following cardiac surgery: a historical cohort study. Pain. 2003;104(1–2):265–73.
13. Hicks F, Simpson KH. Nerve blocks in palliative care. Oxford: Oxford University Press; 2004. 129 p.
14. Kopell HP, Thompson WAL. Peripheral entrapment neuropathies. 2nd ed. Huntington: R. E. Krieger Pub. Co; 1976.
15. Toussaint CP, Perry 3rd EC, Pisansky MT, Anderson DE. What's new in the diagnosis and treatment of peripheral nerve entrapment neuropathies. Neurol Clin. 2010;28(4):979–1004.
16. Menorca RM, Fussell TS, Elfar JC. Nerve physiology: mechanisms of injury and recovery. Hand Clin. 2013;29(3):317–30.
17. Kashuk K. Proximal peripheral nerve entrapment syndromes in the lower extremity. J Am Podiatry Assoc. 1977;67(8):529–44.
18. Vinik A, Mehrabyan A, Colen L, Boulton A. Focal entrapment neuropathies in diabetes. Diabetes Care. 2004;27(7):1783–8.
19. Dellon AL. Diabetic neuropathy: medical and surgical approaches. Clin Podiatr Med Surg. 2007;24(3):425–48, viii.
20. Dellon AL. Susceptibility of nerve in diabetes to compression: implications for pain treatment. Plast Reconstr Surg. 2014;134(4 Suppl 2):142S–50S.
21. Mackinnon SE. Double and multiple "crush" syndromes. Double and multiple entrapment neuropathies. Hand Clin. 1992;8(2):369–90.
22. Maggi SP, Lowe JB, Mackinnon SE. Pathophysiology of nerve injury. Clin Plast Surg. 2003;30(2):109–26.
23. Dellon AL. Clinical grading of peripheral nerve problems. Neurosurg Clin N Am. 2001;12(2):229–40.
24. Rydevik B, Lundborg G, Bagge U. Effects of graded compression on intraneural blood blow. An in vivo study on rabbit tibial nerve. J Hand Surg Am. 1981;6(1):3–12.
25. Dellon AL. The four medial ankle tunnels: a critical review of perceptions of tarsal tunnel syndrome and neuropathy. Neurosurg Clin N Am. 2008;19(4):629–48, vii.
26. Smith EM, Sonstegard DA, Anderson WH. Carpal tunnel syndrome: contribution of flexor tendons. Arch Phys Med Rehabil. 1977;58(9):379–85.
27. Pechan J, Julis I. The pressure measurement in the ulnar nerve. A contribution to the pathophysiology of the cubital tunnel syndrome. J Biomech. 1975;8(1):75–9.
28. Rydevik B, McLean WG, Sjöstrand J, Lundborg G. Blockage of axonal transport induced by acute, graded compression of the rabbit vagus nerve. J Neurol Neurosurg Psychiatry. 1980;43(8):690–8.
29. Fern R, Harrison PJ. The effects of compression upon conduction in myelinated axons of the isolated frog sciatic nerve. J Physiol. 1991;432:111–22.
30. Mackinnon SE, Dellon AL, Hudson AR, Hunter DA. Chronic nerve compression – an experimental model in the rat. Ann Plast Surg. 1984;13(2):112–20.
31. Upton AR, McComas AJ. The double crush in nerve entrapment syndromes. Lancet. 1973;2(7825):359–62.
32. Dellon AL, Mackinnon SE. Chronic nerve compression model for the double crush hypothesis. Ann Plast Surg. 1991;26(3):259–64.
33. Schmid AB, Coppieters MW. The double crush syndrome revisited – a Delphi study to reveal current expert views on mechanisms underlying dual nerve disorders. Man Ther. 2011;16(6):557–62.
34. Lundborg G. The reversed double crush lesion. Am Soc Surg Hand Correspondence Newsletter. 1986.
35. Moghtaderi A, Izadi S. Double crush syndrome: an analysis of age, gender and body mass index. Clin Neurol Neurosurg. 2008;110(1):25–9.
36. Osterman AL. The double crush syndrome. Orthop Clin North Am. 1988;19(1):147–55.
37. Golovchinsky V. Double crush syndrome in lower extremities. Electromyogr Clin Neurophysiol. 1998;38(2):115–20.
38. Wall EJ, Massie JB, Kwan MK, Rydevik BL, Myers RR, Garfin SR. Experimental stretch neuropathy. Changes in nerve conduction under tension. J Bone Joint Surg Br. 1992;74(1):126–9.
39. Walker FO, Cartwright MS, Wiesler ER, Caress J. Ultrasound of nerve and muscle. Clin Neurophysiol. 2004;115(3):495–507.
40. Ikeuchi M, Izumi M, Aso K, Sugimura N, Tani T. Clinical characteristics of pain originating from intra-articular structures of the knee joint in patients with medial knee osteoarthritis. SpringerPlus. 2013;2:628.
41. Kachar SM, Williams KM, Finn HA. Neuroma of the infrapatellar branch of the saphenous nerve a cause of reversible knee stiffness after total knee arthroplasty. J Arthroplasty. 2008;23(6):927–30.
42. Granata G, Padua L, Celletti C, Castori M, Saraceni VM, Camerota F. Entrapment neuropathies and polyneuropathies in joint hypermobility syndrome/Ehlers-Danlos syndrome. Clin Neurophysiol. 2013;124(8):1689–94.
43. Love JM, Chuang TH, Lieber RL, Shah SB. Nerve strain correlates with structural changes quantified by Fourier analysis. Muscle Nerve. 2013;48(3):433–5.
44. Unwin A, Scott J. Nerve palsy after hip replacement: medico-legal implications. Int Orthop. 1999;23(3):133–7.
45. Perkins BA, Olaleye D, Bril V. Carpal tunnel syndrome in patients with diabetic polyneuropathy. Diabetes Care. 2002;25(3):565–9.
46. Dellon AL. A cause for optimism in diabetic neuropathy. Ann Plast Surg. 1988;20(2):103–5.
47. Dellon AL, Muse VL, Nickerson DS, Akre T, Anderson SR, Barrett SL, et al. Prevention of ulceration, amputation, and reduction of hospitalization: outcomes of a prospective multicenter trial of tibial neurolysis in patients with diabetic neuropathy. J Reconstr Microsurg. 2012;28(4):241–6.
48. van Maurik JFMM, van Hal M, van Eijk RPA, Kon M, Peters EJG. Value of surgical decompression of compressed nerves in the lower extremity in patients with painful diabetic neuropathy: a randomized controlled trial. Plast Reconstr Surg. 2014;134(2):325–32.

49. Aszmann OC, Kress KM, Dellon AL. Results of decompression of peripheral nerves in diabetics: a prospective, blinded study. Plast Reconstr Surg. 2000;106(4):816–22.
50. Deramond H, Wright NT, Belkoff SM. Temperature elevation caused by bone cement polymerization during vertebroplasty. Bone. 1999;25(2 Suppl):17s–21s.
51. Raj PP. Intervertebral disc: anatomy-physiology-pathophysiology-treatment. Pain Pract. 2008;8(1):18–44.
52. Morse DR. Endodontic-related inferior alveolar nerve and mental foramen paresthesia. Compend Contin Educ Dent. 1997;18(10):963–8, 70-3, 76-8 passim; quiz 98.
53. Albrecht PJ, Hines S, Eisenberg E, Pud D, Finlay DR, Connolly MK, et al. Pathologic alterations of cutaneous innervation and vasculature in affected limbs from patients with complex regional pain syndrome. Pain. 2006;120(3):244–66.
54. Oaklander AL, Fields HL. Is reflex sympathetic dystrophy/complex regional pain syndrome type I a small-fiber neuropathy? Ann Neurol. 2009;65(6):629–38.
55. Bruehl S. An update on the pathophysiology of complex regional pain syndrome. Anesthesiology. 2010;113(3):713–25.
56. Oaklander AL. Development of CRPS after shingles: it's all about location. Pain. 2012;153(12):2309–10.
57. Goldberg E, Dobransky R, Gill R. Reflex sympathetic dystrophy associated with malignancy. Arthritis Rheum. 1985;28(9):1079–80.
58. Ishibe K, Tamatsu Y, Miura M, Shimada K. Morphological study of the vasa nervorum in the peripheral branch of human facial nerve. Okajimas Folia Anat Jpn. 2011;88(3):111–9.
59. Kim DH, Kline DG. Management and results of peroneal nerve lesions. Neurosurgery. 1996;39(2):312–9; discussion 9–20.
60. Flanigan RM, DiGiovanni BF. Peripheral nerve entrapments of the lower leg, ankle, and foot. Foot Ankle Clin. 2011;16(2):255–74.
61. Peltier AC, Russell JW. Advances in understanding drug-induced neuropathies. Drug Saf. 2006;29(1):23–30.
62. Briemberg HR. Peripheral nerve complications of medical disease. Semin Neurol. 2009;29(2):124–35.

History and Physical Exam

2

Andrea M. Trescot and Daniel Krashin

History

> The diagnosis is hidden in the patient's history, and the cause is elicited by the physical exam. – J. Bart Staal

The history given by the patient, in the patient's own words, is key to making the diagnosis of a peripheral nerve entrapment. Multiple patients use the same words to describe their pain perception ("My testicles are in a vise"). There are reproducible, recognizable patterns of pain that provide clues to the cause of the pain. Unfortunately, many patients with nerve entrapments present with poorly localized or widespread pain. By the time the patient has reached a pain specialist, the pain may have spread beyond its original site "like a forest fire raging out of control," so a careful history of the mechanism of injury, the initial site of pain, and the initial referral pattern are key clues to the etiology of a painful nerve entrapment.

The history of trauma can vary from mild to severe and, at the time of presentation, can be acute, subacute, or chronic. This trauma may have occurred as a result of surgery or fracture, or it may have originally been regarded as "trivial." There may have been abnormal limb positioning, repetitive activity, or recent change in body habitus [1]. The patient often describes the pain as burning, shooting, or lancinating, and there may be associated numbness or tingling. There may also be a history of "clumsiness" and inadvertent injuries [1]. Symptoms are often worse at night, especially when the limb is in the same position for a prolonged period of time.

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There are certain symptoms that point toward a compressive nerve pathology. Peripheral nerves are especially prone to compression, and activities that decrease the entrapment, such as changing position or "shaking it off," will temporarily help. Although initially the pain may be intermittent, the pain usually becomes constant and more intense over time. If postsurgical, the pain can occur immediately after surgery, or it may start weeks to years after the surgery, as the scar cicatrix gradually tightens around the nerve. Pain is usually aggravated by activity or menstrual periods (due to perineural edema, hormone changes, induced modulation of levels of pain neurotransmitters, and increased dorsal horn transmission cell sensitivity). A history of trauma, operations, previous fractures, abnormal limb positioning, repetitive activities, and recent change in body habitus are also clues in the history [1].

However, it is important to remember that many nerve entrapments may present with nonspecific or poorly localized pain or even a false sense of numbness *("nulliness"),* according to Dorman [2]. In this case, the mechanism of injury becomes even more important.

Concomitant symptoms, such as back pain when there is leg pain or neck pain with arm pain, may be significant clues to the diagnosis. The history also gives a clue about potentially more serious conditions; for instance, loss of sphincter tone might represent a spinal cord injury.

Specific questions that should be asked while eliciting the patient's history include:

- Where does it hurt? (Attempting to localize the initial site of the injury, as well as patterns of pain radiation)
- Where and when did it start to hurt?
- What makes it worse? What makes it better? (It is important to ask these questions in this order, since patients will often respond "nothing" to "what makes it better?" if asked that question first.)
- Are there associated weakness or sensory disturbances?
- Are there any changes in the appearance or function of the limb?
- Is there a history of recent or old trauma?

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- Is there a history of other medical conditions that might make the nerve more prone to other injury, including recent weight gain, pregnancy, diabetes, thyroid disease, malignancy, immune suppression, or HIV?
- Does the patient perform repetitive movements at work or during hobbies? (See Table 2.1)

Physical Exam

The physical exam for peripheral nerve entrapments is very specific, directed by the patient pointing to "where it hurts" (Fig. 2.1), followed by identification of known nerve entrapment sites. Normal nerves are almost insensitive to pressure. Beyer described that normal nerves "can be rolled under the thumbnail at will" [4]. The inflamed, entrapped nerve, however, will be extremely sensitive, with the slightest pressure causing the patient "to literally jump out of the chair with pain" [3]. Gentle, open palm probing, specific thumb or finger pressure (Fig. 2.2), tapping (*Tinel's sign*), and "strumming" across the affected structure are the more appropriate palpation techniques for specific identification of tender structures. *Valleix phenomenon* may be present, which is tenderness along the distribution of a peripheral nerve both proximal and distal to the site of entrapment [5]. This focused physical exam becomes even more important when the pain has spread "all over"; having the patient point to where the pain "first started," combined with the pattern of pain ("pattern recognition"), can give important clues as to the etiology.

Sensory testing of the affected area may help clarify the diagnosis, when paired with a good working knowledge of cutaneous innervation patterns and their common variations. Nerve entrapment may cause referred pain in the innervated area; it is also commonly accompanied by decreased sensation. Sensation should be assessed to both light touch and temperature or pinprick; patients will often be aware of areas with decreased sensation to touch but may be surprised to find areas without temperature or pinprick sensation. Mechanical sensitivity can be assessed by a series of graded sizes of plastic filaments called von Frey fibers (Fig. 2.3). Assessment of two-point discrimination is particularly helpful in areas that normally have very good discrimination, such as the hands [6]. If the patient is asked to close their eyes, this approach can also help identify fabricated or embellished symptoms. In those nerves with combined motor and sensory function, motor impairment may also be elicited as a sign.

Knowledge of the common entrapment sites, the pattern of pain, and the specific examination can allow the provider to focus on the likely site of the entrapment, which is key for accurate injection therapy, which then confirms the diagnosis and delivers therapeutic medication to the likely site of the nerve entrapment.

Table 2.1 Information to be obtained in the history [3]

Site of pain	Quality (looking for neuropathic features)
Radiation pattern	Duration
Intensity (at rest and with movement)	Temporal variation
Previous therapies and response	Precipitating factors
Mood	Relieving factors
Activities of daily living	Sleep
Current therapies	Patient beliefs regarding cause of pain

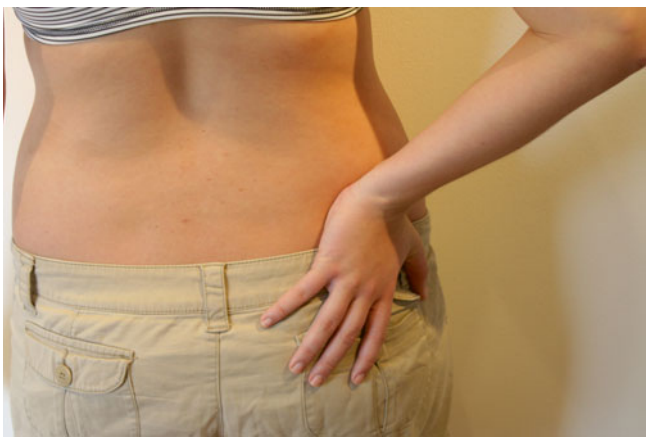

Fig. 2.1 "Where does it hurt?' (Image courtesy of Andrea Trescot, MD)

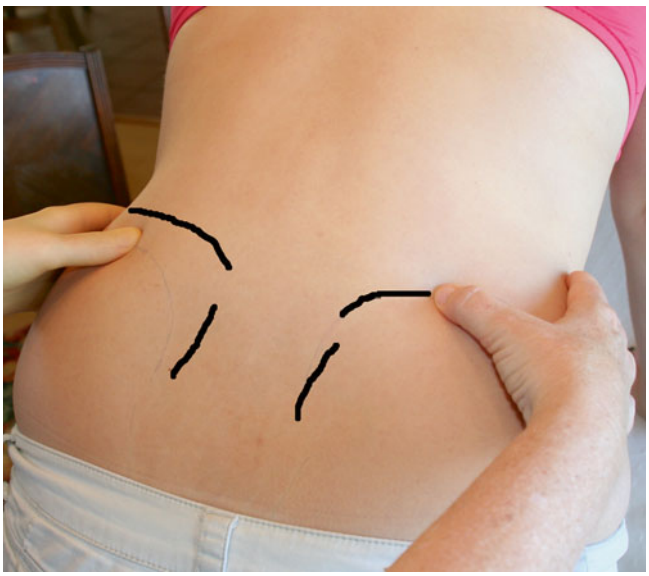

Fig. 2.2 A directed back examination (Image courtesy of Andrea Trescot, MD)

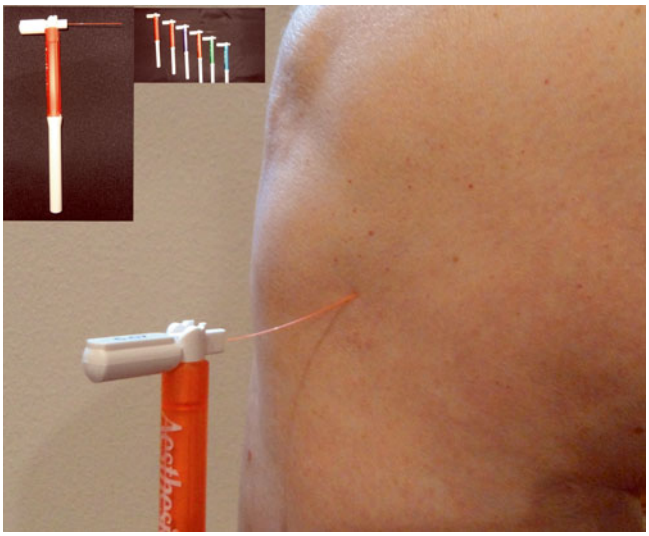

Fig. 2.3 Von Frey fibers of variable stiffness (Image courtesy of Michael Brown, MD)

References

1. deSouza RM, Choi D. Peripheral nerve lesions. Neurosurgery. 2012;3(30):149–54.
2. Dorman TA. Diagnosis and injection techniques in orthopedic medicine. Baltimore: Williams and Wilkins; 1991.
3. Hicks F, Simpson KH. Nerve blocks in palliative care. Oxford: Oxford University Press; 2004. p. 129.
4. Beyer TE. Idiopathic supraorbital neuralgia. Laryngoscope. 1949;59(11):1273–5.
5. Kashuk K. Proximal peripheral nerve entrapment syndromes in the lower extremity. J Am Podiatry Assoc. 1977;67(8):529–44.
6. Dellon AL, Mackinnon SE. Radial sensory nerve entrapment in the forearm. J Hand Surg Am. 1986;11(2):199–205.

Consequences of Peripheral Nerve Entrapment

3

Andrea M. Trescot and Daniel Krashin

Peripheral nerve entrapments can cause a variety of painful conditions as diverse as headache, backache, "sciatica," "endometriosis," and foot pain. Peripheral nerve entrapments can occur at sites where the nerve goes through a tunnel or canal, or pierces through fascia, or makes a sharp change in the direction of its path. Inflammation, edema, or extrinsic compression will trigger entrapment; the sympathetic response to this entrapment leads to more inflammation and edema, leading to more entrapment, creating a vicious cycle.

In nerves with a sensory component, chronic injury often causes increased excitability of the nerve. This can result in both *hyperalgesia* and *allodynia* in the peripheral nerve field supplied by that nerve or the whole sensory dermatome, as is the case with injury to a spinal nerve root.

Nerve entrapments may be present in varying degrees, leading to a variety of clinical presentations. The pain will often have a burning, shooting, or lancinating quality. Although initially the pain may be intermittent, the pain will usually become constant and more intense with time. If post-surgical, the pain can occur immediately after surgery, or it may start weeks or even years after the surgery, because of the scar cicatrix that gradually tightens around the nerve. Pain is usually aggravated by activity, menstruation (due to perineural edema, hormone-induced increased neurotransmitters, and dorsal horn transmission cell sensitivity), or compression. Clinical diagnosis is dependent upon a high index of suspicion and physical exam, but peripheral nerve blocks that provide complete relief, albeit temporary, are the sine qua non for establishing this diagnosis [1].

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Chronic peripheral neuroexcitability may result in major disability. Severe sensitivity of the extremities is particularly impairing, especially with the upper extremities and hands. When mixed motor and sensory nerves are injured, the motor deficit may compound the increased hypersensitivity. For example, an injury to the distal radial nerve may cause hyperalgia and allodynia in the peripheral nerve field of that nerve, including the dorsum of the affected hand, as well as decreased grip and function of that hand. The combination of impaired function and hypersensitivity often leads the patient to avoid using that limb (*kinesiophobia*), even swaddling it in protective gloves or bandages and maintaining that limb in a guarded position. This restricts the patient's ability to engage in rehabilitation and normal social activities and maintain or resume gainful employment [2].

In a study of ulnar nerve entrapment, about half of the patients who did not have surgery were largely asymptomatic, while the rest had varying degrees of paresthesias, pain, and difficulty with fine motor control of the fingers [3]. This was a retrospective, nonrandomized study and included patients who were seen by the surgeons but managed conservatively, likely with milder symptoms.

Strokes or *cerebral vascular accidents (CVAs)* are a common cause of mortality and one of the most common causes of morbidity in the world [4]. Although there can be weakness, paresis, and dysarthria as a major cause of morbidity after CVA, pain can also be a significant cause of distress, and some of that pain comes from entrapment neuropathies. Hunkar and Balci [4] evaluated 32 patients with ischemic or hemorrhagic stroke 6 months after the event and compared them to 10 age-matched controls. Nerve conduction studies showed that 12 patients (37.5 %) had median nerve neuropathy at the wrist, and 12 patients (37.5 %) had ulnar nerve neuropathy at the elbow in the symptomatic extremities. They concluded that entrapment neuropathies could be an important cause for morbidity after CVA.

The most dreaded consequence of peripheral nerve injury is *complex regional pain syndrome* (CRPS), which can be divided into "reflex sympathetic dystrophy" or RSD

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(*CRPS type I*, where there is no underlying nerve injury) and "causalgia" (*CRPS type II,* where there is an identified nerve injury). This condition most commonly develops after a fracture or soft tissue injury of an extremity or after a surgical procedure. For instance, Irwin and Schwartzman published a case report of a patient with chest wall dystonia and CRPS after brachial plexus injury [5]. Factor analysis demonstrates that signs and symptoms of the syndrome cluster into four subgroups: (1) abnormalities in pain processing that cause allodynia, hyperalgesia, and hyperpathia, (2) skin color and temperature change, (3) neurogenic edema and vasomotor and sudomotor abnormalities, and (4) a movement disorder and trophic changes [6]. The movement disorder manifests as a combination of difficulty initiating and maintaining movement, weakness, postural and intention tremor, *myoclonus*, spasm, increased tone, abnormalities of reaching and grasping, and dystonia [7]. *Dystonia* in CRPS is most likely a peripherally induced, focal dystonia [8].

Oaklander et al. [9] and Albrecht et al. [10], both in 2006, felt that some form of initial nerve trauma (and the subsequent ischemia) was "an important trigger for the cascade of events leading to CRPS" [11]. Coderre and Bennett [12] proposed that "the fundamental cause of the abnormal pain sensation is ischemia." They also suggested that ischemia provides a "unifying idea that relates the pathogenesis of CRPS-I to that of CRPS-II," suggesting that the distinction between the two diagnoses is a matter of degree and not pathology.

CRPS associated with *shingles* or *herpes zoster (HZ)* was first reported by Sudeck in 1901 [13] but had been surprisingly unrecognized until recently [13–15]. One hypothesis of this relationship involves the provocation of local tissue inflammation secondary to cytopathic changes induced by the HZ infection. These changes cause local trauma and a potential entrapment of the peripheral nerves [16], contributing to *postherpetic neuralgia (PHN)*, the devastating complication of HZ outbreaks. PHN is most often found in patients who had CRPS-like symptoms [14]. Berry et al. [17] prospectively reviewed 75 patients over 50 years old with pain score ≥20/100 at 2–6 weeks post-HZ onset and followed these patients for 6 months, evaluating for CRPS-like symptoms. Of these patients, 25 had involvement of the extremities; 89 % of those with distal extremity involvement had symptoms of CRPS at 3 months. Although they did not specifically address the cause of these symptoms, inflammation and subsequent entrapment of the peripheral nerve are likely.

Hypermobility may also contribute to both nerve entrapment and nerve stretch injury. Stoler and Oaklander described four patients with joint injuries who developed CRPS; all four also met criteria for Ehlers-Danlos hypermobility syndrome [18]. They hypothesized that the CRPS occurred via stretch injury to nerves crossing hypermobile joints, increased fragility of the nerve connective tissue, or nerve trauma from the more frequent surgeries needed to treat the hypermobile joints.

Patients in the early phases of CRPS are more likely to respond to sympathetic ganglion blocks, as their symptoms are still sympathetically mediated. In a trial of stellate ganglion and lumbar sympathetic ganglion blocks, the duration of pain relief was shown to be more than three times longer in responding CRPS patients using local anesthetic when compared to saline [19]. Aggressive physical therapy is also a mainstay of treatment; in treatment-resistant cases, many other modalities have been used, including indwelling nerve catheters, ketamine infusions, and spinal cord stimulation [20]. Though widely practiced, the evidence base is slim and inconsistent in this area.

However, these therapies presume that the *underlying process* is sympathetically mediated rather than being the *response to the process*. Coderre and Bennett hypothesize that the role of the sympathetic nervous system in CRPS-I "is a factor that is not fundamentally causative, but may have an important contributory role in early-stage disease" [12].

Dellon et al. [21] in 2009 reevaluated this entire concept and proposed that there is no real distinction between RSD (CRPS type I) and causalgia (CRPS type II). Rather, they hypothesized that chronic pain input to the dorsal spinal columns is misdiagnosed as CRPS-I and that chronic nerve compression and inflammation (i.e., CRPS-II) can be the source of these painful dorsal column inputs. They performed a retrospective review of 100 consecutive patients with the diagnosis of "RSD" based on the following criteria from the International Association for Study of Pain [22]:

Absolute: Pain extending outside the area of trauma, impaired extremity function, and either cold or warm perceptions or temperature changes in the affected extremity

Relative: Edema, increased or decreased hair or nail growth, hyperalgesia, allodynia, abnormal skin coloring

They identified 70 of those 100 patients who had documentation of chronic peripheral nerve entrapment based on abnormal sensory testing, a positive Tinel's sign at the site of known anatomic narrowing, and temporary response to a local anesthetic injection (without steroid). Of note was the relatively large volume used (5 cc), which could have contributed to more entrapment. They noted "good" to "excellent" relief in 80 % of the patients after surgical release. In another paper in 2010, Dellon and colleagues [23] reviewed the results of 13 patients treated for lower extremity CRPS with surgical release of peripheral nerve entrapments; 75 % noted good or excellent relief a minimum of 24 month later. In both papers, they concluded that more than 80 % patients with a diagnosis of "CRPS-I" of the upper or lower extremity could have one or more injured and/or compressed peripheral nerves as the source of their continuing dorsal

column painful neural input and should actually have a diagnosis of treatable "CRPS-II."

Summary

Peripheral nerve entrapment can cause a wide number of clinical presentations, many of which are misdiagnosed (and therefore less likely to respond to treatment). Recognition (and appropriate treatment) of the peripheral nerve entrapment can increase the potential for successful treatment.

References

1. Kline DG, Hudson AR. Nerve injuries: operative results for major nerve injuries, entrapments, and tumors. Philadelphia: W.B. Saunders; 1995.
2. Davies E, Pounder D, Mansour S, Jeffery IT. Cryosurgery for chronic injuries of the cutaneous nerve in the upper limb. Analysis of a new open technique. J Bone Joint Surg Br. 2000;82(3):413–5.
3. Padua L, Aprile I, Caliandro P, Foschini M, Mazza S, Tonali P. Natural history of ulnar entrapment at elbow. Clin Neurophysiol. 2002;113(12):1980–4.
4. Hunkar R, Balci K. Entrapment neuropathies in chronic stroke patients. J Clin Neurophysiol. 2012;29(1):96–100.
5. Irwin DJ, Schwartzman RJ. Complex regional pain syndrome with associated chest wall dystonia: a case report. J Brachial Plex Peripher Nerve Inj. 2011;6:6.
6. Harden RN, Oaklander AL, Burton AW, Perez RS, Richardson K, Swan M, et al. Complex regional pain syndrome: practical diagnostic and treatment guidelines, 4th edition. Pain Med. 2013;14(2): 180–229.
7. Schwartzman RJ, Patel M, Grothusen JR, Alexander GM. Efficacy of 5-day continuous lidocaine infusion for the treatment of refractory complex regional pain syndrome. Pain Med. 2009;10(2):401–12.
8. Schott GD. Peripherally-triggered CRPS and dystonia. Pain. 2007;130(3):203–7.
9. Oaklander AL, Fields HL. Is reflex sympathetic dystrophy/complex regional pain syndrome type I a small-fiber neuropathy? Ann Neurol. 2009;65(6):629–38.
10. Albrecht PJ, Hines S, Eisenberg E, Pud D, Finlay DR, Connolly MK, et al. Pathologic alterations of cutaneous innervation and vasculature in affected limbs from patients with complex regional pain syndrome. Pain. 2006;120(3):244–66.
11. Bruehl S. An update on the pathophysiology of complex regional pain syndrome. Anesthesiology. 2010;113(3):713–25.
12. Coderre TJ, Bennett GJ. A hypothesis for the cause of complex regional pain syndrome-type I (reflex sympathetic dystrophy): pain due to deep-tissue microvascular pathology. Pain Med. 2010;11(8):1224–38.
13. Öztop P, Saraçgīl Coşar SN, Ümīt Yemīşcī O, Varol Üre RS. Complex regional pain syndrome associated with herpes zoster: a case report. Turk J Rheumatol. 2011;26(3):254–7.
14. Gimenez-Mila M, Busquets C, Ojeda A, Fauli A, Moreno LA, Videla S. Neuropathic pain with features of complex regional syndrome in the upper extremity after herpes zoster. Pain Pract. 2014;14(2):158–61.
15. Louizos L, Kremastinou F, Panaretou V, Skalistira M, Kouroukli I, Georgiou L. Complex regional pain syndrome after herpes zoster: case report. Eur J Pain. 2006;10(Suppl S1):S204.
16. Ecker A. Pathogenesis of reflex sympathetic dystrophy. Arch Neurol. 1989;46(5):482.
17. Berry JD, Rowbotham MC, Petersen KL. Complex regional pain syndrome-like symptoms during herpes zoster. Pain. 2004; 110(1–2):e1–12.
18. Stoler JM, Oaklander AL. Patients with Ehlers Danlos syndrome and CRPS: a possible association? Pain. 2006;123(1–2):204–9.
19. Price DD, Long S, Wilsey B, Rafii A. Analysis of peak magnitude and duration of analgesia produced by local anesthetics injected into sympathetic ganglia of complex regional pain syndrome patients. Clin J Pain. 1998;14(3):216–26.
20. O'Connell NE, Wand BM, McAuley J, Marston L, Moseley GL. Interventions for treating pain and disability in adults with complex regional pain syndrome. Cochrane Database Syst Rev. 2013;(4):Cd009416.
21. Dellon AL, Andonian E, Rosson GD. CRPS of the upper or lower extremity: surgical treatment outcomes. J Brachial Plex Peripher Nerve Inj. 2009;4:1.
22. Merskey H, Bogduk N. Classification of chronic pain: descriptions of chronic pain syndromes and definitions of pain terms. 4th ed. Seattle: International Association for the Study of Pain (IASP) Press; 1994.
23. Dellon L, Andonian E, Rosson GD. Lower extremity complex regional pain syndrome: long-term outcome after surgical treatment of peripheral pain generators. J Foot Ankle Surg. 2010; 49(1):33–6.

Pharmacological Treatment of Painful Nerve Entrapment Syndrome

4

Tiffany Zhang

Introduction

Clinical presentation of nerve entrapment syndrome (NES) is directly or indirectly related to the compression of or damage to peripheral nerve fibers. Pain is often the first sign, and probably the most disturbing symptom, that patients seek health care for. Alleviating pain is an important goal of nerve entrapment syndrome treatment.

There is limited literature available about pharmacological treatment specifically for NES [1–3]. In principle, the pharmacological treatment of NES is the same as that of neuropathic pain syndrome. The advantage of pursuing medication treatment first is that some NES may spontaneously resolve over time, without the need for more invasive interventions [4].

Both systemic medicine and topical agents, as monotherapy or combined therapy, can be utilized for pain relief associated with NES. Those medications can be categorized as follows:

Systemic medications
- Nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen
- Antidepressants
- Anticonvulsants
- Opioid medication

Topical medications
- Local anesthetic (e.g., lidocaine)
- Capsaicin

NSAIDs
- COX-1 and COX-2

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Other Investigational Topical Compounds

The International Association for the Study of Pain (IASP) recommended first-line agents for neuropathic pain treatment including topical lidocaine, antidepressants such as tricyclics (TCAs), serotonin-norepinephrine reuptake inhibitors (SNRIs), and calcium channel α 2-delta ligands (gabapentin and pregabalin). The second-line agents recommended are tramadol and opioids [5, 6].

Basic pharmacology (as a category), clinical application including recommended dosages, common side effects, and precautions are discussed in the following sections.

Systemic Medications

Nonsteroidal Anti-inflammatory Drugs (NSAIDs) and Acetaminophen

NSAIDs are a group of medications widely used for various painful conditions, particularly for somatic pain. Commonly used agents in this group include aspirin, ibuprofen, naproxen, diclofenac, and newer compounds such as piroxicam and celecoxib.

Acetaminophen is included in this group, although it is not an anti-inflammatory agent. It is commonly combined with opioid medications to reduce the amount of opioid needed.

Pharmacology

The analgesic effect of NSAIDs derives mainly from its peripheral action on the enzyme cyclooxygenase (COX) blockade, resulting in inhibition of prostaglandin synthesis, which plays a central role in inflammatory conditions [7]. NSAIDs do not readily cross blood-brain barrier. NSAIDs are extensively metabolized in the liver, and they have low renal clearance. NSAIDs are subcategorized to nonselective COX inhibitors (COX-1 and COX-2) and selective COX-2 inhibitors (coxibs) [8, 9].

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Table 4.1 Acetaminophen and NSAID dose recommendations

Drug	Analgesic dose schedule	Maximum daily dose
Acetaminophen	325–650 mg every 4–6 h	3,000 mg
		Lower maximum dose threshold for patient with abnormal LFT
Diclofenac	50–75 mg every 8 h	150 mg
Ibuprofen	200–800 mg every 4–6 h	2,400 mg
Meloxicam	7.5–15 mg every 24 h	15 mg
Naproxen	250 mg every 8 h or 500 mg every 12 h	1,000 mg
Piroxicam	10–20 mg every 24 h	20 mg
Celecoxib	100 mg every 12 h or 200 mg every 24 h	400 mg[a]

[a]The cardiac risk associated with celecoxib is dose dependent [11]

The mechanism of acetaminophen as analgesic agent is unclear; it is generally considered a centrally acting agent.

Clinical Applications

There are many NSAIDs available, and there is little evidence of substantial efficacy of one NSAID agent over another [10]. A patient who does not tolerate or respond to a particular NSAID may do well on another. Compliance maybe improved by using a twice a day or once daily schedule. The analgesic dosing recommendation and maximum daily dose of commonly used agents are summarized in Table 4.1.

Medicine Side Effects and Contraindications

Acetaminophen and NSAIDs are generally recommended for short-term use only. The main concern for acetaminophen is its hepatic toxicity; therefore doses should be lowered or avoided entirely in patients with compromised liver function. There are a multitude of side effects associated with NSAIDs, which are summarized in Table 4.2.

Antidepressants

Antidepressants are a diverse group of medications approved for treatment of major depressive disorders or other related psychiatric disorders. Many clinical experiences and studies have also demonstrated their efficacy and superiority in pain treatment, especially with neuropathic pain [6, 13–15]. Antidepressants can improve the depressive symptoms associated with pain, but analgesic effects are also believed to be independent of depression control. Among those, tricyclic antidepressants (TCAs) such as amitriptyline, nortriptyline, desipramine, and imipramine have been the mainstays of neuropathic pain treatment, with efficacy proven in several well-designed studies [14–18]. TCAs are inexpensive, and their long half-life made dosing simple. The sedation side effect is also a desirable therapeutic effect, since many patients with pain have difficulty sleeping.

Table 4.2 Common side effects and contraindications of NSAIDs

Organ systems	Common side effects	Contraindications
Gastrointestinal (GI)	Dyspepsia, peptic ulcer, GI bleeding; risk is higher if concurrent glucocorticoid use	Active or history of peptic ulcer or GI bleeding or bleeding diathesis
Hematology	Decreased hemostasis, surgical bleeding	Immediate perioperative period
Nephrology	Reversible nephrotoxicity	Renal insufficiency or failure
Cardiovascular	Hypertension, fluid retention, increased myocardial infarction with coxibs (dose dependent); risk is lower if concurrent use of aspirin (ASA)	Known history of myocardial infarction or severe coronary artery disease, use in lowest possible dose, especially celecoxib [11]
Drug-drug interaction	Warfarin dose may need to be adjusted due to the platelet effects of NSAIDs; reduced NSAIDs dose is also recommended in patient with severe hypoalbuminemia [12]	

Antidepressants with clinically proven analgesic efficacy are TCAs, SNRIs, dopaminergic bupropion, and tetracyclic maprotiline [14, 16, 17, 19].

Pharmacology

The exact mechanism for analgesic effects of antidepressants is not clear. Putative mechanisms include inhibition of norepinephrine and/or serotonin reuptake at synapses of centrally descending pain modulation pathways, as well as the antagonism of N-methyl-D-aspartate (NMDA) receptors, which mediate hyperalgesia and allodynia [6, 14]. In addition, TCAs are also potent sodium channel blockers, and some have significant sympatholytic effect [20].

Clinical Applications

Analgesic dose range of antidepressants may be different from the dose required for depression treatment. For instance, the analgesic dose of TCAs is generally lower than the dose for depression treatment, while the analgesic dose for duloxetine and venlafaxine is generally higher than the dose for depression treatment [14]. Patients should be educated that it may take weeks to obtain any significant benefit. If one antidepressant failed, either from intolerable side effect or ineffectiveness, another can always be tried.

The recommended analgesic dose range and the maximum daily dose are summarized in Table 4.3.

Table 4.3 Analgesic dose range of antidepressants and the maximum daily dose

Drug	Analgesic dose schedule	Maximum daily dose
TCAs		
Amitriptyline	25–75 mg once daily in the evenings[a]	200 mg
Nortriptyline	25–75 mg once daily in the evenings[a]	150 mg
Imipramine	25–150 mg once daily in the evenings[a]	200 mg
Desipramine	25–150 mg once daily in the evenings[a]	300 mg
SNRIs		
Duloxetine	30–60 mg once daily	120 mg
Venlafaxine	37.5–225 mg/day in 2–3 doses (immediate release) or once daily (extended release)	375 mg
Milnacipran	12.5–50 mg 2 times a day	200 mg
Tetracyclics		
Maprotiline	25–50 mg once daily in the evening	225 mg
Mirtazapine	15–30 mg once daily in the evening	45 mg
Bupropion	Immediate release 100–150 mg 2 or 3 times a day or extended release 150–450 mg once daily (XL)	450 mg 450 mg

[a]TCAs are usually given in the evening due for their profound sedating effect

Medicine Side Effects and Contraindications

Antidepressants are associated with multiple undesirable side effects that vary depending on the individual agent and, often, the dosage. TCAs have the most side effects that limit their clinical use. The more mild side effects, such as dry mouth, mental clouding, and oversedation, may diminish in days or weeks. The dose should be increased gradually to effect or be diminished to limit the intolerable side effects.

The most serious risk of antidepressants is suicide in major depressive patients, especially children, adolescents, and young adults. Antidepressants also have other serious side effects, including serotonin syndrome and cardiac effects (e.g., QT prolongation, AV blocks, arrhythmias). Patients taking monoamine oxidase inhibitors (MAOIs) should avoid taking TCAs, due to the increased risk of serotonin syndrome. TCAs are relatively contraindicated in patients with severe cardiac disease, especially conduction disturbances. The starting dose and maintenance dose should be lower for elderly patients, and cognitive impaired elderly should avoid TCAs completely.

The common side effects and contraindications of antidepressants are summarized in Table 4.4.

Table 4.4 The common side effects and contraindications of antidepressants

Mechanisms	Common side effects	Contraindications
Anticholinergic (TCAs)	Nausea, constipation, urinary retention, dry mucosa, cognitive impairment	Elderly with cognitive impairment; lower starting and maintenance dose in elderly
Antihistamine (TCAs)	Sedation[a], drowsiness, lethargy	
Cardiovascular (TCAs)	Orthostatic hypotension, arrhythmia	Severe cardiac disease, especially conduction disturbances
Serotonin (except bupropion)	Serotonin syndrome	Avoid concurrent MAOI drugs
Others	Sexual dysfunction, weight gain	

[a]Amitriptyline is most sedating; desipramine is least sedating

Anticonvulsants

Anticonvulsants have been used in the management of neuropathic pain for many years [15, 21–26]. Many large-scale clinical studies have demonstrated their efficacies in treating neuropathic pain. Most of the evidence was from studies on postherpetic neuralgia (PHN), painful diabetic peripheral neuropathy (DPN) [15, 21–24], and trigeminal neuralgia [27, 28]. Within the category of anticonvulsants, gabapentin, pregabalin, and carbamazepine are all approved by the FDA for neuropathic pain treatment. Many other anticonvulsants have also been used as analgesics, though there have been only small studies and clinic reports on their effectiveness.

Pharmacology

The analgesic effect of anticonvulsants is considered to be through binding to voltage-gated ion channels and therefore inhibiting neurotransmitter release. The ion channels are either calcium mediated (gabapentin and pregabalin) or sodium mediated (carbamazepine, oxcarbazepine, topiramate). Other proposed mechanisms of analgesic actions may include inhibition of release of excitatory amino acids, augmentation of CNS inhibitory pathways by increasing gamma-aminobutyric acid (GABA)-ergic transmission [14, 15, 24].

Clinical Application

Medications should be initiated at a low dose with gradual increase until pain relief, or until the dose needs to be limited to decrease undesirable side effects. Among the analgesic anticonvulsants, gabapentin is more popular than others for neuropathic pain treatment. This is related to its ease of monitoring, relatively low incidence of serious adverse events, and less drug-drug interaction [14].

Table 4.5 The recommended analgesic dose range and maximum daily dose of anticonvulsants

Drug	Dosage	Maximum daily dose
Gabapentin	600–900 mg 3 times a day	3,600 mg
Pregabalin	50–150 mg 3 times a day	600 mg
Carbamazepine	100–2,400 mg/day divided to 2 doses	2,400 mg

The analgesic dose of gabapentin is at 1,800–2,400 mg, divided to three times a day; however, up to 3,600 mg/day may be required in some patients.

Pregabalin may provide analgesia more quickly than gabapentin, and the therapeutic dose is generally between 300 and 450 mg/day, but a dose of up to 600 mg/day has been deemed safe [29]. Pregabalin can be taken two or three times a day.

Carbamazepine (or its derivative, oxcarbazepine) has been used in trigeminal neuralgia treatment, but there is limited evidence of its use in other neuropathic pain. The dose range for trigeminal neuralgia pain control is as wide as 100–2,400 mg/day [27, 28].

Other anticonvulsants, including topiramate, lamotrigine, and levetiracetam, have been used as adjunct analgesic agents. However, those medications have been utilized anecdotally. So far, there is no robust evidence of their effectiveness in treatment of neuropathic pain [27].

The recommended analgesic dose range and maximum daily dose of anticonvulsants are summarized in Table 4.5.

Medicine Side Effects and Contraindications

Anticonvulsants are generally well tolerated; common side effects include dizziness, ataxia, and somnolence.

Gabapentin and pregabalin doses must be adjusted in patients with renal failure (calculated dose reduction is based on glomerular filtration rate [GFR], or for patients on dialysis, and is available in many online drug sites, for instance, UpToDate at www.uptodate.com [30]). No adjustment is necessary in patients with hepatic disorders, as the drug is excreted through the kidneys unmetabolized.

It should be pointed out that carbamazepine can cause serious and sometimes fatal dermatologic reactions, including Stevens-Johnson syndrome and toxic epidermal necrolysis. Also, a potential rare hematological reaction of irreversible aplastic anemia was reported. Potential hepatic impairment necessitates regular hematologic and hepatic function monitoring.

Opioids

The use of opioids in the treatment of acute and severe pain is well studied and well established [31–34]. However, their utilization in chronic neuropathic pain treatment remains controversial, and they are currently considered a second-line therapy for neuropathic pain [5, 32–35]. Opioid therapy should be reserved for patients with severe pain who have failed non-opioid regimens and have pain that causes a severe impact on function and quality of life. Opioids should be used for as short a duration as possible.

Among the opioid agents, three unique drugs (tramadol, tapentadol, and a partial mu receptor agonist, buprenorphine) have some advantages in management of chronic neuropathic pain and will be discussed in more detail [36–40].

Pharmacology

Opioids exert analgesic effects through binding and activating their receptors. There are three classic opioid receptors – mu, kappa, and delta. Activation of the mu receptor is responsible for the majority of an opioid's analgesic effects, as well as many side effects, such as nausea, constipation, euphoria, and respiratory depression.

Buprenorphine is a unique member of this family in that it is a partial mu receptor agonist combined with kappa antagonism. This offers less abuse potential and a favorable safety profile, such as the "ceiling effect" associated with less respiratory depression risks [38, 39].

Other mechanisms are also involved. For example, tramadol inhibits the reuptake of serotonin and norepinephrine, while tapentadol inhibits norepinephrine reuptake. Therefore, both are relatively effective in neuropathic pain management, although both are weak mu receptor agonists [36, 37]. Methadone is also an NMDA antagonist and inhibits reuptake of serotonin and norepinephrine, in addition to its strong mu receptor agonism.

Clinical Applications

Opioids can be administered through different routes, including oral, intravenous, transdermal, transmucosal, and intrathecal. There are also many different dose forms, immediate-release agents, or extended-release oral tablets available. There are well-established guidelines readily available about opioid therapies [41, 42]. As such, opioids will not be the focus of this chapter, except tramadol and tapentadol, because of their special use in neuropathic treatment, and buprenorphine, because of its relative novelty in the United States and potential usefulness in neuropathic pain management. Buprenorphine transdermal patches are approved by FDA for chronic pain management. Buprenorphine transoral agents (sublingual tablets or films, with or without opioid antagonist naloxone) are approved for substance use disorder treatment (special training and qualification are required to prescribe for addiction treatment) and are used off label for chronic pain management [38–40]. Dosage recommendations are outlined below and summarized in Table 4.6.

Table 4.6 Analgesic dose range and maximum daily dose of tramadol, tapentadol, and buprenorphine

Drug	Dosage	Maximum daily dose
Tapentadol	Immediate release:	Immediate release: 400 mg
	50–100 mg every 6 h	
	Extended release: 100–300 mg once daily	Extended release: 300 mg
Tramadol	Immediate release: 50–100 mg every 6 h	Immediate release: 700 mg
	Extended release: 100–250 mg every 12 h	Extended release: 500 mg
Tramadol/ acetaminophen	37.5/325 mg every 4–6 h	8 tablets (dose limited by acetaminophen)
Buprenorphine transdermal patch (Butrans)	5–20 mcg/h every 7 days	20 mcg/h (dose ≥20 mcg/h increases risk of QTc prolongation) [40]
Buprenorphine sublingual tablets or films (Subutex or Suboxone)	2–24 mg/day, once a day or divided doses	32 mg/day

Both tramadol and tapentadol have immediate-release and extended-release options. Currently, only oral formulations are available. Tapentadol can be given at a 50–100 mg dose every 6 h as needed; an extended-release (ER) formulation, taken every 12 h, is available in multiple strengths (50, 100, 150, 200, and 250 mg). The maximum daily dose is 700 mg for immediate release and 500 mg for extended release. Tramadol can be given at a 50–100 mg dose every 6 h as needed or at a 100 mg extended-release (ER) increment, taken once daily. The maximum daily dose is 400 mg for immediate release and 300 mg for the ER formula. There are also tramadol and acetaminophen combination tablets (tramadol 37.5 mg/acetaminophen 325 mg), with a maximum daily dose of eight tablets.

Buprenorphine is available in an IV formulation (Buprenex) and a transdermal patch (Butrans) at doses from 5 to 20 mcg/h, applied once every 7 days. Sublingual tablet or film (Subutex, buprenorphine; Suboxone, buprenorphine plus naloxone) dosages range from 2 to 24 mg per day, taken once daily or in divided doses.

Medicine Side Effects and Contraindications

Opioids have many side effects. The most common ones are nausea, constipation, cognitive impairment or mental cloudiness, and sedation. However, the most serious side effect is respiratory depression, especially with concurrent use of other respiratory depression drugs. This combination is often the cause of death from drug overdose.

Long-term use often results in drug dependence and tolerance. It may also lead to opioid-induced hyperalgesia including allodynia, which was demonstrated in animal models, although there is not yet strong evidence in clinic relevance [43–45].

Relative contraindication for long-term use of opioids is personal history of past drug or other substance abuse, with an absolute contraindication in patients with ongoing drug abuse.

Additionally, tramadol lowers the seizure threshold (dose dependent). This increases the risk for seizures, particularly in patients concurrently taking drugs that decrease the seizure threshold.

Topical Analgesics

Pain associated with NES is often localized at, or distal to, the area of nerve compression or entrapment, making topical analgesics a viable modality. The IASP guideline recommends topical lidocaine as one of the first-line therapies for neuropathic pain [5].

Topical agents have several advantages over systemic drugs, including higher concentration at the area of nerve damage or dysfunction; minimal systemic side effect and drug-drug interaction, due to minimal systemic absorption and low blood level; and ease of use with, generally, no need to titrate dose.

Currently, the two topical agents approved by the FDA for neuropathic pain treatment are a lidocaine patch 5 % and a capsaicin patch 8 % [46–48]. A third topical agent, diclofenac (gel concentration 1 %, patch concentration 1.3 %), is approved in the United States by the FDA for acute pain caused by minor strains, sprains, and contusions. In other countries, it is also approved for the treatment of osteoarthritis [49]. However, because of its anti-inflammatory nature and analgesic effect, it may have some role in the treatment of neuropathic pain. There are also other investigational topical compounds made by compounding pharmacies. These drugs include clonidine, amitriptyline, baclofen, ASA, and others, either monotherapy or combination; however, there is not enough study supporting their efficacy [46].

Pharmacology

The exact mechanism of action is not entirely clear. The mechanism for topical lidocaine is speculated as a sodium channel blocker, which reduces ectopic nerve discharge in a damaged nerve. Capsaicin decreases transient receptor potential vanilloid-1 (TRPV1) expression in nociceptive nerve endings and reduction in the density of epidermal nerve fibers in the application area [50].

Clinical Use

Capsaicin Patch 8 %

Apply the patch to the most painful area for 60 min. The patch can be cut to match the size and shape of the treatment area, and up to four patches may be applied in a single application. To prevent severe irritation from the high concentration of capsaicin, the area should be pretreated with a topical anesthetic prior to patch application. This treatment may be repeated every 3 months if pain returns [30].

Lidocaine Patch 5 %

Apply the patch to the most painful area. The patch can be cut to match the size and shape of the treatment area, and up to three patches may be applied in a single application. These patches may remain in place for up to 12 h in any 24-h period [30].

Medication Side Effects and Contraindications

The side effects of topical analgesics are most often related to the focal skin reaction to adhesives; systemic side effects are rare, due to the very low level of systemic absorption [46].

The patches should not be applied to mucous membranes or broken or inflamed skin to avoid excess systemic absorption [30].

References

1. Bare AA, Haddad SL. Nerve entrapment syndromes. In: Thordarson DB, editor. Foot and ankle. Philadelphia: Lippincott, Willams & Wilkins; 2004. p. 79–97.
2. Morganti CM, McFarland EG, et al. Saphenous neuritis: a poorly understood cause of medial knee pain. J Am Acad Orthop Surg. 2002;10:130–7.
3. Pecina M, Markiewitz AD, et al. Tunnel syndrome in athletes in tunnel syndromes: peripheral nerve compression syndrome. New York: Taylor & Francis; 2001. p288.
4. Flaigan RM, DiGiovanni BF. Peripheral nerve entrapments of the lower leg, ankle, and foot. Foot Ankle Clin N Am. 2011;16: 255–74.
5. Leon-Casasola OD. New developments in the treatment algorithm for peripheral neuropathic pain. Pain Med. 2011;12:S100–8.
6. Dworkin RH, O'Connor AB, et al. Pharmacologic management of neuropathic pain: evidence-based recommendations. Pain. 2007;132:237–51.
7. McCormack K. Non-steroidal anti-inflammatory drugs and spinal nociceptive processing. Pain. 1994;59:9.
8. Gajraj N. Cyclooxygenase-2 inhibitors. Anesth Analg. 2003;96: 1720–38.
9. Laneuvill O, et al. Differential inhibition of human prostaglandin endoperoxide H synthases-1 and -2 by nonsteroidal anti-inflammatory drugs. J Pharmacol Exp Ther. 1994;271:927–34.
10. Roelofs PD, Deyo RA, et al. Nonsteroidal anti-inflammatory drugs for low back pain: an updated Cochrane review. Spine (Phila PA 1976). 2008;33L:1766.
11. Solomon SD, Hawk E, et al. Cardiovascular risk of celecoxib in 6 randomized placebo-controlled trials: the cross trial safety analysis. Circulation. 2008;117:2104–13.
12. Ballantyne JC, Barna SA. Nonsteroidal anti-inflammatory drugs. In: Ballantyne JC, editor. The Massachusetts General Hospital handbook of pain management. Philadelphia: Lippincott Williams & Wilkins; 2006. p. 97–103.
13. Traynor LM, Thiessen CN, Traynor AP. Pharmacotherapy of fibromyalgia. Am J Health-Syst Pharm. 2011;68:1307.
14. Sullivan M, Robinson JP. Antidepressant and anticonvulsant medication for chronic pain. Phys Med Rehabil Clin N Am. 2006;17:381–400.
15. Singleton JR, Smith AG. The diabetic neuropathies: practical and rational therapy. Semin Neurol. 2012;32:196–203.
16. Saarto T, Wiffen PJ. Antidepressants for neuropathic pain: a Cochrane review. J Neurol Neurosurg Psychiatry. 2010;81:1372.
17. Sindrup SH, Otto M, et al. Antidepressants in the treatment of neuropathic pain. Basic Clin Pharmacol Toxicol. 2005;96:399.
18. Max MB, Muir J, et al. Effects of desipramine. Amitriptyline and fluoxetine on pain relief in diabetic neuropathy. N Engl J Med. 1996;326:1250.
19. Collins SL, Moore RA, et al. Antidepressants and anticonvulsants for diabetic neuropathy and postherpetic neuralgia: a quantitative systematic review. J Pain Symptom Manage. 2000;20:449.
20. Deffois A, Carter C, et al. Inhibition of synaptosomal veratridine-induced sodium influx by antidepressants and neuroleptics used in chronic pain. Neurosci Lett. 1996;220:117.
21. Boyle J, Kerr D, et al. Randomized, placebo-controlled comparison of amitriptyline, duloxetine, and pregabalin in patients with chronic diabetic peripheral neuropathic pain: impact on pain, polysomnographic sleep, daytime functioning, and quality of life. Diabetes Care. 2012;35:2451.
22. Tanenberg RJ, Malcolm SK, et al. Duloxetine, pregabalin, and duloxetine plus gabapentin for diabetic peripheral neuropathic pain management in patients with inadequate pain response to gabapentin: an open-label, randomized, noninferiority comparison. Mayo Clin Proc. 2011;86:615.
23. Gilron I, Lemay J, et al. Pregabalin for peripheral neuropathic pain: a multicenter, enriched enrollment randomized withdrawal placebo-controlled trial. Clin J Pain. 2011;27:185.
24. Backnonia M, Beydoun A, Edwards KR, et al. Gabapentin for the symptomatic treatment of painful neuropathy in patients with diabetes mellitus: a randomized controlled trial. JAMA. 1998;280(21): 1831–6.
25. Rice AS, Maton S. Gabapentin in postherpetic neuralgia: a randomized, double blind, placebo controlled study. Pain. 2001;94:215.
26. Serpell MG. For the neuropathic pain study group: gabapentin in neuropathic pain syndromes: a randomized, double-blind, placebo-controlled trial. Pain. 2002;99:557.
27. Gronseth G, Cruccu G, et al. Practice parameter: the diagnostic evaluation and treatment of trigeminal neuralgia (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology and the European Federation of Neurological Societies. Neurology. 2008;71:1183.
28. Martin WJJ, Forouzanfar T. The efficacy of anticonvulsants on orofacial pain: a systematic review. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2011;111:627.
29. Arezzo JC, Rosenstock J, Lamoreaux L, Pauer L. Efficacy and safety of pregabalin 600mg/day for treating painful diabetic peripheral neuropathy: a double-blind placebo-controlled trial. BMC Neurol. 2008;8:33.
30. Drug information website. www.uptodate.com/lexicomp.
31. Von Korff M, Kolodny A, et al. Long-term opioid therapy reconsidered. Ann Intern Med. 2011;155:325.
32. Ballantyne JC, Mao J. Opioid therapy for chronic pain. N Engl J Med. 2003;349:1943.

33. Chou R, Fanciullo GJ, et al. Clinical guidelines for the use of chronic opioid therapy in chronic noncancer pain. J Pain. 2009;10:113.
34. Furlan AD, Sandoval JA, et al. Opioids for chronic noncancer pain: a meta-analysis of effectiveness and side effects. CMAJ. 2006; 174:1589.
35. Foley KM. Opioids and chronic neuropathic pain. N Engl J Med. 2003;348:1279.
36. Duhmke RM, Cornblath DD, et al. Tramadol for neuropathic pain. Cochrane Database Syst Rev. 2004;2:CD003276.
37. Schwartz S, Etropolski M, Shapiro DY, et al. Safety and efficacy of tapentadol ER in patients with painful diabetic peripheral neuropathy: results of a randomized-withdrawal, placebo-controlled trial. Curr Med Res Opin. 2011;27:151–62.
38. Cote J, Montgomery L. Sublingual buprenorphine as an analgesic in chronic pain: a systematic review. Pain Med. 2014;15:1171.
39. Li X, Kosten TR, et al. Buprenorphine in the treatment of opioid addiction: opportunities, challenges and strategies. Expert Opin Pharmacother. 2014;15:2263.
40. Buprenorphine: drug information. http://www.uptodate.com/contents/buprenorphine-drug-information.
41. American Society of Interventional Pain Physicians (ASIPP) guidelines for responsible opioid prescribing in chronic non-cancer pain: part 2 - guidance. http://www.guideline.gov/content.aspx?id=38257&search=opioid.
42. Managing chronic non-terminal pain in adults including prescribing controlled substances. http://www.guideline.gov/content.aspx?id=25657&search=opioid.
43. Chu LF, Angst MS, et al. Opioid-induced hyperalgesia in humans: molecular mechanisms and clinical considerations. Clin J Pain. 2008;24:479.
44. Fishbain DA, Cole B, et al. Do opioids induce hyperalgesia in humans? An evidence-based structured review. Pain Med. 2009;10:829.
45. Brush DE. Complications of long-term opioid therapy for management of chronic pain: the paradox of opioid-induced hyperalgesia. J Med Toxicol. 2012;8:387–92.
46. Zur E. Topical treatment of neuropathic pain using compounded medications. Clin J Pain. 2014;30(1):73–91.
47. Derry S, Moore RA. Topical capsaicin (high concentration) for chronic neuropathic pain in adults. Cochrane Database Syst Rev. 2013;2:CD 007393.
48. Simpson DM, Brown S, Tobias JK, Vanhove GF. NGX-4010, a capsaicin 8 % dermal patch, for the treatment of painful HIV-associated distal sensory polyneuropathy: results of a 52-week open-label study. Clin J Pain. 2014;30(2):134–42.
49. McCarberg BH, Argoff CE. Topical diclofenac epolamine patch 1.3% for treatment of acute pain caused by soft tissue injury. Int J Clin Pract. 2010;64:1546–53.
50. Vorobeychik Y, Chen L. Combination therapy for neuropathic pain – a review of current evidence. CNS Drugs. 2011;25:1023.

Non-pharmacologic Treatment of Peripheral Nerve Entrapment

5

Helen W. Karl, Heather Tick, and Kris A. Sasaki

Introduction

Many non-pharmacologic treatments for pain, such as manipulation [1], massage therapy [2], and acupuncture, date back thousands of years. With the rise of "Western" biomedicine and increasingly powerful drug companies in the last century, these methods were often sidelined as quackery [3]. The difficulty of evaluating many of these therapies with the gold-standard randomized clinical trials, which are well suited to medication trials, contributed to their marginalization [4]. Because of their efficacy in many situations, however, patients continue to use a variety of non-pharmacologic therapies. Currently, increasingly sophisticated visualization [5–10], anatomic [11–16], biochemical [17–21], and systematic data evaluation [22–30] techniques are able to show objective tissue characteristics and response to therapies.

Myofascial pain syndrome (MFPS) symptoms may mimic other conditions commonly thought to require invasive interventions, such as carpal tunnel syndrome, herniated discs with sciatica, and thoracic outlet syndrome, among others. If the myofascial component is recognized and treated, symptoms often abate, leading to less need for analgesic and anti-inflammatory medication, invasive procedures, and surgery. Unfortunately, many primary care practitioners and pain practitioners have not been taught about this cause of morbidity [31].

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Patients with severe acute injuries may initially need passive modalities (see below) and traditional pharmacologic intervention to allow sufficient reduction of excess inflammation before beginning more intensive manual therapies (Table 5.1). Those with less severe acute injuries should be encouraged to start manipulative intervention early, while those with chronic pain will benefit from the addition of the non-pharmacologic therapies with effects synergistic to those achieved with traditional pharmacology.

Myofascial Pain Syndromes

MFPS are characterized by diffusely aching pain and tenderness in one or more taut and shortened muscles, as well as hypersensitive areas known as *myofascial trigger points* (MTrPs) [32]. The pain is often referred, rarely in the distribution of a peripheral nerve or spinal segment [33], but rather in the predictable pattern mapped by Travell and Simons [32]. Unless palpation of the painful site evokes signs of acute tenderness, the source is likely a MTrP somewhere

Table 5.1 Therapeutic approaches to MFPS, in order of intensity

Manual therapies	Needle therapies
Patient education on postural habits and activity modification	Acupuncture (acupressure, traditional and electroacupuncture)
Therapeutic exercise	Direct or remote intramuscular stimulation (dry needling)
Myofascial mobilization	Local anesthetic injection
Massage (including Swedish, shiatsu, self-massage, structural integration (Rolfing), craniosacral)	
Instrument assisted (Astym®, Graston®)	
Neural mobilization (neural flossing)	
Joint manipulation or mobilization	

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else. Pressure on the most tender area often reproduces the pain pattern, and massage or injection of local anesthetic into that site relieves the pain for a period of time, usually much greater than the duration of the drug [32]. The onset of MFPS is usually a muscle injury that activates an MTrP; this can be due to acute trauma, cumulative repetitive overload, or a peripheral nerve injury. The intensity of the symptoms reflects the degree of irritability of the MTrP rather than the size of the muscle.

In addition to taut and shortened painful muscles, patients with MFPS may have localized weakness and autonomic changes, such as abnormal pilomotor, sudomotor, and vasomotor phenomena. Tendons can become thickened or enthesopathic, with traction on tendon attachments and the potential for compression of other structures, particularly peripheral nerves. Symptoms presenting in a specific individual depend on the surrounding anatomical structures; for example, if myofascial dysfunction causes nerve entrapment, it is likely to result in localized neuropathic pain with radiation and dysesthesias.

Clinical characteristics of the MTrPs in these muscles have been well described [32]. These extremely tender nodules, associated with taut muscle bands, cause local and referred pain, and they produce a spinal cord reflex known as a *local twitch response (LTR)* when stimulated by snapping palpation or needle penetration. Travell and Simons proposed a theory of the etiology of myofascial abnormalities known as the integrated trigger point hypothesis [32]. MTrPs are thought to result from excess acetylcholine at the neuromuscular junction, leading to persistent muscle contraction and an "energy crisis" in this localized area with increased demand and decreased supply. Gunn's postulate that muscle dysfunction is caused by subtle compression of the nerve roots, which creates nerve dysfunction equivalent to partial denervation, remains controversial [34], although nerve root compression likely contributes to the "double crush" phenomenon [35] (see Chap. 1).

The advent of technologies to assess the functional derangements of these anatomic muscle disorders allows more to be learned about their properties. In a selective review of animal studies, Mense described the ways that algesic (pain-causing) agents, most commonly ATP and low tissue pH, excite muscle nociceptors. Muscle spasm and other causes of chronic ischemia, abnormal posture, inflammation, and MTrPs are all associated with low tissue pH. Chronic activation of muscle nociceptors can lead to central sensitization [9, 36]. Sikdar et al. have demonstrated hypoechoic areas on ultrasound that correspond to MTrPs and have used elastography to show that MTrPs are stiffer than normal muscle [7]. Shah et al. have microdialysed the interstitial fluid at MTrPs and found an inflammatory milieu in the trigger points, but not in an unaffected muscle [17].

Investigations at the tissue and cellular level have also been useful to identify mechanisms underlying *fascial dysfunction*. Fascia is a three-dimensional continuous network of cells (mostly fibroblasts) and intercellular fibers which envelops and divides all the other components of the body [37–40]. It supplies structural support, transmits forces between muscles [41, 42], is highly innervated with *mechanoreceptors* and *autonomic fibers* [43], and has *piezoelectric* properties [44, 45]. Fascia may become shortened by acute injuries such as trauma or inflammation and by chronically abnormal posture or repetitive use [44], but it has a remarkable ability to reform [43, 46]. Any kind of mechanical loading affects the extracellular matrix and increases the number and function of embedded fibroblasts. Application of acute or chronic mechanical loads at higher "doses," as seen in acute trauma or repetitive motion strain, leads to the release of the inflammatory mediators described above. In contrast, lower loads, as seen in cyclic mechanical stretch or massage, release anti-inflammatory compounds and stimulate collagen metabolism [47, 48]. Clinically, muscle and fascia are usually injured and treated in concert; fibroblasts appear to provide important connections between them at a subcellular level [48, 49].

Taken together, these and many more objective examinations of muscle and fascial dysfunction provide insight into the complex etiology of MFPS and an evidence base for the success of a variety of clinical approaches to its relief. Manual medical therapies and needle therapies are the two broad approaches found to be effective for the relief of myofascial pain.

Manual Medical Treatments (Table 5.2)

Numerous manipulative techniques have developed over the last two centuries and have been practiced by often competing professionals [1, 20]. Modern scientific publication on manipulative methods began with Travell [50] and quickly progressed to include the benefits of combined therapies, such as manipulation and local anesthetic infiltration [51]. The results of subsequent work have been published by

Table 5.2 Manual therapies

Treatment	Practitioners
Physical modalities	Physical therapist, chiropractor
Patient education	Physical therapist, movement therapist, chiropractor
Massage, self-massage	Massage therapist, structural integration practitioner (Rolfer)
Active release techniques, myofascial release	Physical therapist, chiropractor
Neural mobilization (neural flossing)	Physical therapist, chiropractor, osteopathic physician
Joint manipulation or mobilization	Chiropractor, osteopathic physician, physical therapist

investigators in multiple disciplines, often using different terms for essentially the same maneuvers [52]. This is one reason it has been difficult to demonstrate the short- and long-term benefits of these techniques [53]. As the evidence base increases, it is hoped that practitioners will use the strengths of their particular backgrounds to cooperate at all levels, from diagnosis and treatment of individual patients to research and teaching [22, 39, 52, 53].

Physical Modalities

Application of ice, heat, *contrast baths* (for complex regional pain syndrome (CRPS) symptoms), splinting, compression stockings, and/or electrical stimulation can help decrease pain and inflammation. Application of *low-level laser light* has also been shown to be helpful [54, 55]. The routine use of *ice* to reduce inflammation in acute injuries has recently come into question; its use does temporarily decrease discomfort, but normal inflammation is a constructive part of healing [56–58]. These passive interventions should be used to facilitate active stretching and strengthening exercises to correct biomechanics [59].

Patient Education on Postural Habits, Ergonomics, and Activity Modification

Identification of work-related or lifestyle habits that cause or contribute to MFPS is key to treatment. For long-term relief to be successful, the patient must understand the *ergonomics* of their diagnosis and actively participate in rehabilitation [59]. Body awareness therapies to address postural habits such as *Feldenkrais* and the *Alexander technique* naturally complement education, self-massage, and active therapeutic exercises [60].

Structural Integration (Rolfing)

Dr. Ida Rolf developed in the 1920s a combination of movement training and massage designed to maximize the body's vertical alignment [61–63]. In theory, when the body is not aligned with gravity, additional energy must be used for any task, and affected fascia will shorten and thicken. For example, if the head is shifted forward, its effective weight can more than double, causing pain and fatigue. One study showed improvement of neck pain and range of motion after ten sessions [62].

Massage

Massage therapy has been defined as "soft tissue manipulation by trained therapists for therapeutic purposes" [2]. It encompasses hundreds of different techniques and may be performed with hands alone or with instruments [64]. Skilled practitioners can identify taut muscle bands and MTrPs, improve local blood flow, and reduce pain and disability. A recent meta-analysis of its effectiveness for diverse chronic pain conditions has shown massage alone to be effective for low back pain and progressively less effective for shoulder and headache pain (moderate support), with only modest support to treat fibromyalgia, mixed chronic pain, neck pain, and carpal tunnel syndrome [2]. One area of debate is the optimal location at which to apply pressure or friction. Stecco et al. addressed this question by developing a biomechanical model of the myofascial system (Fascial Manipulation©), dividing this continuous structure into segments, each served by myofascial units defined by specific movements [12, 13, 65–68]. Analysis of abnormal motion allows identification of areas requiring treatment [69].

Therapeutic Mechanical Load

Employing instruments to amplify and concentrate the hands' ability to affect the muscle and fascia have been used for centuries [44, 70]. The Astym® [71] and Graston Technique® are two contemporary, instrument-assisted soft-tissue mobilization methods [64].

Neural Mobilization (NM, Colloquially Termed "Neural Flossing")

NM consists of active or passive exercises to improve the movement of peripheral nerves with respect to the other tissues that surround them. In clinical studies, there is level 3 evidence of effectiveness for the upper quadrant (cervical spine, shoulder, arm) with less conclusive evidence in the lower quadrant (lumbar spine, pelvis, leg) [72]. Authors have reported particular success treating median nerve entrapment at the carpal tunnel (Chap. 37) [73–75], neck and arm pain [76, 77], and the pain of lateral epicondylitis and neurogenic cervicobrachial disorders [78]. Dr. Gabor Racz has also encouraged this type of nerve mobilization after cervical and lumbar epidural adhesiolysis [79, 80]. Recent studies in animals have provided evidence for the mechanisms underlying this practice [18, 19].

Joint Manipulation or Mobilization

Removing restrictions on almost any joint limited in its range of motion can improve joint function and reduce the stress on nearby myofascial structures. Broadly speaking, this can be achieved with manipulation using low-amplitude, high-velocity movements or with mobilization using higher-amplitude, low-

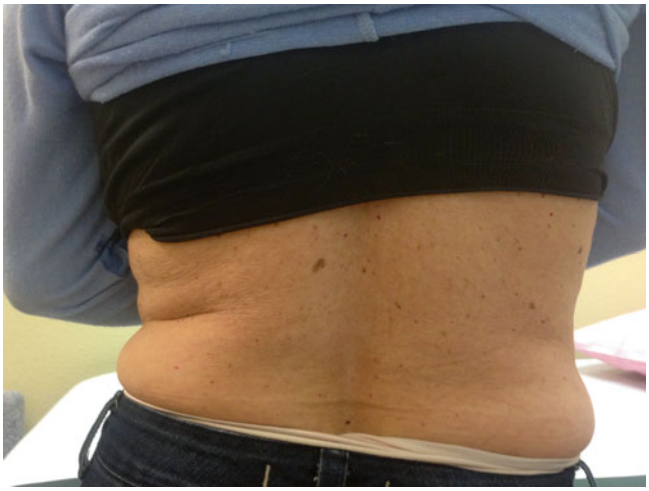

Fig. 5.1 Pelvic obliquity due to sacroiliac dysfunction (Image courtesy of Andrea Trescot, MD)

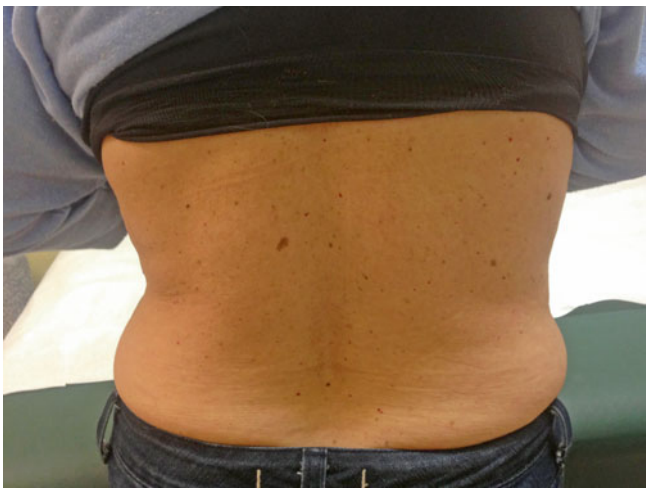

Fig. 5.2 Restoration of pelvic symmetry after sacroiliac self-mobilization (Image courtesy of Andrea Trescot, MD)

velocity measures [43, 59]. Self-mobilization, such as is used for the sacroiliac joint, can effectively restore joint mobility and function (Figs. 5.1 and 5.2). In one long-term follow-up of a randomized clinical trial comparing three standardized regimens for treatment of chronic mechanical spinal pain, only spinal manipulation led to "broad and significant long-term benefit." Acupuncture resulted in consistent, though not statistically significant, improvements, while anti-inflammatory medication was not effective [81]. Rare adverse events associated with cervical manipulation or mobilization have been reported [30, 82].

Needle Therapies

Needle therapies include a wide variety of pain-relieving procedures during which the skin is penetrated with a needle [83]. A recent issue of the journal *Evidence-Based Complementary and Alternative Medicine* was dedicated to these and contained two reviews [84, 85] and seven papers presenting the results of both clinical [86–88] and animal research [89–91].

Acupuncture (Traditional Acupuncture and Electroacupuncture)

Traditional acupuncture is based on an ancient Chinese medicine practice using very fine, solid needles to stimulate specific points in order to increase the flow of energy (qi, chi) and promote healing. It has been shown to decrease signals in the limbic system on functional MRIs [92]. A wide variety of needling techniques (puncture depth, presence or absence of post-insertion needle movement) have been used, presenting one of many challenges in objective evaluation of the therapeutic effects of acupuncture [4]. In order to mitigate one of these, for example, robotic needling techniques have been developed. Comparison of needling cycles and amplitude of bidirectional robotic needle rotation in mouse subcutaneous tissue explants has showed significant effects on fibroblast size [11]. Subjective and objective improvements in myofascial pain and MTrP characteristics after acupuncture treatment have been shown using 2-D ultrasound and ultrasound elastography [8]. Further development of objective protocols and outcome measures should improve the ability of studies to show clear results.

Electroacupuncture is a relatively recent enhancement to traditional acupuncture. Pairs of acupuncture needles are connected to a device that generates a small electric current. The controllability of the current applied is another strategy to improve objective quantification of acupuncture "dose" [92]. Electroacupuncture has been shown to be more effective than simple acupuncture for treatment of injury to the superior cluneal nerves [93] and pain in the head, neck, and/or upper back [8].

Direct Intramuscular Stimulation (Dry Needling)

Direct intramuscular stimulation (dry needling) was developed by Dr. Chan Gunn in the 1970s in response to a request for development of an effective treatment for back pain [94]. After studying back pain patients and a wide variety of therapeutic options, he inserted acupuncture needles into tender and non-tender MTrPs. Needle insertion often results in a local twitch response with reproduction of the patient's pain, followed by muscle relaxation, increased range of motion, and resolution of pain. He called this technique *Intramuscular Stimulation* (IMS), also called *dry needling*. There are many practitioners of IMS, but not all

follow the Gunn protocol, which calls for additional treatment of the paraspinal muscles of the spinal segments involved. A systematic review of the Gunn protocol is currently underway. Other needling therapies include those at local and remote sites, including injection of local anesthetic [95] or botulinum toxin [85], using any of the multiple injection protocols that are available. Many of these approaches have been shown to decrease the abnormal biochemistry associated with MTrPs.

Trigger Point Injections

Travell and Simons codified and popularized the recognition and treatment of *myofascial trigger points* with their landmark book *Myofascial Pain and Dysfunction*: *The Trigger Point Manual* [96]. In that book, the authors described the local and referred patterns of pain seen with myofascial trigger point pathology, reviewed the anatomy of each involved motor unit, identified the contributing and perpetuating factors, and illustrated the techniques of "spray and stretch" (using a vapocoolant on the muscle as it is placed in a position of tension). They also described the technique of injection of local anesthetic into the muscle for diagnosis and treatment. Many of these trigger point pathologies entrap nerves, such as the greater occipital nerve by the trapezius muscle (Chap. 17), the axillary nerve within the quadrangular space (Chap. 31), and the sciatic nerve by the piriformis muscle (Chap. 65). In these syndromes, treatment of the myofascial spasm will result in release of the entrapment.

Recognition of the potential myofascial contribution to pain conditions can decrease misdiagnosis and, therefore, ineffective treatments. As an example, Calandre et al. [97] identified several patients with "cluster headaches" (a condition felt to be caused by intracranial blood vessel pathology) who responded well to a series of trigger point injections. Similarly, migraines [98], chest pain [99], abdominal pain [100], and pelvic pain [101] have all been successfully treated with trigger point injections.

Conclusion

Examples of successful non-pharmacologic treatment of nerve entrapments include the median nerve entrapped in the pronator teres muscle and/or carpal tunnel (Chap. 37) [102], the sciatic nerve entrapped by the piriformis muscle (Chap. 65) [103], and cervicogenic headaches (Sect. 2) [104]. Understanding of the anatomy and importance of the myofascial continuum in health and disease is increasing. A multidisciplinary physical approach to the treatment of peripheral nerve entrapments provides the best opportunity for healing in early stages of the process and should be attempted before more invasive methods are employed.

References

1. Pettman E. A history of manipulative therapy. J Man Manip Ther. 2007;15(3):165–74.
2. Tsao JC. Effectiveness of massage therapy for chronic, non-malignant pain: a review. Evid Based Complement Alternat Med. 2007;4(2):165–79.
3. Simpson CA. Complementary and alternative medicine. In: Fishman SM, Ballantyne JC, Rathmell JP, editors. Bonica's management of pain. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2010.
4. Langevin HM, Hammerschlag R, Lao L, Napadow V, Schnyer RN, Sherman KJ. Controversies in acupuncture research: selection of controls and outcome measures in acupuncture clinical trials. J Altern Complement Med. 2006;12(10):943–53.
5. Ballyns JJ, Shah JP, Hammond J, Gebreab T, Gerber LH, Sikdar S. Objective sonographic measures for characterizing myofascial trigger points associated with cervical pain. J Ultrasound Med. 2011;30(10):1331–40.
6. Love JM, Chuang TH, Lieber RL, Shah SB. Nerve strain correlates with structural changes quantified by Fourier analysis. Muscle Nerve. 2013;48(3):433–5.
7. Sikdar S, Shah JP, Gebreab T, Yen RH, Gilliams E, Danoff J, et al. Novel applications of ultrasound technology to visualize and characterize myofascial trigger points and surrounding soft tissue. Arch Phys Med Rehabil. 2009;90(11):1829–38.
8. Müller CE, Aranha MF, Gavião MB. Two-dimensional ultrasound and ultrasound elastography imaging of trigger points in women with myofascial pain syndrome treated by Acupuncture and Electroacupuncture: a Double-Blinded Randomized Controlled Pilot Study. Ultrason Imaging. 2015;37(2):152–67.
9. Niddam DM, Chan RC, Lee SH, Yeh TC, Hsieh JC. Central representation of hyperalgesia from myofascial trigger point. Neuroimage. 2008;39(3):1299–306.
10. Filler AG, Haynes J, Jordan SE, Prager J, Villablanca JP, Farahani K, et al. Sciatica of nondisc origin and piriformis syndrome: diagnosis by magnetic resonance neurography and interventional magnetic resonance imaging with outcome study of resulting treatment. J Neurosurg Spine. 2005;2(2):99–115.
11. Langevin HM, Bouffard NA, Churchill DL, Badger GJ. Connective tissue fibroblast response to acupuncture: dose-dependent effect of bidirectional needle rotation. J Altern Complement Med. 2007;13(3):355–60.
12. Stecco C, Gagey O, Macchi V, Porzionato A, De Caro R, Aldegheri R, et al. Tendinous muscular insertions onto the deep fascia of the upper limb. First part: anatomical study. Morphologie. 2007; 91(292):29–37.
13. Stecco C, Gagey O, Belloni A, Pozzuoli A, Porzionato A, Macchi V, et al. Anatomy of the deep fascia of the upper limb. Second part: study of innervation. Morphologie. 2007;91(292): 38–43.
14. Stecco C, Corradin M, Macchi V, Morra A, Porzionato A, Biz C, et al. Plantar fascia anatomy and its relationship with Achilles tendon and paratenon. J Anat. 2013;223(6):665–76.
15. Macchi V, Tiengo C, Porzionato A, Stecco C, Sarasin G, Tubbs S, et al. The cubital tunnel: a radiologic and histotopographic study. J Anat. 2014;225(2):262–9.
16. Benjamin M. The fascia of the limbs and back – a review. J Anat. 2009;214(1):1–18.
17. Shah JP, Danoff JV, Desai MJ, Parikh S, Nakamura LY, Phillips TM, et al. Biochemicals associated with pain and inflammation are elevated in sites near to and remote from active myofascial trigger points. Arch Phys Med Rehabil. 2008;89(1):16–23.
18. Santos FM, Silva JT, Giardini AC, Rocha PA, Achermann AP, Alves AS, et al. Neural mobilization reverses behavioral and cellular changes that characterize neuropathic pain in rats. Mol Pain. 2012;8:57.

19. Santos FM, Grecco LH, Pereira MG, Oliveira ME, Rocha PA, Silva JT, et al. The neural mobilization technique modulates the expression of endogenous opioids in the periaqueductal gray and improves muscle strength and mobility in rats with neuropathic pain. Behav Brain Funct: BBF. 2014;10:19.
20. Standley PR, Meltzer K. In vitro modeling of repetitive motion strain and manual medicine treatments: potential roles for pro- and anti-inflammatory cytokines. J Bodyw Mov Ther. 2008; 12(3):201–3.
21. Meltzer KR, Cao TV, Schad JF, King H, Stoll ST, Standley PR. In vitro modeling of repetitive motion injury and myofascial release. J Bodyw Mov Ther. 2010;14(2):162–71.
22. Gross A, Miller J, D'Sylva J, Burnie SJ, Goldsmith CH, Graham N, et al. Manipulation or mobilisation for neck pain. Cochrane Database Syst Rev. 2010;(1):CD004249.
23. Loew LM, Brosseau L, Tugwell P, Wells GA, Welch V, Shea B, et al. Deep transverse friction massage for treating lateral elbow or lateral knee tendinitis. Cochrane Database Syst Rev. 2014;(11):CD003528.
24. Chaibi A, Russell MB. Manual therapies for primary chronic headaches: a systematic review of randomized controlled trials. J Headache Pain. 2014;15:67.
25. Yu H, Côté P, Southerst D, Wong JJ, Varatharajan S, Shearer HM, et al. Does structured patient education improve the recovery and clinical outcomes of patients with neck pain? A systematic review from the Ontario Protocol for Traffic Injury Management (OPTIMa) Collaboration. Spine J: Off J N Am Spine Soc. 2014:on line. doi:10.1016/j.spinee.2014.03.039.
26. Verhagen AP, Bierma-Zeinstra SM, Burdorf A, Stynes SM, de Vet HC, Koes BW. Conservative interventions for treating work-related complaints of the arm, neck or shoulder in adults. Cochrane Database Syst Rev. 2013;(12):CD008742.
27. Bryans R, Decina P, Descarreaux M, Duranleau M, Marcoux H, Potter B, et al. Evidence-based guidelines for the chiropractic treatment of adults with neck pain. J Manipulative Physiol Ther. 2014;37(1):42–63.
28. Jäkel A, von Hauenschild P. A systematic review to evaluate the clinical benefits of craniosacral therapy. Complement Ther Med. 2012;20(6):456–65.
29. Page MJ, O'Connor D, Pitt V, Massy-Westropp N. Exercise and mobilisation interventions for carpal tunnel syndrome. Cochrane Database Syst Rev. 2012;(6):CD009899.
30. Carlesso LC, Macdermid JC, Gross AR, Walton DM, Santaguida PL. Treatment preferences amongst physical therapists and chiropractors for the management of neck pain: results of an international survey. Chiropr Man Ther. 2014;22(1):11.
31. Simons DG. Review of enigmatic MTrPs as a common cause of enigmatic musculoskeletal pain and dysfunction. J Electromyogr Kinesiol. 2004;14(1):95–107.
32. Simons DG, Travell JG, Simons LS. Travell & Simons' myofascial pain and dysfunction:the trigger point manual. 2nd ed. Philadelphia: Lippincott Williams & Wilkins; 1999.
33. Travell J, Bigelow NH. Referred somatic pain does not follow a simple "segmental" pattern. Fed Proc. 1946;5(1 Pt 2):106.
34. Dommerholt J, Shah JP. Myofascial pain syndrome. In: Fishman SM, Ballantyne JC, Rthmell JP, editors. Bonica's management of pain. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2010.
35. Dellon AL, Mackinnon SE. Chronic nerve compression model for the double crush hypothesis. Ann Plast Surg. 1991;26(3):259–64.
36. Mense S. Muscle pain: mechanisms and clinical significance. Dtsch Arztebl Int. 2008;105(12):214–9.
37. Stecco C. Why are there so many discussions about the nomenclature of fasciae? J Bodyw Mov Ther. 2014;18(3):441–2.
38. Chaitow L. The fascia debate. J Bodyw Mov Ther. 2014;18(3):443.
39. Myers T. Myers' response to Stecco's fascial nomenclature editorial. J Bodyw Mov Ther. 2014;18(3):445–6.
40. Myers TW. Anatomy trains: myofascial meridians for manual and movement therapists. 3rd ed. New York: Churchill Livingstone: Elsevier; 2014.
41. Huijing PA, Baan GC. Myofascial force transmission: muscle relative position and length determine agonist and synergist muscle force. J Appl Physiol (Bethesda, Md : 1985). 2003; 94(3):1092–107.
42. Huijing PA, Yaman A, Ozturk C, Yucesoy CA. Effects of knee joint angle on global and local strains within human triceps surae muscle: MRI analysis indicating in vivo myofascial force transmission between synergistic muscles. Surg Radiol Anat. 2011;33(10):869–79.
43. Simmonds N, Miller P, Gemmell H. A theoretical framework for the role of fascia in manual therapy. J Bodyw Mov Ther. 2012; 16(1):83–93.
44. Findley T, Chaudhry H, Stecco A, Roman M. Fascia research – a narrative review. J Bodyw Mov Ther. 2012;16(1):67–75.
45. Rivard M, Laliberté M, Bertrand-Grenier A, Harnagea C, Pfeffer CP, Vallières M, et al. The structural origin of second harmonic generation in fascia. Biomed Opt Express. 2010;2(1):26–36.
46. Schleip R. Fascial plasticity – a new neurobiological explanation: part 1. J Bodyw Mov Ther. 2003;7(1):11–9.
47. Wang JH, Jia F, Yang G, Yang S, Campbell BH, Stone D, et al. Cyclic mechanical stretching of human tendon fibroblasts increases the production of prostaglandin E2 and levels of cyclooxygenase expression: a novel in vitro model study. Connect Tissue Res. 2003;44(3–4):128–33.
48. Hicks MR, Cao TV, Campbell DH, Standley PR. Mechanical strain applied to human fibroblasts differentially regulates skeletal myoblast differentiation. J Appl Physiol (Bethesda, Md : 1985). 2012;113(3):465–72.
49. Hicks MR, Cao TV, Standley PR. Biomechanical strain vehicles for fibroblast-directed skeletal myoblast differentiation and myotube functionality in a novel coculture. Am J Physiol Cell Physiol. 2014;307(8):C671–83.
50. Travell W, Travell J. Technic for reduction and ambulatory treatment of sacroiliac displacement. Archaeol Phys Ther. 1942;23: 222–46.
51. Travell J, Travell W. Therapy of lowback pain by manipulation and of referred pain in the lower extremity by procaine infiltration. Arch Phys Med Rehabil. 1946;27:537–47.
52. Standley PR. Towards a Rosetta Stone of manual therapeutic methodology. J Bodyw Mov Ther. 2014;18(4):586–7.
53. Clar C, Tsertsvadze A, Court R, Hundt GL, Clarke A, Sutcliffe P. Clinical effectiveness of manual therapy for the management of musculoskeletal and non-musculoskeletal conditions: systematic review and update of UK evidence report. Chiropr Man Ther. 2014;22(1):12.
54. Hashmi JT, Huang YY, Osmani BZ, Sharma SK, Naeser MA, Hamblin MR. Role of low-level laser therapy in neurorehabilitation. PM R. 2010;2(12 Suppl 2):S292–305.
55. Chung H, Dai T, Sharma SK, Huang YY, Carroll JD, Hamblin MR. The nuts and bolts of low-level laser (light) therapy. Ann Biomed Eng. 2012;40(2):516–33.
56. Takagi R, Fujita N, Arakawa T, Kawada S, Ishii N, Miki A. Influence of icing on muscle regeneration after crush injury to skeletal muscles in rats. J Appl Physiol (Bethesda, Md : 1985). 2011;110(2):382–8.
57. Bleakley CM, McDonough SM, MacAuley DC, Bjordal J. Cryotherapy for acute ankle sprains: a randomised controlled study of two different icing protocols. Br J Sports Med. 2006;40(8):700–5; discussion 5.
58. Tseng CY, Lee JP, Tsai YS, Lee SD, Kao CL, Liu TC, et al. Topical cooling (icing) delays recovery from eccentric exercise-induced muscle damage. J Strength Cond Res. 2013;27(5):1354–61.

59. McLean JP, Chimes GP, Press HM, Hearndon ML, Willick SE, Herring SA. Basic concepts in biomechanics and musculoskeletal rehabilitation. In: Fishman SM, Ballantyne JC, Rathmell JP editors. Bonica's Management of Pain. 4th ed. Philadelphia: Wolters Kluwer/Lippincott/WIlliams & Wilkins; 2010. p. 1294–312.
60. Jain S, Janssen K, DeCelle S. Alexander technique and Feldenkrais method: a critical overview. Phys Med Rehabil Clin N Am. 2004;15(4):811–25, vi.
61. Jacobson E. Structural integration, an alternative method of manual therapy and sensorimotor education. J Altern Complement Med. 2011;17(10):891–9.
62. James H, Castaneda L, Miller ME, Findley T. Rolfing structural integration treatment of cervical spine dysfunction. J Bodyw Mov Ther. 2009;13(3):229–38.
63. Jones TA. Rolfing. Phys Med Rehabil Clin N Am. 2004;15(4):799–809, vi.
64. Hammer WI. The effect of mechanical load on degenerated soft tissue. J Bodyw Mov Ther. 2008;12(3):246–56.
65. Day JA, Stecco C, Stecco A. Application of fascial manipulation technique in chronic shoulder pain – anatomical basis and clinical implications. J Bodyw Mov Ther. 2009;13(2):128–35.
66. Stecco C, Pavan PG, Porzionato A, Macchi V, Lancerotto L, Carniel EL, et al. Mechanics of crural fascia: from anatomy to constitutive modelling. Surg Radiol Anat. 2009;31(7):523–9.
67. Stecco A, Masiero S, Macchi V, Stecco C, Porzionato A, De Caro R. The pectoral fascia: anatomical and histological study. J Bodyw Mov Ther. 2009;13(3):255–61.
68. Stecco C, Day JA. The fascial manipulation technique and its biomechanical model: a guide to the human fascial system. Int J Ther Massage Bodyw. 2010;3(1):38–40.
69. Day JA, Copetti L, Rucli G. From clinical experience to a model for the human fascial system. J Bodyw Mov Ther. 2012;16(3):372–80.
70. Nielsen A. Gua sha research and the language of integrative medicine. J Bodyw Mov Ther. 2009;13(1):63–72.
71. Gehlsen GM, Ganion LR, Helfst R. Fibroblast responses to variation in soft tissue mobilization pressure. Med Sci Sports Exerc. 1999;31(4):531–5.
72. Ellis RF, Hing WA. Neural mobilization: a systematic review of randomized controlled trials with an analysis of therapeutic efficacy. J Man Manip Ther. 2008;16(1):8–22.
73. Baysal O, Altay Z, Ozcan C, Ertem K, Yologlu S, Kayhan A. Comparison of three conservative treatment protocols in carpal tunnel syndrome. Int J Clin Pract. 2006;60(7):820–8.
74. Pinar L, Enhos A, Ada S, Güngör N. Can we use nerve gliding exercises in women with carpal tunnel syndrome? Adv Ther. 2005;22(5):467–75.
75. Akalin E, El O, Peker O, Senocak O, Tamci S, Gülbahar S, et al. Treatment of carpal tunnel syndrome with nerve and tendon gliding exercises. Am J Phys Med Rehabil. 2002;81(2):108–13.
76. Nee RJ, Vicenzino B, Jull GA, Cleland JA, Coppieters MW. Neural tissue management provides immediate clinically relevant benefits without harmful effects for patients with nerve-related neck and arm pain: a randomised trial. J Physiother. 2012;58(1):23–31.
77. Nee RJ, Vicenzino B, Jull GA, Cleland JA, Coppieters MW. Baseline characteristics of patients with nerve-related neck and arm pain predict the likely response to neural tissue management. J Orthop Sports Phys Ther. 2013;43(6):379–91.
78. Coppieters MW, Stappaerts KH, Wouters LL, Janssens K. The immediate effects of a cervical lateral glide treatment technique in patients with neurogenic cervicobrachial pain. J Orthop Sports Phys Ther. 2003;33(7):369–78.
79. Sizer Jr PS, Phelps V, Dedrick G, Matthijs O. Differential diagnosis and management of spinal nerve root-related pain. Pain Pract. 2002;2(2):98–121.
80. Racz GB, Day MR, Heavner JE, Smith JP, Scott J, Noe CE, et al. Epidural lysis of adhesions and percutaneous neuroplasty. In: Razcz GB, Noe CE, editors. Pain management – Current issues and opinions. on line: InTech; 2012.
81. Muller R, Giles LG. Long-term follow-up of a randomized clinical trial assessing the efficacy of medication, acupuncture, and spinal manipulation for chronic mechanical spinal pain syndromes. J Manipulative Physiol Ther. 2005;28(1):3–11.
82. Carlesso LC, Gross AR, Santaguida PL, Burnie S, Voth S, Sadi J. Adverse events associated with the use of cervical manipulation and mobilization for the treatment of neck pain in adults: a systematic review. Man Ther. 2010;15(5):434–44.
83. Hong CZ. Needling therapy for myofascial pain control. Evid Based Complement Alternat Med. 2013;2013:946597.
84. Chou LW, Kao MJ, Lin JG. Probable mechanisms of needling therapies for myofascial pain control. Evid Based Complement Alternat Med. 2012;2012:705327.
85. Climent JM, Kuan TS, Fenollosa P, Martin-Del-Rosario F. Botulinum toxin for the treatment of myofascial pain syndromes involving the neck and back: a review from a clinical perspective. Evid Based Complement Alternat Med. 2013;2013:381459.
86. Mayoral O, Salvat I, Martín MT, Martín S, Santiago J, Cotarelo J, et al. Efficacy of myofascial trigger point dry needling in the prevention of pain after total knee arthroplasty: a randomized, double-blinded, placebo-controlled trial. Evid Based Complement Alternat Med. 2013;2013:694941.
87. Chen KH, Hsiao KY, Lin CH, Chang WM, Hsu HC, Hsieh WC. Remote effect of lower limb acupuncture on latent myofascial trigger point of upper trapezius muscle: a pilot study. Evid Based Complement Alternat Med. 2013;2013:287184.
88. Lin MT, Chou LW, Chen HS, Kao MJ. Percutaneous soft tissue release for treating chronic recurrent myofascial pain associated with lateral epicondylitis: 6 case studies. Evid Based Complement Alternat Med. 2012;2012:142941.
89. Hsieh YL, Yang SA, Yang CC, Chou LW. Dry needling at myofascial trigger spots of rabbit skeletal muscles modulates the biochemicals associated with pain, inflammation, and hypoxia. Evid Based Complement Alternat Med. 2012;2012:342165.
90. Domingo A, Mayoral O, Monterde S, Santafé MM. Neuromuscular damage and repair after dry needling in mice. Evid Based Complement Alternat Med. 2013;2013:260806.
91. Fu Z, Hsieh YL, Hong CZ, Kao MJ, Lin JG, Chou LW. Remote subcutaneous needling to suppress the irritability of myofascial trigger spots: an experimental study in rabbits. Evid Based Complement Alternat Med. 2012;2012:353916.
92. Napadow V, Makris N, Liu J, Kettner NW, Kwong KK, Hui KK. Effects of electroacupuncture versus manual acupuncture on the human brain as measured by fMRI. Hum Brain Mapp. 2005;24(3):193–205.
93. Tao Y. Eighty cases of injury of the superior cluneal nerve treated by electroacupuncture. J Tradit Chin Med = Chung i tsa chih ying wen pan/sponsored by All-China Association of Traditional Chinese Medicine, Academy of Traditional Chinese Medicine. 2000;20(2):132–3.
94. Gunn CC, Milbrandt WE, Little AS, Mason KE. Dry needling of muscle motor points for chronic low-back pain: a randomized clinical trial with long-term follow-up. Spine (Phila Pa 1976). 1980;5(3):279–91.
95. Hong CZ. Lidocaine injection versus dry needling to myofascial trigger point. The importance of the local twitch response. Am J Phys Med Rehabil. 1994;73(4):256–63.
96. Travell JG, Simons LS. Myofascial pain and dysfunction: the trigger point manual. Philadelphia: Lippincott Williams & Wilkins; 1992.

97. Calandre EP, Hidalgo J, Garcia-Leiva JM, Rico-Villademoros F, Delgado-Rodriguez A. Myofascial trigger points in cluster headache patients: a case series. Head Face Med. 2008;4:32.
98. Calandre EP, Hidalgo J, Garcia-Leiva JM, Rico-Villademoros F. Trigger point evaluation in migraine patients: an indication of peripheral sensitization linked to migraine predisposition? Eur J Neuro. 2006;13(3):244–9.
99. Chen SM, Chen JT, Kuan TS, Hong CZ. Myofascial trigger points in intercostal muscles secondary to herpes zoster infection of the intercostal nerve. Arch Phys Med Rehabil. 1998;79(3):336–8.
100. Sharpstone D, Colin-Jones DG. Chronic, non-visceral abdominal pain. Gut. 1994;35(6):833–6.
101. Slocumb JC. Chronic somatic, myofascial, and neurogenic abdominal pelvic pain. Clin Obstet Gynecol. 1990;33(1):145–53.
102. Huisstede BM, Hoogvliet P, Randsdorp MS, Glerum S, van Middelkoop M, Koes BW. Carpal tunnel syndrome. Part I: effectiveness of nonsurgical treatments – a systematic review. Arch Phys Med Rehabil. 2010;91(7):981–1004.
103. Boyajian-O'Neill LA, McClain RL, Coleman ML, Thomas PP. Diagnosis and management of piriformis syndrome: an osteopathic approach. JAOA. 2006;108(11):657–64.
104. Borg-Stein J. Cervical myofascial pain and headache. Curr Pain Headache Rep. 2002;6(4):324–30.

Diagnostic Evaluation

6

Daniel Krashin and Andrea M. Trescot

Part of the success of the treatment of peripheral nerve disorders is based on the accuracy of the diagnosis. Peripheral neuropathy and peripheral nerve entrapment can present in a similar fashion, and some authors [1] recommend a basic evaluation to rule out peripheral neuropathy. This includes thyroid function studies, fasting blood glucose, vitamin B_{12}, and folate, nerve conduction studies, and potentially imaging (such as MRI or ultrasound). However, the history and the physical exam should support the diagnosis, and, for many patients, further diagnostic studies are not necessary.

Electromyography and Nerve Conduction Studies

Entrapments of specific peripheral nerves can be confirmed and localized using *electromyography (EMG)* and *nerve conduction studies (NCS),* also known as *nerve conduction velocities (NCV)*. It is important to note that these studies are not a substitute for history and clinical examination but serve as a method of clarifying and confirming suspected diagnoses [2]. These studies are generally less useful for thoracic, pelvic, or abdominal entrapments. EMG and NCS are typically performed at the same time, and the results are interpreted with clinical correlation. In the many combined motor and sensory nerves of the extremities, electromyography may demonstrate sequelae of nerve entrapment, including denervation of distal muscles, which can be seen as *positive sharp waves*, fibrillations, and giant *motor unit potentials (MUPs*). For example, in an EMG study of median nerve entrapment, all 11 patients with entrapment at both the wrist and elbow showed evidence of denervation on EMG, while half of the patients with only carpal tunnel syndrome showed evidence of denervation, including both fibrillations and positive sharp waves [3]. Negative EMG testing of adjacent muscles with different innervation within the same myotome can help rule out radiculopathy.

NCS are particularly useful for localizing and characterizing the nature of a nerve injury. Evidence of demyelination, such as slowed conduction velocity or block, is sought across suspected nerve segments. Some locations are extremely common, such as entrapment of the ulnar nerve at the elbow, the median nerve at the wrist, the radial nerve at the spiral groove, or the peroneal nerve at the fibular neck. If other specific locations are suspected due to the patient's exam, previous trauma, or surgical history, it is essential to share this information with the electromyographer to help guide the evaluation. Conduction studies can assess both motor and sensory function, but they only evaluate large myelinated fibers.

Ultrasound

Peripheral nerve ultrasound was first described nearly two decades ago to evaluate carpal tunnel entrapment [4]. Although there are currently no formal standards established for the use of ultrasound in the diagnosis of peripheral nerve entrapments, it is recommended that a high-frequency (>12 MHz) linear probe be used for most peripheral nerves [5]. Lower-frequency transducers (10–15 MHz) may be needed for nerves more than 4 cm below the skin's surface. Since most of the nerves of interest travel with blood vessels, Doppler imaging may aid in nerve identification. Swelling of the nerve with entrapment [6], as well as increased echogenicity of muscles in CRPS [7], can be seen. Ultrasound can be used to confirm pathology and/or aid in needle localization for diagnostic injections.

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Magnetic Resonance Imaging (MRI)

Magnetic resonance imaging (MRI) of the peripheral nerves, until recently, had limited application. MRI affords superb differentiation of soft tissue and assessment of the perineural tissue and bone. However, nerves can be difficult to discern on conventional MRI, and even small amounts of patient movement can render the image unhelpful due to motion artifact. MRI is particularly useful in the assessment of deeper structures that are beyond the range of high-resolution ultrasound. MRI has also been helpful in distinguishing nerve entrapment from other focal nerve lesions, including invasive tumors and intrinsic nerve lesions such as schwannomas. MRI may show perineural structures impinging on the nerve, and it may also show abnormalities within the nerve itself such as focal enlargement, hyperintensity on STIR images, and altered fascicular patterns [8]. In addition, a hyperintense signal of the denervated muscle can usually be identified when entrapment is acute, while fatty infiltration and muscle atrophy are the signs of chronic neuropathy in long-standing cases [9].

Employment of MRI with particular emphasis on increased structural resolution and optimized nerve T2 contrast may be termed m*agnetic resonance neurography* (MRN) [10]. The T2 signal is known to increase after various mechanical and nonmechanical forms of experimental nerve injury, and it has now been shown to strongly correlate with NCV findings [10]. Specific nerve entrapments that have been extensively studied using MRN include the suprascapular nerve, proximal sciatic nerve, and pudendal nerve, as well as many extremity entrapments in locations such as the popliteal fossa, tarsal tunnel, and Guyon's canal. Research into new forms of MRN is ongoing, with both new scanning protocols and new contrast media being actively studied.

Summary

There are multiple tools available to aid in the diagnosis of peripheral nerve entrapment. Choice of technique depends on the structure to be studied and the tools available.

References

1. DeSouza RM, Choi D. Peripheral nerve lesions. Neurosurgery. 2012;3(30):149–54.
2. Fuller G. How to get the most out of nerve conduction studies and electromyography. J Neurol Neurosurg Psychiatry. 2005;76 Suppl 2:ii41–6.
3. Buchthal F, Rosenfalck A, Trojaborg W. Electrophysiological findings in entrapment of the median nerve at wrist and elbow. J Neurol Neurosurg Psychiatry. 1974;37(3):340–60.
4. Buchberger W, Schon G, Strasser K, Jungwirth W. High-resolution ultrasonography of the carpal tunnel. J Ultrasound Med. 1991;10(10):531–7.
5. Hobson-Webb LD. Neuromuscular ultrasound in polyneuropathies and motor neuron disease. Muscle Nerve. 2013;47(6):790–804.
6. Tagliafico A, Bignotti B, Miguel Perez M, Reni L, Bodner G, Martinoli C. Contribution of ultrasound in the assessment of patients with suspect idiopathic pudendal nerve disease. Clin Neurophysiol. 2014;125(6):1278–84.
7. Vas LC, Pai R, Radhakrishnan M. Ultrasound appearance of forearm muscles in 18 patients with complex regional pain syndrome 1 of the upper extremity. Pain Pract. 2013;13(1):76–88.
8. Kim S, Choi JY, Huh YM, Song HT, Lee SA, Kim SM, et al. Role of magnetic resonance imaging in entrapment and compressive neuropathy – what, where, and how to see the peripheral nerves on the musculoskeletal magnetic resonance image: part 1. Overview and lower extremity. Eur Radiol. 2007;17(1):139–49.
9. Dong Q, Jacobson JA, Jamadar DA, Gandikota G, Brandon C, Morag Y, et al. Entrapment neuropathies in the upper and lower limbs: anatomy and MRI features. Radiol Res Pract. 2012;2012, 230679.
10. Kollmer J, Baumer P, Milford D, Dombert T, Staub F, Bendszus M, et al. T2-signal of ulnar nerve branches at the wrist in guyon's canal syndrome. PLoS One. 2012;7(10), e47295.

Peripheral Nerve Entrapment: Injection Techniques

7

Andrea M. Trescot and Natalia Murinova

Introduction

Peripheral nerve blocks (PNBs) can offer both diagnosis and treatment of peripheral nerve entrapments. These injections have a unique role in the management of peripheral nerve entrapments. PNBs with local anesthetic help localize and confirm nerve entrapment diagnoses. Injections can also treat the nerve entrapment, with putative mechanisms including hydrodissection, the anti-inflammatory effect of injected corticosteroids, and the dilution and flushing out of inflammatory mediators. Precise and atraumatic injection techniques are essential to maximize the informational and treatment value of any nerve injection for peripheral nerve entrapment.

Bogduk [1, 2] has reported that nerve blocks are not specific, in that they do not identify the actual source of pain, but only show that pain is caused or mediated by the nerve that is anesthetized. However, if the injection is done at the site of the entrapment, the injection can be diagnostic as well as therapeutic. *Controlled blocks* (using a short-acting local anesthetic and then a long-acting local anesthetic, with the patient blinded as to which local anesthetic is being used), which have also been promoted by Bogduk [3], can minimize the placebo effect. However, the use of diagnostic and prognostic blocks is still hampered by false-positive results, unequal sensitivity to the two local anesthetics [4], patient expectations, the use of concurrent therapy with numerous drugs, and the subjective nature of pain.

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Injection Medications

The therapeutic injectate usually consists of a long-lasting local anesthetic and a depo-steroid. The response to the *local anesthetics* is key to the diagnosis, and therefore it may be prudent to test the response to a variety of local anesthetics prior to the diagnostic injection. *Local anesthetic resistance* is an under-recognized cause of injection failure. In 2003, Trescot interviewed 1,198 consecutive patients; 250 patients noted failure of relief from an injection of bupivacaine or had a history of difficulty getting numb at the dentist [4]. Skin testing with lidocaine, bupivacaine, and mepivacaine was performed to identify the most effective local anesthetic (i.e., the local anesthetic that caused the most skin numbness). Ninety of those patients (7.5 % of the total patients, but 36 % of the test group) were numb only to mepivacaine, and an additional 43 patients (3.8 % of the total patients, but 17 % of the test group) only got numb to lidocaine. Thus, 133 of 250 patients with a history of difficulty with local anesthetic analgesia (53 %), and 11 % of the total patients, did not get numb with bupivacaine (the most commonly used anesthetic), suggesting a significant potential false-negative response to diagnostic injections.

Glucocorticosteroids are often used to decrease inflammation that both causes and accompanies nerve entrapment. However, these steroids need careful consideration and judicious use. Not only is there a risk of superficial skin injury and atrophy (see Complications – Chap. 13) but large doses of steroids can cause suppression of the hypothalamus-pituitary-adrenal axis, leading to the potential for Cushing's syndrome, which has been reported to occur with even a single dose of 60 mg methylprednisolone [5].

Because entrapment of the nerve is usually the underlying pathology, care must be used to avoid further entrapment with large volumes of injectate. Methylprednisolone (Depo-Medrol®) is the steroid of choice for at least one author (Trescot), because of its high lipophilic nature (to enter the myelin sheath) and its high concentration (80 mg/cc). The

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total dose of steroid would normally be limited to 80 mg methylprednisolone (or equivalent), with no more than 40 mg at any one site (even less should be used if the skin is thin or the injection is superficial, because of the risk of steroid-induced skin atrophy).

Sarapin® (High Chemical Company, Levittown, PA), a sterile extract distilled from *Sarracenia purpurea* (the purple pitcher plant), is a biologic injection material that has been used to treat neuropathic pain for more than 70 years. It has been described as a nontoxic, reversible neurolytic as well as an anti-inflammatory agent, and it has been used to treat a variety of painful conditions, including sciatica, meralgia paresthetica, and trigeminal neuralgia.

Special Considerations

Special procedural considerations may be necessary in certain patient populations, including pregnant patients, the elderly, and those with local anesthetic allergy (usually from the *methyl parabens* used as a preservative), prior vasovagal attacks, an open skull defect, and cosmetic concerns [6]. Antiplatelet/anticoagulant use may be an issue when injections are close to large blood vessels or closed spaces, but most of the injections in this book are done with small needles in areas where any potential bleeding should be easily controlled. Therefore, the risk of bleeding is a relative one, and the decision to stop anticoagulation needs to be balanced with the risk of the condition for which the medication was prescribed.

Types of Injections

Landmark-Guided Nerve Blocks

Anesthesiologists have used *landmark-guided ("blind") injections* for more than 100 years, and the subspecialty of regional anesthesia dramatically expanded the role of injections performed with palpable landmarks and a working knowledge of the superficial and deep anatomy. Francis Rynd (1801–1861) performed the first hypodermic injections in May 1844; interestingly, the patient described lancinating face pain (consistent with nerve entrapment), and she was injected with "morphia" dissolved in creosote into the supraorbital, temporal, malar, and buccal nerves with immediate and excellent relief [7]. The first textbook of regional anesthesia (*Regional Anesthesia: Techniques and Clinical Applications*) was published in 1922 by Gaston Labat (1876–1934) [8].

Once the landmarks have been indentified, the site is prepped, usually with alcohol or Betadine. Many people are taught to hold the syringe like a dart, and then changing hands to inject. Instead, Trescot teaches that the syringe is held as an extension of the injecting hand, and the target is stabilized with the non-injecting hand (Fig. 7.1). Because these are small (27–25 gauge), sharp needles, the needle does not need to be "stabbed." Rather, it is advanced slowly and smoothly, in a tangential angle to the target area (Fig. 7.2).

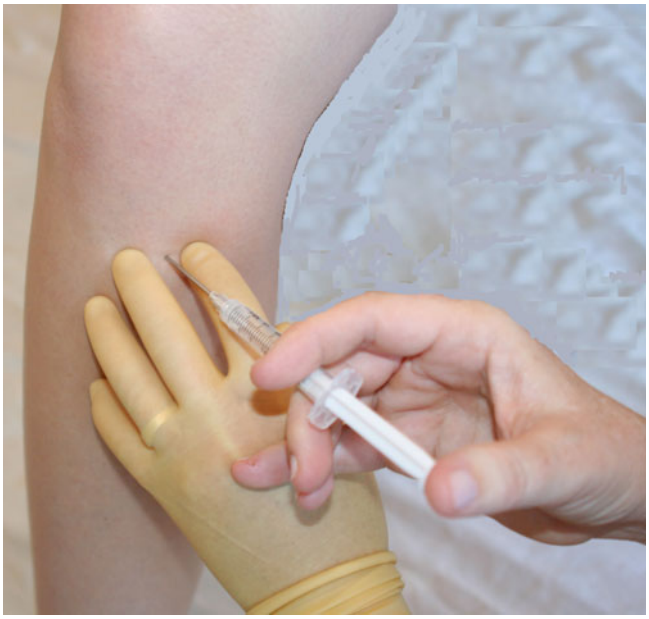

Fig. 7.1 Injection technique: the non-injecting hand is used to stabilize the target, and the injecting hand holds the needle at the phalanges between the index and middle fingers, with the thumb on the plunger and the other fingers resting on the non-injecting hand (Image courtesy of Andrea Trescot, MD)

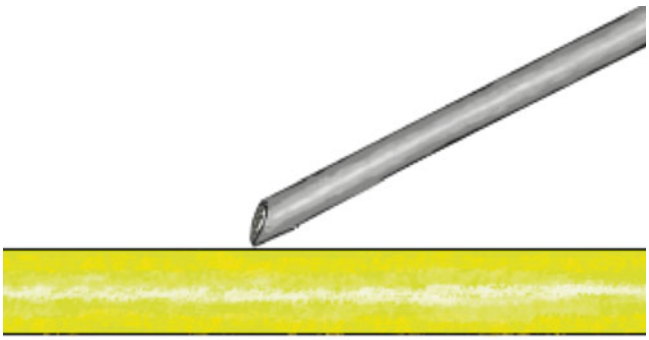

Fig. 7.2 Angle of injection (Image courtesy of Michael Brown, MD)

Use of Peripheral Nerve Stimulator for Nerve Blocks

Nerve stimulation for neural blockade was first described in 1912 [9]. Instead of "poking around with the needle" to elicit a paresthesia, the use of a peripheral stimulator has been the "gold standard" for PNBs for more than a decade [10].

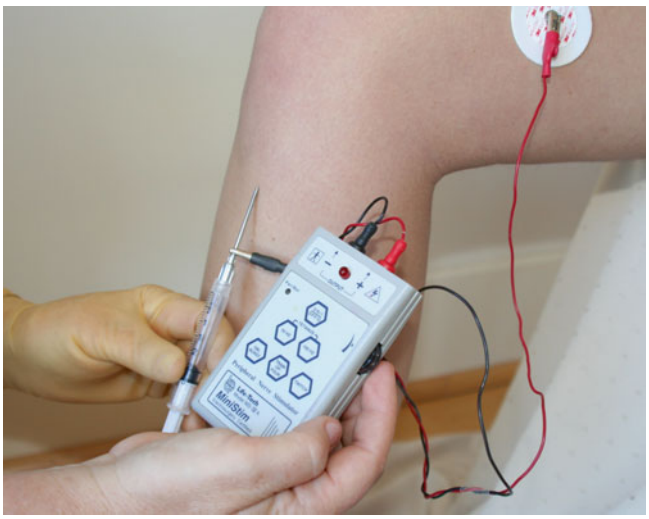

Fig. 7.3 Peripheral nerve stimulator (*PNS*) with EKG pad as a grounding pad (Image courtesy of Andrea Trescot, MD)

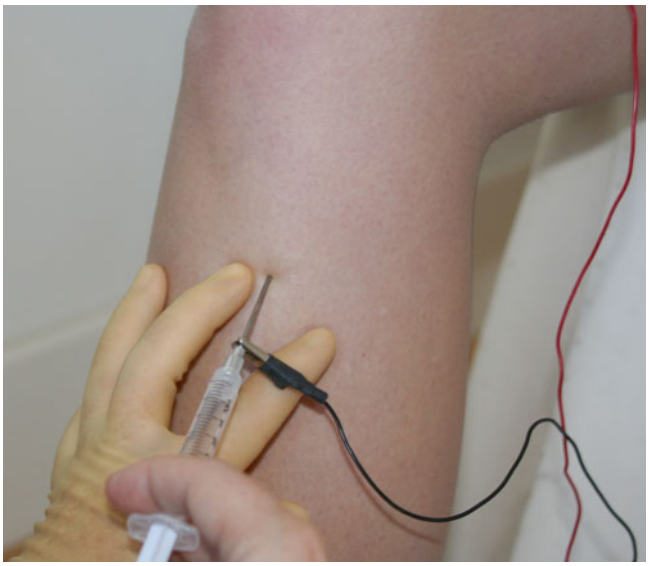

Fig. 7.4 Infrapatellar saphenous nerve injection with PNS (Image courtesy of Andrea Trescot, MD)

Although there are several specialized nerve stimulation systems available that use special, expensive needles, one author (Trescot) uses a simple system that incorporates an anesthesia neuro-blockade stimulator, an EKG pad as a grounding pad, an alligator clip ("negative to needle"), and a standard needle (Fig. 7.3). This PNS technique allows the needle to be advanced close to the target nerve without actually hitting the nerve. An added benefit is that the patient will feel an increasing "jumping" sensation along the path of the nerve (even with pure sensory nerves) as the needle approaches the nerve, allowing the patient to confirm the "that's it" sensation (Fig. 7.4).

Fluoroscopy-Guided Nerve Blocks

The use of *fluoroscopy* has dramatically improved the efficacy and safety of spinal injections. Fluoroscopy also offers the ability to "take a picture of where it hurts"; placing a radiopaque marker on the skin at the site of pain allows a correlation between physical exam and anatomic location (Fig. 7.5). Many of the procedures noted in this book have something in common – they may not be routinely performed under fluoroscopic control – and yet, perhaps they should be.

There are important issues to remember when performing a procedure under fluoroscopy. *Radiation safety* is of paramount concern. Minimizing exposure, particularly the time of exposure, is the easiest way to help improve safety. In order to minimize time of exposure, knowledge of radiographic anatomy is required. If one knows "what nerve is where," it is much easier to be accurate in performing an injection. The focus in this book will therefore be on providing anatomic locations for key significant nerves in these areas.

Ultrasound-Guided Nerve Blocks

Over the last several years, *ultrasound* (US) has moved out of the radiology suite, into the regional anesthesia world, and from there, into pain medicine. US is particularly well suited for regional anesthesia, since many of the anesthesia targets (e.g., brachial plexus, femoral nerve) have large blood vessels that travel with the target nerves. Abrahams et al. [11] performed a meta-analysis comparing PNS and US for regional anesthetic injections. Injections performed using US guidance were more likely to be successful, took less time to perform, had faster onset, and lasted longer. Although these were regional anesthesia injections, it is likely that similar results would be seen for chronic pain nerve injections; the authors felt that US could improve block success rates, especially for anesthetists who do not frequently perform peripheral nerve blocks, or for those supervising trainees. In fact, as little as 1 h of simulation, US training increased the injection success rate of second year anesthesiology trainees from 40 % proficiency to 80 % [12].

However, in pain medicine, the targets are usually much smaller, and they rarely have easily identified large blood vessels. In this book, we have tried to include US images of most of the nerves, though the US image is not always useful. The US probe can be oriented parallel to the nerve (Fig. 7.6), which shows the nerve longitudinally (Fig. 7.7) or perpendicular to the nerve (Fig. 7.8), which shows the nerve in cross section (Fig. 7.9). Brown described using a paperclip (or similar object) over the nerve to help localization under

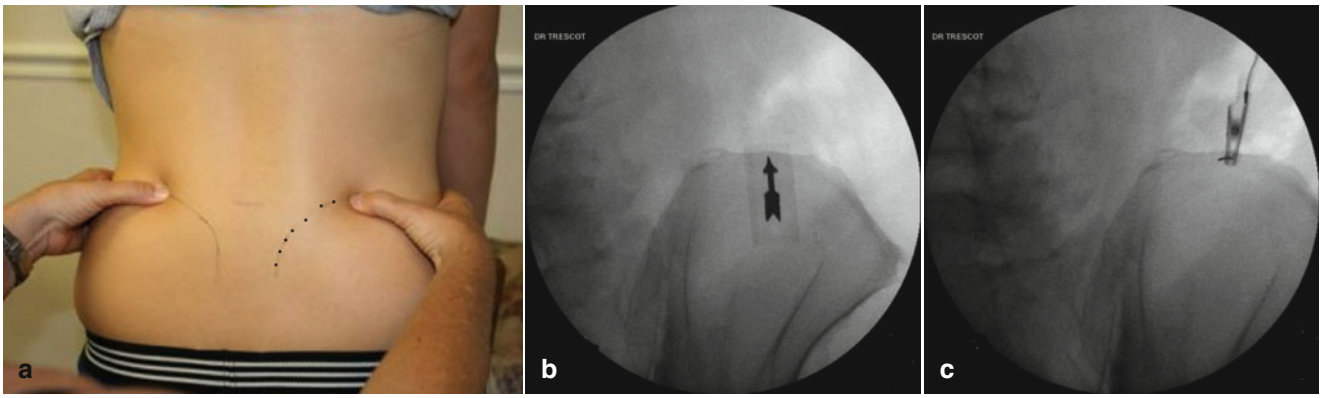

Fig. 7.5 Landmark location (**a**), fluoroscopic location (**b**), and fluoroscopy-guided injection (**c**) of the cluneal nerve (Image courtesy of Andrea Trescot, MD)

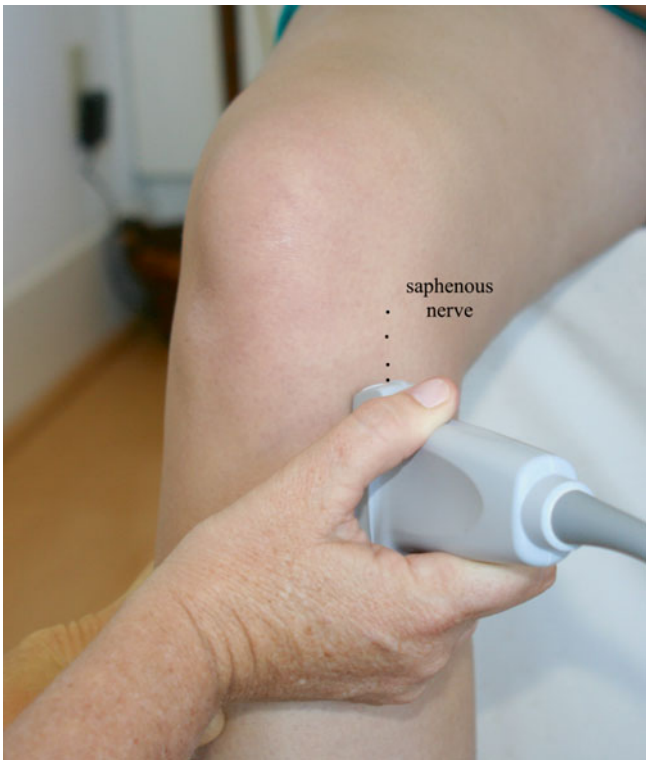

Fig. 7.6 Longitudinal (*long axis*) ultrasound probe placement (Image courtesy of Andrea Trescot, MD)

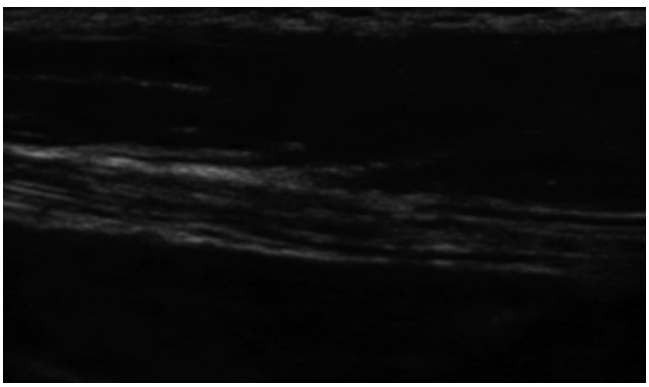

Fig. 7.7 Longitudinal (*long axis*) ultrasound image (Image courtesy of Andrea Trescot, MD)

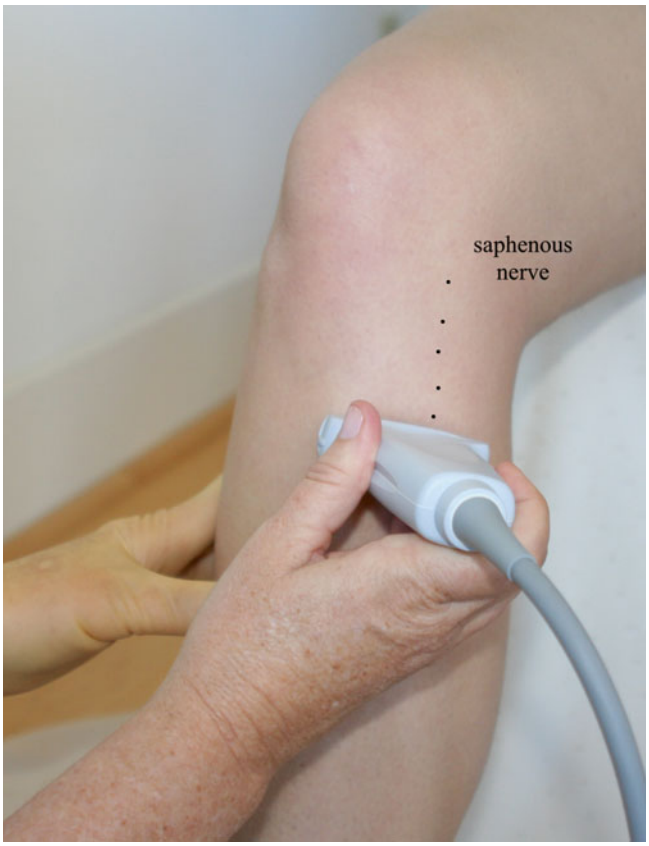

Fig. 7.8 Cross-sectional (*short axis*) ultrasound probe placement (Image courtesy of Andrea Trescot, MD)

ultrasound (Fig. 7.10) [13]. *Out-of-plane injections* (the needle perpendicular to the probe) result in the shortest path of the needle (Fig. 7.11), but the needle tip cannot be easily seen. The *in-plane injection* approach to the nerve allows visualization of the path of the needle but requires a longer injection path (Fig. 7.12).

Although it is often said that the "bone is your friend" for both fluoroscopy and ultrasound, perhaps a better saying for peripheral nerve injections is that "the vessel is your friend" [Thiago Nouer Fredrerico, MD – personal communication],

as most of the nerves have small blood vessels that travel with the nerve, visualized on US with practice.

Coaxial Tomography (CT)-Guided Nerve Blocks

Although the use of a *CT scan* for needle placement is likely more accurate than fluoroscopy, the radiation exposure can be much higher, and it is not possible to watch contrast in real time to look for vascular uptake. However, newer CT machines with faster spins and lower radiation doses may be changing this paradigm. Wagner [14] described over 2,000 epidurals done under CT guidance with only three intrathecal injections (early in his experience); he documented approximately 0.1 mrem per procedure to the operator, though he did not specifically measure the radiation dose to the patient.

Magnetic Resonance Imaging (MRI or MR Imaging)-Guided Nerve Blocks

MRI provides excellent visualization of soft tissues, but the detail is not usually enough to see the small nerves that are often entrapped. Newer technology (*MR neurography*), however, has increased the level of nerve detail seen and allowed for more accurate MRI-directed injections [15]. Needle placement in a standard MRI machine is relatively difficult because of the need to move the patient in and out of

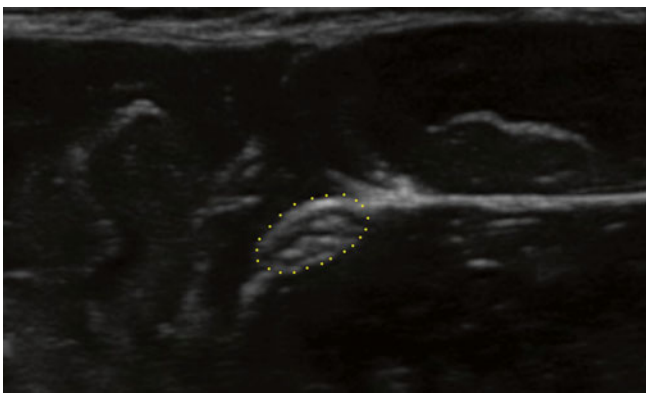

Fig. 7.9 Cross-sectional (*short axis*) ultrasound image (Image courtesy of Andrea Trescot, MD)

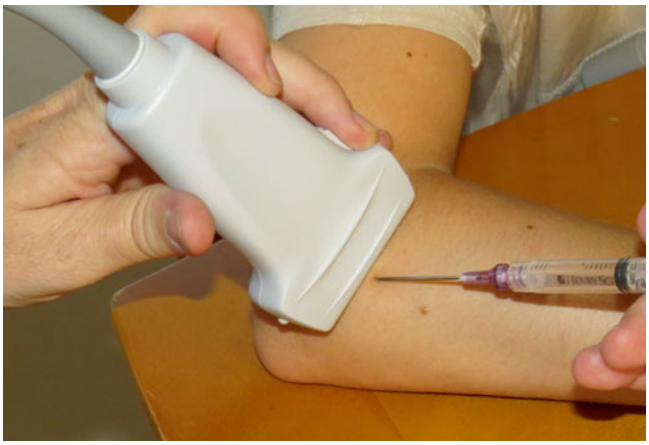

Fig. 7.11 Example of out-of-plane injection (Image courtesy of Andrea Trescot, MD)

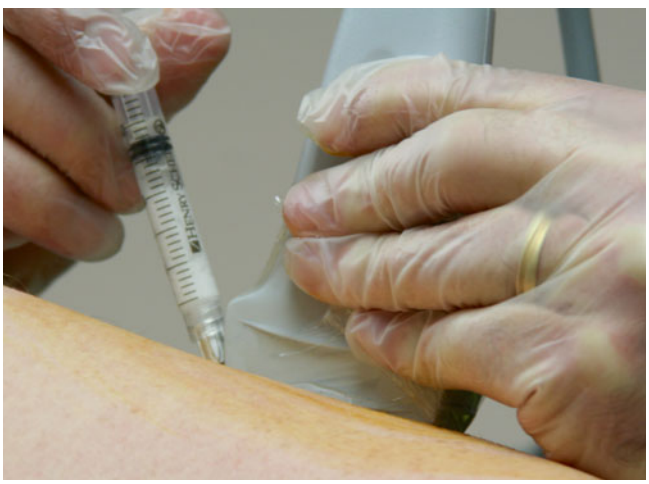

Fig. 7.12 Example of in-plane injection (Image courtesy of Michael Brown, MD)

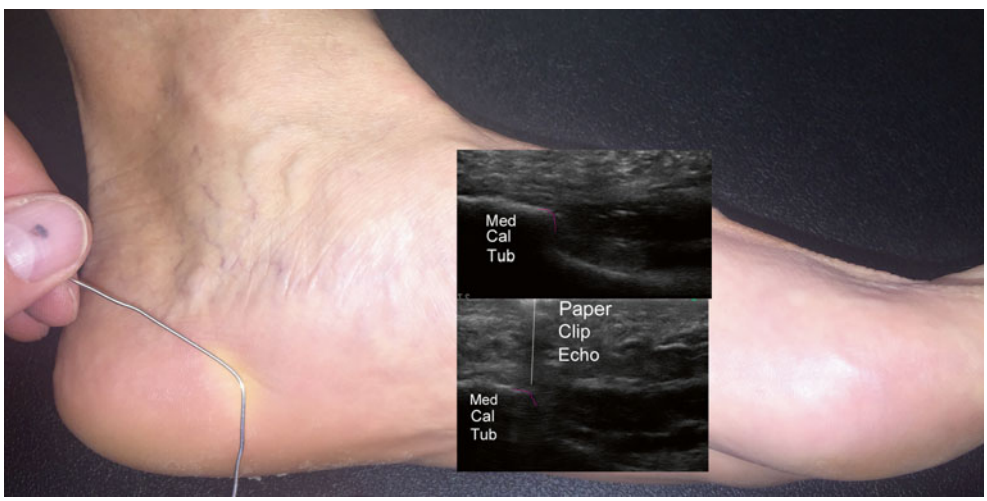

Fig. 7.10 Use of a paperclip to localize injection site (Image courtesy of Michael Brown, MD)

the bore of the machine. As such, injections perhaps would be easier to perform in open MRIs.

Hydrodissection

Hydrodissection is a technique of injections into scars or fascia to release entrapped nerves. The skin is supposed to move smoothly over the fascia, but often these scars are similar to adhesions in the epidural space, tethering the cutaneous and deep nerves and causing pain and autonomic dysfunction. *Neural therapy* (NT) was originally described for treatment of superficial scars. Local anesthetic, sometimes with small doses of steroids, is injected subdermally to separate the tissue plans, creating a hydrodissection of the tissues (Fig. 7.13).

Lyftogt described a number of case presentations using an injection of dextrose 5 % in sterile water (*D5W)* around a variety of nerves [16, 17]. The proposed mechanism suggested by Lyftogt was that dextrose seemed to bind to presynaptic calcium channels and inhibit the release of substance P and CGRP, thus having a beneficial effect of decreasing neurogenic inflammation. This was thought to have neurotrophic effects on growth factors that ultimately might provide a mechanism for subsequent nerve repair and decreased pain [18].

This suggestion has led a number of physicians skilled in ultrasonography to use US to isolate nerves, and to use saline or D5W to hydrodistend and hydrodissect tissues around the perineural region, mechanically decompressing the nerve. The use of D5W delivers dextrose to the perineural soft tissues, which may aid recovery of the nerve after decompression. More recently, the effects of "nanogram" dosing of dexamethasone have been reported in the stem cell literature as a means to promote differentiation of mesenchymal stem cells [19, 20]; as a result, some physicians add 30 nanograms of dexamethasone to the injection solution (personal communication, Michael Brown, MD).

Regenerative Injection Therapy

There are a number of basic regenerative technologies that can be used in some patients with peripheral nerve entrapment neuropathies and nerve injury. Once one gains an understanding of the critical anatomy as well as the ultrasonography skills required to target nerves, there are a variety of injectates available for injection techniques. For example, one author in this book (Michael Brown, MD) has used an autologous "platelet lysate" to hydrodissect around a focal entrapment neuropathy or nerve injury. A *platelet lysate* (PL) is a platelet-rich plasma (PRP) that has had the platelets lysed and the cell membranes and debris removed by filtration. PL has been shown to have natural repair proteomes to support human mesenchymal stem cells [21, 22] and numerous growth factors such as TGF-β, VEGF, PDGF, FGF, and EGF, which provide a paradigm for healing and repair [19]. The PL can be made from a patient's autologous PRP in a number of ways. A preparation of PRP can be mixed with patient's autologous thrombin in serum, or one can simply add calcium chloride to the test tube to activate the platelets in the PRP. A clot is formed, the platelets are lysed, and the growth factors contained within the platelets are discharged into the platelet-poor plasma, becoming plasma that is rich in platelet-rich growth factors (i.e., platelet lysate). This PL can then be injected in a similar fashion to the hydrodissection described previously.

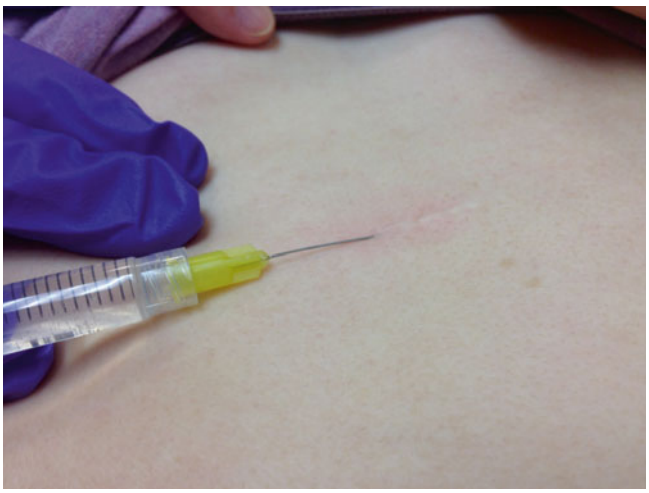

Fig. 7.13 Subcutaneous hydrodissection of a superficial scar (neural therapy) (Image courtesy of Andrea Trescot, MD)

Needles

The classic needle used for injections is the same that was used for spinal anesthetics, a sharp, beveled needle, such as a *Quinke* or *Chiba* needles, in a variety of lengths. Unfortunately, these sharp, cutting needles have been associated with a variety of nerve injuries and intravascular disasters (see Chap. 13). To decrease the risk of nerve damage from the long bevel of these needles, a shorter needle (*B-bevel*) was developed. Pencil-point needles (such as *Whitacre* and *Sprotte* needles) may decrease the intravascular risk, but they are difficult to steer. There are now injection needles specifically designed for peripheral nerve injections – some are insulated except at the very tip, some have introducers because of their blunt tips, and some are curved to improve "steer ability" (Fig. 7.14). The blunt-tipped needles should decrease the risk of nerve damage, unexpected vascular injections, and pneumothorax (see Chap. 13).

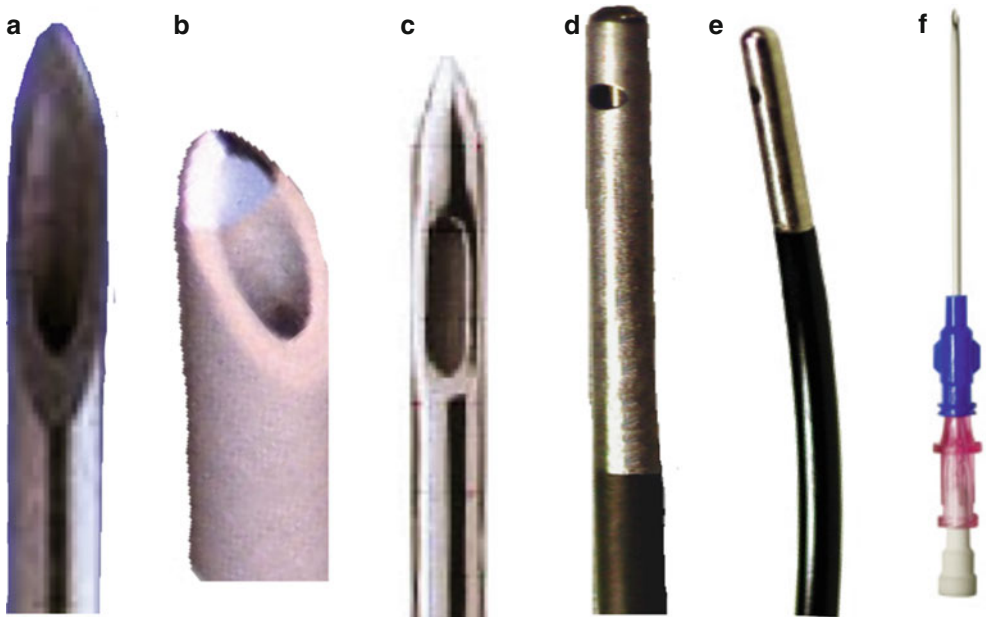

Fig. 7.14 Examples of types of needles used in peripheral nerve injections. (**a**) Quinke, (**b**) B-bevel, (**c**) Sprotte, (**d**) straight blunt tipped, (**e**) curved blunt tipped, (**f**) introducer for blunt-tipped needles (Image courtesy of Andrea Trescot, MD)

Complications

A discussion of complications is found in Chap. 13.

Summary

For peripheral nerve entrapment, there are multiple injection techniques, involving the medication as well as the guidance technique. Injections can help to localize and confirm nerve entrapments, delivering medications directly to the appropriate site. Landmarks, peripheral nerve stimulators, fluoroscopy, CT, MRI, or ultrasound can direct these injections, and a variety of medications can be used. By matching the technique to the patient, the clinician can provide the best standard of care.

References

1. Bogduk N. Cervicogenic headache: anatomic basis and pathophysiologic mechanisms. Curr Pain Headache Rep. 2001;5(4):382–6.
2. Bogduk N. Role of anesthesiologic blockade in headache management. Curr Pain Headache Rep. 2004;8(5):399–403.
3. Barnsley L, Lord S, Bogduk N. Comparative local anaesthetic blocks in the diagnosis of cervical zygapophysial joint pain. Pain. 1993;55(1):99–106.
4. Trescot AM. Local anesthetic "resistance". Pain Physician. 2003;6(3):291–3.
5. Tuel SM, Meythaler JM, Cross LL. Cushing's syndrome from epidural methylprednisolone. Pain. 1990;40(1):81–4.
6. Blumenfeld A, Ashkenazi A, Napchan U, Bender SD, Klein BC, Berliner R, et al. Expert consensus recommendations for the performance of peripheral nerve blocks for headaches – a narrative review. Headache. 2013;53(3):437–46.
7. Rynd F. Neuralgia – introduction of fluid to the nerve. Dublin Med Press. 1845;13:167–8.
8. Labat G. Regional anesthesia: techniques and clinical applications. Philadelphia: WB Saunders; 1922.
9. Loughnane F. Peripheral nerve block materials. In: Harmon DC, Barrett J, Loughnane F, Finucane B, Shorten GD, editors. Peripheral nerve blocks and peri-operative pain relief. 2nd ed. Philadelphia: Saunders Elsevier; 2011. p. 41–6.
10. Fanelli G, Casati A, Garancini P, Torri G. Nerve stimulator and multiple injection technique for upper and lower limb blockade: failure rate, patient acceptance, and neurologic complications. Study Group on Regional Anesthesia. Anesth Analg. 1999;88(4):847–52.
11. Abrahams MS, Aziz MF, Fu RF, Horn JL. Ultrasound guidance compared with electrical neurostimulation for peripheral nerve block: a systematic review and meta-analysis of randomized controlled trials. Br J Anaesth. 2009;102(3):408–17.
12. Niazi AU, Haldipur N, Prasad AG, Chan VW. Ultrasound-guided regional anesthesia performance in the early learning period: effect of simulation training. Reg Anesth Pain Med. 2012;37(1):51–4.
13. Brown MN. Intra-articular joint injections. In: Diwan S, Staats PS, editors. Atlas of pain management procedures. New York: McGraw-Hill; 2014.
14. Wagner AL. CT fluoroscopy-guided epidural injections: technique and results. AJNR Am J Neuroradiol. 2004;25(10):1821–3.
15. Fritz J, Chhabra A, Wang KC, Carrino JA. Magnetic resonance neurography-guided nerve blocks for the diagnosis and treatment of chronic pelvic pain syndrome. Neuroimaging Clin N Am. 2014;24(1):211–34.
16. Lyftogt J. Subcutaneous prolotherapy for achilles tendinopathy: the best solution? Aust Musculoskelet Med. 2007;12(2):107–9.
17. Lyftogt J. Subcutaneous prolotherapy treatment of refractory knee, shoulder and lateral elbow pain. Aust Musculoskelet Med. 2007;12(2):110–2.
18. Lyftogt J. The Hackett Hemwall Foundation and the Italian Society for Prolotherapy sponsored. "Neural Prolotherapy Workshop meeting." Ferrara. 2010.

19. Xiao Y, Peperzak V, van Rijn L, Borst J, de Bruijn J. Dexamethasone treatment during the expansion phase sustains stemness of mesenchymal stem cells from human bone marrow. Cell Res. 2008;18:s116.
20. Wang H, Pang B, Li Y, Zhu D, Pang T, Liu Y. Dexamethasone has variable effects on mesenchymal stromal cells. Cytotherapy. 2012;14(4):423–30.
21. Crespo-Diaz R, Behfar A, Butler G, Padley D, Sarr M, Bartunek J, et al. Platelet lysate consisting of a natural repair proteome supports human mesenchymal stem cell proliferation and chromosomal stability. Cell Transplant. 2011;20:797–811.
22. Awidi A, Abu harfeil N, Jafar H, Abu A, Ismail S, Kailani M. The in vitro effect of autologous platelet lysate on different types of mesenchymal stem cells. Regen Res. 2012;1(1):25–32.

Neurolytic Techniques

8

Brett Lockman, Andrea M. Trescot, and Daniel Krashin

Introduction

Once a nerve has been identified as causing pain, one of the ways of treating that pain involves the injection of nerve-killing medicines (*chemoneurolysis*), or the application of thermal forces – heat (radiofrequency lesioning) or cold (cryoneuroablation) – for nerve destruction.

Current guidelines do little to truly guide a physician presented with a subtle case of peripheral nerve entrapment. Also, should the clinician ultimately make the diagnosis of intractable pain due to peripheral nerves, the current literature largely fails to direct a specific and effective treatment. This may indirectly prolong patient suffering or lead to less effective interventions.

Sadly, the unwillingness to perform neurolysis for chronic peripheral somatic neuralgia is so high that the techniques are rarely taught, even in well-respected pain fellowship programs. This is unfortunate, as their use, however narrow, is often the optimal choice for the well-selected patient. This book is an attempt to remind the reader of the multitude of entrapment neuropathies that can otherwise baffle clinicians and to present options for treatment, including the rarely needed but extremely effective techniques of chemoneurolysis, cryoneuroablation, and radiofrequency lesioning. Each of these techniques has indications and contraindications, which will be discussed separately.

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Chemoneurolytic Techniques

Much has been written about chemoneurolysis for treating end-of-life, cancer-related pain, but little is published in modern medical literature on the safe and effective use of the technique for chronic, nonmalignant, peripheral pain syndromes. In fact, because of the heightened risk that chemoneurolysis poses in *non-selected* peripheral nerve pathologies, the method has largely been dismissed for *any* nonmalignant pain application.

History of Chemoneurolysis

Chemoneurolysis is the earliest form of targeted, minimally invasive nerve ablation used in the era of modern medicine. After the development of the modern *hypodermic needle* and the glass syringe by Dr. Alexander Wood in 1853, injection therapy was explored for many medical ailments of the time. By 1863, Luton had performed the first documented chemoneurolysis by injecting *silver nitrate* and *hypertonic saline* solutions for the treatment of chronic sciatica [1]. Varying degrees of success with other solutions were reported in the years following Luton's efforts, but as with silver nitrate, they were often accompanied by unacceptable sequelae. *Ethyl alcohol* treatment for chronic nonmalignant pain was first utilized by Schlosser in 1903 to treat convulsive trigeminal neuralgia, with significant lasting benefit [2]. Doppler, Dogliotti [3], and Swetlow [4] used alcohol neurolysis in the 1920s with much success. This prompted investigation into other compounds (such as phenol) to create vascular ablation and eventually nerve ablation, with less periprocedural pain and greater C and Aδ fiber specificity than alcohol. Though not without inherent risks, careful application of these and other chemicals (such as *glycerin*, *ammonium salts*, and, to a lesser degree, *hypertonic saline* and *chlorocresol*) formed the basis of long-lasting, nonsurgical, analgesia/neurolytic blocks for decades thereafter, partly due to the enthusiasm of

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the relatively new field of regional anesthesia and also due to the relative paucity of other non-opioid analgesia options.

In the 1960s, significant advances were made in pharmacotherapy, neurosurgery, *epidurals, opioids*, *cryoneuroablation*, and *radiofrequency*, so that chemoneurolysis fell out of favor for peripheral nerve applications. Still, it maintained a strong presence in the field of malignant pain treatment, where the benefit of broader, liquid application to deep autonomic ganglia or epidural infusion often afforded better results (or was the sole viable option) with "acceptable" side effects in an otherwise agonized, often bed-bound patient, near the end of life.

As the field of interventional pain has grown and physicians from different backgrounds have entered the specialty, there has been a resurgence of interest in chemoneurolysis as treatment for peripheral nerve entrapment pain as well as chronic spasticity following central neurologic injury.

Chemoneurolytic Agents

Alcohol

Colorless, pharmaceutical-grade, water-miscible, dehydrated *ethyl alcohol* is typically available in 95–98 % concentrations ("*absolute alcohol*") in 1 or 5 ml, single-use ampules for neurolysis application. Ethyl alcohol is hypobaric to cerebrospinal fluid, though for most peripheral applications, this is not relevant. However, every neurolytic solution's baricity must be considered in cases of potential CSF communication (as with trigeminal, gasserian, or intercostal neurolytic applications) to avoid distant spread.

Concentrated alcohol is painful upon injection, so a preliminary anesthetic block is in order. Alcohol quickly diffuses from the injection site and is oxidatively metabolized primarily via a hepatically mediated *alcohol dehydrogenase* reaction shortly thereafter. Patients with alcohol dehydrogenase deficiency (common in Asians) or those taking beta-lactam antibiotics, metronidazole, or disulfiram may have increased intoxication-like side effects of flushing, stumbling, dizziness, etc. [5].

Upon injection and contact with nervous tissue, alcohol exerts a dehydrating effect by extracting cholesterol and precipitating lipoprotein from the nerve cell lipid membranes [6]. Whether this affects immediate and long-term outcomes is clinically difficult to ascertain, as outcome varies even when exposed nerves are directly painted with neurolytic solutions [7].

Complete (or near complete) analgesia may be obtained via injection of a high concentration of ethanol perineurally, and relief should endure for 12 months or longer. Although blocks may be repeated, efficacy appears to diminish following numerous treatments, likely secondary to neurogenic sprouting or arborization.

In addition to the potential injectate spread to nontargeted structures, risks of alcohol neurolysis include chemical neuritis (typically from incomplete lysis), neuroma formation, and tissue necrosis with wound formation if too concentrated a solution is placed superficially. Localization under image guidance is highly recommended, and adjunctive nerve stimulator use is also beneficial. To minimize neuritis, B. Lockman (personal data, not published) allows sufficient time for the alcohol to take effect, follows with a small dose of steroid (6–8 mg of betamethasone or 10–15 mg of triamcinolone acetonide), and then clears the needle with 0.5 % bupivicaine before needle removal. Adding a small dose of steroid reduced neuritis incidence from 5–6 % to ~1 % (assessed 1 week post-injection, B. Lockman's personal data, not published).

Efficacy of multiple, low-concentration (4 %) injections was reported by Dockery [8]. B. Lockman (personal data, not published) produced similar results with not only Morton's neuroma treatment but also with the most dorsal cutaneous branches of the superior and middle cluneal nerves, as described by Maigne [9] and Lyftogt [10]. Arguably, repeat low-dose application of alcohol or phenol in combination with other diluents exits the realm of true neurolysis and enters into prolotherapy, neural prolotherapy, or simply prolonged regional anesthesia.

A recent study using a nerve catheter to provide multiple celiac plexus blocks using alcohol showed that even this technique, which provides higher alcohol concentrations, was safe and well tolerated [11].

Phenol

Phenol, also known as *carbolic acid* or *phenylic acid*, is a weakly acidic aromatic compound first discovered in 1834 and purified in 1841. A 5 % carbolic acid solution was the first antiseptic used by Joseph Lister and led to his pivotal work *Antiseptic Principle of the Practice of Surgery*, in 1867. It was first used for neurolytic analgesia by *Putnam* and Hampton in 1936 [12].

In pure form, phenol is crystalline and will burn skin on contact. It oxidizes and turns red upon exposure to sunlight or air. It is freely soluble in alcohol and somewhat less so in glycerin. It may be prepared by compounding pharmacies or purchased directly from medical supply houses in various "aqueous" concentrations or in glycerin. If refrigerated and protected from light, its shelf life is 12–18 months. The addition of glycerin imparts greater viscosity to the solution, requiring larger-gauge needles. It mixes well with iodinated contrast agents, which, with fluoroscopic guidance, can be used to monitor peri-injection medication spread.

Phenol's mechanism of neurolysis is directly dependent upon the concentration of phenol used, the axonal diameter of the treated nerve, and whether the nerve is myelinated. At concentrations of 1–2 %, phenol acts as a long-lasting

anesthetic with additional pro-inflammatory properties. It is also used at these doses in combination with osmotic and chemotactic agents such as dextrose and glycerin as a *prolotherapy* solution [13, 14]. Upon injection of small volumes of 3–5 % phenol, there can be an immediate burning sensation followed rapidly by a painless warmth, followed by complete anesthesia of small unmyelinated C fibers [15]. Phenol in small volumes can therefore act as its own local anesthetic agent, if injected slowly and if appropriate time for neurolysis is allowed. Protein denaturation follows, as does segmental demyelination of Aδ fibers. Smaller nerves are more fully destroyed, as they are more susceptible to phenol's ablative effect on surrounding neural microvasculature. Large myelinated nerves may sustain patchy, less predictable blockade. From a neurolytic perspective, 3 % phenol approximates the effect of a 35–40 % concentrated ethanol solution.

At phenol concentrations greater than 5 %, true neurolysis occurs, and phenol concentrations between 5 % and 15 % are widely used. At higher concentrations, phenol causes pan-axonal damage (i.e., axonal and *Wallerian degeneration*) via protein coagulation and precipitation, *Schwann cell destruction* affecting the neurolemma basal lamina, focal vasculitis, and neurogenic edema of the residual nerve. It will also cause generalized tissue necrosis [16]. Phenol actually has a dual action: it has an immediate local anesthetic effect (which anesthetizes myelinated nerves) and a longer-term effect of denaturing the proteins within nerves [17]. This effect takes time to establish itself, reaching its peak after 2 weeks. Despite its broader effect on the nerve, phenol may affect a less permanent block on larger myelinated nerves, as it may not ablate the nerve cell body as well as alcohol. A second treatment is often needed after waiting 2 weeks to note the final result of the initial block. However, because it more effectively ablates the endoneurium, the small unmyelinated C fibers may remain permanently ablated following the initial treatment.

Regeneration can occur around 4–5 months. Phenol at its usual concentration is heavier than water (or CSF), and the patient should be positioned accordingly.

There may be some post-injection aching and pruritic dysesthesias for a few days following injection.

Phenol Metabolism and Toxicity

Because of its use as a disinfecting agent and in plastic manufacture, some clinicians shy away from phenol, and thus its safety profile and metabolism deserve greater discussion. As serum phenol levels increase, an initial neuroexcitatory response of tachycardia, tachypnea, mild tremor, and vasodilation/constriction occurs. Because these symptoms mimic local anesthetic toxicity and since a pretreatment anesthetic block is usually performed, care should be taken to limit the target site (and volume injected) to a small focus, performing any additional treatments at a later date. Phenol's putative selectivity for cardiac sodium channel subtypes over skeletal muscle sodium channels is responsible for its arrhythmogenic effect [18]. Severe toxicity (>5 g) may progress rapidly to seizure, renal failure, and cardiopulmonary collapse. Ventilator support, hemodialysis, and charcoal hemoperfusion [19] have all been used for effective resuscitation and symptom management until natural clearance normalized. Nomoto [20] reported phenol elimination half-lives of 64.0 ± 7.3 min for conjugated phenol and 30.3 ± 2.8 min for unconjugated phenol. Peak concentration occurred at ~19 min, and after 8 h, conjugated phenol urinary excretion was 52 ± 5 %.

Generally, the recommended dose of injected phenol should not exceed 120 mg to avoid even mildly toxic responses. However, it is unlikely that serious effects would occur at six to eight times that amount. For reference, over-the-counter Cepastat® Extra Strength lozenges contain 29 mg of phenol per lozenge. The maximum recommended adult dose (24 lozenges per day) results in a 696 mg total oral dose.

Phenol is hepatically metabolized primarily via three separate enzymatic pathways. Initial sulfation by cytosolic sulfotransferases results in a phenyl sulfate metabolite, which, at low phenol doses, may be the predominantly excreted marker. The cytochrome P450 2E1 (CYP2E1) oxidative pathway also acts to convert phenol to carbon dioxide and water. Approximately 50 % of phenol metabolism occurs via CYP2E1, which also produces hydroquinone and, to a lesser degree, catechol [21]. With higher doses, additional phenol enters the uridine diphosphate glucuronosyltransferase (UGT) conjugation pathway, resulting in a phenyl glucuronide metabolite. Peroxidation may also occur and produce small quantities of benzoquinone or biphenol. There is no evidence that this biphenol metabolite has any endocrine-disrupting estrogen affinity [22]. Final renal excretion is primarily via urine. Pitrowsky [22] showed that phenol is in fact a natural product of human metabolism and is found in both free and conjugated forms in the urine, with 8.7 ± 2.0 mg/day total daily excretion rate. It appears in lower concentrations in those with raw vegan diets [23], and its metabolism is age dependent. Though far from benign, to date, phenol is considered noncarcinogenic in humans, even with chronic exposure.

Though phenol is rarely used for nerve ablation in children, it is worth noting that the CYP2E1 isozymes are developmentally regulated. Thus, children receiving a phenol injection for cerebral palsy spasticity could potentially tolerate higher weight-based dosing, since diminished CYP2E1-mediated oxidative stress would presumably attenuate toxic metabolite production [24]. However, the specific pharmacokinetics of phenol in children is unknown.

Unique Phenol Precautions

Concentrated phenol will immediately burn skin and mucous membranes. Eye protection is a must, and spill contamination precautions should be taken. These include Luer-lok syringe/connector tubing (avoid use of slip connections which may easily separate, resulting in phenol splatter to skin or eyes) and surrounding the injection site with gauze or maintaining water-soaked gauze at the ready, in the event of spillage. Use of protective latex or vinyl gloves by staff when assisting with phenol solution mixing is crucial, and maintaining a polyethylene glycol (PEG) solution (such as MiraLAX®) at hand is prudent in the event of skin contamination with higher concentrations, since PEG will neutralize carbolic acid more quickly than water or an isopropyl alcohol swab.

There are numerous reports that phenol exhibits higher affinity than alcohol for perivascular sympathetic nerve fibers, mucous membranes, and vascular tissue itself. However, Lema's postmortem analysis of patients who received over 3 g of intrapleural phenol for esophageal neoplastic pain failed to reveal vascular or even parenchymal damage [25]. If chemoneurolysis *is* desired in a highly vascular area, discretion merits use of serial low-concentration ethanol treatments as a medicolegal precaution, given the often cited report of phenol's vascular affinity.

Thermal Neurolysis Techniques

Radiofrequency Ablation

Egyptians were using heat to destroy tissue as early as 3000 BC [26]. In 400 BC, Hippocrates described the use of heat to treat shoulder dislocations [27]. Starting in the 1870s, direct current electricity (DC current) was used experimentally to produce discrete tissue lesions in addition to powering devices. In the 1920s, Cushing and Bovie [28] introduced DC current into clinical practice as a means to produce surgical thermocoagulation for hemostasis. In 1931, Kirschner [29] introduced the coagulation of the Gasserian ganglion in patients with trigeminal neuralgia. In the 1950s, Sweet and Mark [30] adapted this technique to generate neural lesions. Unfortunately, these generators produced unpredictable and irregular lesions that varied in size as much as fourfold. The first commercial generators, developed by Aranow [31] and Cosman [32], were released in the 1950s and used higher frequency (in the range of 300–500 KHz) to produce more reliable lesions. Since high frequencies in this range were also used in radio transmitters, the term "radiofrequency lesioning" (RF) was coined.

Radiofrequency ablation (RFA) is sometimes misunderstood as "cooking" or direct heating of tissue. In fact, conventional radiofrequency neurolysis relies on heating of tissue due to the electrical resistance of tissue between poles

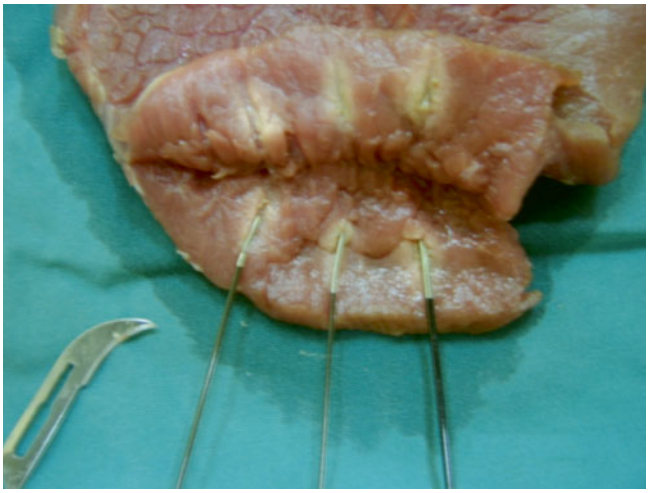

Fig. 8.1 Radiofrequency lesion in raw chicken (Image courtesy of Agnes Stogicza, MD)

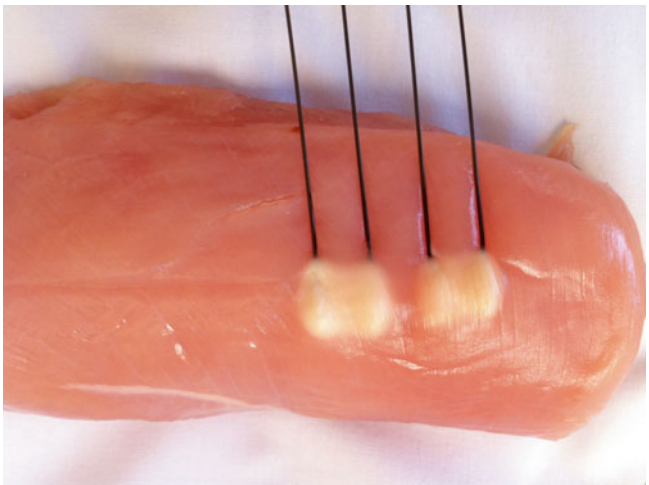

Fig. 8.2 Radiofrequency bipolar lesion in raw chicken (Image courtesy of Agnes Stogicza, MD)

of the electrode. This heat causes tissue destruction in a controllable and predictable manner, in an elliptical pattern along and parallel to the exposed area of the radiofrequency catheter's exposed tip (Fig. 8.1) [33]. A radiofrequency circuit consists of a generator, an active electrode that delivers the current, a thermistor to measure tissue temperature, and a passive or dispersing electrode (which is usually a large grounding pad). A reusable or disposable cannula is advanced to the target area, and then the RF probe is advanced through the cannula. Each cannula is available in a variety of lengths (usually 50, 100, and 150 mm) and gauges (18–22 g), is insulated except for the active tip (5–20 mm), and is either sharp or blunt tipped, straight or curved.

Anatomic landmarks, with or without nerve stimulation, can be used to place the cannula on the target tissue, and either *bipolar* (two probes placed close together with the current running between them) (Fig. 8.2) or *unipolar* (the

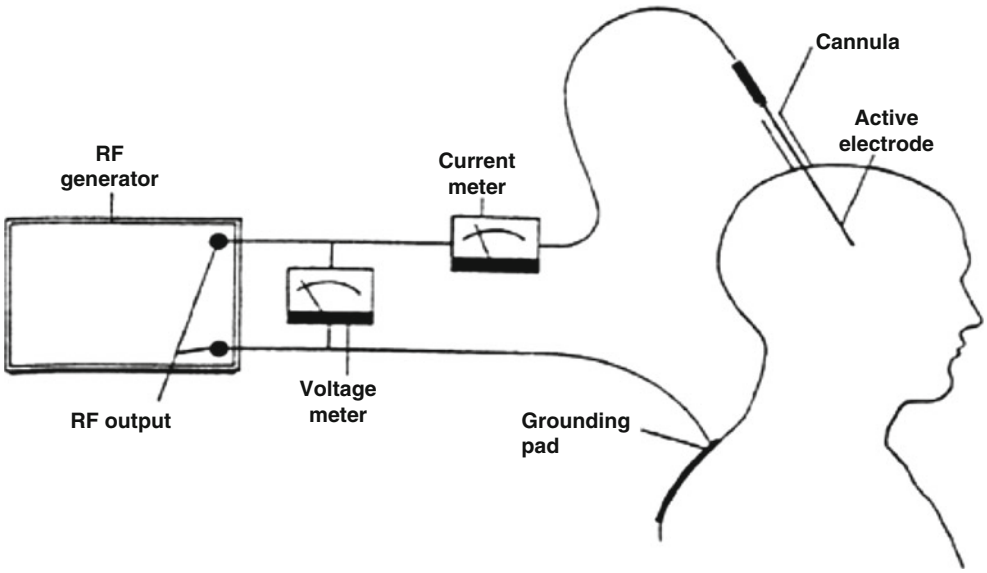

Fig. 8.3 Radiofrequency circuit for unipolar lesioning (Image courtesy of Andrea Trescot, MD, from Trescot and Hansen [65], with permission)

current passes from the tip of the probe to the dispersive electrode) (Fig. 8.3) lesions are then created when the current passes through the tissue. The size of the lesion, which is usually shaped as an inverted cone, does not correlate well with the length of time of the lesioning or the temperature. The lesion radius is maximal at the most proximal portion of the active tip, which means that the distal tip might not even be incorporated into the lesion. A nerve in perpendicular contact with the probe may only be partially lesioned. This has led to attempts to place the probe tangentially to the nerve when possible (Fig. 8.4).

This thermocoagulation is painful and usually requires sedation and analgesia, although judicious use of local anesthetic through the RF cannula can obviate that need. For larger areas of neurolysis, *cooled radiofrequency probes* (cooled RF) are available. Paradoxically, the cooled probes result in a larger thermocoagulation lesion, since they prevent the area adjacent to the probe from being excessively heated.

Pulsed radiofrequency (PRF) treatment relies on high-frequency pulses of electric current. This has been shown to have a select effect on sensory nerves while sparing motor nerves and other vital structures [34], and there are multiple case reports of its use for nerve entrapments and neuropathic pain [35–40]. PRF lesioning, unlike conventional RF, does not apparently disrupt the myelin sheath and therefore has been used successfully on large nerves. Unfortunately, since it does not cause thermocoagulation of nerve tissue and is therefore not technically neurodestructive, the payment codes for neurolysis cannot be used, and pulsed radiofrequency is usually not covered by health insurance within the United States.

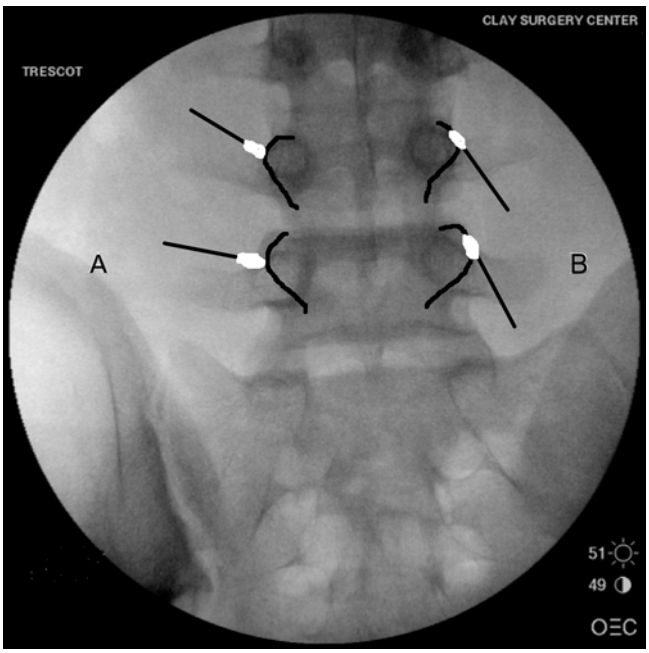

Fig. 8.4 Radiofrequency probe placement for medial branch lesioning. *A* perpendicular to the nerve; *B* parallel to the nerve (Image courtesy of Andrea Trescot, MD)

Cryoneurolysis

The use of cold for pain relief has been observed and used clinically since the dawn of medicine, initially with the application of ice to injuries, then as *ether spray* for topical anesthesia. In 1917, Trendelenburg was the first to report that cooling nerves produces prolonged and reversible loss of its function [41]. However, modern cryoneurolysis (also known as *cryoneuroablation*, *cryoanalgesia*, *cryoablation*, or *cryosurgery*) dates to 1961, when Cooper developed the technique of using liquid nitrogen traveling down a hollow probe [42]. This was later improved with the introduction of cryotherapy probes cooled by carbon dioxide [43] and then by nitrous oxide [44]. It was Lloyd and his colleagues that coined the term *cryoanalgesia* for its use in pain management [44].

The cryoprobe consists of a hollow tube with a smaller inner tube. A high-pressurized gas (usually CO_2 or N_2O) at 600–800 psi goes through the smaller tube and is released into the larger, low-pressure, outer tube through a microscopic aperture (0.002 mm) (Fig. 8.5). The gas expands quickly at the distal tip in an adiabatic process, dropping the distal tip to a temperature as low as −70 °C (Joule-Thompson effect) [16], creating an ice ball (Fig. 8.6). The gas then travels back to the machine where it is scavenged through a ventilated outlet, making no contact with the patient tissues. Most cryo machines have an incorporated nerve stimulator and thermistor to measure temperature in the tissue (Fig. 8.7a, b), though there are some devices that do not have a nerve stimulator (Fig. 8.7c, d).

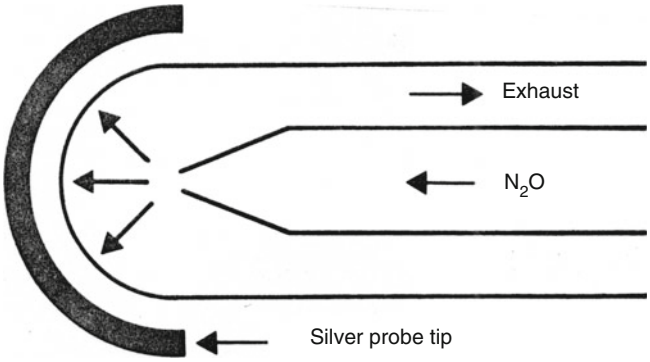

Fig. 8.5 Diagram of Joules-Thompson effect (Image courtesy of Epimed®)

Cryoneurolysis exerts its effect by freezing the nerve and disrupting the vasa nervorum, which causes disruption and Wallerian degeneration of the axon but preserves the myelin sheath and endoneurium (Fig. 8.8) [45]. *Neurotropin* is not released from exposed nerve endings during cryoneurolysis procedures, unlike with other techniques such as surgical or electrocautery transection; it is apparently neurotropin release that leads to the formation of painful neuromas [46]. Thus, the nerve regrows along the same pathway without neuroma formation. In rats, full recovery of sensory and motor nerve function has been observed after three rounds of cryoneurolysis with time to recover between each session (Fig. 8.9) [47]. This return of function is unique to cryoneurolysis, and it allows a period of prolonged analgesia, usually 6–8 months, which allows for a window of

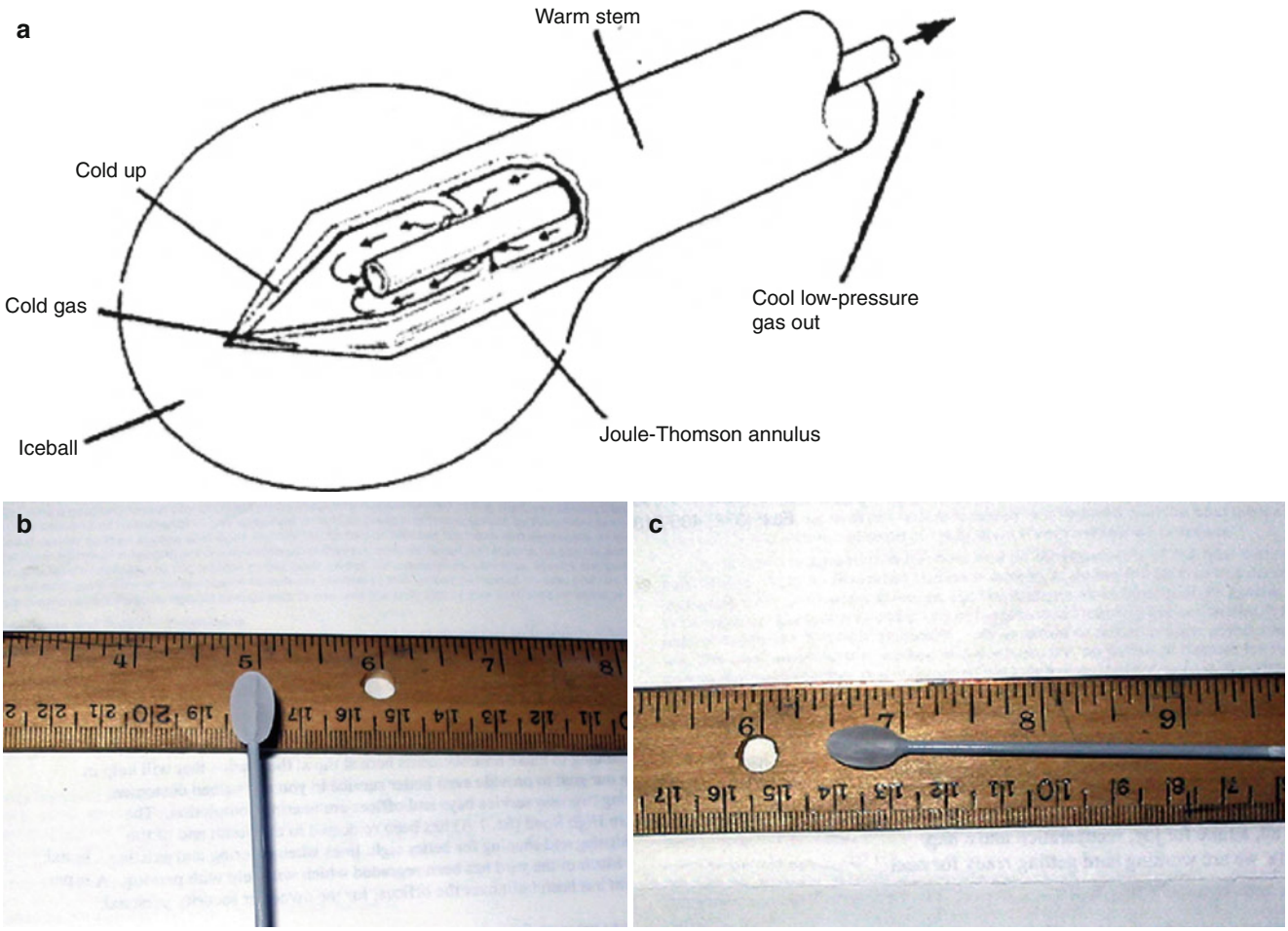

Fig. 8.6 Formation of ice ball. (**a**) Mechanism ice ball formation; (**b**, **c**) size of ice ball (Image courtesy of Epimed®)

aggressive rehabilitation and then return of normal function (Fig. 8.10). Wang described cryoneurolysis performed on 12 patients, reporting pain relief for 1–12 months, but specifically noting that the patients resumed normal activities during this period of remission [48].

Cryoanalgesia is beneficial for lasting pain relief from many peripheral nerves, with good results reported in many different areas, including cranial nerves [49], occipital nerves [46], thoracic nerves [50], ilioinguinal [51] and genitofemoral nerves [52], foot nerves [53], phantom limb pain [54], and many others [55]. The nerve will regenerate but usually without the pain. Repeat freezing of the nerves can yield results as good as the primary attempt [56].

The cryoprobe can be placed using landmarks (Fig. 8.11), though for deeper structures, it is useful to direct the cryoprobe under fluoroscopy or ultrasound guidance. Placing the

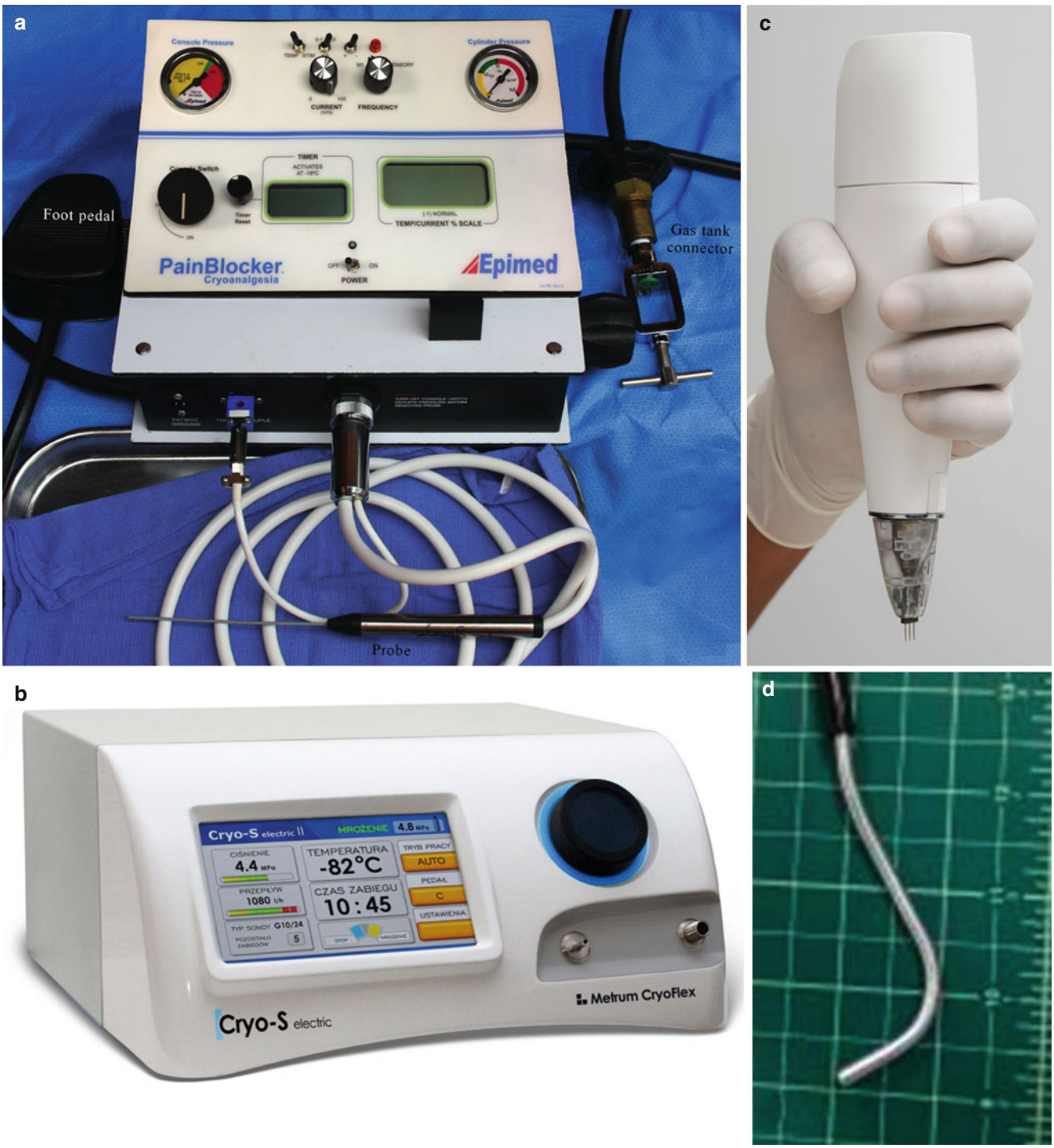

Fig. 8.7 Cryoneurolysis machines. (**a**) Wallach/Epimed Pain Blocker machine (Image courtesy of Epimed); (**b**) Metrum Cryoflex Cryo-S machine (Image courtesy of Metrum Cryoflex); (**c**) Myoscience Iovera machine (Image courtesy of Myoscience); (**d**) Atricure cryoneurolysis probe (Image courtesy of Atricure)

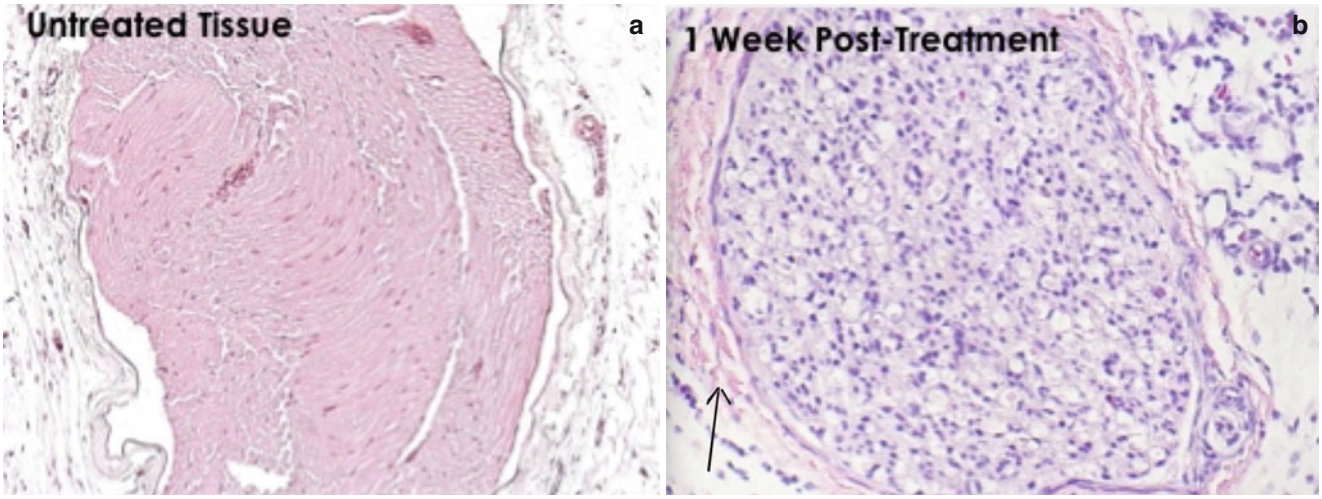

Fig. 8.8 Pathology slide of nerve tissue showing untreated tissue on the *left* and 1 week after cryoneurolysis on the *right*. Note the intact myelin sheath (*arrow*) (Image courtesy of Myoscience)

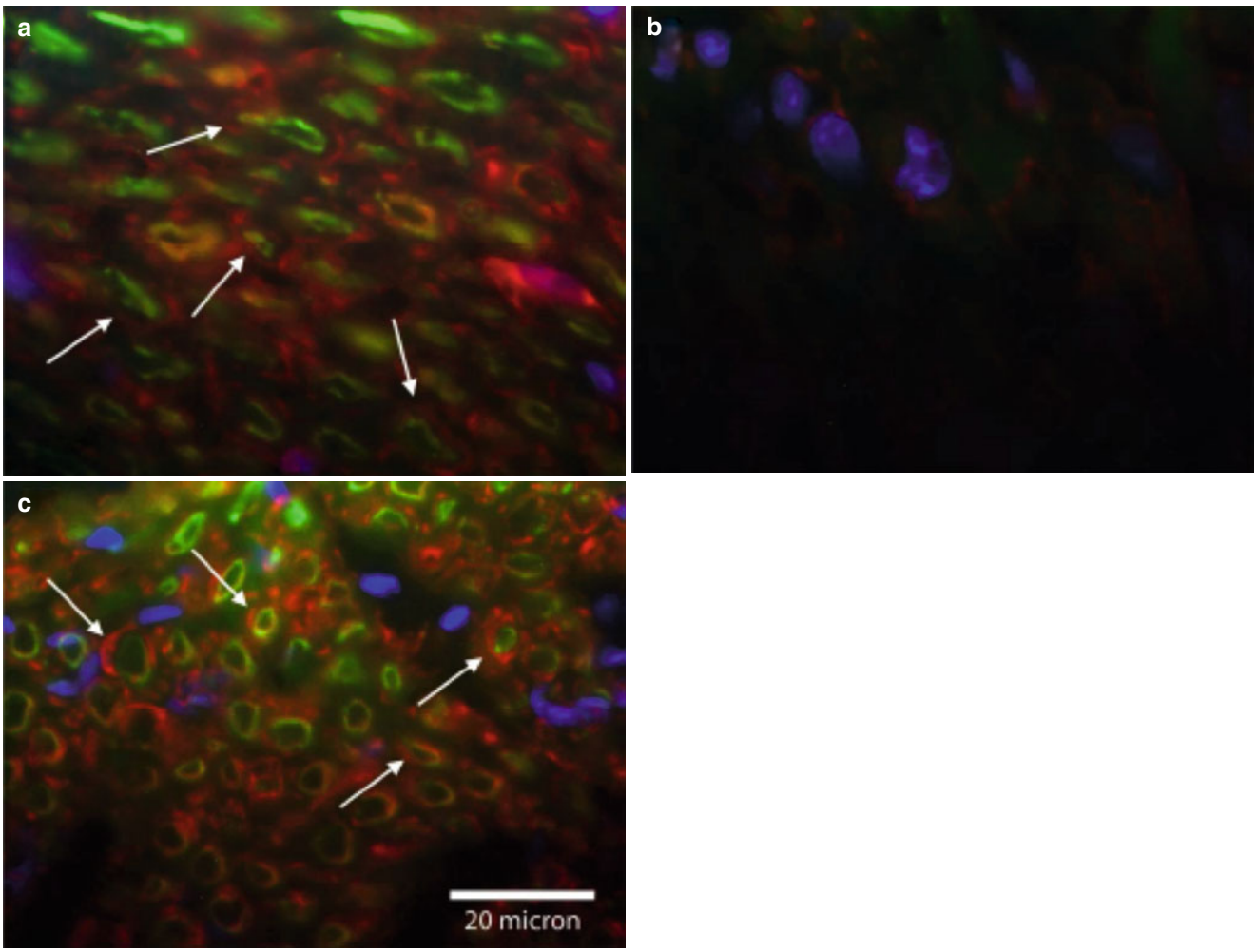

Fig. 8.9 Immunofluorescence staining of nerve tissue. *Green* neuron, *red* Schwann cell, *blue* nuclei. (**a**) untreated control tissue; (**b**) 1 week after cryoneurolysis showing complete loss of neurons, (**c**) 32 weeks after multiple refreezes, showing complete regeneration (Image courtesy of Myoscience, from Hsu and Stevenson [66])

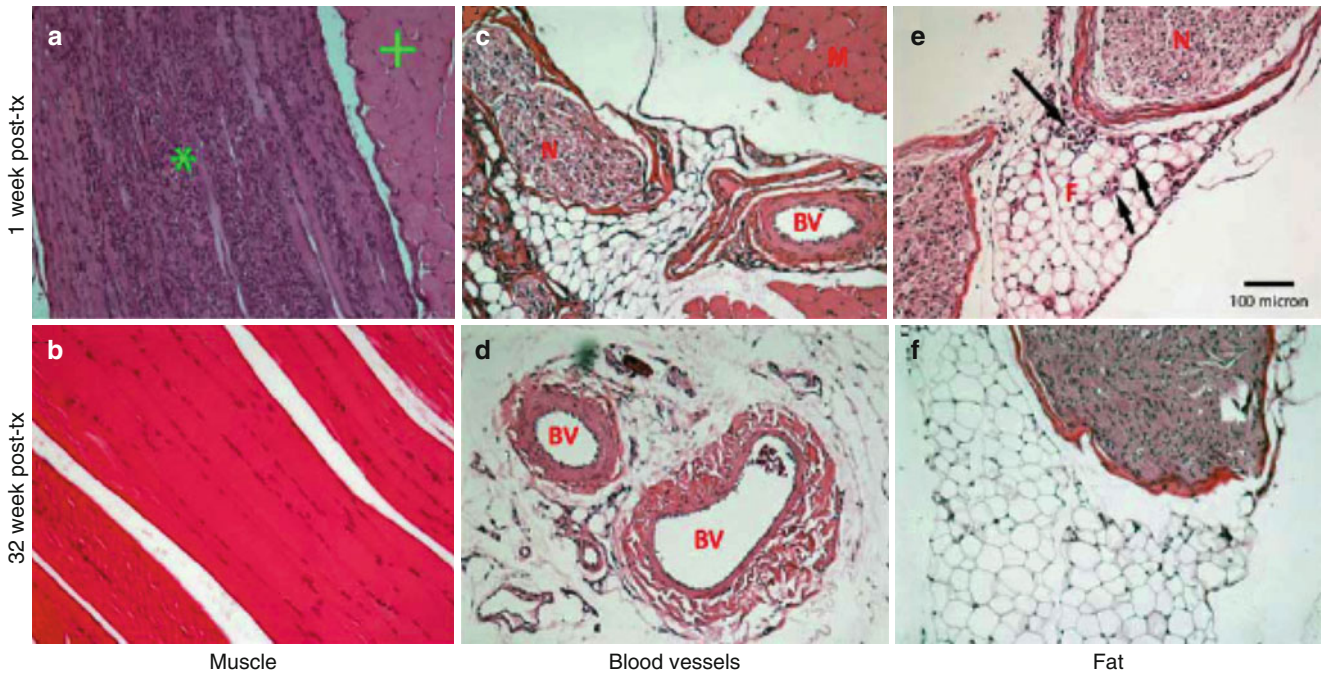

Fig. 8.10 Tissue histology 1 week and 32 weeks after cryoneurolysis. (**a**, **b**) show muscle tissue; note the cellular infiltration in (**a**) marked by a *, while undamaged muscle in (**a**) is marked by a **+,** with (**b**) showing complete resolution. (**c**) shows the blood vessel (*BV*) next to the treated nerve (*N*) and nearby muscle (*M*), while (**d**) shows no damage 32 weeks later. (**e**, **f**) show fat tissue; (**e**) shows necrotic fat (*arrows*) next to treated nerve (*N*), resolved in (**f**) (Image courtesy of Myoscience, from Hsu and Stevenson [66])

probe as close to the nerve as possible is key to the success of this technique, so the use of sensory stimulation, as well as ultrasound or fluoroscopy, can aid in the accurate placement. Because the ice ball itself can insulate the nerve from the cryoprobe, it is recommended to perform several freeze-defrost-freeze-defrost cycles. This creates the largest ice ball to most effectively incorporate the nerve within the ice ball. Patients usually describe a burning pain in the beginning seconds of the first freezing cycle, which usually resolves within 30 s.

The cryoprobe can also be placed under direct vision, such as during intrathoracic surgery (Fig. 8.12), or during nerve release or neuroma resection. Davies [56] reported on the excellent response after long-term follow-up of six patients treated with cryoneurolysis of upper extremity nerves under surgical exposure.

Indications for Neurolysis

Peripheral neurolysis, both thermal and chemical, when performed expertly, provides effective and enduring analgesia. However, chemoneurolysis has unique risks that are not associated with most other interventional procedures. Precise knowledge of anatomy and sequelae management is imperative to the effective use of neurolysis. Even with adherence to strict precautions, nerve ablation carries significant risk and should be used as a late, if not last, resort. Potential candidates for treatment include patients who suffer severe, focal, chronic somatic pain that is refractory to more conservative treatments (such as osteopathic or chiropractic manipulation, physical therapy, NSAIDs, orthotics, TENS, steroid injection, and low-dose opioids).

A confirmatory, diagnostic local anesthetic injection is required for pain mapping. Concordant analgesia after short-term local anesthetic blockade is required for, but not fully predictive of, both proper target and beneficial outcome from neurolysis. Detailed consideration of all possible structural anatomic deficits and their treatments should guide any intervention decision but particularly those that destroy tissue as a means to an end. Precise anatomic localization on exam and a precise, small-volume anesthetic block are imperative; imaging for needle guidance is highly recommended.

Proper pain diagnosis is key, as there are multiple causes of pain (muscles, ligaments, disks, bursas, and nerves), but neurolysis is used only for nerve-related pain. Thus, the foremost consideration in determining the appropriate neurolysis candidate is not a technical plan for "how to do the procedure" but a thoughtful, differential diagnosis that not only considers "what is the pathology?" but also "why is there pathology?" In particular, answering the latter question will remarkably improve clinical outcomes in all patients, regardless of their diagnosis, and symptom relief will follow. A baseline psychological evaluation should be considered

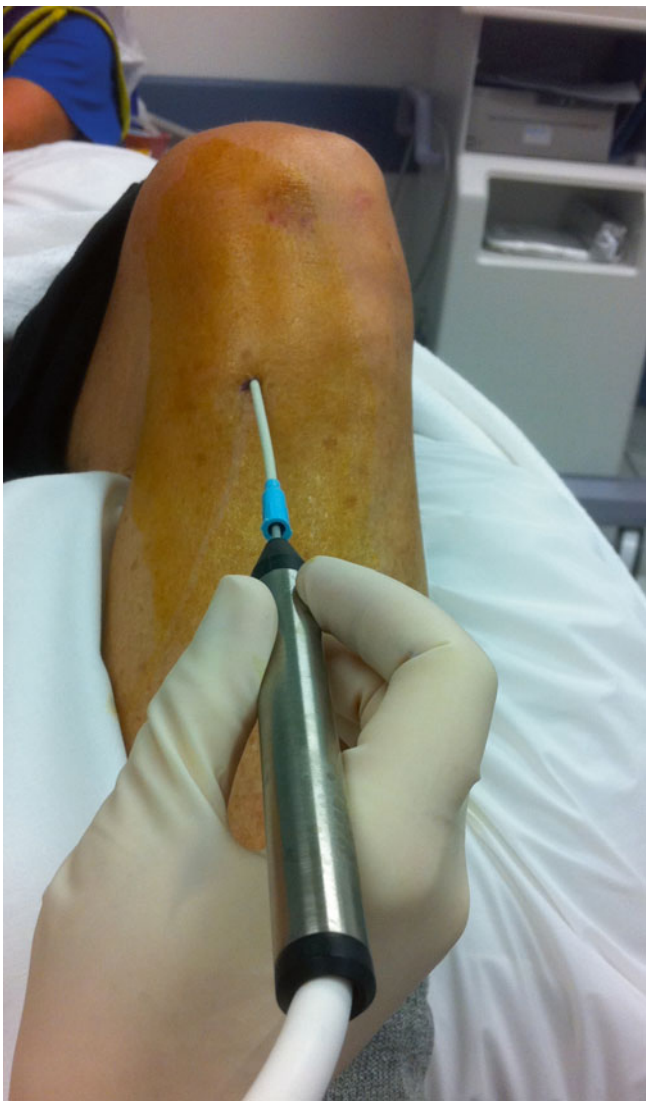

Fig. 8.11 Cryoneuroablation of the infrapatellar saphenous nerve using landmarks and peripheral nerve stimulation (Image courtesy of Andrea Trescot, MD)

for all pain patients before evaluating any intervention, and this is especially true when considering neurolysis.

Once chronic refractory neuralgia is identified, the decision to surgically decompress, or ablate via open neurotomy, or perform neurolysis (cryosurgery, radiofrequency ablation, or chemoneurolysis) must be made. Each method has its merits and shortfalls, and the choice must consider these in concert with the patient and family wishes after their full understanding of the risks. Additionally, available equipment, negative sequelae recovery, and, with ever-tightening insurance coverage, the patient's financial stability must all be considered. If prior opioid therapy was initiated, tapering is indicated following successful neurolysis so as to prevent overdose or withdrawal sequelae.

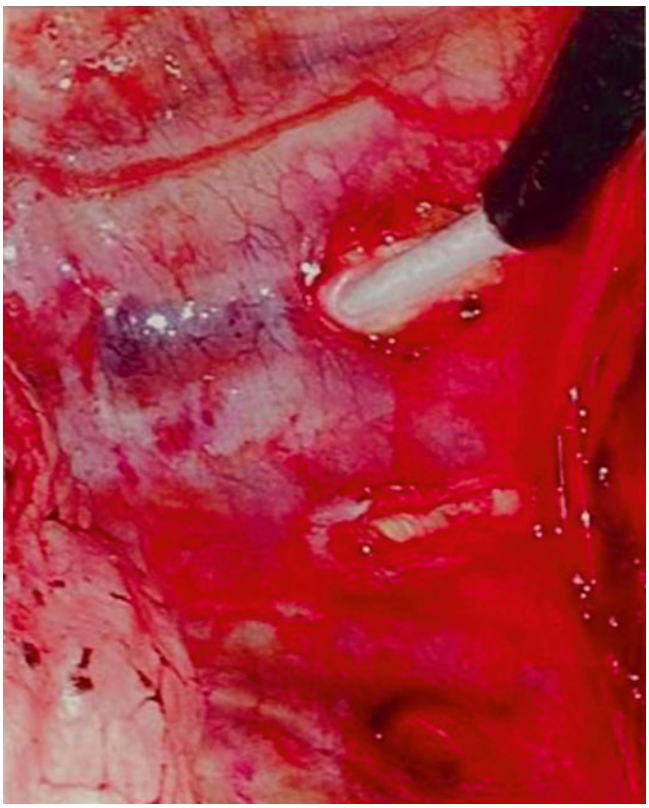

Fig. 8.12 Cryoprobe placed on the intercostal nerve during open thoracotomy (Image courtesy of Vasant Jayasankar, MD)

Chemoneurolysis is often preferable in the following scenarios:

- The nerve is superficial, and the risk of tissue damage is higher with cryoneurolysis or radiofrequency probes. Serial low-dose neurolytic injections are warranted in such cases as Morton's neuroma [57].
- The target nerve is motor-sensory mixed, and chemical concentrations for sensory-fiber selectivity may be utilized. (Pulsed RFA or cryoneuroablation may also be an option.)
- There is no focal "named-nerve" entrapment responsible for the patient's pain and thus no easily identified lesion to surgically decompress (e.g., postsurgical cicatrix, hardware pain, or neuroma).
- Nerve arborization has occurred secondary to previous neurolytic treatments by other means (such as radiofrequency ablation). However, while chemoneurolysis works well in such scenarios, the most common sites of RFA are the dorsal primary rami medial branches, and the risk of adjacent neurovascular damage from chemoneurolytic spread is high, particularly in the cervicothoracic regions. Cryoneuroablation, with its large ice ball, may be an option in this circumstance. Newer "cooled" RFA probes and machines are the latest attempt to accommodate the arborization issue and, though costly, may be preferable.

- In cases where a discrete *regional* neuralgia presides but is *not* attributable to an isolated nerve amenable to radiofrequency or cryoprobe localization. This scenario is similar to arborization, although the pain in question is due not to entrapment but to small fiber nociception from chronic connective tissue tensile failure following repetitive strain or trauma. In this case, regenerative options such as platelet-rich plasma (PRP) should be considered as a primary intervention to restore tissue integrity. Unfortunately, limited insurance coverage for PRP may deter the patient, and chemoneurolysis is an excellent alternative choice with site-specific concentrations. It is tragic that, despite its near 100-year history of use, insurance companies often deny chemoneurolysis as "experimental" as a cost avoidance measure.
- In cases of spasticity where botulinum toxin doses would be too high or would be cost prohibitive to the patient
- When no other superior treatment options exist and the benefits outweigh the risks

Radiofrequency lesioning is indicated for the denervation of small or unmyelinated nerves of the facets, dorsal root ganglion (DRG), periosteum, joints, disks, and sympathetic system. The trigeminal nerve has also been the target of radiofrequency lesioning, using a technique of successive lesions to decrease the risk of neuroma formation or complete denervation. It is critical that small-volume diagnostic injections be used to confirm the site of pathology prior to any denervation procedure. Heat radiofrequency lesioning is contraindicated for large, myelinated nerves, since the coagulation of proteins can lead to *anesthesia dolorosa* and neuroma formation. Some have questioned the utility of thermal lesioning in the presence of neuropathic pain [58] because of the possible neuroma formation as a result of nerve damage/Wallerian degeneration and subsequent nerve regeneration [33, 59]. Although it has been used for enthesopathies (such as plantar fasciitis or lateral epicondylitis), bursitis, and myofascial trigger points, facet denervation with radiofrequency has been used most commonly to treat spondylosis in the cervical, thoracic, and lumbar regions. Due to the potential unintended spread to the spinal nerve roots and epidural/intrathecal space, chemoneurolysis is rarely used for this indication.

Cryoneuroablation, on the other hand, is primarily indicated for the treatment of neuralgia of large, myelinated nerves, though it has been used for plantar fasciitis and symphysis pubis pain [60] as well as facet denervation [61, 62].

Risks of Chemoneurolysis

The specific properties of alcohol and phenol are described in their respective sections, but, as a rule, the fluid state of chemoneurolytic agents is a double-edged sword. Despite policy statements by insurance companies that chemical ablation is outdated and RFA is preferred, the distribution afforded by a liquid solution through a 25-gauge needle can prove valuable in accessing otherwise difficult-to-target tissue, where more rigid cryoprobes or RF cannulae cannot easily reach without repeated tip re-localization, associated tissue trauma, and time expenditure, often with a lesser result. That said, inadvertent neurolytic flow to surrounding unintended tissue may cause necrosis and potential catastrophic outcomes if the liquid reaches CNS structures, major mixed nerve roots, or critical vasculature, such as the artery of Adamkiewicz.

A case of peripheral injection causing catastrophic CNS outcome was reported in 2002 following phenol intercostal neurolysis for chronic nonmalignant pain [63]. It was postulated that the tracking of the 7.5 % concentrated phenol along the intercostal nerve sheath reached the associated neuroforamen and diffused to the central spinal canal, effectively ablating portions of the spinal cord and cauda equina inferiorly, resulting in permanent neurogenic bowel and bladder and paraplegia. In these authors' opinion, the 6 ml volume used seems excessive for a single nerve. Slowly injected volumes of 0.75–1.5 ml will usually suffice for most small- to medium-sized peripheral nerves. Of note, but not mentioned in the report, was the lack of fluoroscopic imaging and appropriate dye study, as the technique was performed blindly with only landmark identification. The study authors reflect that all aspirations for heme were negative; upon this, they based their deduction that the tracking occurred along the nerve sheath. While that may be the case, seemingly negative heme aspiration can deceive. For this reason, even with image guidance, these authors do not use concentrated chemoneurolysis for intercostal ablations, preferring cryoneuroablation for both its efficacy [64] and its safety measure in avoiding potential central neurologic spread.

Risks of Radiofrequency Ablation

Although a more controlled and therefore potentially safer method of neurolysis than alcohol or phenol, the technique of RFA demands a careful patient selection and meticulous technique. The most common complications of radiofrequency include those related to the placement of the needle and those related to the neurolysis. The majority of these problems are short-lived and self-limited, and they include local swelling, pain at the site of the needle insertion, and neuritis. Other general complications may include, based on the location of the needle placement, pneumothorax, dural puncture, spinal cord trauma, subdural injection, neural trauma, placement of the needle into the intervertebral foramen, and hematoma formation; infectious complications including abscess and bacterial meningitis and side effects

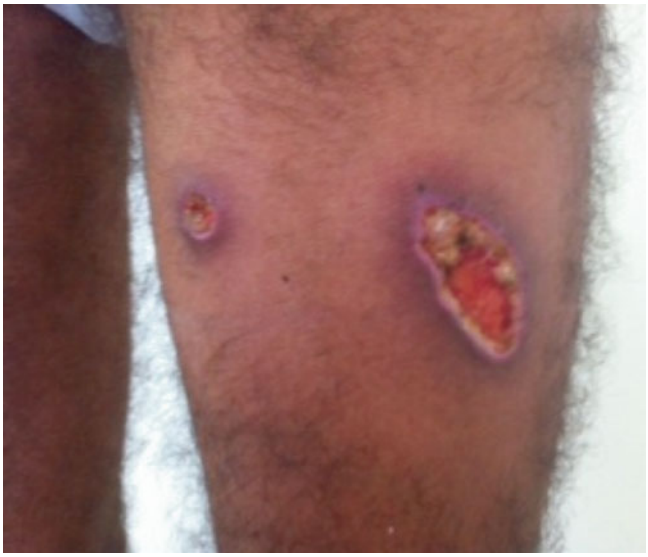

Fig. 8.13 Third-degree burn after sacroiliac radiofrequency denervation (Image courtesy of Charles Amaral de Oliveira, MD)

related to administration of local anesthetic and/or steroids and other drugs can also occur. Minor complications include light-headedness, flushing, sweating, nausea, hypotension, syncope, and non-postural headaches.

Other reported complications of radiofrequency thermoneurolysis include a worsening of the usual pain, burning or dysesthesias, decreased sensation and allodynia in the paravertebral skin of the facets denervated, transient pain, and inadvertent lesioning of the spinal nerve or ventral ramus resulting in motor deficits, sensory loss, and possible deafferentation pain. Third-degree burns can also occur at the dispersive electrode, especially if care has not been used in confirming even placement on the skin or avoiding placement on hairy areas (Fig. 8.13). There is also at least the theoretic risk that the RF electrical current can trigger a defibrillation charge from an implantable defibrillator, if the defibrillator mistakes the RF current for ventricular fibrillation.

Risks of Cryoneuroablation

Because many of the targets of cryoneuroablation are superficial peripheral nerves, there is a potential for "frostbite" if the ice ball formed too close to the surface of the skin. Care must be taken during the freezing process to inspect the site for the development of a white, waxy appearance to the skin, similar to that seen on chicken that has suffered "freezer burn." This can usually be managed by warming the skin with wet gauze or saline dripped onto the site, or with the physician's fingers, but it may necessitate stopping the procedure and repositioning the probe. Because the electrical current is used only for finding the nerve (and not for creating the lesion), there is less theoretic risk of unintended defibrillation discharge.

Although not technically a complication, incomplete or inadequate freezing of the nerve will result in very temporary analgesia, on the order of 1–10 days. This is usually corrected by a repeat lesioning session, taking care to use meticulous technique to place the probe as close as possible to the nerve. And, because of the size of the probe, there is a risk of damage to the viscera (such as pneumothorax) while placing the probe.

Summary

Once the diagnosis of nerve entrapment has been made and initial injection techniques have given good, but only temporary, relief, thought must turn to the next step. In selective cases, alcohol, phenol, cryoneurolysis, or radiofrequency lesioning may be appropriate. Other options such as peripheral stimulation (Chap. 9) and surgery (Chap. 10) are discussed elsewhere.

References

1. Racz GB. Techniques of neurolysis. Boston: Kluwer Academic Publishers; 1989.
2. Schloesser H. Heilung periphärer Reizzustände sensibler und motorischer Nerven. Klin Monatsbl Augenheilkd. 1903;41:244.
3. Dogliotti AM. Traitment des syndromes douloureux de la peripherie par l'alcoolisation sus-arachnoidienne des racines posterieures à leur emergence de la moelle epinière. Press Med (Paris, France: 1983). 1931;39:1249–54.
4. Swetlow GI. Paravertebral alcohol block in cardiac pain. Am Heart J. 1926;1:393.
5. Umeda S, Arai T. Disulfiram-like reaction to moxalactam after celiac plexus alcohol block. Anesth Analg. 1985;64(3):377.
6. Rumsby MG, Finean JB. The action of organic solvents on the myelin sheath of peripheral nerve tissue. I. Methanol, ethanol, chloroform and chloroform-methanol (2:1, v-v). J Neurochem. 1966;13(12):1501–7.
7. Woolsey RM, Taylor JJ, Nagel JH. Acute effects of topical ethyl alcohol on the sciatic nerve of the mouse. Arch Phys Med Rehabil. 1972;53(9):410–4.
8. Dockery GL. The treatment of intermetatarsal neuromas with 4% alcohol sclerosing injections. J Foot Ankle Surg. 1999;38(6):403–8.
9. Maigne JY, Doursounian L. Entrapment neuropathy of the medial superior cluneal nerve. Nineteen cases surgically treated, with a minimum of 2 years' follow-up. Spine (Phila Pa 1976). 1997;22(10):1156–9.
10. Lyftogt J. Prolotherapy for recalcitrant lumbago. Australas Musculoskelet Med. 2008;13(1):18–20.
11. Yang FR, Wu BS, Lai GH, Wang Q, Yang LQ, He MW, et al. Assessment of consecutive neurolytic celiac plexus block (NCPB) technique outcomes in the management of refractory visceral cancer pain. Pain Med. 2012;13(4):518–21.
12. Putnam TJ, Hampton AO. The technic of injection into the Gasserian ganglion under roentgenographic control. Arch Neurol Psychiatry. 1936;35:92–8.
13. Ongley MJ, Klein RG, Dorman TA, Eek BC, Hubert LJ. A new approach to the treatment of chronic low back pain. Lancet. 1987;2:143–6.

14. Klein RG, Eek BC, DeLong WB, Mooney V. A randomized double-blind trial of dextrose-glycerine-phenol injections for chronic, low back pain. J Spinal Disord. 1993;6(1):23–33.
15. Lyness WH. Pharmacology of neurolytic agents. In: Racz GB, editor. Technique of neurolytisis. Boston: Kluwer Academic Publishers; 1989. p. 13–25.
16. Nathan PW, Sears TA, Smith MC. Effects of phenol solutions on the nerve roots of the cat: an electrophysiological and histological study. J Neurol Sci. 1965;2(1):7–29.
17. Felsenthal G. Pharmacology of phenol in peripheral nerve blocks: a review. Arch Phys Med Rehabil. 1974;55(1):13–6.
18. Zamponi GW, French RJ. Arrhythmias during phenol therapies: a specific action on cardiac sodium channels? Circulation. 1994;89(2):914.
19. Christiansen RG, Klaman JS. Successful treatment of phenol poisoning with charcoal hemoperfusion. Vet Hum Toxicol. 1996;38(1):27–8.
20. Nomoto Y, Fujita T, Kitani Y. Serum and urine levels of phenol following phenol blocks. Can J Anaesth. 1987;34(3 (Pt 1)):307–10.
21. Toxicology profile for phenol. Atlanta, GA: U.S. Department of Health and Human Services,; 2008. Available from: http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=148&tid=27.
22. Piotrowski JK. Evaluation of exposure to phenol: absorption of phenol vapour in the lungs and through the skin and excretion of phenol in urine. Br J Ind Med. 1971;28(2):172–8.
23. Ling WH, Hanninen O. Shifting from a conventional diet to an uncooked vegan diet reversibly alters fecal hydrolytic activities in humans. J Nutr. 1992;122(4):924–30.
24. Kowalewski R, Schurch B, Hodler J, Borgeat A. Persistent paraplegia after an aqueous 7.5% phenol solution to the anterior motor root for intercostal neurolysis: A case report. Arch PMR. 2002;83(2):283–5.
25. Lema MJ, Myers DP, De Leon-Casasola O, Penetrante R. Pleural phenol therapy for the treatment of chronic esophageal cancer pain. Reg Anesth. 1992;17(3):166–70.
26. Breasted J. The Edwin Smith surgical papyrus. Ancient records of Egypt. III. Chicago: University of Chicago Oriental Institute Publications; 1930. p. 54.
27. Hippocrates. On the articulations. The genuine works of Hippocrates. Clin Orthop Relat Res. 2002;400:19–25.
28. Cushing H, Bovie WT. Electro-surgery as an aid to the removal of intracranial tumors. With a preliminary note on a new surgical-current generator by W. T. Bovie. Surg Gynecol Obstet. 1928;47:751–85.
29. Kirschner M. Zur Elektrochirurgie. Arch Klin Chir. 1931;167:761.
30. Sweet WH, Mark VH. Unipolar anodal electrolyte lesions in the brain of man and rat: report of five human cases with electrically produced bulbar or mesencephalic tractotomies. AMA Arch Neurol Psychiatry. 1953;70:224–34.
31. Aranow S. The use of radiofrequency power in making lesions in the brain. J Neurosurg. 1960;17:431–8.
32. Cosman BJ, Cosman ER. Guide to radio frequency lesion generation in neurosurgery. Burlington: Radionics; 1974.
33. Racz GB, Ruiz-Lopez R. Radiofrequency procedures. Pain Pract. 2006;6(1):46–50.
34. Sluijter ME, Cosman ER, Rittman WB, Kleef MV. The effects of pulsed radiofrequency fields applied to the dorsal root ganglion – a preliminary report. Pain Clin J. 1998;11(2):109–18.
35. Restrepo-Garces CE, Marinov A, McHardy P, Faclier G, Avila A. Pulsed radiofrequency under ultrasound guidance for persistent stump-neuroma pain. Pain Pract. 2011;11(1):98–102.
36. Choi HJ, Choi SK, Kim TS, Lim YJ. Pulsed radiofrequency neuromodulation treatment on the lateral femoral cutaneous nerve for the treatment of meralgia paresthetica. J Korean Neurosurg Soc. 2011;50(2):151–3.
37. Akkaya T, Ozkan D. Ultrasound-guided pulsed radiofrequency treatment of the intercostal nerve: three cases. J Anesth. 2013;27(6):968–9.
38. Gofeld M, Restrepo-Garces CE, Theodore BR, Faclier G. Pulsed radiofrequency of suprascapular nerve for chronic shoulder pain: a randomized double-blind active placebo-controlled study. Pain Pract. 2013;13(2):96–103.
39. Park HG, Park PG, Kim WJ, Park YH, Kang H, Baek CW, et al. Ultrasound-assisted mental nerve block and pulsed radiofrequency treatment for intractable postherpetic neuralgia: three case studies. Korean J Pain. 2014;27(1):81–5.
40. Rhame EE, Levey KA, Gharibo CG. Successful treatment of refractory pudendal neuralgia with pulsed radiofrequency. Pain Physician. 2009;12(3):633–8.
41. Trendelenburg W. Über langdauernde nervenausschaltung mit sicherer regenerationsfähigkeit. Z Gesamte Exp Med. 1917;5:371–4.
42. Cooper IS, Lee AS. Cryostatic congelation: a system for producing a limited, controlled region of cooling or freezing of biologic tissues. J Nerv Ment Dis. 1961;133:259–63.
43. Amoils SP. The Joules Thompson cryoprobe. Arch Ophthalmol. 1967;78:201–7.
44. Lloyd JW, Barnard JDW, Glynn CJ. Cryoanalgesia, a new approach to pain relief. Lancet. 1976;2:932–4.
45. Mazur P. Freezing of living cells: mechanisms and implications. Am J Physiol Cell Physiol. 1984;247:C125–42.
46. Overbaugh RH, Compton CH. Ultrasound guided cryoablation for treatment of occipital neuralgia (abstract). Reg Anes Pain Med. 2010;35(5):E1.
47. Hsu M, Stevenson FF. Wallerian degeneration and recovery of motor nerves after multiple focused cold therapies. Muscle Nerve. 2015;51(2):268–75.
48. Wang JK. Cryoanalgesia for painful peripheral nerve lesions. Pain. 1985;22(2):191–4.
49. Nally FF. A 22-year study of paroxysmal trigeminal neuralgia in 211 patients with a 3-year appraisal of the role of cryotherapy. Oral Surg Oral Med Oral Pathol. 1984;58(1):17–23.
50. Green CR, de Rosayro AM, Tait AR. The role of cryoanalgesia for chronic thoracic pain: results of a long-term follow up. J Natl Med Assoc. 2002;94(8):716–20.
51. Fanelli RD, DiSiena MR, Lui FY, Gersin KS. Cryoanalgesic ablation for the treatment of chronic postherniorrhaphy neuropathic pain. Surg Endosc. 2003;17(2):196–200.
52. Campos NA, Chiles JH, Plunkett AR. Ultrasound-guided cryoablation of genitofemoral nerve for chronic inguinal pain. Pain Physician. 2009;12(6):997–1000.
53. Noback CR. Podiatric treatment of Morton's neuroma with cryoneurolytic technique. Jacksonv Med J. 2002;3(2):10–2.
54. Moesker AA, Karl HW, Trescot AM. Treatment of phantom limb pain by cryoneurolysis of the amputated nerve. Pain Pract. 2014;14(1):52–6.
55. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
56. Davies E, Pounder D, Mansour S, Jeffery IT. Cryosurgery for chronic injuries of the cutaneous nerve in the upper limb. Analysis of a new open technique. J Bone Joint Surg Br. 2000;82(3):413–5.
57. Hughes RJ, Ali K, Jones H, Kendall S, Connell DA. Treatment of Morton's neuroma with alcohol injection under sonographic guidance: follow-up of 101 cases. AJR Am J Roentgenol. 2007;188(6):1535–9.
58. Kline MT, Yin W. Radiofrequency techniques in clinical practice. In: Waldman SD, editor. Interventional pain management. Philadelphia: WB Saunders; 1996. p. 243–93.
59. Geurts JW, van Wijk RM, Stolker RJ, Groen GJ. Efficacy of radiofrequency procedures for the treatment of spinal pain: a systematic review of randomized clinical trials. Reg Anesth Pain Med. 2001;26(5):394–400.
60. Glynn CJ, Carrie LE. Cryoanalgesia to relieve pain in diastasis of the symphysis pubis during pregnancy. Br Med J (Clin Res Ed). 1985;290(6486):1946–7.

61. Birkenmaier C, Veihelmann A, Trouillier HH, Hausdorf J, von Schulze Pellengahr C. Medial branch blocks versus pericapsular blocks in selecting patients for percutaneous cryodenervation of lumbar facet joints. Reg Anesth Pain Med. 2007;32(1):27–33.
62. Wolter T, Deininger M, Hubbe U, Mohadjer M, Knoeller S. Cryoneurolysis for zygapophyseal joint pain: a retrospective analysis of 117 interventions. Acta Neurochir (Wien). 2011; 153(5):1011–9.
63. Wang GK, Vladimirov M, Shi H, Mok WM, Thalhammer JG, Anthony DC. Structure-activity relation of N-alkyl tetracaine derivatives as neurolytic agents for sciatic nerve lesions. Anesthesiology. 1998;88(2):417–28.
64. Byas-Smith MG, Gulati A. Ultrasound-guided intercostal nerve cryoablation. Anesth Analg. 2006;103(4):1033–5.
65. Trescot A, Hansen H. Radiofrequency neurolysis. In: Manchikanti L, Singh V, editors. Interventional techniques in chronic non-spinal pain. Paducah: ASIPP Publishing; 2009. p. 59–68.
66. Hsu M, Stevenson FF. Wallerian degeneration and recovery of motor nerves after multiple focused cold therapies. Muscle Nerve. 2015;51(2):268–75.

Neuromodulation for Treatment of Nerve Entrapment Syndromes

9

Amitabh Gulati, Helen W. Karl, Tiffany Zhang, and Andrea M. Trescot

Introduction

When considering surgical options for the treatment of nerve entrapment syndromes, two modalities are available: *surgical decompression* of the affected nerve and *neuromodulation* of the neural pathways. Chapter 10 focuses on the use of surgical decompression and nerve repair.

Neuromodulation techniques more directly involve therapeutic alteration of activity in the nervous system [1], anywhere along the signal's pathway, from peripheral nerves up to the central nervous system. Using electrical fields, these systems reversibly alter the signal pathway so that noxious stimuli are minimized or inhibited [1]. One of the advantages of neuromodulation is that the nerve is usually left intact, allowing for motor movements to remain, while sensory and pain transmissions are altered.

The advent of neurostimulation therapy for peripheral neuropathies began in 1967 when Wall and Sweet first described the use of neurostimulation therapy, spinal cord stimulation (SCS), and peripheral nerve stimulation (PNS) for neuropathic pain [2]. Since then, there have been significant advances in the development of devices, methods, and tools in this field. The applications of this therapy have expanded to include many neuropathic conditions. A recent comprehensive review by the International Association for the Study of Pain Neuropathic Pain Special Interest Group (ISP NeuPSIG) found few high-quality clinical trials of neuromodulation, but the group did recommend SCS for *failed back surgery syndrome* (FBSS) and *complex regional pain syndrome* (CRPS). They called for increased research in this area, particularly with randomized clinical trials and documentation in pain registries [3].

Current neuromodulation systems [4] include *central nervous system devices* (*deep brain stimulation* (DBS), *motor cortex stimulation* (MCS), *spinal cord stimulation* (SCS), and *intrathecal drug delivery* (ITDD)) and *peripheral nerve devices* (*spinal nerve root stimulation* (SNRS), *peripheral nerve stimulation* (PNS) [5], *peripheral nerve field stimulation* (PNFS) (5), and *transdermal electrical nerve stimulation* (TENS)) [1, 6]. The focus of this chapter will be on neuromodulation therapies pertinent to peripheral nerve entrapment syndromes.

Principal Indications

The current recommendation is that implantable neuromodulation therapy be applied for severe neuropathic pain refractory to more conservative and conventional treatment, including medication (Chap. 4), physical therapy (Chap. 5), neurolytics (Chap. 8), and surgery (Chap. 10). Factors that predict a successful outcome include the lack of overuse of medications (specifically opioids), evaluation and treatment of psychological disorders, and a good response to percutaneous stimulation [4]. Deciding when to implant and which device and frequency to use is an ongoing challenge, as algorithms for treating neuropathic pain syndromes continue to evolve.

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Transcutaneous Electrical Nerve Stimulation

Transcutaneous electrical nerve stimulation (TENS) is an external form of peripheral nerve stimulation that has been available for decades (Fig. 9.1) [6]. Application of TENS allows for treatment of pain syndromes with an external device that is noninvasive and flexible. Usually, TENS pads are applied on the skin close to the area of pain (but not areas with sensory changes), and patients are taught techniques to utilize the device during exacerbations of pain [7]. While data remains controversial, TENS as a goal-directed therapy has been shown to improve pain in patients with neuropathic pain syndromes such as sciatica and post-thoracotomy pain syndrome [7]. Recently, a specific transcutaneous nerve stimulation placed over the supraorbital nerve for headache treatment has become available; more than 2,500 patients in Europe tried the device, and >50 % found the device to be useful in managing their headaches [8].

Spinal Cord Stimulation

Spinal cord stimulation (SCS) usually involves placement of leads in the thoracic (Fig. 9.2) or cervical epidural space (Fig. 9.3) along the posterior aspect of the spinal cord (dorsal column), using electrical impulses to interrupt pain signaling at the spinal cord level [1]. The leads can be cylindrical or a flat paddle (Fig. 9.4), and they can be placed through a percutaneous needle or through a surgical laminotomy. The percutaneous leads are usually cylindrical so they can pass through a needle, but, because of their shape, a significant amount of the current is directed away from the spinal cord. The paddle leads can direct all of the current to the cord, and they usually require a surgical placement, though there is a paddle lead that can be placed through a needle. The leads can have electrodes that are close together or wide apart;

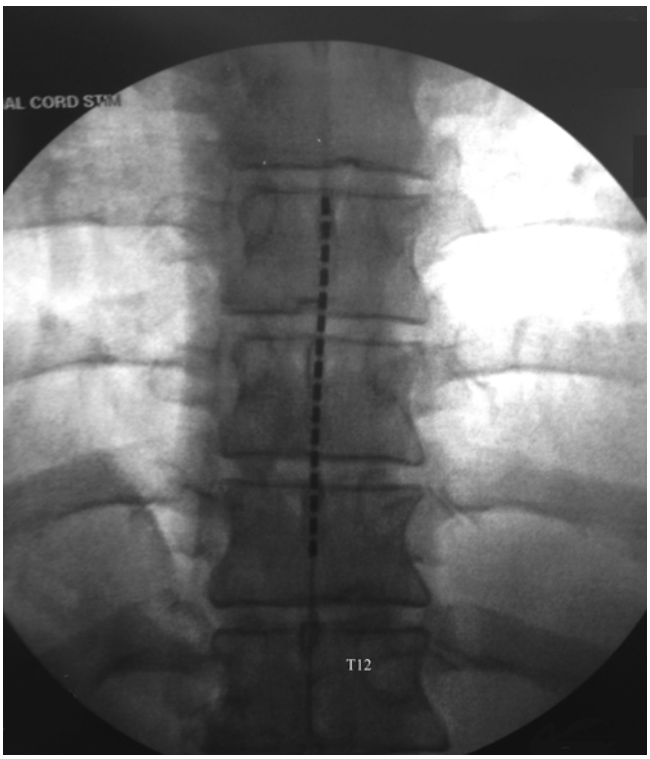

Fig. 9.2 Spinal cord stimulator placement into the thoracic epidural space (Image courtesy of Andrea Trescot, MD)

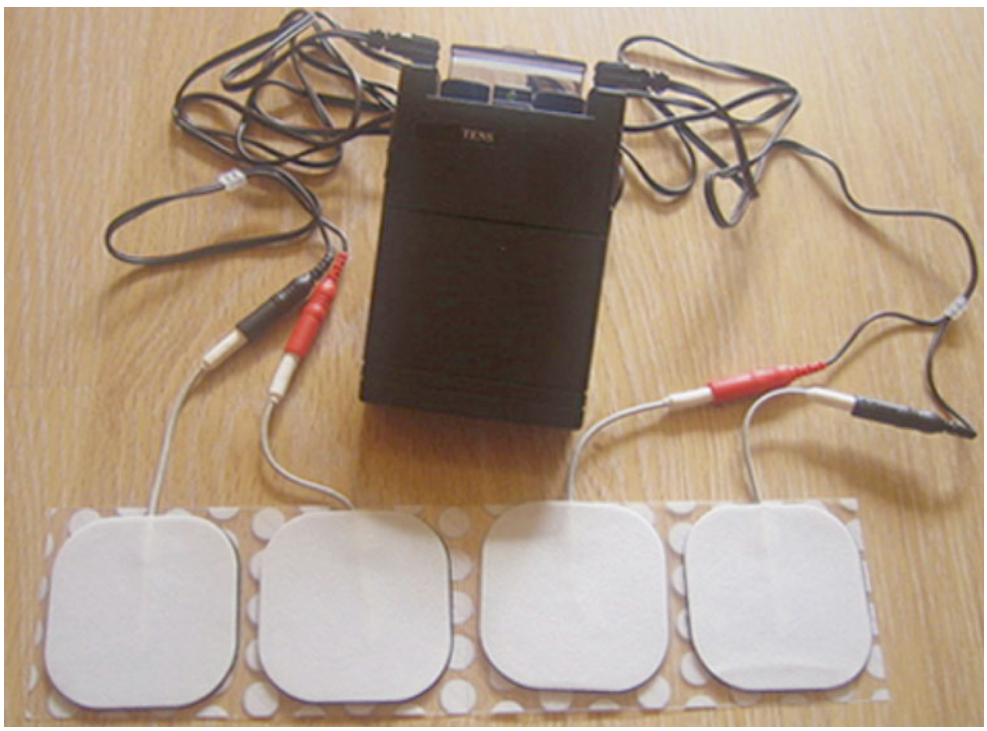

Fig. 9.1 Transcutaneous electrical nerve stimulator (TENS) unit

there can be 4, 8, or 16 electrodes on each lead, and 1, 2, 3, or even 4 leads can be placed into the epidural space.

These leads are typically placed caudad to cephalad ("antegrade") (Fig. 9.5), and there can be one or multiple leads placed (Fig. 9.6). SCS is usually indicated for, but not limited to, cervical and lumbar radiculopathies [9, 10], failed back surgery syndrome (FBSS) [9], peripheral neuropathy (including nerve entrapment) [11, 12], and complex regional pain syndrome (CRPS) [9, 13]. Other indications include unstable angina [9], abdominal pain [14], pelvic pain [11], and peripheral vascular disease [9, 15]. The type, position, and spinal level of the lead placement often determine the pain coverage area and pattern. For example, cervical level is used for upper extremity coverage, while mid to lower thoracic levels are used for low back and lower extremity coverage. The coverage of stimulation is usually over a large area – an entire arm or leg or from the waist down. However, the unneeded areas of stimulation, such as into the chest or abdomen, can be quite uncomfortable for the patient. Patients often need reprogramming of the stimulation during the first few months after the implant to optimize the coverage of the painful area.

In general, selected patients are first subjected to a short trial of a temporarily placed device, usually for several days and up to a week, in an outpatient setting. The percutaneous leads are inserted to the targeted area (epidural space or along the peripheral nerves) and secured to the skin (Fig. 9.7); the proximal end is connected to an external stimulus generator (a battery-operated programmer). The patients are then discharged with the temporarily placed device. Upon return at the end of trial phase, if the patient reports subjective successful pain relief (≥50 % pain relief), the patient may choose to have surgery for implant of permanent leads and a power source, either an implantable pulse generator (IPG) (which can be recharged in some systems) or a radiofrequency receiver (Fig. 9.8) [1]. Recently, a wireless power system has also become available (Fig. 9.9).

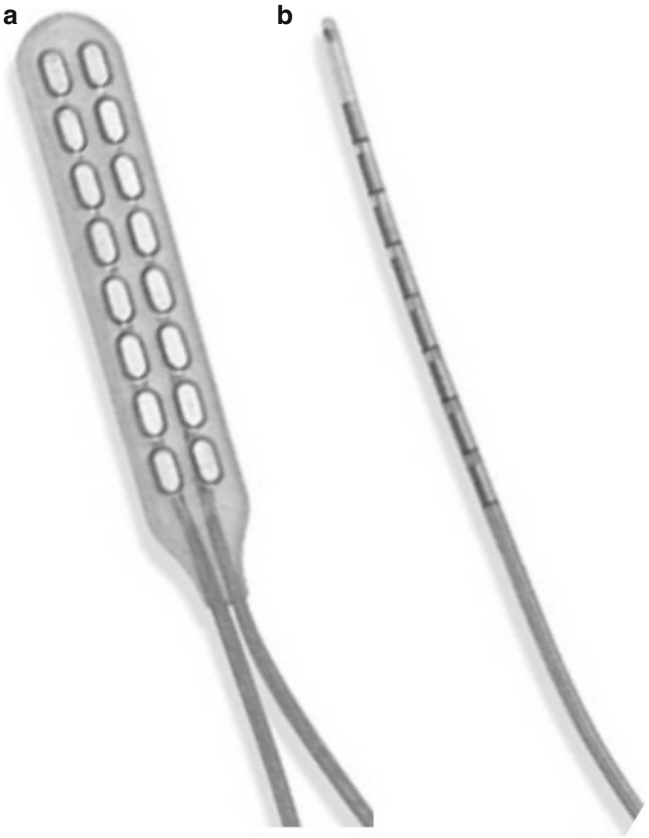

Fig. 9.4 Types of spinal cord stimulator leads. (**a**) Paddle-type lead; (**b**) cylindrical-type lead

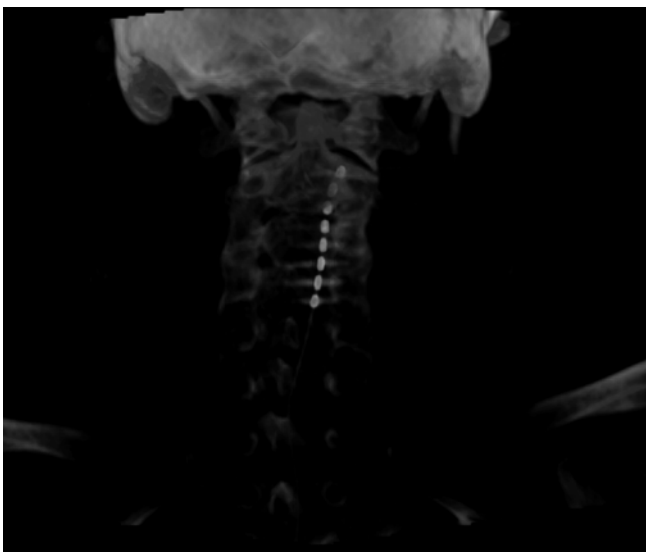

Fig. 9.3 3D imaging of a spinal cord stimulator lead in the cervical epidural space (Image courtesy of Andrea Trescot, MD)

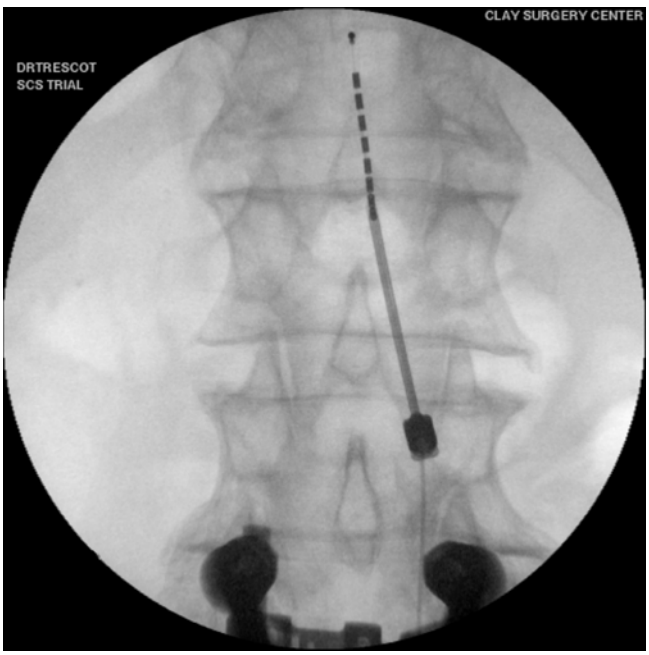

Fig. 9.5 Antegrade placement of a thoracic spinal cord stimulator (Image courtesy of Andrea Trescot, MD)

The intensity, rate, pulse width, and frequency can all be modified, allowing stimulation that can range from a "thumping" sensation to a frequency so high that it is unperceivable. There can be fixed stimulation parameters (the patient can

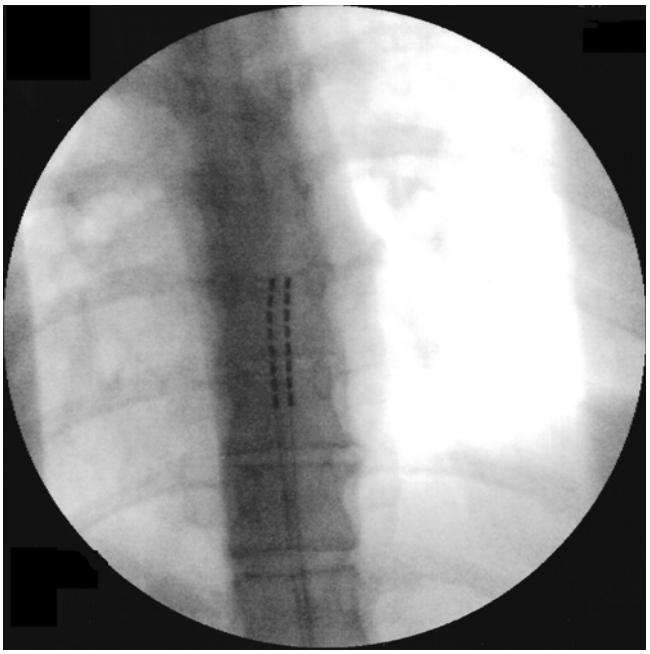

Fig. 9.6 Dual thoracic spinal cord stimulator leads (Image courtesy of Andrea Trescot, MD)

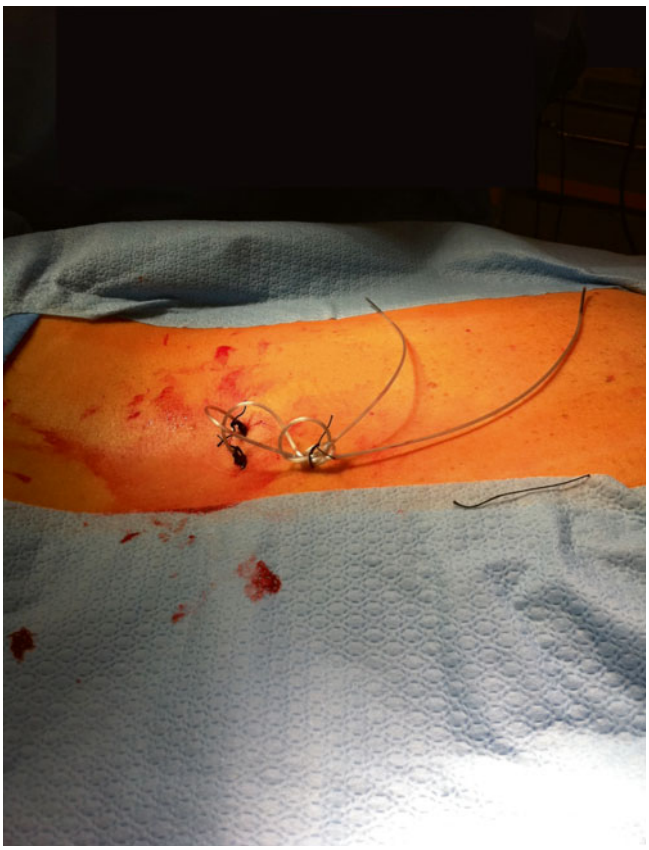

Fig. 9.7 Percutaneous neurostimulation lead trial with leads secured to the skin with suture (Image courtesy of Andrea Trescot, MD)

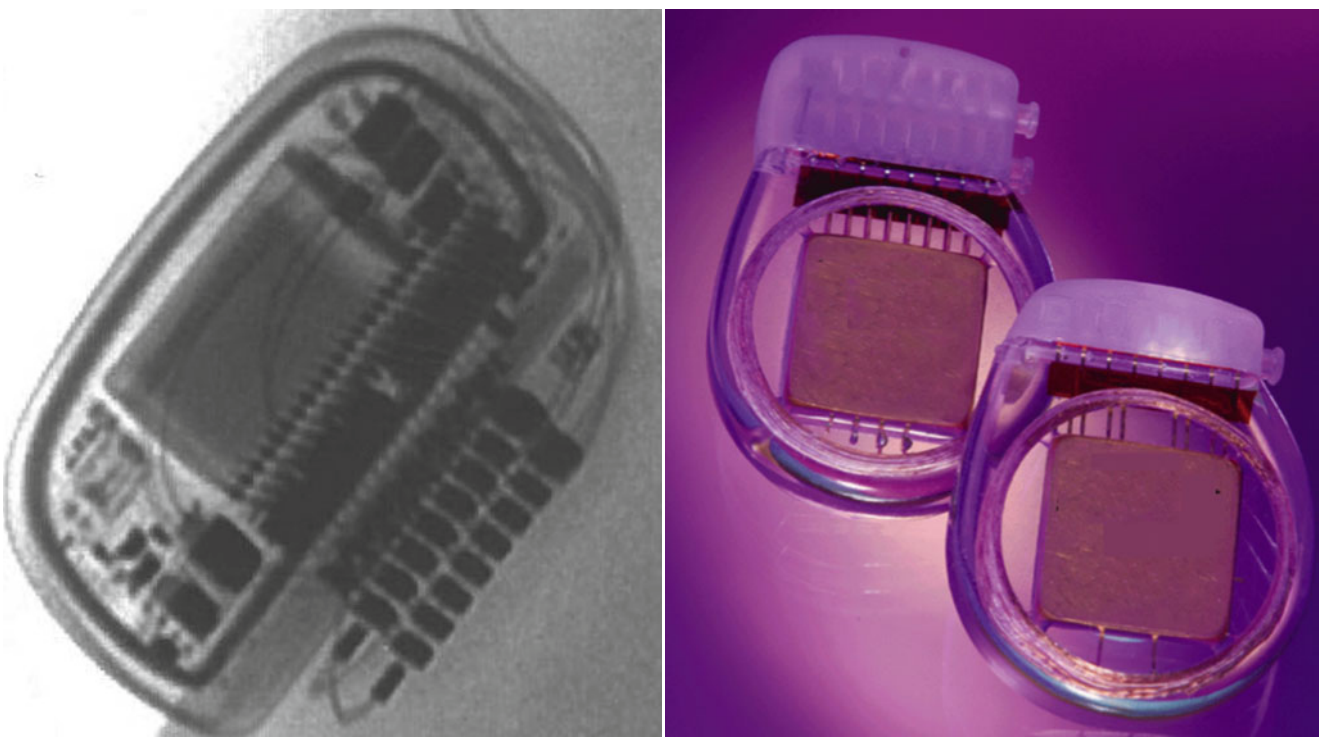

Fig. 9.8 Implanted power for stimulators. (**a**) Implanted power generator (IPG), (**b**) radiofrequency receiver (RF receiver) (Image courtesy of Andrea Trescot, MD)

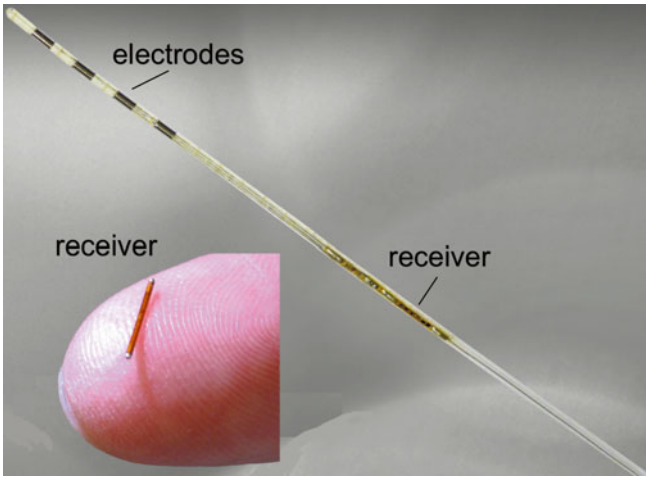

Fig. 9.9 Stimulator lead with incorporated receiver; insert shows the size of the receiver (Image courtesy of StimWave®)

turn the stimulation on/off or up/down) or patient-controlled parameters (changing the pattern of stimulation by changing program channels or shifting the distribution of current). Recently, accelerometers incorporated into the power source allow for automatic changes in power with changes in position. Deciding when to implant and which device to use is a future challenge for physicians, as algorithms develop for treating neuropathic pain syndromes.

Transforaminal Nerve Stimulation

Transforaminal nerve root stimulation (TFNS), also known as *spinal nerve root stimulation* (SNRS) [1, 16–18], is a newer technique in which stimulator electrodes are placed along the spinal nerve roots, targeting a specific radicular pain without the unwanted/unnecessary paresthesias beyond the desired distribution [18]. The electrodes are placed in an anterograde or retrograde fashion, either in the lateral aspect of the spinal canal overlying the desired dorsal rootlets (Fig. 9.10) or out the nerve root foramen from a retrograde epidural approach (Fig. 9.11), or in an retrograde fashion through the foramen (Fig. 9.12). This technique has been shown to be effective for treatment of post-herniorrhaphy and post-traumatic ilioinguinal neuropathies [19], postherpetic neuralgia [20, 21], foot pain [17], trigeminal neuralgia [21], and other peripheral neuropathies [17].

Peripheral Nerve Stimulation and Peripheral Nerve Field Stimulation

Peripheral nerve stimulation (PNS) is not new, having been described by Wall and Sweet in the 1960s [2], but it was "rediscovered" in 1999 when Weiner and Reed published on the percutaneous implantation of an electrode onto the occipital nerve [22]. PNS is indicated when pain is confined to a specific distribution of either a single nerve or a limited

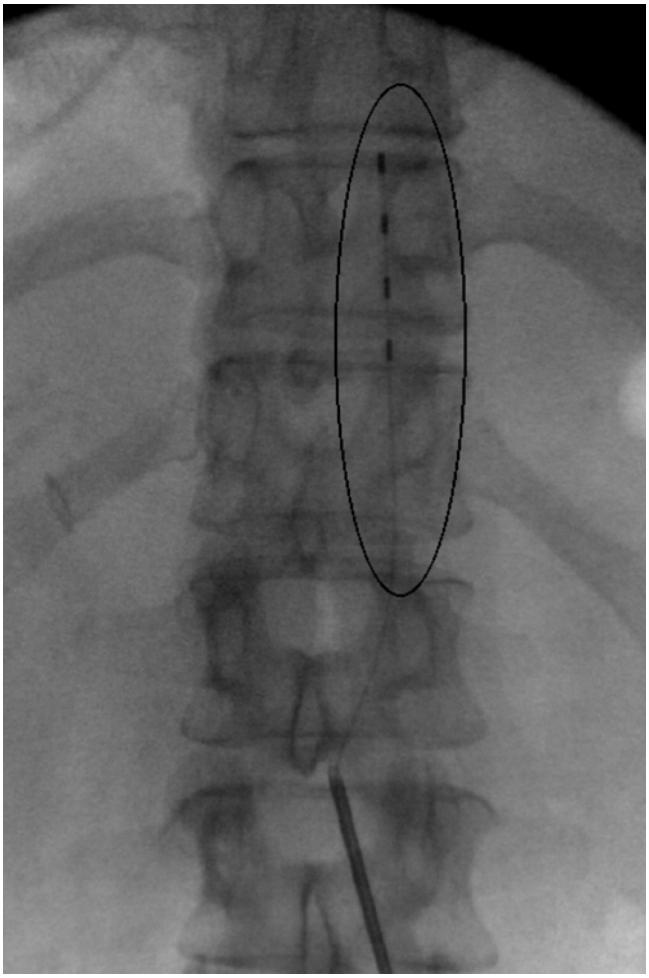

Fig. 9.10 Lateral placement of the spinal cord stimulator to stimulate the dorsal root ganglia of the genitofemoral and ilioinguinal nerves (Image courtesy of Andrea Trescot, MD)

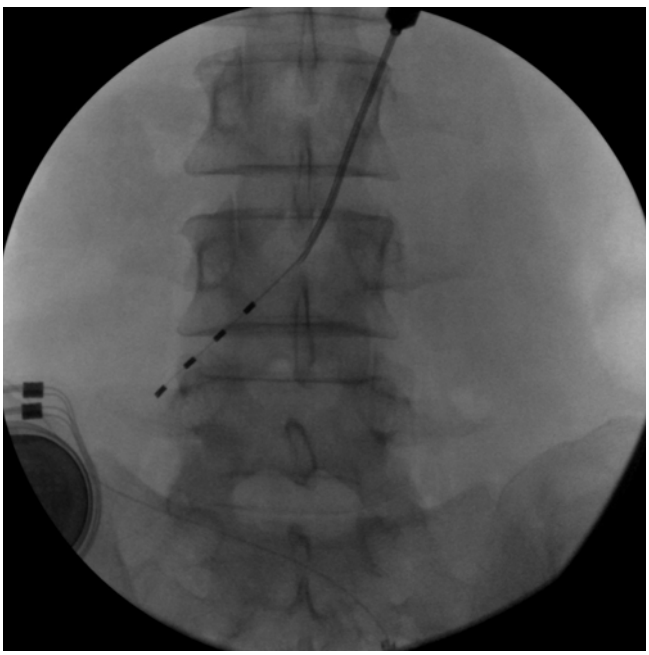

Fig. 9.11 Retrograde epidural spinal cord stimulator placed out the left L45 foramen (Image courtesy of Andrea Trescot, MD)

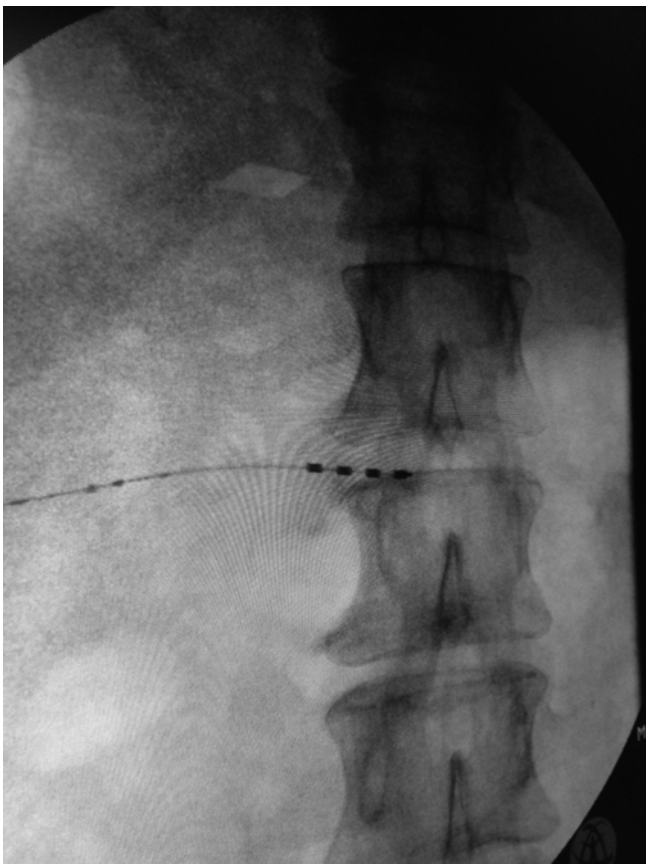

Fig. 9.12 Transforaminal retrograde nerve root stimulation (Image courtesy of StimWave®)

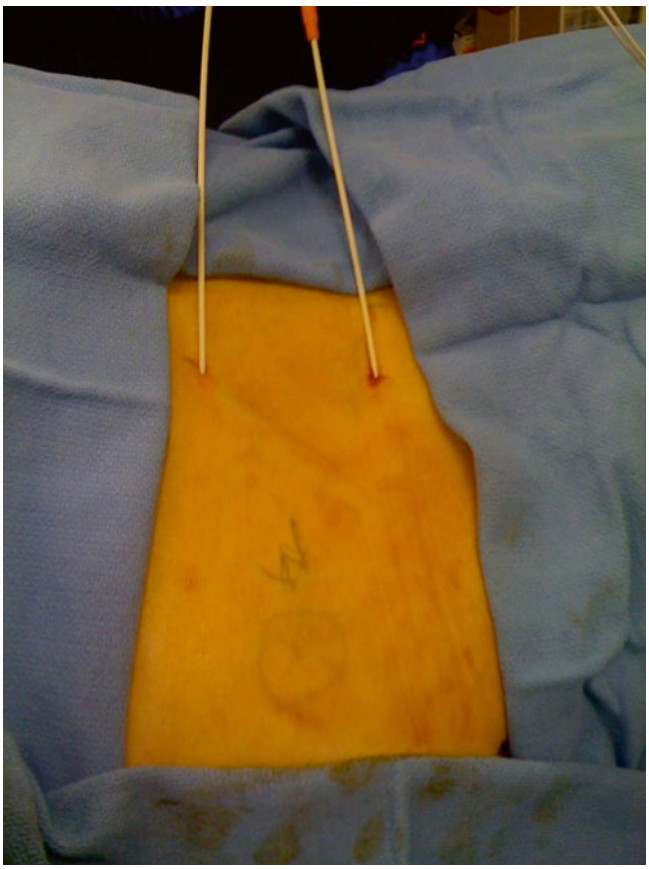

Fig. 9.13 Percutaneous placement of inguinal peripheral nerve stimulator leads (Image courtesy of Andrea Trescot, MD)

number of nerves. It involves implanting a percutaneous lead along the desired nerve, with the aim to produce paresthesia along the entire trajectory of the stimulated nerve (Figs. 9.13 and 9.14) [23]. Unfortunately, PNS is sometimes limited by difficult surgical access to the target nerves or by body locations that might promote lead migrations (such as across the elbow or hip joint).

On the other hand, *peripheral nerve field stimulation* (PNFS) uses leads placed in the area of the pain, independent of the nerve path (Figs. 9.15 and 9.16) [5]. Stimulation can occur between the leads ("cross-talk"), potentially stimulating across large areas [24]. Ultrasound has greatly facilitated the accurate percutaneous placement of leads, which also allows for simple percutaneous trials (Fig. 9.17) [25, 26]. There are many examples of PNS and PNFS for peripheral pain treatment.

Orofacial pain Trigeminal neuralgia is a facial pain that affects 150,000 patients per year in the United States and is characterized by sudden, severe lancinating pain along the path of the trigeminal nerve. Complex craniofacial neuralgias (trigeminal neuralgia, supraorbital, and infraorbital nerve entrapment, or mandibular neuralgias) have been treated with PNS. Slavin et al. [27] used trigeminal and occipital PNS to treat complex facial pain (Fig. 9.18).

Headache Chronic headaches (more than 180 headaches per year), which include migraines and cluster headaches, affect as much as 4 % of the adult population [28]. Occipital neuralgia is one of the neuralgias successfully treated with PNS [29, 30]. Schwedt et al. [31] described a retrospective study of 15 severe headache patients who underwent implantation of occipital stimulation; the mean percent decrease in pain was 52 %. Approaching the pain in a different manner, Shaparin et al. [32] described the use of PNS of the supraorbital and infraorbital nerves to treat chronic headaches.

Post-traumatic headaches Persistent headaches after head trauma affect 30–90 % of patients after even mild head trauma; they are also common after blast trauma (such as in

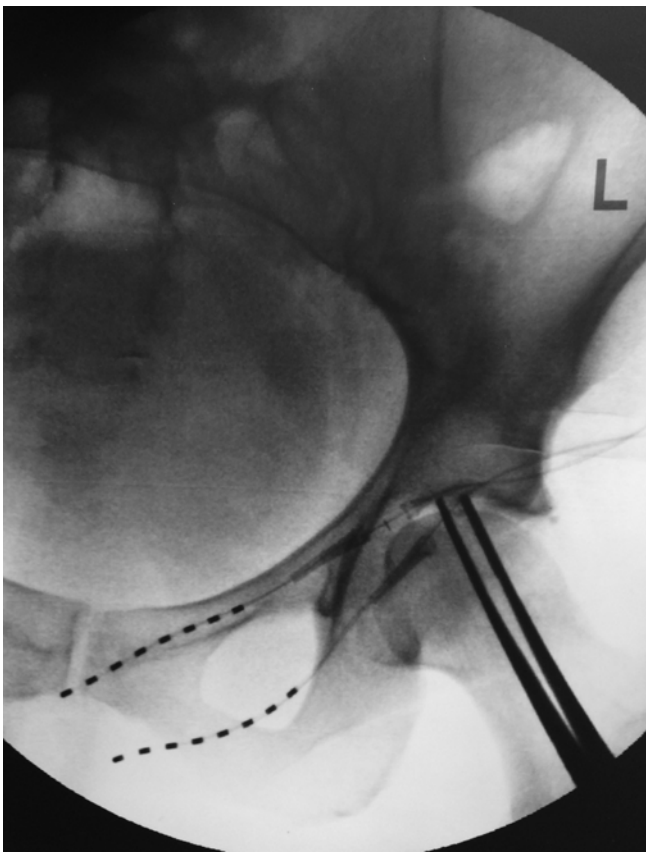

Fig. 9.14 Percutaneous placement of stimulator leads along the path of the ilioinguinal and iliohypogastric nerves (Image courtesy of Gladstone McDowell, MD)

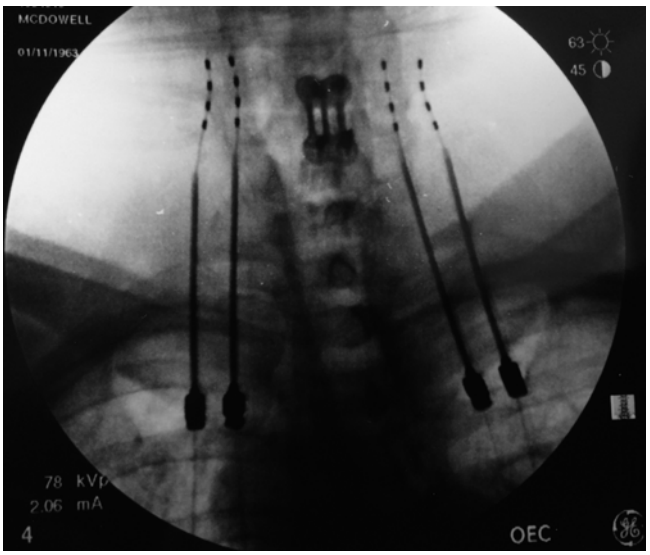

Fig. 9.15 Field stimulation of the cervical region (Image courtesy of Gladstone McDowell, MD)

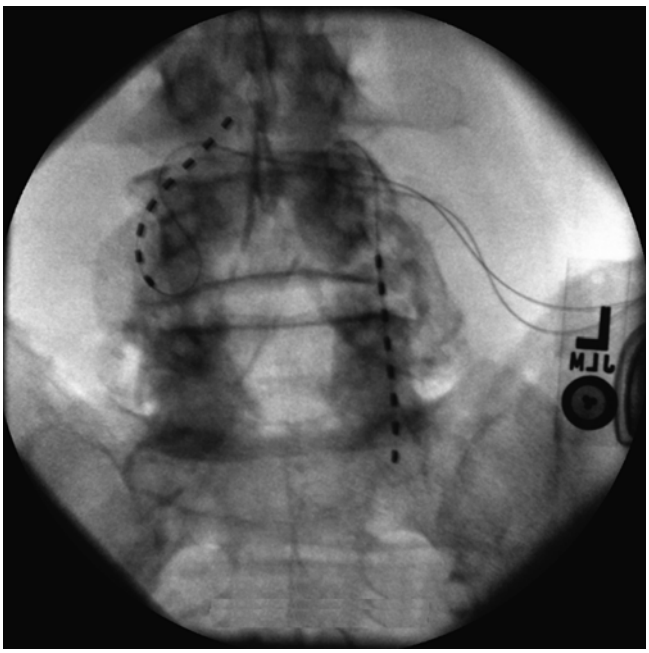

Fig. 9.16 Field stimulation of the lumbar region (Image courtesy of Andrea Trescot, MD)

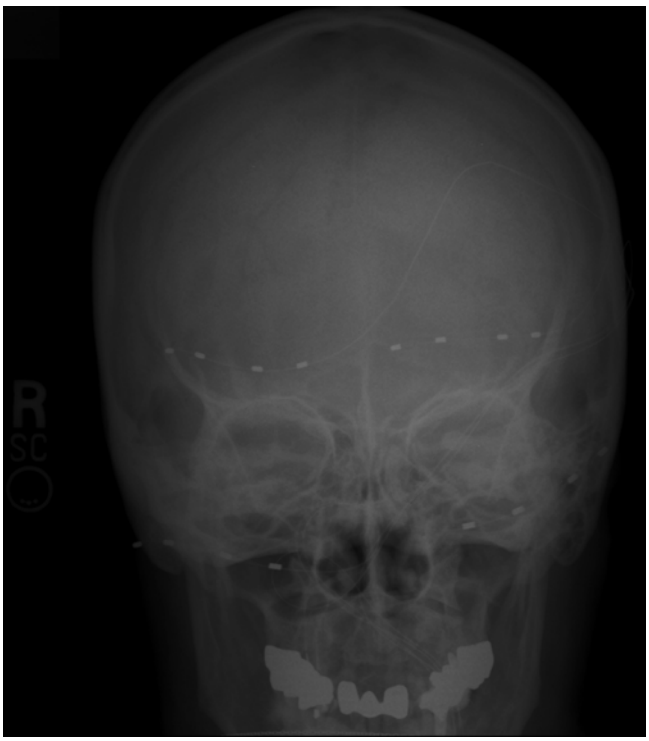

Fig. 9.17 Fluoroscopic image of supraorbital and occipital stimulator trial (Image courtesy of Rafael Justiz, MD)

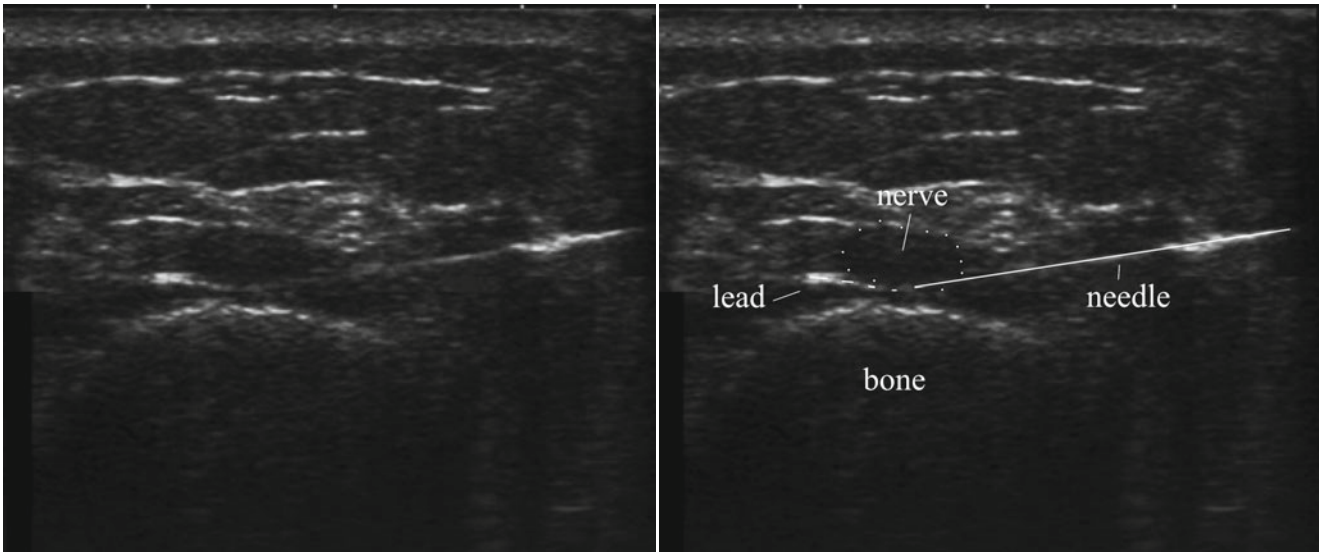

Fig. 9.18 Ultrasound-directed peripheral nerve stimulator placement (Image from Elahi et al. [58], with permission)

the military), athletic trauma, and craniotomies [33]. Stimulation of the occipital region for headache relief after trauma can also be achieved with a high cervical epidural SCS [34] or greater auricular nerve stimulation [35].

Hip pain It has been estimated that at least 25 % of the patients that undergo total hip replacement have persistent pain for at least a year post procedure, and more than 10 % have pain that limits activities of daily living [36]. Yakovlev et al. [37] described 12 patients with post total hip replacement hip pain, treated with PNFS leads placed in the area of greatest pain, parallel to the surgical scar; all 12 patients were still noting >50 % pain relief at 12 months.

Groin pain Persistent groin pain after inguinal hernia repair is, unfortunately, a common and debilitating occurrence, with a prevalence of 15–53 % [38]. Inguinal neuralgias may be one of the causes of pain after hernia repair and pelvic surgeries [25, 39, 40]. It is often difficult to surgically identify the offending nerves within the scar tissue, both for injection treatment or surgical excision, so the leads are placed in the groin region, usually above and below the scar. Rosendal et al. [40] reported on a patient with testicular pain after a hernia repair successfully treated with peripheral nerve stimulation of the ilioinguinal and genitofemoral nerves. Carayannopoulos et al. described two patients with persistent groin pain after hernia surgery in whom the PNS was successfully implanted under ultrasound guidance [25].

Chest wall pain The incidence of chest wall pain after thoracotomy (*post-thoracotomy syndrome*) has been estimated as high as 60 % of patients, even after 1 year [41]. Intercostal neuralgia can be treated by placing stimulating leads along the rib nerves. McJunkin et al. [42] described the use of peripheral field stimulation to treat a patient with pain after a thoracotomy.

Low back pain There are many causes of low back pain. For pain after surgery (*post-laminectomy syndrome*, also called *failed back syndrome*), the standard approach includes SCS, with stimulation of the low back and one or both legs. This technique is also used for lumbar radiculopathy and spinal stenosis [43]. However, *sacroiliac pathology* can mimic radiculopathy and occurs in at least 20 % of low back pain patients [44]. Guentchev et al. [45] described 12 patients with severe sacroiliac joint (SIJ) pathology treated with PNS leads placed parallel to the joint; after 1 year, 6 of the patients considered their treatment a success.

CRPS The nerve injuries and trauma associated with CRPS can cause devastating pain and dysfunction of the extremities. Various neuromodulation modalities have been used for CRPS, most commonly SCS or intrathecal drug delivery system [46, 47]. The ISP NeuPSIG recommended SCS for CRPS [3], though PNS or PNFS may be indicated in well-localized CRPS. As examples, there have been reports of the use of PNS to treat CRPS of the median nerve [48] and brachial plexus [49].

Postherpetic neuralgia Postherpetic neuralgia (PHN) is the dreaded complication of shingles [50]. One of the most recent studies of the incidence of PHN [51] showed that 20 % of the patients with shingles still had significant pain at 3 months, and 9 % had pain at 6 months. Twelve percent of

the lesions were in the cranial region, 15 % in the cervical region, 59 % in the thoracic region, 14 % in the lumbar region, and 11 % in the sacral region (the results equal more than 100 % because of multiple areas involved). Each of these areas may be a candidate for SCS, PNS, or PNS treatment [52]. High-frequency stimulation may work best for PHN [53].

Intracranial Stimulation

For severe refractory pain cases, where conservative or less invasive therapies have failed, intracranial neurostimulation, i.e., *motor cortex stimulation* (MCS) [54] and *deep brain stimulation* (DBS) or *thalamic stimulation* may be considered [55, 56]. Boccard et al. [55] reported that 85 patients who received DBS for various pain problems had a 66 % improvement 6 months after surgery, some of them with long-lasting relief (30 % improvement in pain and several other measures for >42 months).

However, current literatures on MCS and DBS are mostly case reports and noncontrolled clinical trials; therefore, insufficient data exists to make definite recommendations. Also, MCS and DBS are not FDA approved for pain management, and their use for pain treatment is considered "off-label" and often not reimbursed by insurances [4].

General Contraindications [15]

- Patients with bleeding disorders or those on anticoagulation that cannot be withheld in the perioperative period
- Major cognitive impairment or untreated psychiatric conditions
- Severe immunosuppression or active infections
- Patients who need routine MR imaging

Complications

Complications from neuromodulation can be divided into placement, migration, infection, and equipment failure:

Placement of SCS can result in spinal cord injury, CSF leak, epidural hematoma, and nerve injury. Placement of PNS can cause more nerve injury, trauma, and bleeding, while PNFS can be associated with nerve bleeding and trauma to nearby structures.

Migration of the leads is a chronic problem for SCS, PNS, and PNFS, because of movement, though the brain stimulators rarely migrate. Migration of the leads, mostly related to failure of the anchor, results in a loss of stimulation as well as potential trauma to nearby structures.

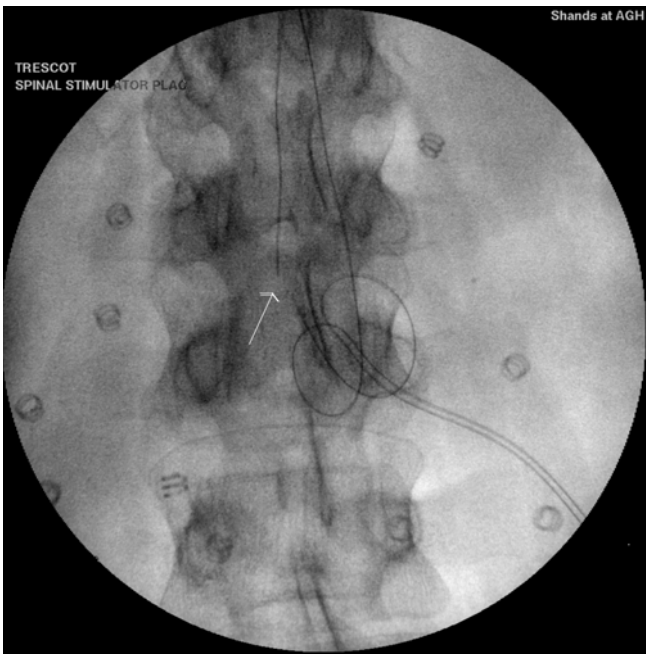

Fig. 9.19 Lead fracture seen at the *white arrow* (Image courtesy of Andrea Trescot, MD)

Infection is a potential complication of any implantation, though injection in the spinal canal has the added risk of meningitis as well as mechanical compression on the spinal cord from an epidural abscess. There can be dehiscence of the surgical wound or erosion of the equipment through the skin. These infections usually require equipment removal.

Equipment failure can be caused by lead fracture (Fig. 9.19) (though this is less of a problem with newer technology), disconnection from power source, and power failure. Although the use of a trial period theoretically should increase the success of the permanent placement, there is still a significant failure of success over time, which can be related to fibrosis. Caution must be used when performing RF (especially conventional RF) in the presence of a nearby spinal cord stimulator (SCS), because the electrical field could induce heating of the implanted SCS leads, causing damage to the SCS system as well as heat damage to unintended neural tissues [57].

Summary

Neuromodulation is a treatment option available for refractory nerve entrapment pain. As techniques and technology improve, recognizing appropriate subjects and choosing the most effective modalities can be challenges for providers.

References

1. Stuart RM, Winfree CJ. Neurostimulation techniques for painful peripheral nerve disorders. Neurosurg Clin N Am. 2009;20(1): 111–20.
2. Wall PD, Sweet WH. Temporary abolition of pain in man. Science. 1967;155:108–9.
3. Dworkin RH, O'Connor AB, Kent J, Mackey SC, Raja SN, Stacey BR, et al. Interventional management of neuropathic pain: NeuPSIG recommendations. Pain. 2013;154(11):2249–61.
4. Levy RM. The past, present, and future of neuromodulation: 2012 and beyond. Neuromodulation. 2012;15(5):411–5.
5. Deogaonkar M, Slavin KV. Peripheral nerve/field stimulation for neuropathic pain. Neurosurg Clin N Am. 2014;25(1):1–10.
6. Johnson MI, Bjordal JM. Transcutaneous electrical nerve stimulation for the management of painful conditions: focus on neuropathic pain. Expert Rev Neurother. 2011;11(5):735–53.
7. Carrol EN, Badura AS. Focal intense brief transcutaneous electric nerve stimulation for treatment of radicular and postthoracotomy pain. Arch Phys Med Rehabil. 2001;82(2):262–4.
8. Magis D, Sava S, d'Elia TS, Baschi R, Schoenen J. Safety and patients' satisfaction of transcutaneous Supraorbital NeuroStimulation (tSNS) with the Cefaly(R) device in headache treatment: a survey of 2,313 headache sufferers in the general population. J Headache Pain. 2013;14(1):95.
9. Lee AW, Pilitsis JG. Spinal cord stimulation: indications and outcomes. Neurosurg Focus. 2006;21(6):E3.
10. Burchiel KJ, Anderson VC, Brown FD, Fessler RG, Friedman WA, Pelofsky S, et al. Prospective, multicenter study of spinal cord stimulation for relief of chronic back and extremity pain. Spine. 1996;21(23):2786–94.
11. Rigoard P, Delmotte A, Moles A, Hervochon R, Vrignaud T, Misbert L, et al. Successful treatment of pudendal neuralgia with tricolumn spinal cord stimulation: case report. Neurosurgery. 2012;71(3):E757–62; discussion E63.
12. Barna SA, Hu MM, Buxo C, Trella J, Cosgrove GR. Spinal cord stimulation for treatment of meralgia paresthetica. Pain Physician. 2005;8(3):315–8.
13. Kemler MA, de Vet HC, Barendse GA, van den Wildenberg FA, van Kleef M. Spinal cord stimulation for chronic reflex sympathetic dystrophy – five-year follow-up. N Engl J Med. 2006;354(22):2394–6.
14. Kapural L, Nagem H, Tlucek H, Sessler DI. Spinal cord stimulation for chronic visceral abdominal pain. Pain Med. 2010;11(3): 347–55.
15. Deogaonkar M, Zibly Z, Slavin KV. Spinal cord stimulation for the treatment of vascular pathology. Neurosurg Clin N Am. 2014;25(1):25–31.
16. Kellner CP, Kellner MA, Winfree CJ. Spinal nerve root stimulation. Prog Neurol Surg. 2011;24:180–8.
17. Haque R, Winfree CJ. Transforaminal nerve root stimulation: a technical report. Neuromodulation. 2009;12(3):254–7.
18. Slavin KV. In: Lunsford LD, editor. Peripheral nerve stimulation. Pittsburgh: Karger; 2011.
19. Haque R, Winfree CJ. Spinal nerve root stimulation. Neurosurg Focus. 2006;21(6):E4.
20. Yanamoto F, Murakawa K. The effects of temporary spinal cord stimulation (or spinal nerve root stimulation) on the management of early postherpetic neuralgia from one to six months of its onset. Neuromodulation. 2012;15(2):151–4; discussion 4.
21. Lynch PJ, McJunkin T, Eross E, Gooch S, Maloney J. Case report: successful epiradicular peripheral nerve stimulation of the C2 dorsal root ganglion for postherpetic neuralgia. Neuromodulation. 2011;14(1):58–61; discussion.
22. Weiner RL, Reed KL. Peripheral neurostimulation for control of intractable occipital neuralgia. Neuromodulation. 1999;2(3): 217–21.
23. Abejon D, Perez-Cajaraville J. Peripheral nerve stimulation: definition. Prog Neurol Surg. 2011;24:203–9.
24. Falco FJ, Berger J, Vrable A, Onyewu O, Zhu J. Cross talk: a new method for peripheral nerve stimulation. An observational report with cadaveric verification. Pain Physician. 2009;12(6):965–83.
25. Carayannopoulos A, Beasley R, Sites B. Facilitation of percutaneous trial lead placement with ultrasound guidance for peripheral nerve stimulation trial of ilioinguinal neuralgia: a technical note. Neuromodulation. 2009;12(4):296–301.
26. Huntoon MA, Burgher AH. Ultrasound-guided permanent implantation of peripheral nerve stimulation (PNS) system for neuropathic pain of the extremities: original cases and outcomes. Pain Med. 2009;10(8):1369–77.
27. Slavin KV, Colpan ME, Munawar N, Wess C, Nersesyan H. Trigeminal and occipital peripheral nerve stimulation for craniofacial pain: a single-institution experience and review of the literature. Neurosurg Focus. 2006;21(6):E5.
28. Scher AI, Stewart WF, Liberman J, Lipton RB. Prevalence of frequent headache in a population sample. Headache. 1998;38(7):497–506.
29. Kapural L, Sable J. Peripheral nerve stimulation for occipital neuralgia: surgical leads. Prog Neurol Surg. 2011;24:86–95.
30. Abhinav K, Park ND, Prakash SK, Love-Jones S, Patel NK. Novel use of narrow paddle electrodes for occipital nerve stimulation – technical note. Neuromodulation. 2013;16(6):607–9.
31. Schwedt TJ, Dodick DW, Hentz J, Trentman TL, Zimmerman RS. Occipital nerve stimulation for chronic headache – long-term safety and efficacy. Cephalalgia. 2007;27(2):153–7.
32. Shaparin N, Gritsenko K, Garcia-Roves DF, Shah U, Schultz T, DeLeon-Casasola O. Peripheral neuromodulation for the treatment of refractory trigeminal neuralgia. Pain Res Manag: J Canadian Pain Society J Soc Can Traitement Douleur. 2015;20(2):63–6.
33. Evans RW. Posttraumatic headaches in civilians, soldiers, and athletes. Neurol Clin. 2014;32(2):283–303.
34. Elahi F, Reddy C. High cervical epidural neurostimulation for posttraumatic headache management. Pain Physician. 2014;17(4): E537–41.
35. Elahi F, Reddy C. Neuromodulation of the great auricular nerve for persistent post-traumatic headache. Pain Physician. 2014;17(4): E531–6.
36. Nikolajsen L, Brandsborg B, Lucht U, Jensen TS, Kehlet H. Chronic pain following total hip arthroplasty: a nationwide questionnaire study. Acta Anaesthesiol Scand. 2006;50(4):495–500.
37. Yakovlev AE, Resch BE, Karasev SA. Treatment of intractable hip pain after THA and GTB using peripheral nerve field stimulation: a case series. WMJ: Off Publ State Med Soc Wis. 2010; 109(3):149–52.
38. Poobalan AS, Bruce J, Smith WC, King PM, Krukowski ZH, Chambers WA. A review of chronic pain after inguinal herniorrhaphy. Clin J Pain. 2003;19(1):48–54.
39. Paicius RM, Bernstein CA, Lempert-Cohen C. Peripheral nerve field stimulation in chronic abdominal pain. Pain Physician. 2006;9(3):261–6.
40. Rosendal F, Moir L, de Pennington N, Green AL, Aziz TZ. Successful treatment of testicular pain with peripheral nerve stimulation of the cutaneous branch of the ilioinguinal and genital branch of the genitofemoral nerves. Neuromodulation. 2013;16(2):121–4.
41. Maguire MF, Ravenscroft A, Beggs D, Duffy JP. A questionnaire study investigating the prevalence of the neuropathic component of chronic pain after thoracic surgery. Eur J Cardiothorac Surg. 2006;29(5):800–5.

42. McJunkin TL, Berardoni N, Lynch PJ, Amrani J. An innovative case report detailing the successful treatment of post-thoracotomy syndrome with peripheral nerve field stimulation. Neuromodulation. 2010;13(4):311–4.
43. Kamihara M, Nakano S, Fukunaga T, Ikeda K, Tsunetoh T, Tanada D, et al. Spinal cord stimulation for treatment of leg pain associated with lumbar spinal stenosis. Neuromodulation. 2014;17(4):340–4; discussion 5.
44. Schwarzer AC, Aprill CN, Bogduk N. The sacroiliac joint in chronic low back pain. Spine. 1995;20(1):31–7.
45. Guentchev M, Preuss C, Rink R, Peter L, Wocker EL, Tuettenberg J. Technical note: treatment of sacroiliac joint pain with peripheral nerve stimulation. Neuromodulation. 2015;18(5):392–6.
46. Geurts JW, Smits H, Kemler MA, Brunner F, Kessels AG, van Kleef M. Spinal cord stimulation for complex regional pain syndrome type I: a prospective cohort study with long-term follow-up. Neuromodulation. 2013;16(6):523–9; discussion 9.
47. Goto S, Taira T, Horisawa S, Yokote A, Sasaki T, Okada Y. Spinal cord stimulation and intrathecal baclofen therapy: combined neuromodulation for treatment of advanced complex regional pain syndrome. Stereotact Funct Neurosurg. 2013;91(6):386–91.
48. Jeon IC, Kim MS, Kim SH. Median nerve stimulation in a patient with complex regional pain syndrome type II. J Korean Neurosurg Soc. 2009;46(3):273–6.
49. Stevanato G, Devigili G, Eleopra R, Fontana P, Lettieri C, Baracco C, et al. Chronic post-traumatic neuropathic pain of brachial plexus and upper limb: a new technique of peripheral nerve stimulation. Neurosurg Rev. 2014;37(3):473–9.
50. Weaver BA. Herpes zoster overview: natural history and incidence. J Am Osteopath Assoc. 2009;109(6 Suppl 2):S2–6.
51. Bricout H, Perinetti E, Marchettini P, Ragni P, Zotti CM, Gabutti G, et al. Burden of herpes zoster-associated chronic pain in Italian patients aged 50 years and over (2009–2010): a GP-based prospective cohort study. BMC Infect Dis. 2014;14(1):637.
52. Levy RM. Evidence-based review of neuromodulation for complex regional pain syndrome: a conflict between faith and science? Neuromodulation. 2012;15(6):501–6.
53. Lerman IR, Chen JL, Hiller D, Souzdalnitski D, Sheean G, Wallace M, et al. Novel high-frequency peripheral nerve stimulator treatment of refractory postherpetic neuralgia: a brief technical note. Neuromodulation. 2015;18(6):487–93.
54. Henderson JM, Lad SP. Motor cortex stimulation and neuropathic facial pain. Neurosurg Focus. 2006;21(6):E6.
55. Boccard SG, Pereira EA, Moir L, Aziz TZ, Green AL. Long-term outcomes of deep brain stimulation for neuropathic pain. Neurosurgery. 2013;72(2):221–30; discussion 31.
56. Moore NZ, Lempka SF, Machado A. Central neuromodulation for refractory pain. Neurosurg Clin N Am. 2014;25(1):77–83.
57. Jeon HY, Shin JW, Kim DH, Suh JH, Leem JG. Spinal cord stimulator malfunction caused by radiofrequency neuroablation -a case report. Korean J Anesthesiol. 2010;59(Suppl):S226–8.
58. Elahi F, Reddy C, Ho D. Ultrasound guided peripheral nerve stimulation implant for management of intractable pain after inguinal herniorrhaphy. Pain Physician. 2015;18(1):E31–8.

Surgical Treatment of Nerve Entrapment Syndromes

10

Amitabh Gulati, Daniel Krashin, and Helen W. Karl

Introduction

Surgical options for the treatment of nerve entrapments include decompression, transposition, and, rarely, transfer or grafting of the affected nerve. The choice among them depends on the degree of nerve injury. Most entrapments result in focal damage to the myelin sheath alone (*neuropraxic injury*) and will often resolve with nonoperative therapies. Very severe or prolonged nerve compression can lead to damage to the axon with variable damage to the surrounding layers (*axonotmesis)* or complete disruption of the axon (*neurotmesis*) [1, 2]. It is important that such lesions be recognized early, so that surgical intervention can be carried out before permanent changes develop in the denervated muscles. Surgical treatment of localized compression in selected patients, even in the presence of a systemic neuropathy due to diabetes mellitus or chemotherapy, has been shown to improve their quality of life [3]. Several recent books [4, 5] and chapters [6, 7] provide much greater detail than can be provided here, and the interested reader is encouraged to consult these texts.

Before the inevitable additional tissue injury of surgery is considered, however, failure of nonoperative therapies and substantial interference with the patient's function should be documented [8] (Table 10.1).

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Table 10.1 Preoperative considerations

Exhaust nonoperative therapies, usually at least 6 months	Duration and kind depend on the extent that the injury interferes with function (see Chap. 3)
Identify the nerve	Understanding of anatomical variations
	Careful physical examination (see Chap. 2)
	Tinel's sign or electrodiagnostics to document chronic nerve injury
	Rational nerve block (see Chap. 7)
Patient education	Expected areas of sensory and/or motor dysfunction
	Compliance with the rehabilitation program

Surgical Decompression and Repair

Techniques for Decompression

Perhaps the most common technique to relieve pressure on a nerve is *open decompression*, often termed *external neurolysis*, with direct visualization and release of all the tissues entrapping the nerve. Details of the techniques vary, but all include an incision to expose the nerve and resect all tissue encroaching on it [9]. Release of the median nerve in the carpal tunnel is one of the most common treatments [9, 10], and it has been shown to improve pain and reduce numbness by 70 % or more [11].

Some compression injuries lead to *intraneural edema*, hemorrhage, and fibrosis and produce a physiologic obstruction to nerve conduction [1], which may be relieved by *internal neurolysis* (*interfascicular dissection*). Internal neurolysis involves opening the *epineurium* and removing abnormal tissue. However, entering the epineurium increases the risk of further nerve damage, and its use remains controversial [12].

As surgical technology has improved, endoscopic methods for nerve decompression have become widespread [13]. *Endoscopic decompression* techniques involve smaller incisions, are considered minimally invasive, and may lead to

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faster recovery times and better satisfaction scores [14, 15]. However, endoscopic approaches carry a greater risk of damage, particularly to anatomically variant structures [16], and the advantages of endoscopic versus open decompression are still being debated [17, 18].

Some sites of nerve entrapment may not be appropriate for extensive open decompression. One such example is vascular decompression of the trigeminal ganglion (See Chaps. 23 and 24). In these cases, other methods to relieve entrapment must be used, the most common of which are *balloon compression* of the vascular bundle around the trigeminal ganglion and *microvascular decompression* [19]. Both techniques have been effective in treating trigeminal neuralgia [19, 20]. Innovative techniques have also been described for other entrapment syndromes, such as pudendal neuralgia [21, 22].

Transposition

In an effort to improve the outcomes of surgical treatment of some entrapments, such as the ulnar nerve in the cubital tunnel, a change in the path of the intact nerve (*transposition*) can be performed [23, 24]. Variations in transposition techniques may lead to better outcomes, as seen, for example, in the treatment of inferior alveolar [25] or ulnar [23] nerve entrapment. Transposition may even be considered to prevent possible future entrapment syndromes, such as changing the course of the radial nerve after fixation of a humeral fracture [26].

Nerve Transfer/Nerve Grafting

Nerve transfers usually involve the relocation of less important nerves near the afflicted nerve to the area of the dysfunction [27]. For example, intercostal nerves can be transferred to the deltoid and triceps muscles to improve function of those muscles [28]. Functional education is necessary to stimulate the transferred nerve's new role and should lead to a return of at least some function that was lost due to the injured nerve [29]. As these techniques improve, *grafting* of an entire nerve, in addition to transfers of a partial nerve, may allow return of function for injuries that once led to permanent loss of function, especially for brachial plexus injuries [30].

Nerve Conduits

Using *nerve conduits* to bridge gaps less than 3 cm in sensory nerves is a recent surgical development. These very thin tubes of biocompatible material provide a pathway for nerve regrowth. Veins were the first nerve conduits, and the FDA recently approved several additional materials [31]. The intact myelin sheath and endoneurium that remain after cryoneuroablation are an additional example of a physiologic conduit (see Chap. 8) [32].

The Role of Imaging (See Chap. 6)

With radiologic evaluation, especially ultrasonography and MRI, surgeons can better identify soft tissue lesions causing nerve entrapment [33–35]. Visualization of the nerves, perhaps in conjunction with electrophysiological studies, can improve surgical diagnosis and intervention [16, 36, 37]. Ultrasound can be used to locate specific anatomical variations, leading to the entrapment, that otherwise would be difficult to clinically ascertain [38, 39]. In addition to evaluating the detailed location of compression or injury, MRI can show pathology within the nerve itself [34, 40]. Finally, postoperative imaging has contributed evidence for postoperative management [41] and rational treatment selection [42].

Specific surgical treatments of nerve entrapments are outlined in each chapter.

Conclusion

Many surgical options are available for refractory pain and weakness resulting from nerve entrapment. Thorough preoperative evaluation and planning will lead to optimal outcomes.

References

1. Menorca RM, Fussell TS, Elfar JC. Nerve physiology: mechanisms of injury and recovery. Hand Clin. 2013;29(3):317–30.
2. Wong AH, Pianta TJ, Mastella DJ. Nerve transfers. Hand Clin. 2012;28(4):571–7.
3. Aszmann OC, Kress KM, Dellon AL. Results of decompression of peripheral nerves in diabetics: a prospective, blinded study. Plast Reconstr Surg. 2000;106(4):816–22.
4. MacKinnon SE. Nerve Surgery. New York: Thieme; 2015.
5. Kim DH, Hudson AR, Kline DG. Atlas of Peripheral Nerve Surgery. 2nd ed. Philadelphia, PA: Elsevier; 2013.
6. Wang AC, Campbell JN, Patil PG. Surgery of the peripheral nervous system as a treatment for pain. In: Fishman SM, Ballantyne JC, Rathmell JP, editors. Bonica's Management of Pain. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.
7. Dorsi MJ, Lenz FA. Neurosurgical approaches to the treatment of pain. In: McMahon SB, Koltzenburg M, Tracey I, Turk D, editors. Wall and Melzack's textbook of pain. 6th ed. Philadelphia: Elsevier Saunders; 2013.
8. Dellon AL, Mackinnon SE. Radial sensory nerve entrapment in the forearm. J Hand Surg Am. 1986;11(2):199–205.
9. Steinberg DR. Surgical release of the carpal tunnel. Hand Clin. 2002;18(2):291–8.
10. Huang JH, Zager EL. Mini-open carpal tunnel decompression. Neurosurgery. 2004;54(2):397–9; discussion 9–400.

11. Thurston A, Lam N. Results of open carpal tunnel release: a comprehensive, retrospective study of 188 hands. Aust N Z J Surg. 1997;67(5):283–8.
12. Mackinnon SE, McCabe S, Murray JF, Szalai JP, Kelly L, Novak C, et al. Internal neurolysis fails to improve the results of primary carpal tunnel decompression. J Hand Surg Am. 1991;16(2):211–8.
13. Uchiyama S, Itsubo T, Nakamura K, Kato H, Yasutomi T, Momose T. Current concepts of carpal tunnel syndrome: pathophysiology, treatment, and evaluation. J Orthop Sci. 2010;15(1):1–13.
14. Hwang PY, Ho CL. Minimally invasive carpal tunnel decompression using the KnifeLight. Neurosurgery. 2007;60(2 Suppl 1):ONS162–8; discussion ONS8-9.
15. Trumble TE, Diao E, Abrams RA, Gilbert-Anderson MM. Single-portal endoscopic carpal tunnel release compared with open release: a prospective, randomized trial. J Bone Joint Surg Am. 2002;84-A(7):1107–15.
16. Vinding MT, Tarnowski JR, Benyahia M. A rare anatomical variant of the thenar branch discovered during open decompression of the median nerve. J Plast Surg Hand Surg. 2010;44(6):322–4.
17. Thoma A, Veltri K, Haines T, Duku E. A systematic review of reviews comparing the effectiveness of endoscopic and open carpal tunnel decompression. Plast Reconstr Surg. 2004;113(4):1184–91.
18. Dützmann S, Martin KD, Sobottka S, Marquardt G, Schackert G, Seifert V, et al. Open vs retractor-endoscopic in situ decompression of the ulnar nerve in cubital tunnel syndrome: a retrospective cohort study. Neurosurgery. 2013;72(4):605–16; discussion 14–6.
19. Stomal-Słowińska M, Słowiński J, Lee TK, Uitti RJ, Deen HG, Reimer R, et al. Correlation of clinical findings and results of percutaneous balloon compression for patients with trigeminal neuralgia. Clin Neurol Neurosurg. 2011;113(1):14–21.
20. Jagannath PM, Venkataramana NK, Bansal A, Ravichandra M. Outcome of microvascular decompression for trigeminal neuralgia using autologous muscle graft: a five-year prospective study. Asian J Neurosurg. 2012;7(3):125–30.
21. Forbes J, Cooper C, Jermakowicz W, Neimat J, Konrad P. Microvascular decompression: salient surgical principles and technical nuances. J Vis Exp. 2011;53:e2590.
22. de Bisschop E, Nundlall R. Surgical decompression of pudendal nerve by transperineal approach using a probe with a small balloon. J Gynecol Obstet Biol Reprod (Paris). 2011;40(3):225–30.
23. Henry M. Modified intramuscular transposition of the ulnar nerve. J Hand Surg Am. 2006;31(9):1535–42.
24. Macadam SA, Gandhi R, Bezuhly M, Lefaivre KA. Simple decompression versus anterior subcutaneous and submuscular transposition of the ulnar nerve for cubital tunnel syndrome: a meta-analysis. J Hand Surg Am. 2008;33(8):1314.e1–12.
25. Khajehahmadi S, Rahpeyma A, Bidar M, Jafarzadeh H. Vitality of intact teeth anterior to the mental foramen after inferior alveolar nerve repositioning: nerve transpositioning versus nerve lateralization. Int J Oral Maxillofac Surg. 2013;42(9):1073–8.
26. Chamseddine AH, Zein HK, Alasiry AA, Mansour NA, Bazzal AM. Trans-fracture transposition of the radial nerve during the open approach of humeral shaft fractures. Eur J Orthop Surg Traumatol. 2013;23(6):725–30.
27. Tung TH, Mackinnon SE. Nerve transfers: indications, techniques, and outcomes. J Hand Surg Am. 2010;35(2):332–41.
28. Malungpaishrope K, Leechavengvongs S, Witoonchart K, Uerpairojkit C, Boonyalapa A, Janesaksrisakul D. Simultaneous intercostal nerve transfers to deltoid and triceps muscle through the posterior approach. J Hand Surg Am. 2012;37(4):677–82.
29. McRae MC, Borschel GH. Transfer of triceps motor branches of the radial nerve to the axillary nerve with or without other nerve transfers provides antigravity shoulder abduction in pediatric brachial plexus injury. Hand (N Y). 2012;7(2):186–90.
30. Bertelli JA, Ghizoni MF. Nerve root grafting and distal nerve transfers for C5–C6 brachial plexus injuries. J Hand Surg Am. 2010;35(5):769–75.
31. Deal DN, Griffin JW, Hogan MV. Nerve conduits for nerve repair or reconstruction. J Am Acad Orthop Surg. 2012;20(2):63–8.
32. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
33. Bruyn GA, Moller I, Klauser A, Martinoli C. Soft tissue pathology: regional pain syndromes, nerves and ligaments. Rheumatology (Oxford). 2012;51 Suppl 7:vii22–5.
34. Hochman MG, Zilberfarb JL. Nerves in a pinch: imaging of nerve compression syndromes. Radiol Clin North Am. 2004;42(1):221–45.
35. Grant GA, Goodkin R, Maravilla KR, Kliot M. MR neurography: diagnostic utility in the surgical treatment of peripheral nerve disorders. Neuroimaging Clin N Am. 2004;14(1):115–33.
36. Zhong W, Zhang W, Zheng X, Li S, Shi J. The high-resolution ultrasonography and electrophysiological studies in nerve decompression for ulnar nerve entrapment at the elbow. J Reconstr Microsurg. 2012;28(5):345–8.
37. Lee D, Dauphinée DM. Morphological and functional changes in the diabetic peripheral nerve: using diagnostic ultrasound and neurosensory testing to select candidates for nerve decompression. J Am Podiatr Med Assoc. 2005;95(5):433–7.
38. Harvie P, Patel N, Ostlere SJ. Ulnar nerve compression at Guyon's canal by an anomalous abductor digiti minimi muscle: the role of ultrasound in clinical diagnosis. Hand Surg. 2003;8(2):271–5.
39. Lucchetta M, Liotta GA, Briani C, Marquez EM, Martinoli C, Coraci D, et al. Ultrasound diagnosis of peroneal nerve variant in a child with compressive mononeuropathy. J Pediatr Surg. 2011;46(2):405–7.
40. Grant GA, Britz GW, Goodkin R, Jarvik JG, Maravilla K, Kliot M. The utility of magnetic resonance imaging in evaluating peripheral nerve disorders. Muscle Nerve. 2002;25(3):314–31.
41. Baltodano PA, Tong AJ, Chhabra A, Rosson GD. The role of magnetic resonance neurography in the postoperative management of peripheral nerve injuries. Neuroimaging Clin N Am. 2014;24(1):235–44.
42. Vögelin E, Nüesch E, Jüni P, Reichenbach S, Eser P, Ziswiler HR. Sonographic follow-up of patients with carpal tunnel syndrome undergoing surgical or nonsurgical treatment: prospective cohort study. J Hand Surg Am. 2010;35(9):1401–9.

The Why and What of Integrative Pain Medicine

11

Heather Tick

Introduction

The medical system in the USA is the most expensive in the world by a large margin, spending almost twice as much as the second costliest system. Despite this great cost, usually involving expensive drugs and procedures, the USA does not have the best outcomes when compared to other developed nations. The Institute of Medicine (IOM) reported in *US Healthcare*: In International Perspective: Shorter Lives Poorer Health [1] a comparison of the US healthcare system to other developed nations and found that the USA has "a healthcare disadvantage." This deficit in health and longevity is not explained by economic and educational status. Even wealthy Americans with access to health services were not as healthy as their counterparts in other countries. Conventional hospital care in the USA is ranked as the third leading cause of death [2] and has been the subject of study by the Institute of Medicine (IOM) [3] and other large reviews [4].

Commonly used pain medications are the source of a significant portion of the morbidity and mortality. Nonsteroidal anti-inflammatory drugs (NSAIDs) were found to kill 16,500 Americans each year [5]. The only diagnostic categories studied in this review were osteoarthritis and rheumatoid arthritis; therefore, this is likely a gross underestimation of the actual death toll. In fact, NSAIDs outstrip HIV as a cause of death. A report by Volkow et al. stated: "In 2010 alone, prescription opioids were involved in 16,651 overdose deaths, whereas heroin was implicated in 3,036" [6].

The Institute for Healthcare Improvement (IHI) has outlined "the triple aim"

1. Improving the patient experience of care (including quality and satisfaction)
2. Improving the health of populations
3. Reducing the per capita cost of healthcare [9]

It is statistics like these that, in part, fuel interest in integrative medicine (IM), which involves the integration of all available and appropriate healthcare strategies and disciplines for the patient's benefit. Ideally IM and Integrative Pain Medicine (IPM) involve integration at many levels: the coordination of the services provided, collaboration among the disciplines involved, a patient-centered focus with acceptance of the patient as a member of the team, attention to predictive and preventative models of care, and, finally, inclusion of social, psychological, and physical factors into the assessment of patients. The conventional allopathic medical system engages patients around a problem or disease and focuses extensively on the management of diseases, rather than preventative measures or health promotion. Both the World Health Organization (WHO) [7] and the Institute of Medicine (IOM) [8] have affirmed that health is more than the absence of disease, and the IOM Summit on Integrative Medicine and the Health of the Public outlined key features of IM.

For the IHI, integration is seen as a key component of erasing the US health deficit, mitigating the risk of the current system while lowering costs and improving outcomes. In specifically addressing the problem of chronic pain and the failure of the commonly available drugs, procedures, and surgeries to stem the tide of rising numbers of people affected by pain and opioid overdoses, of the Army Surgeon General Task Force Report prescribed IPM. Additionally, it recommended that the therapeutic order of interventions be flipped to begin with yoga,

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acupuncture, chiropractic work, and massage as the first-order interventions, instead of drugs, surgeries, ablations, and injections [10].

The Medicine of What You Eat, Drink, Think, Feel, and Do

In this short chapter, it is impossible to cover all the facets that can be included in an integrative approach to pain medicine. Instead, a few key factors will be reviewed, and references for further reading will be included.

Eat, Drink

There is a growing literature documenting the effects of lifestyle choices on overall health outcomes by changing host susceptibility and propensity to heal. The European Prospective Investigation Into Cancer and Nutrition-Potsdam Study (EPIC) evaluated the effects of four lifestyle factors on health (never smoking, BMI under 30, physical activity for at least 3.5 h a week, and eating a healthy diet with vegetables, fruit, whole grain bread, and low meat consumption) and revealed benefits that no drugs or procedures can remotely approximate. Participants with all four factors at baseline had a 78 % lower risk of developing a chronic disease – diabetes, 93 %; myocardial infarction, 81 %; stroke, 50 %; and cancer, 36 % – than participants without a healthy factor [11]. The common denominator of all these chronic conditions is inflammation, and the four factors studied all improve body-wide inflammation. Inflammation influences both the experience of pain and the recovery from it.

In North America, grains and their processed derivative foods are heavily subsidized, but fresh, whole foods are not. Therefore, it is cheaper to buy fast food and bags of chips and cookies than it is to eat broccoli or apples. There are food deserts in our country where people cannot find stores that stock fresh foods. Americans each eat an average of 150 lb of sugar and 150 lb of refined flour each year. It is becoming clearer that our epidemic of obesity and diabetes, with all their attendant health and pain complications, is a food-borne illness fostered by a heavily processed and sweetened diet.

High glycemic foods that spike blood sugar also increase inflammation. There is an association between inflammatory biomarkers and the level of pain experienced [12]. Additionally, high glycemic diets are associated with diabetes and obesity, both of which adversely affect health, life expectancy, and cause pain. There is ample medical literature citing diabetes and obesity as risk factors for adverse outcomes with interventional procedures. A processed food diet also promotes low pH, which in turn reduces the activity of essential physiological enzyme reactions in the body [13]. Refined foods, sugar, meat, and dairy all lower the body's pH level, while green vegetables, lentils, and most fruits raise the pH.

Adopting a health focus, even once patients have diseases including chronic pain, can improve outcomes, both interventional and noninterventional, through the reduction of inflammation and improved metabolism. We spend 75 cents of every healthcare dollar on diet-preventable diseases, and pain figures highly in that calculation.

Think, Feel, Do

There is lengthy literature on exercise, stress reduction, cognitive behavioral, and mind-body interventions that affect mood, pain perception, opioid use, and improvements in function. Kabat-Zinn et al. [14] showed that participants in a 10-week mindfulness course reduced opioid consumption and improved self-esteem compared with a control group. Many scientists have been able to show how the brain changes in pain states, how emotion and attention affect pain processing, and the impact of mind-body mechanisms [15, 16]. Exercise and physical activity are well known to improve the outcomes for pain patients who are often phobic of movement, in part because they confuse hurt and harm, have a depressed mood, or possibly as a side effect of many medications. Social isolation and the loss of relationships in the realms of work, friendship, and intimacy also take their toll. Shelley Taylor's work on the beneficial effects of oxytocin on our biochemistry as result of social support and even touch explains why the social aspects of pain care need attention [17]. Elizabeth Blackburn, who shared a Nobel Prize for her research on telomeres and telomerase, has shown that distress shortens our telomeres and is associated with decreased longevity [18]. Further studies are showing that telomere length can be preserved through stress reduction strategies.

Summary

As high-cost and high-risk interventions become less economically sustainable in US healthcare, the interest in IM and IPM increases. The Summit on Integrative Medicine and the Health of the Public [19] went on to outline several key features of IM: person-centered care with integration "across the lifespan to include personal, predictive, preventive, and participatory care," integration of teams that included all disciplines including CAM, and "seamless engagement of the full range of established health factors—physical, psychological, social, preventive, and therapeutic."

Resources

Here are some of the data we have on nutrition and lifestyle and pain.

Diet, Inflammation, Obesity, and Disease

http://lpi.oregonstate.edu/infocenter/inflammation.html

Dietrich M, Jialal I: The effect of weight loss on a stable biomarker of inflammation, C-reactive protein. Nutr Rev 2005; 63(1): 22–8.

Nutrition

C. Colantuoni et al., "Excessive Sugar Intake Alters Binding to Dopamine and Mu-Opioid Receptors in the Brain," Neuroreport 12, no. 16 (2001): 3549–52.

Alan W. Barclay et al., "Glycemic Index, Glycemic Load, and Chronic Disease Risk — a Meta-analysis of Observational Studies," American Journal of Clinical Nutrition 87, no. 3 (March 2008): 627–37.

A. S. Grimaldi et al., "25(OH) Vitamin D Is Associated with Greater Muscle Strength in Healthy Men and Women," Medicine and Science in Sports and Exercise 45, no. 1 (January 2013): 157–62.

Turmeric

V. Kuptniratsaikul et al., "Efficacy and Safety of Curcuma domestica Extracts in Patients with Knee Osteoarthritis," Journal of Alternative and Complementary Medicine 15, no. 8 (August 2009): 891–97.

K. A. Agarwal et al., "Efficacy of Turmeric (Curcumin) in Pain and Postoperative Fatigue after Laparoscopic Cholecystectomy: A Double-Blind, Randomized Placebo-Controlled Study," Surgical Endoscopy 25, no. 12 (December 2011): 3805–10.

Meditation and Mind Body Interventions

Jon Kabat-Zinn, Full Catastrophe Living: Using the Wisdom of Your Body and Mind to Face Stress, Pain, and Illness (New York: Delta, 2005).

R. Sapolsky, Why Zebras Don't Get Ulcers: A Guide to Stress, Stress-Related Diseases, and Coping (New York: Henry Holt, 2004).

J. F. Thayer and E. Sternberg, "Beyond Heart Rate Variability," Annals of the New York Academy of Sciences 1088 (November 2006): 361–72.

Integrative Pain Medicine

Heather Tick, *Holistic Pain Relief*, New World Library, Novato CA, 2013.

References

1. Institute of Medicine. Shorter lives poorer health: panel on understanding cross-national health differences among high-income countries. Washington, DC: National Academy of Sciences Press; 2013.
2. Starfield B. Is US, health really the best in the world? JAMA J Am Med Assoc. 2000;284(4):483–5.
3. Institute of Medicine. To err is human. Washington, DC: National Academy of Sciences Press; 2000.
4. James JT. A new, evidence-based estimate of patient harms associated with hospital care. J Patient Saf. 2013;9(3):122–8.
5. Singh G. Gastrointestinal complications of prescription and over-the-counter nonsteroidal anti-inflammatory drugs: a view from the ARAMIS database. Arthritis, Rheumatism, and Aging Medical Information System. Am J Ther. 2000;7(2):115–21.
6. Volkow ND, Frieden TR, Hyde PS, Cha SS. Medication-assisted therapies – tackling the opioid-overdose epidemic. N Engl J Med. 2014;370(22):2063–6.
7. World Health Organization (WHO). WHO launches the first global strategy on traditional and alternative medicine. 2002 [23 Mar 2014]. Available from: http://www.who.int/mediacentre/news/releases/release38/en/.
8. Institute of Medicine. Integrative medicine and the health of the public: a summary of the February 2009 summit. Washington, DC: The National Academy of Sciences Press; 2009.
9. The Institute for Healthcare Improvement. IHI Triple aim initiative. [11 Feb 14]. Available from: http://www.ihi.org/Engage/Initiatives/TripleAim/Pages/default.aspx.
10. Pain Management Task Force Report: Office of the Surgeon General of the US Army Medical Department. 2010. [11 Mar 14]. Available from: http://www.regenesisbio.com/pdfs/journal/pain_management_task_force_report.pdf.
11. Ford ES, Bergmann MM, Kroger J, Schienkiewitz A, Weikert C, Boeing H. Healthy living is the best revenge: findings from the European Prospective Investigation into Cancer and Nutrition-Potsdam study. Arch Intern Med. 2009;169(15):1355–62.
12. Wilander AM, Monica K, Axmon A, Nordander C. Inflammatory biomarkers in serum in subjects with and without work related neck/shoulder complaints. BMC Musculoskelet Disord. 2014;15:103–10.
13. Schwalfenberg GK. The alkaline diet: is there evidence that an alkaline pH diet benefits health? J Environ Public Health. 2012; 2012:727630.
14. Kabat-Zinn J, Lipworth L, Burney R. The clinical use of mindfulness meditation for the self-regulation of chronic pain. J Behav Med. 1985;8(2):163–90.
15. Bushnell MC, Ceko M, Low LA. Cognitive and emotional control of pain and its disruption in chronic pain. Nat Rev Neurosci. 2013;14(7):502–11.
16. Villemure C, Bushnell MC. Cognitive modulation of pain: how do attention and emotion influence pain processing? Pain. 2002;95(3):195–9.
17. Taylor SE, Gonzaga GC, Klein LC, Hu P, Greendale GA, Seeman TE. Relation of oxytocin to psychological stress responses and hypothalamic-pituitary-adrenocortical axis activity in older women. Psychosom Med. 2006;68(2):238–45.
18. Blackburn EH. Telomeres and telomerase: the means to the end (Nobel lecture). Angew Chem Int Ed Engl. 2010;49(41): 7405–21.
19. Weisfeld V. Summit on integrative medicine & the health of the public: issue background & overview 2009 [11 Mar 14]. Available from: http://www.iom.edu/~/media/Files/ActivityFiles/Quality/IntegrativeMed/IM20Summit20Background20Paper20Weisfeld2022309.pdf.

Peripheral Nerve Blocks in Palliative Care for Cancer Patients

12

Zbigniew (Ben) Zylicz

Introduction

Pain control in patients with cancer is undergoing constant evolution. In the 1960s, it was obvious that effective pain-controlling drugs like opioids were not frequently enough used, because of fear of addiction and rapid development of tolerance. Morphine was available only parenterally, and oral administration was seen as ineffective. A lot of effort has been put into changing these attitudes. Since the 1980s, morphine, and later other opioids, dominated on the pain market. Morphine (equivalent) consumption became a recognized parameter of the quality of palliative care [1]. Since this time, pain physicians concentrated more and more on chronic nonmalignant pain, leaving cancer patients to palliative care physicians. Many countries broke their legislative barriers and considerably increased their opioid use. In these countries, however, it became clear that opioid consumption alone was unable to solve the problem of cancer pain. A systematic review of 52 studies revealed that equal numbers of patients with advanced cancer, as well as patients actively treated by oncologists, are suffering from pain now in comparison to 40 years ago [2]. This is occurring despite the rapid development of modern oncology, which for most patients is palliative treatment. The reason for this phenomenon is still unknown, and few people try to investigate this.

A couple of factors are candidates to explain this paradox. First of all, patients with cancer live much longer in comparison to four decades ago. They live longer, but they suffer pain longer and need to use pain-controlling measures much longer than before. The second factor could be that doctors became used to the term "cancer pain" and see all pains tormenting patients with cancer as cancer pain. It is reasonable to suggest that doctors treat tumor-related pain much better with modern oncological technologies and classic analgesics, but become more and more desperate with types of pain that have nothing to do with cancer, such as increasing debility, post-therapy toxicity, lack of movement, and cachexia. Those pains may arise as a result of nerve compression and nerve stretching, overuse of atrophic muscles, myofascial trigger points, sores, and stiffness of joints and tendons [3]. If these pains are less sensitive to opioids and to other analgesics, calling them "cancer pain" will result in more intensive treatment with opioids and, as a result, more opioid toxicities and treatment failures. One of the common adverse effects of opioids used in such a way is opioid-induced hyperalgesia (OIH), which may be much more frequent now than in the past [4]. OIH pain, which may be virtually "invisible" for the patient, can be accompanied by periodic, diffuse pain all over the body. This is why we should concentrate on history taking and meticulous pain diagnosis and differentiation. This virtue is nearly lost when doctors decide that all pain is "cancer pain," and cancer pain should be treated with opioids.

Etiology of Nerve Compression Pain in Cancer Patients

In fact, many of those nonmalignant pains in cancer patients will be caused by nerve compression against bony edges, impingement between the ligaments and tendons, or simply when powerful muscles are atrophic and do not protect the joints and their excursions increase, stretching the nerve. The prevalence of these entrapment syndromes is different from those seen in the healthier, noncancer population. Most of the painful sites due to nerve compression are at the rear and sides of the body, as the patients compress these nerves while lying supine or on the side for long periods of time. The nerves of cachectic patients lose their supporting tissue (fat pads, muscles, connective tissue) and come closer to more firm structures such as bone, ligaments, or tendons.

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Examination

During the physical examination, it may appear that the patient suffers pain on palpation in some typical sites, usually suboccipitally, on the shoulders, or on the pelvis. The localized pain may be unilateral or bilateral. Unilateral points usually suggest peripheral compression. For example, a patient with hepatomegaly due to liver metastases lies preferentially on the right side and compresses a number of small cutaneous nerves against the iliac crest. When the points are distributed symmetrically, the damage may be located more centrally (e.g., at the level of nerve roots), and physicians should consider imaging this area with an MRI or CT. Compression of the nerve root may cause neural edema and nerve entrapment in a distal narrow space, the so-called "double crush" phenomenon [5]. Multilevel pains may be a sign of increased spinal sensitization and can be better treated with, among other options, epidural steroids [6, 7].

During the physical examination, attention should be paid to muscle tension and spasm. For instance, after a vertebral fracture, increased muscle tension stabilizes the fractured area. The pain is caused not by the fracture but by tense muscles and entrapped nerves, which pierce these muscles. Treating the entrapped nerves and muscles may relieve pain but may also destabilize the fracture, with potentially severe consequences.

Scope of the Chapter

This chapter focuses on purely peripheral lesions, which can be treated by non-anesthesiologists at the bedside. Why non-anesthesiologists? Solely because these procedures are rarely done by modern pain clinics; they are too simple and are hardly ever mentioned in anesthesiology literature [8]. Compressed nerves are "under the radar" of sophisticated imaging techniques, and "what is not seen is not recognized." Also, cancer patients, once declared terminally ill, tend to dislike hospital procedures and operating rooms. They frequently refuse to be transported back to hospitals and prefer simple methods of treatment. Institutions that provide palliative care are often closer to where the patients live, as the visits of family and friends belong undoubtedly to the "quality of life" concept of palliative care. This chapter will not concentrate on anatomy or the technique of infiltration, as that is described in more detail elsewhere.

Technique and Contraindications

The techniques that provide the best results are infiltrations of the painful site with local anesthetics (usually bupivacaine) mixed with steroids, most often methylprednisolone (Depo-Medrol®). This procedure is simple, does not need a fluoroscopy, can be done at the bedside, and can be repeated after a couple of weeks or months. Doctors attempting these infiltrations should observe the following rules:

1. Infiltrations done by the non-anesthesiologists should be on a distance from the median line of the spine, as damaging nerve roots or even the spinal cord may have serious consequences and increase patient suffering.
2. The painful spot should be palpable, and, for the patients with abundant fat above the painful site, ultrasound should be used to localize the bone. The skin around the painful spot should be unchanged. Avoid infections and areas with lymphedema. Blood coagulation is not such an issue as with other anesthesiology procedures, but the puncture sites should sometimes be compressed for a longer while, post procedure. For obvious reasons, avoid puncturing varicose veins or arteries. Also, caution should be used for nerve infiltration of the lower body parts in case of inferior vena cava syndrome.

The technique of injection is usually simple. After puncturing the skin, try to make contact with the bone and "walk" the needle tip until the patient indicates that you are in the vicinity of the nerve. The patient will be keen to tell you when you have made contact with the nerve. Next, retract the needle a couple of millimeters to avoid injecting drugs into the nerve, as you may damage it and increase pain for the patient. In areas like the suboccipital region (see Chap. 17: Greater occipital nerve) where there is little anatomical space, you may try to inject local anesthetic first, and add the steroids in the second injection, 20–30 min later. You may have problems with localizing the nerve again, but it is certainly less painful for the patients.

A number of patients will be "needle phobic," too healthy to get repeated steroid injections, or have another contraindication mentioned above. For these patients, there is a treatment procedure with low-level laser [9]. This technique is efficacious, but the patient needs to get the treatment daily and wait for 1–2 weeks to achieve results. This time can be too long and too cumbersome for the terminally ill.

Evaluation

The physician performing infiltrations should evaluate the results and carefully describe them. The first (but never only) evaluation should be done 30–60 min after the injection. The patient will notice the immediate local anesthetic effect. This will indicate whether or not you have infiltrated the right spot. The patient may experience numbness around the injection site but may already notice decrease of the pain. The second evaluation should be done about 24 h later. At this time, the patient may tell you that pain control was excellent overnight, but the pain has now returned. The effect of steroids may be nonapparent for another day or two, especially if the drug needs to diffuse a long way before it reaches

the nerve. The patient needs your encouragement at this stage. It may be possible that the patient experienced the first (local anesthetic) effect, but never the second (steroid) effect. This may mean that the cause of nerve compression is higher, for example, at the nerve root level. Injections can be repeated, although frequent injections may cause local atrophy. Some patients will tell you that the pain has not gone away, but has certainly decreased. This may mean that not all painful spots were identified and/or infiltrated. At this stage, you should repeat the examination again.

Most terminally ill patients will need one or at most two injections (the effect usually lasts for couple of weeks to months), where the danger of local atrophy is close to zero. Remember that methylprednisolone may suppress the adrenals, as all corticosteroids do, and a patient with adrenal insufficiency may develop serious problems afterwards.

Some Other Safety Aspects Relevant to the Procedure

Prior to infiltrations, many patients are being treated with high doses of opioids and other analgesics. Rapid relief of pain may theoretically cause increased opioid toxicity, especially respiratory depression. There are no protocols as to how to deal with these situations. The number one rule is to decrease the dose of the opioid as much as possible before the procedure. "As much as possible" means that the patient should not experience more pain because of the reduction. Usually, a decrease of the opioid dose by one-third is feasible. Some patients who are already experiencing opioid-induced hyperalgesia may even feel better afterwards. After the procedure had been completed and seems to be effective (evaluation after one hour), opioid reduction by another third can be attempted. The last third of the dose is dependent on the other pains in this patient, as well as the long-term effects of nerve infiltrations. It is not unusual that opioids are reduced to 10–20 % of the original dose. Too fast a reduction of the dose my precipitate symptoms of withdrawal. Also, some patients will suffer bouts of diarrhea for a while, so please warn them ahead of time.

Injections of steroids may aggravate existing diabetes. Increased blood glucose can be observed during the first week, but not later on. Bupivacaine can be cardiotoxic; in the case of heart failure, reduce the dose accordingly or avoid the procedure completely.

Most Often Performed Procedures in Palliative Care

Infiltration of the Greater Occipital Nerve (GON)

Many patients with cachexia who wish to stay active and read books or newspapers in bed will overuse their occipital muscles trying to keep their head straight. This may result in pain in the neck, sometimes radiating to the front of the head and causing one-sided, tension-like headaches (see Chap. 17). Patients are also prone to this syndrome after neck dissection for head and neck tumors. The greater occipital nerve pierces the suboccipital muscles, where the nerve can become entrapped. Changes may be uni- or bilateral. You can diagnose this syndrome by standing in front of the patient, taking his/her head in your hands, and palpating the suboccipital area with one finger. Reproduction of pain with or without radiation to the front of the head is diagnostic. Rarely, the branches of the GON are re-entrapped against the prominent *superior nuchal line*, at the place of attachment of the nuchal muscles. This happens in the case of prolonged disease and lying in bed with a firm pillow. Inject the local anesthetics first (usually 1–2 ml of bupivacaine 0.5 %) and methylprednisolone 30 min later.

Suprascapular Nerve Entrapment (SNE)

One of the most common nerve entrapment syndromes in palliative care is suprascapular nerve entrapment (SNE) [10]. In healthy people, the shoulder blade is fixed to the chest and spine with powerful muscles. In patients with cachexia, these muscles become weak and atrophic. The excursions of the shoulder blade increase, which may cause tension at the *suprascapular nerve*, which enters the shoulder blade through the narrow suprascapular notch (see Chap. 28). Not surprisingly, shoulder pain due to SNE is most often seen on the dominant side. Patients who do everything to stay at home are of higher risk; using crutches or propelling a wheel chair also makes patients vulnerable. Women who suffer from arm lymphedema after treatment for breast cancer may have an extremity so heavy that they stretch the suprascapular nerve. Some patients use their auxiliary chest muscles to improve ventilation. They may support their body on their elbows, greatly increasing traction of the suprascapular nerves on both sides. These patients will be very vulnerable for respiratory depression when the pain is suddenly relieved, because of relative opioid overdose. In this case, it is recommended to perform these procedures twice, with 1 or 2 days in between, simultaneously reducing opioids. Another group of patients who are of high risk of suprascapular nerve entrapment are the patients treated over a long period of time with oral corticosteroids, especially dexamethasone. Dexamethasone causes proximal muscle atrophy in the area of the shoulders and hips [11]. Diagnosis is made by palpating, usually with a thumb, in the suprascapular fossa. The painful spot is localized above the distal one-third portion of the shoulder blade crest. The injection should first aim at the crest and should not be in the craniocaudal direction, to avoid puncturing the pleura. Five mililiters of a mixture of bupivacaine and methylprednisolone will likely fill the whole fossa, so an accurate localization of the nerve with the needle is not

strictly necessary. The suprascapular artery lies close to the nerve, which increases the danger of complications, such as hematoma. In rare cases, it is advised to use ultrasound to visualize the fossa.

Notalgia Paresthetica

Notalgia paresthetica is a common localized itch, mostly not painful, affecting mainly the T2–T6 dermatomes on the back, but occasionally with a more widespread distribution, involving the shoulders, back, and upper chest. Typically, only the medial half of the dermatome will be involved. It is thought that this syndrome is caused by entrapment of the median branch of the spinal nerve in the paraspinal muscles. Not infrequently, these contracted paraspinal muscles are fixing spine pathology. Relieving this tension will probably not destabilize the fracture, but may, paradoxically, increase pain.

Intercostal 11th or 12th Entrapment; "Kissing Syndrome"

In many older patients, there is a shortening of the lumbar spine due to vertebral osteoporosis. Standing behind the patient in upright position, try to insert your hand between the lowest ribs and the iliac crest. Usually four fingers of your hand fit in this space. However, when the space is limited to one or no fingers and palpation is tender, you may expect the "kissing syndrome," since the two bones touch each other. Typically, patients complain of flank pain while walking or sitting, but the pain is relieved while lying in bed. To infiltrate the nerve(s), lay the patient on a firm surface, not in the bed, and put a firm pillow under the pelvis. In this case, infiltration should be "wide," as there is usually not one specific spot, but the whole nerve trajectory is involved (see Chap. 29).

Obturator Nerve Entrapment

This is a rare entrapment syndrome, but it may be significant for couple of reasons. This entrapment is not on the "rear" side of the body and likely has nothing to do with the deterioration hypothesis presented above. The nerve can be damaged or entrapped higher, due to L2–L3 pathology or pathology within the pelvis (see Chap. 47). Proper imaging is necessary before doing the infiltration. The patient may complain of pain on the medial aspect of the thigh. The tender spot can be identified on the side of pubic bone, where the nerve leaves the obturator foramen. This is one of the few infiltrations/blocks for which you should ask the help of a skilled anesthesiologist. He or she may need to perform the block at the root level.

Meralgia Paresthetica (MP)

Meralgia paresthetica (MP) is a well-known entrapment syndrome caused by dysfunction of the *lateral femoral cutaneous nerve* of the thigh (see Chap. 56) [12]. The patient suffers from pain on the outer thigh. Differentiation of MP and pain due to hip fracture is necessary, but is not usually very difficult. Patients suffering from MP will have palpable pain several centimeters medially and below the anterior superior iliac spine. Infiltration may not be easy, as the nerve is entrapped between ligaments, not bone.

Ramus Cutaneous of the 12th Intercostal Nerve and Ramus Cutaneous of the Iliohypogastric Nerve

This is one of the most common entrapment syndromes in palliative care. The two tender points are localized on the side of the iliac crest and are typically 7–8 cm from each other (see Chap. 50) [13]. Patients who are forced to lie on one side and patients with the sacral sores, pleural effusion, or hepatomegaly are especially vulnerable. The pain can be a horror for the patient, as he or she cannot change position in bed. The patients cannot sleep and typically use huge doses of opioids and/or sedatives. Often, both sites are tender simultaneously. Sometimes patients experience excruciating attacks of pain in the ipsilateral chest, since the intercostal nerves are all connected with each other in a peripheral network. To diagnose, the iliac crest needs to be carefully palpated from the rear to the front. Painful spots should be marked on the skin. Not infrequently, patients experience hyperalgesia in the whole lateral part of the thigh. They usually say "I have a hip pain," not infrequently after total hip prosthesis implantation. Infiltration of the anterior site (ramus cutaneous of the 12th intercostal nerve) is rarely a problem. A more posteriorly located site is sometimes more difficult to find with the needle, especially in overweight patients.

Superior Cluneal Nerve Entrapment

Superior cluneal nerves, of which there may be more than one, are the terminal ends of the posterior rami of lumbar spinal nerves (L1, L2, L3) (see Chap. 50). Typically, cluneal nerves pierce the *quadratus lumborum* muscle and cross the iliac crest some 7–8 cm from the median line at the level of the L5 vertebrae. Patients lying for a long time in bed in a

supine position and cachectic patients with prominent pelvic bones are prone to this mononeuropathy. The diagnosis is made by palpation of the iliac crest from the rear at both sides at the same time. Palpation of the lumbar muscles, as well as percussion of the spinous processes of the lumbar vertebrae, should be performed to exclude pathology there. Tender points frequently appear symmetrically on both sides, suggesting vertebral pathology. In this case, destabilization of a vertebral fracture (see above) should be avoided.

Sacral Nerve Entrapment

Sacral sensory branches (posterior rami) leave the sacrum through the dorsal sacral foramina. Patients may complain of severe burning pain at their sacrum. Typically, this type of pain occurs in patients with marked cachexia, especially with lung pathology. Patients with dyspnea tend to sit and sleep upright in bed, a position in which they compress the sacral cutaneous nerves. The pain can be excruciating, and it is rare that only one level or one side is involved. Infiltration is difficult, as too deep a penetration of the sacral foramen may damage other sacral nerves. Usually, the needle should penetrate not deeper than 0.5–1 cm from the foramen edge. Ideally, a skilled interventionalist under fluoroscopic control should do these infiltrations. As several places should be infiltrated at the same time, the maximum doses of bupivacaine and methylprednisolone will be easily reached, making a repeat procedure impossible. A caudal epidural might also provide simple relief.

Other Infiltrations That May Be Useful in Control of Pain or Other Symptoms

Occasionally, patients may experience severe pain in a small area of a scar. This may be a transected nerve growing into the scar tissue. Infiltration of this site with local anesthetic and methylprednisolone may be diagnostic and therapeutic.

Pain may be experienced at the insertions of the muscles, for example, at the shoulder blade. The most common pathology here is *levator scapulae syndrome*. The pain is localized in the upper median part of the scapula and increases with the shoulder movement. Palpation should involve not only the suprascapular fossa but all edges of the shoulder blade. Injection should first be directed to the shoulder blade bone, and the infiltration should be wide, with a considerable volume of injectate. Sometimes it needs to be infiltrated in the space between shoulder blade and ribs.

In some terminally ill patients, impaction of stool in the rectum may be a significant problem. Removal of the hard fecal masses (fecal stones) may be extremely unpleasant and painful, especially when the patient is suffering from anal fissure. In these patients, infiltrate through the sacral hiatus, just above the coccyx bone with a low volume caudal epidural. This is the only "midline" infiltration advised to do at the bedside, and only then in a desperate situation with a terminally ill patient. The patient is usually lying on the side, since lying on the abdomen is impossible for many patients. A 25 g needle is bent into a hook of 45° about 25 mm from the tip. This will avoid puncturing the dural sac, but, if fluid is aspirated, the procedure should be stopped immediately. Usually lidocaine is used instead of bupivacaine in this procedure. The use of steroids is not indicated here. After anesthetizing the lower sacral nerves, the anus opens painlessly for a period of 1–2 h, and the hard fecal masses can be removed from the rectum.

Conclusion

Infiltration of the peripheral nerves is a safe and effective method of pain control in terminally ill patients. Knowledge of anatomy and some skills are necessary; however, most of the procedures can be performed at the bedside, after disinfection of the skin. The principle here is that localized pain should be treated locally. Many patients, however, come to our attention on very high doses of opioids and other analgesics. In these cases, attention should be paid to appropriate dose reduction. Too rapid a reduction may precipitate withdrawal symptoms, while too slow a reduction may bring the danger of respiratory depression. Peripheral injections, of which palliative care physicians should learn not more than a dozen, are a welcome complementary technique to control pain and other symptoms.

References

1. Silbermann M. Current trends in opioid consumption globally and in Middle Eastern countries. J Pediatr Hematol Oncol Off J Am Soc Pediatr Hematol Oncol. 2011;33 Suppl 1:S1–5.
2. van den Beuken-van Everdingen MH, de Rijke JM, Kessels AG, Schouten HC, van Kleef M, Patijn J. Prevalence of pain in patients with cancer: a systematic review of the past 40 years. Ann Oncol. 2007;18:1437–49.
3. Zylicz Z. Noncancer related pain in daily practice. In: Hanna M, Zylicz Z, editors. Cancer pain. London: Springer; 2013. p. 191–202.
4. Zylicz Z, Twycross R. Opioid-induced hyperalgesia may be more frequent than previously thought. J Clin Oncol Off J Am Soc Clin Oncol. 2008;26:1564.
5. Childs SG. Double crush syndrome. Orthop Nurs. 2003;22:117–21; quiz 22–3.
6. Abdi S, Datta S, Trescot AM, et al. Epidural steroids in the management of chronic spinal pain: a systematic review. Pain Physician. 2007;10:185–212.
7. Manchikanti L. Role of neuraxial steroids in interventional pain management. Pain Physician. 2002;5:182–99.
8. Chambers WA. Nerve blocks in palliative care. Br J Anaesth. 2008;101:95–100.

9. Tumilty S, Munn J, McDonough S, Hurley DA, Basford JR, Baxter GD. Low level laser treatment of tendinopathy: a systematic review with meta-analysis. Photomed Laser Surg. 2010;28(1):3–16.
10. Zylicz Z, Haijman J. Suprascapular nerve entrapment: a neglected cause of shoulder pain in cachectic patients? J Pain Symptom Manage. 2000;20:315–7.
11. Dekhuijzen PN, Decramer M. Steroid-induced myopathy and its significance to respiratory disease: a known disease rediscovered. Eur Respir J. 1992;5:997–1003.
12. Grossman MG, Ducey SA, Nadler SS, Levy AS. Meralgia paresthetica: diagnosis and treatment. J Am Acad Orthop Surg. 2001;9:336–44.
13. Maigne JY, Lazareth JP, Guerin Surville H, Maigne R. The lateral cutaneous branches of the dorsal rami of the thoraco-lumbar junction. An anatomical study on 37 dissections. Surg Radiol Anat. 1989;11:289–93.

Complications of Nerve Entrapment Injection

13

Daniel Krashin and Natalia Murinova

Introduction

Most providers are ill at ease when confronted by a patient with a complication from treatment. Unfortunately, procedure textbooks seldom stress how to approach patient education in the context of sharing the information about potential complications and risks. The key to a good relationship with patients is to be a good listener. However rushed you may be, it is vital that the patient feels as though he has the whole of your time and attention during the interview. You must also be constantly aware that majority of your patients are extremely scared and anxious, even though their outward behavior may not show this. It is also essential to avoid the presumption that because the patient was referred to you for a procedure, they actually want to have the procedure or understand the risks entailed. Every effort should be made to put the patient at ease. Repeatedly checking the cell phone or watch, avoiding eye contact, or continuous interruptions all put the patient at a disadvantage and impede the development of trust and a sense of partnership and respect. Many patients and their loved ones dread needles, procedures, and pain, and they often do not understand neuroanatomy or anesthesia.

It is very important not to paint a rosy picture of the procedural outcome but to rather present a balanced picture of positive and negative outcomes. In order for the patients to believe the information presented to them, they need to feel cared for, and physicians need to convey their empathy. It is important to understand the emotional state of the patient and how they perceive risks. A patient about to undergo an intervention needs relevant information about those options that are considered most effective and safest for them. Naturally, every patient wishes to maximize benefit while minimizing risk, and patients often have trouble knowing how to interpret the information presented by their provider. When told that there is a small probability of a disastrous outcome, for example, some patients may discount it based on the small probability, while others will become fearful of even a small risk of a disaster. As the sophistication, diversity, and invasiveness of pain treatments continue to grow, there may be many options to present to patients. Often, our ability to quantify risk and benefit is limited by the paucity of well-designed, randomized clinical trials in the field, and it is important to share with patients the limitations of our predictions. While it may take years and many cases to demonstrate benefit, physicians owe it to their patients to reduce the known risks as much as possible.

Bleeding Complications

Any procedure involving injection carries a risk of *bleeding*. Within the field of regional anesthesia, the most dreaded complication is a spinal hematoma associated with neuraxial anesthesia. A deep injection has the potential to cause bleeding that may go undetected for some time with hematoma formation in a deep space of the body or within a muscle. This may occur in the absence of any *anticoagulation* treatment or bleeding diathesis; for example, pelvic hematoma has been described after ilioinguinal nerve block in otherwise healthy and uncoagulated patients [1]. This *hematoma* may cause nerve injury or impair the function of other vital organs.

Risk factors for bleeding complications include the intensity of anticoagulant effect (antithrombotic or antiplatelet therapy), increased age, female gender, history of gastrointestinal bleeding, concomitant aspirin use, and length of

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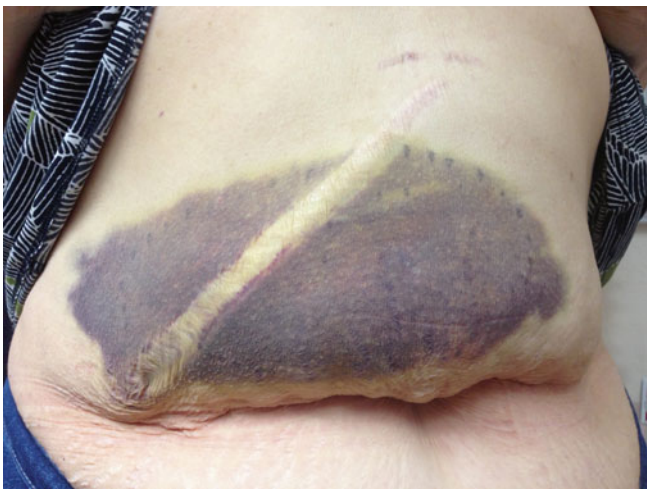

Fig. 13.1 Ecchymosis after injection (Image courtesy of Andrea Trescot, MD)

Table 13.1 Risk factors associated with bleeding complications in regional anesthesia

Modifiable risk factors	Non-modifiable risk factors
Anticoagulant therapy with warfarin	Increased age
Anticoagulant therapy with heparin	Female gender → osteoporosis → spinal stenosis
Patient's sensitivity to anticoagulants → 20 % of patients will have increased prothrombin time after single dose (3–5 mg) of warfarin	Comorbid liver disease → increased anticoagulant effect
Patient's diet may affect sensitivity to anticoagulants	Comorbid kidney disease
Herbal medications – garlic, ginkgo, ginseng (discontinue for at least 36 h prior to procedure)	Preexisting medical conditions
Traumatic needle or catheter placement	
Larger gauge needles	
Continuous catheter technique	

therapy [2]. It should be noted that newer antiplatelet drugs have variable half-lives, and no convenient blood test is available to assess their effect on hemostasis. Therefore, the guidelines for stopping these therapies prior to injection should be observed (see below). For elective procedures for the relief of pain, there should always be time to check with the patient's primary care provider, neurologist, or cardiologist who is prescribing these agents to obtain their agreement with the plan to suspend treatment.

American Society of Regional Anesthesia and Pain Medicine (ASRA) guidelines suggest that physicians apply the same recommendations for nerve blocks that apply to neuraxial injections regarding anticoagulation [3]. Some providers may choose to liberalize these recommendations when performing nerve injections in areas that are superficial or compressible. The blood may travel along fascial planes, with bruising appearing in areas distant to the injection site (Fig. 13.1). In any case, it is essential to monitor patients after injections and to counsel them on signs and symptoms of hematoma. Evaluation of a suspected hematoma may be possible with ultrasound imaging, but frequently CT or MRI imaging is required to fully assess the size and extent, and in rare cases, surgical evacuation may be necessary. Risk factors are discussed in Table 13.1.

Infection

Infectious complications may occur after any regional anesthesia technique, but the greatest concern is with procedures near the neuraxis. Bacterial infection in the neuraxis may present either as increased pain (Fig. 13.2), meningitis, or cord compression secondary to abscess formation. In peripheral injections, the risk of *infection* with single-injection technique is thought to be small compared to that associated with nerve catheters. The best prevention is rigorous adherence to aseptic technique in the performance of injections and administration of injections. The usage of sterile gloves, face masks, and hats and skin preparation are recommended [4]. The recent outbreak of *fungal meningitis* linked to epidural steroid injections using compounded steroids [5] reminds us that the medications themselves, as well their administration, must also be strictly aseptic [6]. Usually, the patient and provider can easily detect infectious complications at peripheral sites because of pain and erythema at the injection site (Fig. 13.3). In some cases, imaging may be needed to assess for abscess formation, and antibiotics are usually adequate treatment. However, the reported case of a patient dying due to *necrotizing fasciitis* arising from an axillary block is a reminder of the potentially disastrous consequences [7]. Below are sources of infection and possible risk factors for infectious complications.

Infectious Source

Exogenous – contaminated equipment or contaminated medication

Endogenous – seeding from a bacterial source to the needle or catheter site

Possible Risk Factors for Infectious Complications After Procedures

Sepsis
- Diabetes
- Immunocompromised patient
- Steroid therapy
- Localized bacterial colonization or infection
- Chronically ill patients
- Prolonged catheter in situ
- Breaks in aseptic technique
- Preexisting infection of the skin, soft issues, or spine at the insertion site
- Epidural placement (versus spinal)

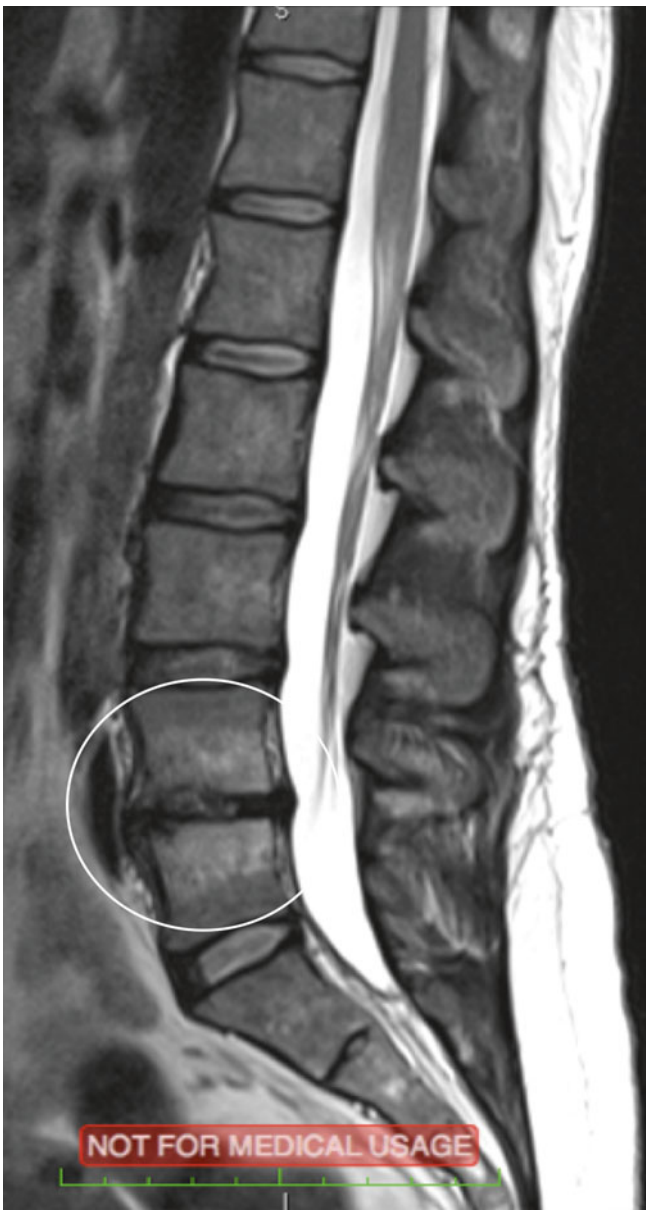

Fig. 13.2 Discitis after discogram presenting as increasing pain (Image courtesy of Andrea Trescot, MD)

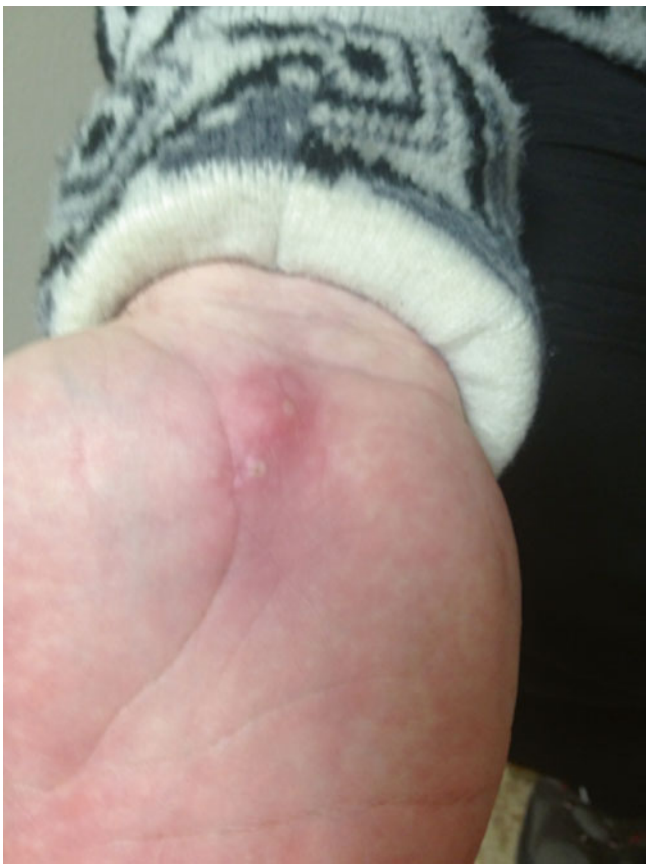

Fig. 13.3 Peripheral abscess after carpal tunnel injection (Image courtesy of Andrea Trescot, MD)

Nerve Injury

In the regional anesthesia literature, about 15 % of all patients receiving regional anesthesia will experience at least mild *paresthesias*, which typically resolve in days to weeks, though 0.2–1 % of patients will suffer significant neurologic damage [8]. This study was concluded in 2004, and improved technique and ultrasound availability have likely decreased this number. More recently, in 2012, a group of orthopedic surgeons surveyed their patients postoperatively and reported that 38.14 % of patients who underwent a preoperative peripheral nerve block for postoperative pain had neurogenic complaints, compared to 9.43 % of patients that did not receive a peripheral nerve block [9].

The *landmark-guided* ("blind") nerve block technique is considered more likely to be associated with needle contact with the nerve and thus has a greater potential for nerve injury; ultrasound-guided technique should reduce this risk by allowing direct visualization of the needle tip's position relative to the nerve. Long experience suggests that even penetration of the nerve by the needle tip does not always result in injury, but injection into the epineurium or perineurium may cause intraneural hemorrhage and neural lysis, particularly with high concentrations of local anesthetic or

mixtures containing epinephrine. Animal studies suggest that intrafascicular local anesthetic injections showed significant loss of large-diameter fiber, even at traditional concentrations [10].

Steroids themselves may also have a *neurotoxic* effect. According to a study by Mackinnon et al. [11], only intrafascicular injections caused damage; *dexamethasone* caused minimal damage, while *hydrocortisone* and *triamcinolone* caused widespread axonal and myelin degeneration.

When injecting near a nerve, a sudden severe increase in pain reported by the patient should be grounds for suspicion of a nerve injury and should be a reason to stop the injection and reassess. After the procedure, any abnormal sensation beyond the expected area of anesthesia should be noted and documented. Paresthesia or anesthesia in the distribution of the nerve that was treated may be addressed initially with reassurance and reassessment, but persistent dysesthesia, pain, or loss of motor function should be closely examined and documented. In unclear cases, referral to a neurologist or physiatrist for specific assessment and testing may be helpful to clarify the diagnosis, as well as to guide the rehabilitation efforts. Most peripheral nerve injuries resolve completely in time.

Local Anesthetic Systemic Toxicity (LAST)

Local anesthetics have a variety of toxicities, including *myotoxicity* and *myonecrosis*, usually minor though occasionally severe [12]. *Intravascular* injection of local anesthetics can cause systemic toxicity. The symptoms may appear immediately after injection or may occur with delayed onset and usually present in the central nervous system before the cardiovascular system. Central neurological symptoms include a metallic taste in the mouth, auditory changes, circumoral numbness, confusion, coma, and seizure; cardiovascular symptoms include tachycardia, hypertension, and arrhythmia. It is best to avoid complications by prevention, using excellent technique, and aspiration before injection, combined with attempts to use small total doses of local anesthetic, particularly bupivacaine. In some cases, it is advised to perform a test injection with epinephrine. It is incumbent on all providers who perform nerve block injections to recognize and treat LAST appropriately. A study of ultrasound-guided nerve blocks in regional anesthesia found LAST to be very uncommon in a series of over 12,000 procedures [13]. ASRA published a revised checklist for LAST management, which is freely downloadable at www.asra.com [14]. The standard of care now includes airway management to prevent hypoxia and acidosis, treatment of seizures, and early use of lipid infusions [15]. However, the volume of local anesthesia used in pain management is usually very low (less than 2 ccs), and therefore the risk of LAST should be exceedingly low, unless the local anesthetic is injected directly into a cerebral artery (carotid or vertebral).

Vascular Compromise

Recently, especially after transforaminal injections, there has been an increase in devastating spinal injuries attributed to *particulate steroids* [16]. Preprocedure evaluation of imaging [17] and the use of real-time fluoroscopy, digital subtraction angiography (Fig. 13.4), blunt-tipped needles, and particulate-free steroids have been advocated as techniques to avoid catastrophic vascular injections.

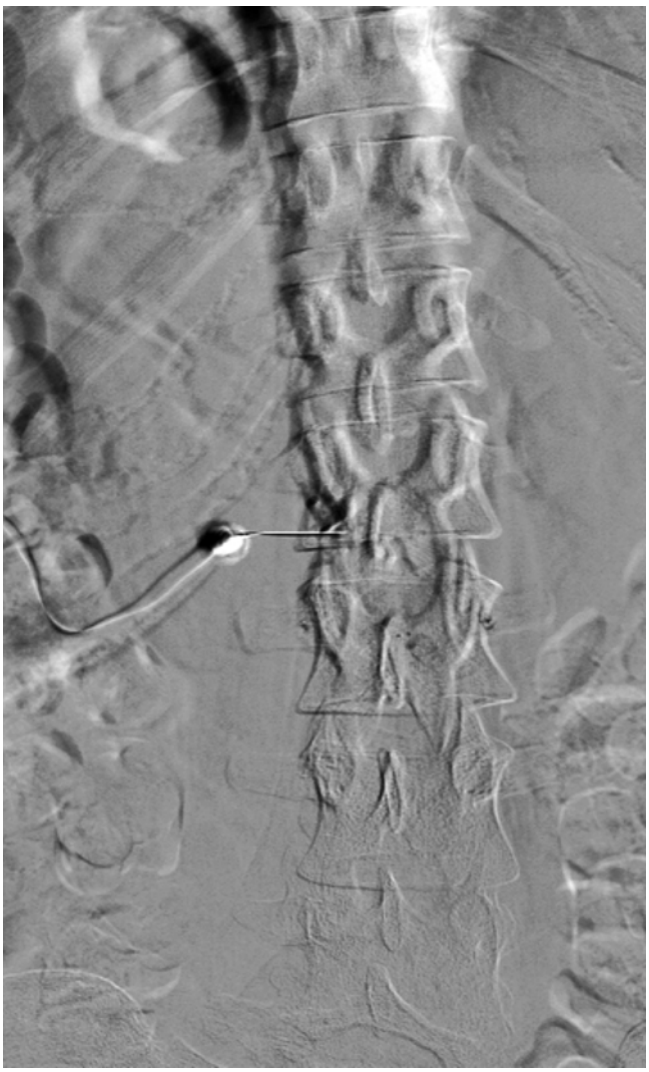

Fig. 13.4 Vascular needle location seen with digital subtraction angiography (Image courtesy of Andrea Trescot, MD)

Steroid Effects

Corticosteroids are frequently included in nerve block injections in order to decrease local inflammation and promote healing. Corticosteroids vary widely in their duration and potency of effect and can have both local and systemic effects. Common errors in administering steroids include giving excessive doses, injecting depot forms into inappropriate areas, and giving doses too frequently.

The literature suggests that more superficial injections that involve the layer of subcutaneous fat and injections of triamcinolone are more likely to be associated with atrophy of the adipose tissue and skin than injections which are deeper and are done with more water-soluble corticosteroids (Fig. 13.5). However, occipital nerve blocks are a particularly common cause of these problems, possibly because the layers of the skin and fat are so delicate at that region [18].

Although steroids are an integral component of the treatment of peripheral nerve entrapment, a microscopic study of nerves after steroid injections showed histologic evidence of neurotoxicity [11]. However, only intrafascicular injections caused damage, and the amount of damage was dependent upon the actual steroid used. Dexamethasone, as a non-particulate steroid, caused minimal damage to the vasa nervorum, while triamcinolone and hydrocortisone caused widespread axonal and myelin degeneration. These findings are strikingly similar to the damage seen recently with intravascular injections during transforaminal epidural steroid injections.

Neuritis from Neurolysis

Radiofrequency nerve ablation (RFA) has a small but notable rate of complications, including persistent post-procedure pain, neuroma formation, dysesthesia, impaired sensation, and motor impairment. These outcomes are mainly anticipated with conventional RFA, as pulsed RFA does not result in a lesion or motor impairment. The incidence of neuropathic pain after cooled RFA in a retrospective chart review of 193 lesions revealed only a 0.7 % rate per lesion, and these were all transient [19]. However, as described by Bogduk [20], there is always the risk of inducing anesthesia dolorosa by cutting or traumatizing these nerves.

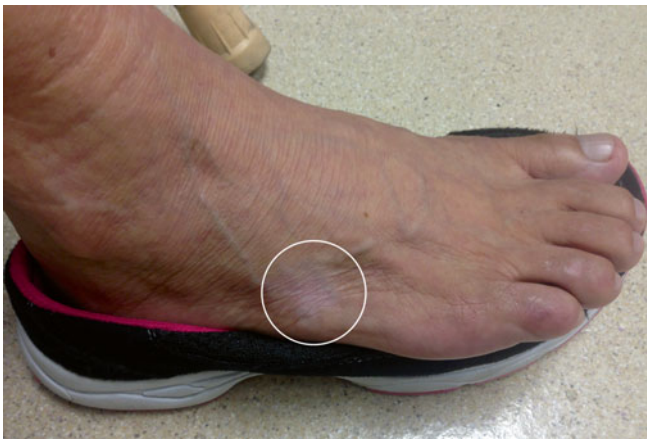

Fig. 13.5 Skin atrophy after superficial steroid injection (Image courtesy of Andrea Trescot, MD)

Chemical neurolysis using substances such as *phenol* and *alcohol* results in powerful neurolysis; however, this is not usually permanent, and the duration may be unpredictable. There are case reports of paraplegia resulting from phenol neurolysis of peripheral nerves near the spine, including celiac plexus block [21] and intercostal block [22]. Similar misadventures have occurred when using alcohol for celiac plexus blocks [23]. For safety, it is important to recognize that aqueous phenol solutions and alcohol can spread within the body and tend to settle or rise, respectively, due to their specific gravities versus body fluids. Neurolytic substances can also spread to nerves other than the target.

The primary complications of peripheral neurolysis are motor deficits when a mixed motor and sensory nerve is treated and the risk of developing dysesthesia or deafferentation pain after treatment which can, in some cases, be more disabling that the initial pain complaint. Pain conditions such as *neuritis* and *deafferentation pain* are rare but may be more common when alcohol is used to block myelinated nerves; however, exact rates are not known. In peripheral neurolysis of the tibial nerve performed for spasticity, a 10 % rate of dysesthesia was reported [24].

Burns

Patients often use heating pads for chronic pain, but sleeping on a heating pad can cause second- and third-degree *burns* (Fig. 13.6). The increased use of radiofrequency lesioning, especially with the higher-powered cooled RF systems, can result in increased energy delivered to the dispersive pad, which can cause burns (Fig. 13.7) [25]. Making sure that the dispersive pad is well applied to the skin on a non-hairy area should decrease the risk of burns.

Pneumothorax

Any injection around the chest and lower cervical region can be associated with a potential *pneumothorax*. The American Society of Anesthesiologists Closed Claim Project results for 2014 reviewed 5475 malpractice claims. Pneumothorax was the main complaint in 51 % of all block claims and the most common complication of trigger point injections and "non-epidural injections" [26].

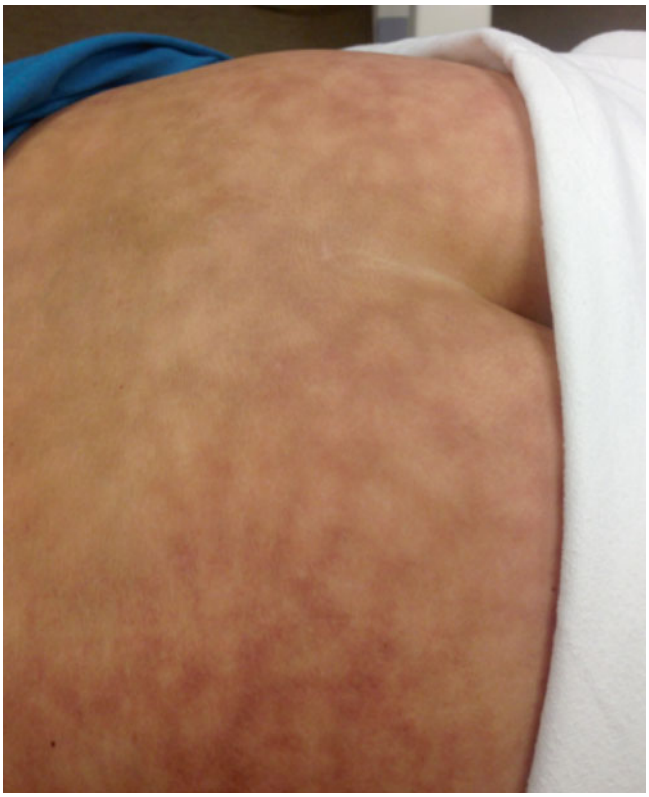

Fig. 13.6 Heating pad burns (Image courtesy of Andrea Trescot, MD)

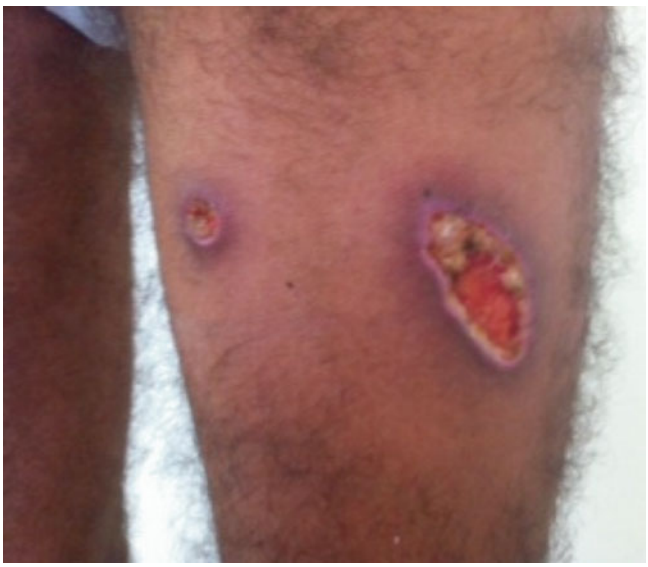

Fig. 13.7 Third-degree burn after radiofrequency lesioning (Image courtesy of Charles Amaral de Oliveira)

Conclusion

The list of potential complications is daunting, and it is surprisingly difficult to demonstrate that the risks of these complications have decreased over time. Therefore, it is important that the provider recognizes the risk of these complications and also appreciates factors, which may make the procedure more risky in the specific patient for whom it is planned. The careful assessment of risks will enable a determination of the risk/benefit ratio to guide the clinical decision, as well as provide accurate information to the patient who is to undergo the treatment.

Recognition of potential complications may also help prevent them, by spurring efforts to modify and avoid risk. The wider field of anesthesia has made great strides in this regard with standardizing treatments and procedures and developing evidence-based guidelines. Some of these, such as the ASRA guidelines on regional anesthesia and anticoagulation, are of direct relevance to pain practice and have been mentioned in the appropriate sections. Individual providers and groups of providers can help this process by participating in registries and by publishing their own experiences of complications.

In addressing the possibility of complications, the provider must seek to prevent them through using excellent technique, reducing variance as much as possible through standardization of the technique, through equipment and approach, and through coordination with the rest of the treatment team. In addition, the provider must have a robust system in place to ensure that the patient is counseled on recognizing serious complications and is reassessed prior to discharge and that emergency care is available in case of delayed onset of symptoms of a complication.

References

1. Vaisman J. Pelvic hematoma after an ilioinguinal nerve block for orchialgia. Anesth Analg. 2001;92(4):1048–9.
2. Schulman S, Beyth RJ, Kearon C, Levine MN. Hemorrhagic complications of anticoagulant and thrombolytic treatment. American College of Chest Physicians Evidence-Based Clinical Practice guidelines. Chest J. 2008;133(6 suppl):257S–98.
3. Horlocker TT, Wedel DJ, Rowlingson JC, Enneking FK, Kopp SL, Benzon HT, et al. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine evidence-based guidelines. Reg Anesth Pain Med. 2010;35(1):64–101.
4. Hebl JR, Horlocker TT. You're not as clean as you think! The role of asepsis in reducing infectious complications related to regional anesthesia. Reg Anesth Pain Med. 2003;28(5):376–9.
5. Kerkering TM, Grifasi ML, Baffoe-Bonnie AW, Bansal E, Garner DC, Smith JA, et al. Early clinical observations in prospectively followed patients with fungal meningitis related to contaminated epidural steroid injections. Ann Intern Med. 2013;158(3):154–61.
6. Pettit AC, Kropski JA, Castilho JL, Schmitz JE, Rauch CA, Mobley BC, et al. The index case for the fungal meningitis outbreak in the United States. N Engl J Med. 2012;367(22):2119–25.
7. Nseir S, Pronnier P, Soubrier S, Onimus T, Saulnier F, Mathieu D, et al. Fatal streptococcal necrotizing fasciitis as a complication of axillary brachial plexus block. Br J Anaesth. 2004;92(3):427–9.
8. Borgeat A, Blumenthal S. Nerve injury and regional anaesthesia. Curr Opin Anaesthesiol. 2004;17(5):417–21.

9. Mellecker C, Albright J, Clark R. Peripheral nerve blocks and incidence of post-operative neurogenic complaints and pain scores. Iowa Orthop J. 2012;32:83–9.
10. Farber SJ, Saheb-Al-Zamani M, Zieske L, Laurido-Soto O, Bery A, Hunter D, et al. Peripheral nerve injury after local anesthetic injection. Anesth Analg. 2013;117(3):731–9.
11. Mackinnon SE, Hudson AR, Gentili F, Kline DG, Hunter D. Peripheral nerve injection injury with steroid agents. Plast Reconstr Surg. 1982;69(3):482–90.
12. Hogan Q, Dotson R, Erickson S, Kettler R, Hogan K. Local anesthetic myotoxicity: a case and review. Anesthesiology. 1994;80(4): 942–7.
13. Sites BD, Taenzer AH, Herrick MD, Gilloon C, Antonakakis J, Richins J, et al. Incidence of local anesthetic systemic toxicity and postoperative neurologic symptoms associated with 12,668 ultrasound-guided nerve blocks: an analysis from a prospective clinical registry. Reg Anesth Pain Med. 2012;37(5):478–82.
14. Neal JM, Mulroy MF, Weinberg GL. American Society of Regional Anesthesia and Pain Medicine checklist for managing local anesthetic systemic toxicity: 2012 version. Reg Anesth Pain Med. 2012;37(1):16–8.
15. Neal JM, Bernards CM, Butterworth IV JF, Di Gregorio G, Drasner K, Hejtmanek MR, et al. ASRA practice advisory on local anesthetic systemic toxicity. Reg Anesth Pain Med. 2010;35(2): 152–61.
16. Benny B, Azari P, Briones D. Complications of cervical transforaminal epidural steroid injections. Am J Phys Med Rehabil. 2010;89(7):601–7.
17. Nishio I. Cervical transforaminal epidural steroid injections: a proposal for optimizing the preprocedural evaluation with available imaging. Reg Anesth Pain Med. 2014;39(6):546–9.
18. Shields K, Levy M, Goadsby P. Alopecia and cutaneous atrophy after greater occipital nerve infiltration with corticosteroid. Neurology. 2004;63(11):2193–4.
19. Stolzenberg D, Gordin V, Vorobeychik Y. Incidence of neuropathic pain after cooled radiofrequency ablation of sacral lateral branch nerves. Pain Med. 2014;15(11):1857–60.
20. Bogduk N. The clinical anatomy of the cervical dorsal rami. Spine. 1982;7(4):319–30.
21. Galizia E, Lahiri S. Paraplegia following coeliac plexus block with phenol. Br J Anaesth. 1974;46(7):539–40.
22. Kowalewski R, Schurch B, Hodler J, Borgeat A. Persistent paraplegia after an aqueous 7.5% phenol solution to the anterior motor root for intercostal neurolysis: a case report. Arch Phys Med Rehabil. 2002;83(2):283–5.
23. Dongen R, Crul B. Paraplegia following coeliac plexus block. Anaesthesia. 1991;46(10):862–3.
24. Kirazli Y, On AY, Kismali B, Aksit R. Comparison of phenol block and botulinus toxin type A in the treatment of spastic foot after stroke: a randomized, double-blind trial. Am J Phys Med Rehabil. 1998;77(6):510–5.
25. Walega D, Roussis C. Third-degree burn from cooled radiofrequency ablation of medial branch nerves for treatment of thoracic facet syndrome. Pain Pract. 2014;14(6):e154–8.
26. Fitzgibbon DR, Posner KL, Domino KB, Caplan RA, Lee LA, Cheney FW. Chronic pain management: American Society of Anesthesiologists Closed Claims Project. Anesthesiology. 2004; 100(1):98–105.

Part II

Headache

Esther Rawner and Matthew P. Rupert

Introduction

Headaches are one of the most prevalent neurologic disorders. They affect 28 million people in the USA [1] and account for millions of dollars in medical costs, lost labor costs, and associated burdens on our society. As with many medical events, headaches are complex, and our understanding of them is an evolving science. Although often seen as the primary pathology, headaches are fundamentally a symptom and have numerous proven origins and causes. There are many types of headaches, and no two headaches are the same. Despite the individual variations, one can see distinctive patterns of pain that can be easily recognized over and over again. Using pattern recognition, many headaches can be quickly diagnosed, treated, and, with proper care, put to an end entirely.

Collectively, headache patterns have provided practitioners and researchers with basic guidelines to further understand and treat this often-debilitating condition. One of the commonly followed classification schemes for headaches is the International Classification of Headache Disorders (ICHD) [2]. This classification has defined many types of head pain from the viewpoint that headaches are either primary or secondary. The ICHD focuses on the categorization and description of syndromes of headache patterns. While thorough and extremely useful, this lexicon only generally describes certain peripheral nerve contributions to headaches as "Other Terminal Branch Neuralgias." The ICHD does little to delineate the specific pain patterns of most of the individual extracranial nerve pathologies in the head and neck. The study of the origins of pain, as a function of extracranial peripheral nerve entrapments and their dysfunction, reveals a great deal of overlap between many of the ICHD-defined headaches and the nerve entrapments potentially causing these pain patterns [3]. The study of headaches, as a subspecialty in the field of neurology, incorporates evidence from closely associated disciplines such as pain management, which become an invaluable tool in the growing repertoire available to headache practitioners.

Here, in this section, we propose that headaches, including migraines, are not always an isolated intracranial phenomenon. Rather, they can be an interaction between the intracranial components of the brain and the extracranial nerves. In 2003, Evans and Pareja [4] proposed the term "epicrania" for headaches triggered by extracranial causes. Plastic surgeons at the beginning of this century noted the relief of migraines with both corrugator muscle resection [5] and injection of botulinum toxin [6], suggesting a peripheral headache trigger. Headache specialists repeatedly see patients with severe disabling migrainous headaches after a head or neck injury, and these headaches respond only modestly to migraine-specific pain medications. These headaches may have an extracranial origin, and the pain is likely a result of a peripheral nerve irritation and entrapment in the head or neck that was sustained during the injury. Moreover, this peripheral nerve irritation may have secondarily activated the migraine centers in the brain to bring about the associated migrainous symptoms. The treatment, therefore, would be to primarily inhibit the nerve irritation utilizing interventional pain techniques and thus turn off the pain origin, which subsequently turns off the activated migraine centers.

A common example of a simple nerve entrapment is seen with pathology of the posterior auricular nerve (Chap. 16). These patients usually have a diagnosis of "migraine," and they suffer from a severe migraine headache whenever they wear tight glasses. Taking off the glasses will give almost immediate relief of the pain and associated symptoms. Thus, the glasses have entrapped the posterior auricular nerve, an extracranial nerve entrapment, and this nerve entrapment has activated the migraine centers. That activation triggers a typical severe, throbbing migraine headache with the associated symptoms of light sensitivity, sound sensitivity, nausea, or vomiting, meeting the IHCD classification of a "migraine." Removing the primary stimulus will terminate both the headache and the associated symptoms. This immediate response is what we see when any of the entrapped nerves in the head and neck are injected with local anesthetic, yielding almost immediate relief of the pain and of the associated symptoms.

The phenomenon of nerve entrapment may happen to all major nerves of the head and neck, leading to severe disabling headaches. Calandre et al. [7] studied the specific areas of the scalp that could be manually palpated to elicit migraines. They found that 42 % of the triggering sites were in the temporal area, and 33.4 % were in the suboccipital area. Thus, it is important to understand these extracranial nerve entrapments and their contribution to headache pain, as one is unable to end the suffering of these headache patients without a proper diagnosis.

Recent studies have challenged the traditional teaching of exclusive centrally mediated activation of migraine headaches, suggesting instead a major contribution from the peripheral trigger sites [5]. Four such peripheral trigger sites have been described. Three of these areas (frontal, temporal, and occipital) correspond to a particular sensory nerve (supraorbital/supratrochlear, zygomaticotemporal, and greater occipital, respectively) that are thought to be the cause of migraine symptoms originating from those locations. The fourth trigger point corresponds to the nasal septum and turbinates [8].

When diagnosing these nerve entrapments, one must not only be proficient at general neurologic examination, but also be adept at palpating and localizing nerve entrapments, and recognizing pain patterns of these peripheral nerves of the head and neck. We will be discussing the anatomy, diagnostic techniques, and interventional treatment options for several peripheral nerve entrapments contributing to headaches in this section, while Part III focuses on some of the nerves that cause the related symptoms of face and neck pain.

References

1. Janis JE, Hatef DA, Ducic I, Ahmad J, Wong C, Hoxworth RE, et al. Anatomy of the auriculotemporal nerve: variations in its relationship to the superficial temporal artery and implications for the treatment of migraine headaches. Plast Reconstr Surg. 2010;125(5):1422–8.
2. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition (beta version). Cephalalgia. 2013;33(9):629–808.
3. Dalkara T, Zervas NT, Moskowitz MA. From spreading depression to the trigeminovascular system. Neurol Sci. 2006;27 Suppl 2:S86–90.
4. Evans RW, Pareja JA. Expert opinion. Supraorbital neuralgia. Headache. 2009;49(2):278–81.
5. Guyuron B, Varghai A, Michelow BJ, Thomas T, Davis J. Corrugator supercilii muscle resection and migraine headaches. Plast Reconstr Surg. 2000;106(2):429–34; discussion 35–7.
6. Gobel H, Heinze A, Heinze-Kuhn K, Jost WH. Evidence-based medicine: botulinum toxin A in migraine and tension-type headache. J Neurol. 2001;248 Suppl 1:34–8.
7. Calandre EP, Hidalgo J, Garcia-Leiva JM, Rico-Villademoros F. Trigger point evaluation in migraine patients: an indication of peripheral sensitization linked to migraine predisposition? Eur J Neurol Off J Eur Federat Neurol Soc. 2006;13(3):244–9.
8. Dash KS, Janis JE, Guyuron B. The lesser and third occipital nerves and migraine headaches. Plast Reconstr Surg. 2005;115(6):1752–8; discussion 9–60.

Supraorbital Nerve Entrapment

14

Rafael Justiz and Andrea M. Trescot

Introduction

Supraorbital neuralgia (SN), first described by Beyer in 1949 [1], is a rare cause of extracranial headache, with a reported incidence of 4 % [2]. It is often characterized by continuous or intermittent forehead pain in the area innervated by the distribution of the *supraorbital nerve* (SON), often presents unilateral with tenderness over the *supraorbital notch* or over the distribution of the nerve, and responds to local anesthetic blockade or ablation of the nerve [3]. The etiology of SN can be multifactorial, with the most common being trauma such as hitting a windshield with the forehead or receiving a blow/punch to the face leading to a nerve entrapment. Other causes include tight-fitting goggles or motorcycle helmets, sinus infections leading to inflammation over the region where the nerve travels, and fluid retention [4]. Entrapment of the nerve often occurs in the supraorbital notch, but it can also occur above the notch after trauma. Treatment for supraorbital neuralgia involves removing any provoking stimulus (better-fitting goggles or helmets), local anesthetic and steroid nerve block, medications, cryoneuroablation, neuromodulation, and surgical decompression [2, 5].

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Clinical Presentation

SN is defined by the International Headache Society (IHS) as a localized headache in the forehead region with the following criteria: paroxysmal or constant pain in the region supplied by the supraorbital nerve [9]. SN has multiple etiologies (Table 14.1) and can sometimes be confused with migraine-type headaches, cluster headaches, or sinusitis. It presents as supraorbital, retro-orbital, and/or forehead pain, unilateral or bilateral, sharp and/or throbbing (Fig. 14.1). It may be spontaneous or the result of palpation of the supraorbital area. Occasionally, patients may complain of blurred vision, nausea, and photophobia, thereby confusing the diagnosis [10]. Triptans can help but usually only temporary, presumably by vasoconstricting the blood vessels traveling with the nerve, thereby decreasing the entrapment. Symptoms include continuous or intermittent unilateral pain above the eye that occasionally radiates distally along the SON to the vertex of the skull.

The SON can also serve as a trigeminal "trigger zone" for tic douloureux. The symptoms may worsen with time, especially if they are associated with progressive scarring around the nerve. Headache symptoms are often exacerbated with excessive squinting or frowning (when the orbicularis oculi entraps the nerve), direct pressure (such as with swimming goggles), the head held lower than the heart (because of increased blood flow), or fluid retention associated with pre-menses and excessive salt consumption (which causes swelling of the nerve in its canal). All these factors can lead to further compression of the nerve. Like *hemicrania continua* and *cluster headaches*, the headache from supraorbital nerve entrapment can have associated ipsilateral conjunctival injection, lacrimation, nasal congestion, rhinorrhea, and frequent "attacks." In fact, hemicrania continua has been successfully treated with supraorbital and supratrochlear nerve injections [11]. And as with migraines, there may be associated photophobia, phonophobia, nausea, and emesis. The pain is almost

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Table 14.1 Occupation/exercise/trauma history relevant to supraorbital nerve entrapment

Trauma	Hitting the windshield, punched in the face, supraorbital fracture [6], previous surgery
Compression	Swimming goggles [4]
	Motorcycle helmet
Infection	Sinus infection (especially frontal)
Calcium deficiency	Possible orbicularis spasm [7]
Neoplasm, chemotherapy, radiation therapy	
Nerve enlargement	Hansen's disease [8]
Relationship to menstrual cycle and salt intake	Water retention may lead to swelling near supraorbital foramen

Fig. 14.1 Patient pain complaint from supraorbital nerve entrapment (Image courtesy of Andrea Trescot, MD)

always unilateral and can be severe enough to lead to suicidal thoughts [12].

Anatomy

The SON is one of five peripheral branches of the ophthalmic division of the t*rigeminal nerve* (cranial nerve V). The *trigeminal ganglion* (TG) gives rise to three divisions, *ophthalmic* (V1), *maxillary* (V2), and *mandibular* (V3). While the V2 and V3 divisions branch downward, the V1 division branches superior and medially from the TG (Table 14.2) until it enters the orbit posteriorly via the *superior orbital fissure* (Fig. 14.2). The nerve then divides into three, continuing on as the *lacrimal nerve*, the *nasociliary nerve*, and the *frontal nerve*. The frontal nerve then splits into the *supraorbital nerve* (SON), which exits the orbit with the *supraorbital artery* via the *supraorbital notch*, and the *supratrochlear nerve* (STN), which exits with the *supratrochlear artery* via the *supratrochlear groove or notch* and runs medially in a small groove at the junction of the ocular ridge and the nares (Fig. 14.3). The SON then branches into a medial and lateral branch over the forehead (Fig. 14.4). Separate exits for the medial and lateral SON branches were observed in 8 of the 28 nerves (29 %) examined by Anderson et al. [13]. The medial branch continues superiorly up to the vertex of the skull, while the lateral branch moves superior-laterally; both branches supply sensory fibers to the forehead, upper eyelid, and anterior scalp, up to the lambdoidal suture (Fig. 14.5), and both were associated with arteries (often intimately entwined). Janis et al. found that 73 % of the specimens they examined had the SON passing through the corrugator muscle [14].

Table 14.2 Supraorbital nerve anatomy

Origin	Trigeminal ganglion
General route	V1 through the supraorbital fissure, through the supraorbital notch, onto the forehead
Sensory distribution	Forehead, upper eyelid, nose, and anterior scalp
Motor innervation	None
Anatomic variability	Separate exits for the medial and lateral branches
Other relevant structures	Supratrochlear notch (vs. supratrochlear foramen), supratrochlear nerve

The STN supplies sensation to the skin and soft tissues of the lower medial forehead, the upper eyelid, and the glabella, as well as the conjunctiva. The STN runs medially along the roof of the orbit, exiting between the supraorbital notch and the trochlea. A supratrochlear artery was observed in 30 of the 50 orbits (72 %) evaluated by Janis et al. [14]. In three cadavers, the STN shared a notch with the SON, and in 84 % of the cadavers, the STN entered the corrugator muscle.

The supraorbital notch may be covered by a small ligament (creating a foramen), which can become thickened or calcified. Fallucco and colleagues called this ligament-covered foramen a "miniature carpal tunnel" [15]. In dissections done by Anderson et al. [13], 20 of 28 nerves (71 %) exited through the foramina (with bony or connective tissue bridges) instead of notches. Fallucco et al. [15] dissected 50 SONs and found that 43 of the 50 (86 %) had fascial bands across the notch. These were further divided into "simple" bands (22 of 43 or 51.2 %), "partial bony" (13 of 43 or

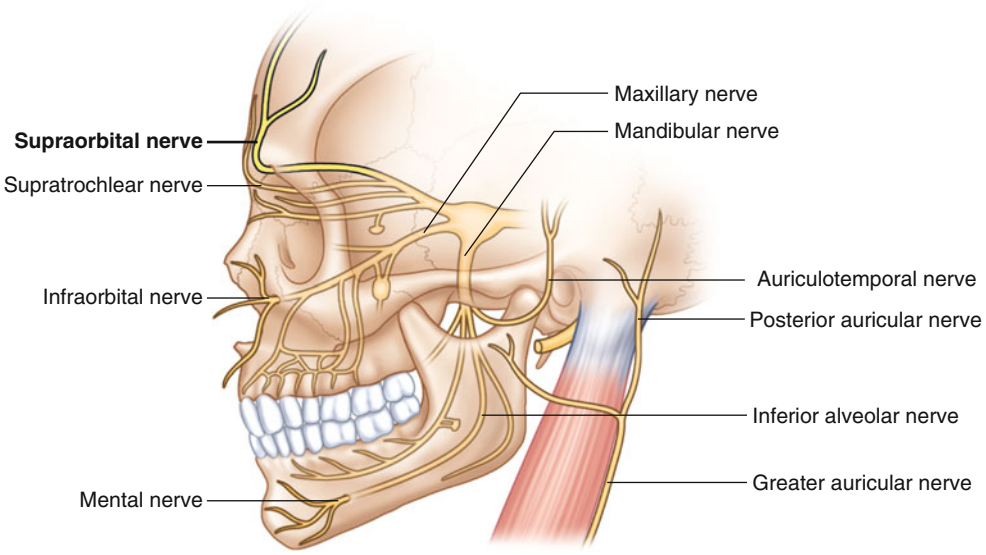

Fig. 14.2 Supraorbital anatomy (Image courtesy of Springer)

Fig. 14.3 Dissection of the supraorbital rim showing the supraorbital and supratrochlear nerves and arteries (Dissection by Gabor Balsa, MD, Semmelweis University, Laboratory for Applied and Clinical Anatomy, Budapest, Hungary; image courtesy of Andrea Trescot, MD)

30.2 %), and "septums," which could be horizontal (4 of 43 or 9.3 %) or vertical (4 of 43 or 9.3 %). There was a high degree of variability, and the authors noted that the anatomic difference and variable diameters could account for unilateral migraine symptoms.

Entrapment

Entrapment occurs primarily at the supraorbital notch as the nerve leaves the orbit, passing through the supraorbital notch, the orbicularis oculi muscle, the glabellar muscle, and the corrugator muscle, and it can be exacerbated by frowning and squinting (perhaps the reason that some "migraines" respond to the use of eyeglasses or *botulinum toxin*). Tight-fitting goggles [4] or an anesthesia mask [16] can compress the nerve. There can also be trauma to the nerve from surgery in the frontal region [16]. This headache will often worsen with menses or increased salt intake, perhaps by causing swelling of the nerve in its canal. In addition, there can be entrapment by scarring [16] as well as by thickening or calcification of the supraorbital ligament.

Physical Exam

A thorough history should first be obtained to identify any previous trauma or inciting events that could lead to SN. This is then followed by a visual inspection and physical examination of the forehead, eyebrow, nasal region, and supraorbital foramen. There will be tenderness to palpation over the supraorbital foramen with possible radiation of

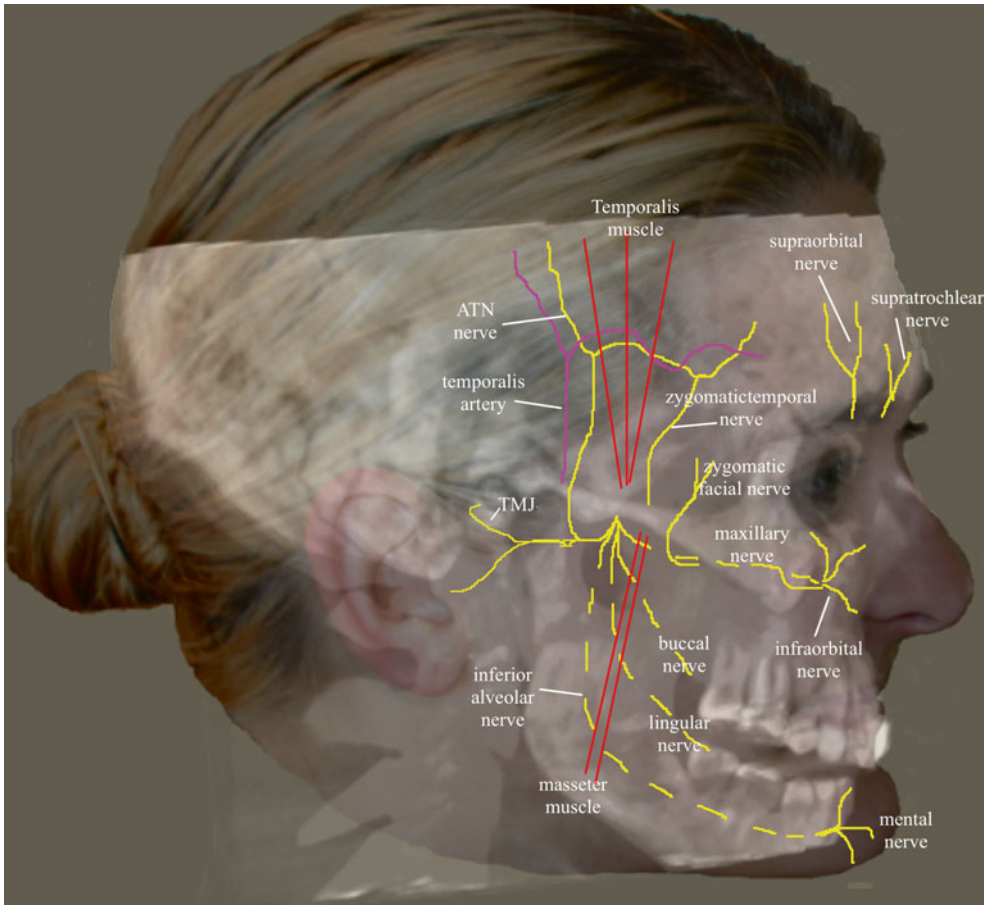

Fig. 14.4 Nerves of the face (Image created by Andrea Trescot, MD)

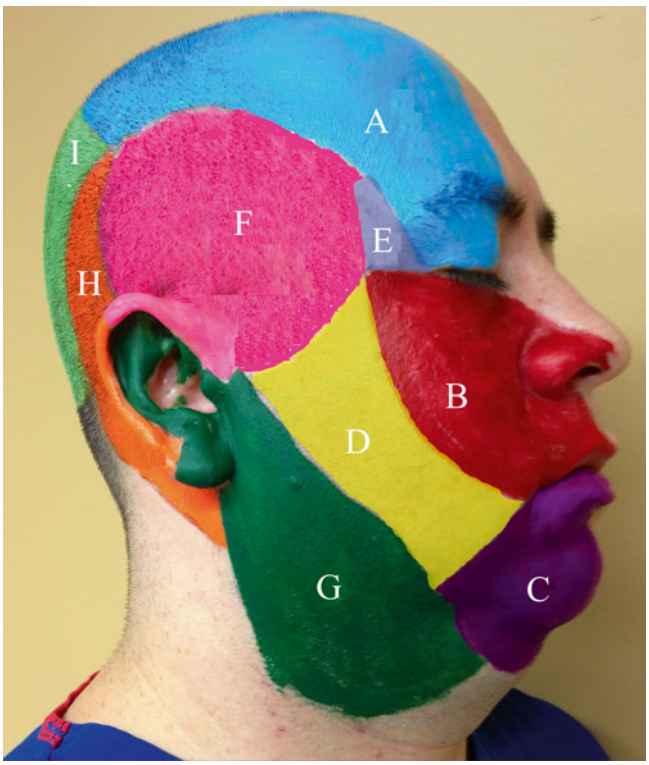

Fig. 14.5 Sensory areas of the trigeminal and cervical nerve branches: *A* supraorbital nerve, *B* infraorbital nerve, *C* mental nerve, *D* buccal nerve, *E* lacrimal nerve, *F* auriculotemporal nerve, *G* superficial cervical plexus, *H* posterior auricular nerve/great auricular nerve, *I* occipital nerve (Image courtesy of Terri Dallas-Prunskis, MD)

Fig. 14.6 Supraorbital examination (Image courtesy of Andrea Trescot, MD)

symptoms along the nerve distribution of the affected side. Support the head with the non-examining hand, then palpate over the orbital rim, feeling for the supraorbital notch (Fig. 14.6) (Video 14.1). Paresthesias should replicate the pain complaints, and with careful palpation, the examiner should be able to identify the slim, string-like vertical SON structure. Move the thumb medially to feel the supratrochlear groove.

In 2005, Sjaastad and colleagues reported on the supraorbital exam of 1,828 of the inhabitants of Vågå, Norway [12]. Only 24 persons (1.3 %) did not have a palpable SON; 98 % of the individuals had a palpable SON, and 5.4 % of the inhabitants (106 of the 1,828 people examined) had increased tenderness over the supraorbital notch. Twelve of these had a clinical presentation of SN; ten of the 12 had a history of forehead trauma.

Differential Diagnosis

The etiology of SN is often elicited clinically. Although previous trauma is common, other potential causes (see Table 14.1) must be considered. The differential diagnosis is extensive and easily confused (see Table 14.3). Additional causes include a neoplastic process (e.g., invasion of the bone by tumor, nerve compression) or complications of cancer treatment (e.g., radiation fibrosis, chemotherapy-induced neuropathy). The various tests for SON entrapment are listed in Table 14.4. Beyer felt that tenderness over the supraorbital and supratrochlear notch was "pathognomonic" [1].

Hemicrania continua (HC) is a headache that by definition needs to have a complete response to indomethacin, but Guerrero et al. [19] described 14 HC patients who underwent supraorbital and/or occipital nerve blocks (see Chap. 17) due to *indomethacin intolerance*. Nine of these patients noted total or partial improvement from 2 to 10 months, and the authors recommended that HC patients be examined for possible injection therapy.

Table 14.3 Differential diagnosis of supraorbital pain

Condition	Potential distinguishing features
Chronic migraine	No response to supraorbital nerve blocks
Shingles	Lesions in a V1 distribution (if no lesions, consider checking herpes varicella-zoster IgM titers)
Tic douloureux (trigeminal neuralgia)	Spontaneous, lancinating pain, trigger zone; very rarely on the forehead [12]
Sinus infection (especially frontal sinus)	Fever, purulent discharge, clouding of sinus on X-ray. MRI or CT can confirm sinus infiltration
Paget's disease	X-rays show bony overgrowth of the supraorbital foramen
Postherpetic neuralgia	History of shingles
Trochleitis	CT shows soft tissue enhancement at the trochlea [17]
Hemangioma or dilatation of the supraorbital artery	MRI, MRA, or arteriogram will show dilated vessel [18]
Intracranial tumor	MRI or CT will show mass
Temporal arteritis	Systemically ill, elevated ESR
Hemicrania continua	Usually improves with carbamazepine and indomethacin

Table 14.4 Diagnostic tests for supraorbital neuralgia

	Potential distinguishing features
Physical exam	Tenderness over the supraorbital notch that replicates pain
Diagnostic injection	Resolution of pain after diagnostic injection at the supraorbital notch
Ultrasound	Enlarged nerve at notch [20]
MRI	The supraorbital artery can be identified with high resolution MRI [21]
Arteriography	May show dilated supraorbital artery
X-ray	Not useful for diagnosis
Electrodiagnostic studies	Blink reflex [22]

Identification and Treatment of Contributing Factors

The most common cause of pain along the supraorbital nerve is an entrapment of that nerve. SON entrapment can occur from multiple reasons (see Tables 14.1 and 14.3), but trauma may be the most common inciting event. On an interesting note, Janiri et al. [7] have been studying the relationship between "*chronic constitutional tetany*" and supraorbital

neuralgia. They have identified a group of patients with neuromuscular hyperexcitability, anxiety, dysautonomia, and supraorbital/oculofrontal headaches, noting that calcium and parathyroid hormone levels were significantly decreased, and phosphorus and beta-endorphin levels were significantly increased. Entrapment of the SON by tetany of the *orbicularis oculi* may be the etiology.

Treatment of perpetuating factors for SN involves removing provoking stimuli (such as using better-fitting goggles or helmets). Squinting can entrap the SON, so if poor vision or bright light sensitivity is the trigger, an optometrist evaluation or sunglasses may help. Frowning can also entrap the SON, so botulinum toxin (see below) can relieve the entrapment as well as treat the perpetuating factor. Since many of these headaches are perimenstrual in nature, a short course of diuretics (25 mg HCTZ or 20 mg furosemide by mouth daily) for 4–5 days prior to menses has been useful in preventing entrapment [Andrea Trescot, MD, personal communication].

Injection Technique

Landmark-Guided Technique

The patient is placed in a supine or sitting position (with the head supported). The supraorbital notch is palpated, and the skin is cleaned and prepped in sterile fashion. The supraorbital notch is typically located 2 cm to 2.5 cm lateral to the bridge of the nares. The non-injecting index and middle fingers are placed to straddle the notch, moving the skin cephalad to avoid injecting in the eyebrow itself (Fig. 14.7). With the target identified, a 27- or 30-gauge 1.5-in needle is inserted at an oblique angle to the skin until bony contact is made (Video 14.2); this area is very tender, and the patient often will involuntarily withdraw. The needle is then slightly withdrawn and redirected medially and slightly cephalad. Care is taken during needle insertion as not to insert the needle tip into the supraorbital notch itself, as this will cause paresthesias along the distribution of the supraorbital nerve, and injection of the medication could cause more entrapment. Following negative aspiration, a volume of 0.5–1 ml of local anesthetic with or without steroid solution is injected. Some physicians recommend using larger volumes of injectate such as 3–4 cc [23]; however, the authors recommend smaller volumes (0.5–1 cc) to avoid the potential increase in entrapment from the injectate. This procedure can be repeated multiple times, but with recurrence of pain, other longer-lasting procedures such as cryoneurolysis or neuromodulation can be performed (see below). The supratrochlear nerve may also need to be treated.

Fig. 14.7 Supraorbital landmark-guided injection (Image courtesy of Andrea Trescot, MD)

Capulli and Firetto [24] treated 27 intractable migraine patients with occipital (see Chap. 17) and/or supraorbital nerve blocks, based on tenderness to palpation, using 0.5–1 cc of 0.5 % bupivacaine (without steroid). Twenty-three patients (85 %) noted 50 % or better improvement in their headaches over a 6-month period. They hypothesized that "the anesthetic blocks extinguished presumed foci of nociceptor discharges maintained by perivascular neurogenic inflammation, thereby reestablishing normal central neurone [sic] sensitivity."

Fluoroscopy-Guided Technique

Fluoroscopy can be used to visualize the supraorbital foramen. The C-arm should be placed in a cephalad rotation to identify the foramen (Fig. 14.8). Once identified, the procedure is then performed in the same fashion as the landmark-guided technique with needle placement just medial to the supraorbital foramen.

Ultrasound-Guided Technique

Using ultrasound (US) to block the supraorbital nerve can be beneficial over the landmark-guided or fluoroscopic approaches. The linear probe is placed horizontally across the supraorbital notch (Fig. 14.9). With direct visualization of the supraorbital foramen, proper needle placement can be achieved as well as avoiding inadvertent needle placement into the foramen. In a recent cadaveric study, Spinner and colleagues [25] demonstrated that ultrasound-guided blocks of the supraorbital and infraorbital nerves (see Chap. 22) had greater accuracy than conventional

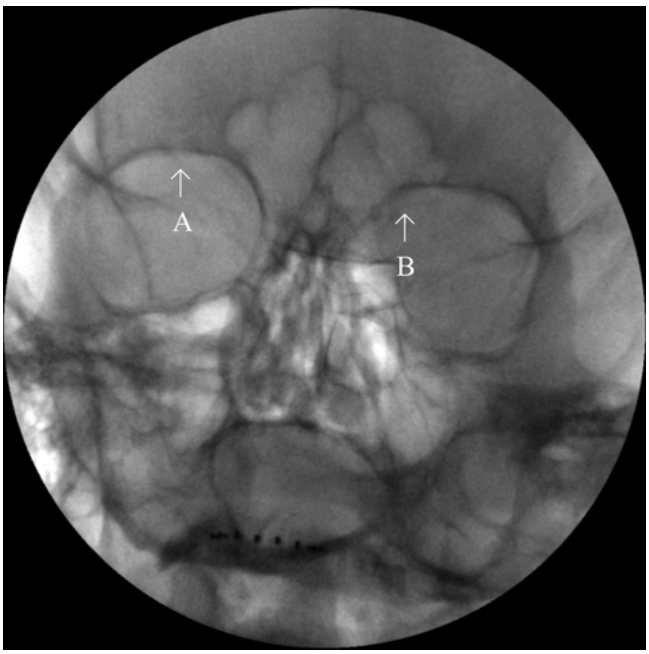

Fig. 14.8 3D image of the skull. *Arrows* show supraorbital notch and supratrochlear groove. *A* supraorbital notch, *B* supratrochlear groove (Image courtesy of Andrea Trescot, MD)

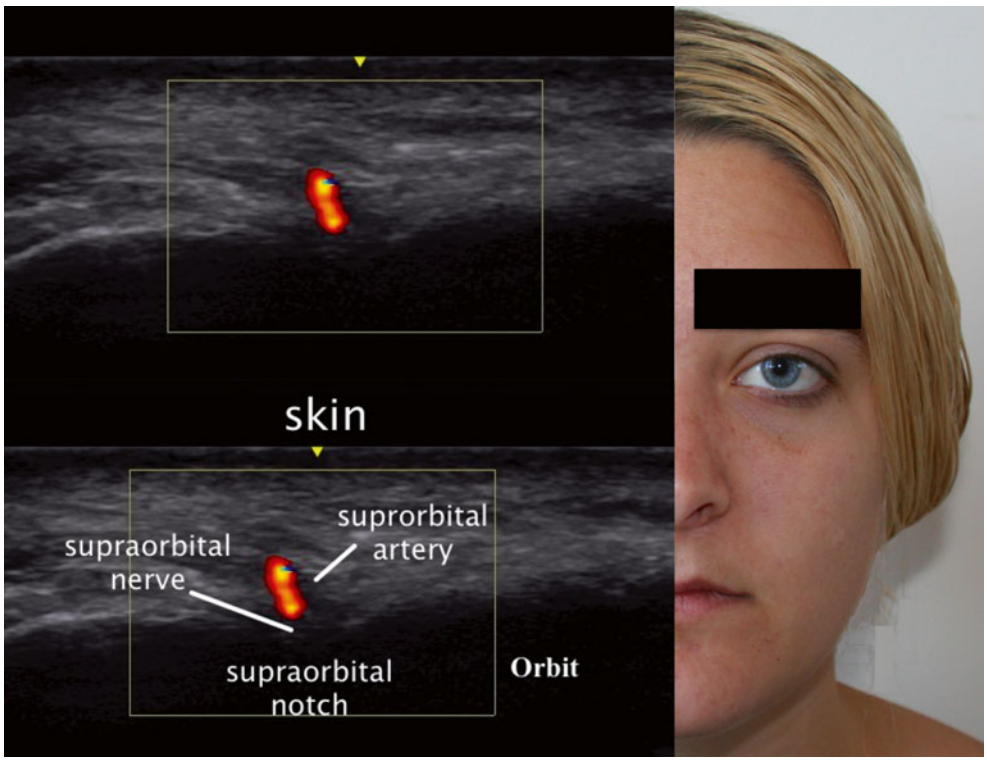

Fig. 14.9 Ultrasound image of the supraorbital nerve (Ultrasound image courtesy of David Spinner, MD; composite created by Andrea Trescot, MD)

landmark-based techniques. The results of the study showed that the US accuracy rate was 100 % (18 of 18) for the in-plane approach and 94 % (17 of 18) for the out-of-plane approach. Thirty-five injections were considered accurate (97 %) with overflow, and one injection was inaccurate. The study concluded that ultrasound-guided injections had a higher degree of accuracy versus the standard techniques used today. The US technique would be performed the same as the conventional landmark-guided technique with improved visualization of the target.

Neurolytic/Surgical Techniques

Cryoneuroablation

Cryoneuroablation has been an effective therapeutic technique for SN. Trescot described the use of cryoneuroablation for the treatment of craniofacial pain syndromes including SN [10]. Figure 14.10 depicts the use of cryoneuroablation in the treatment of the SON.

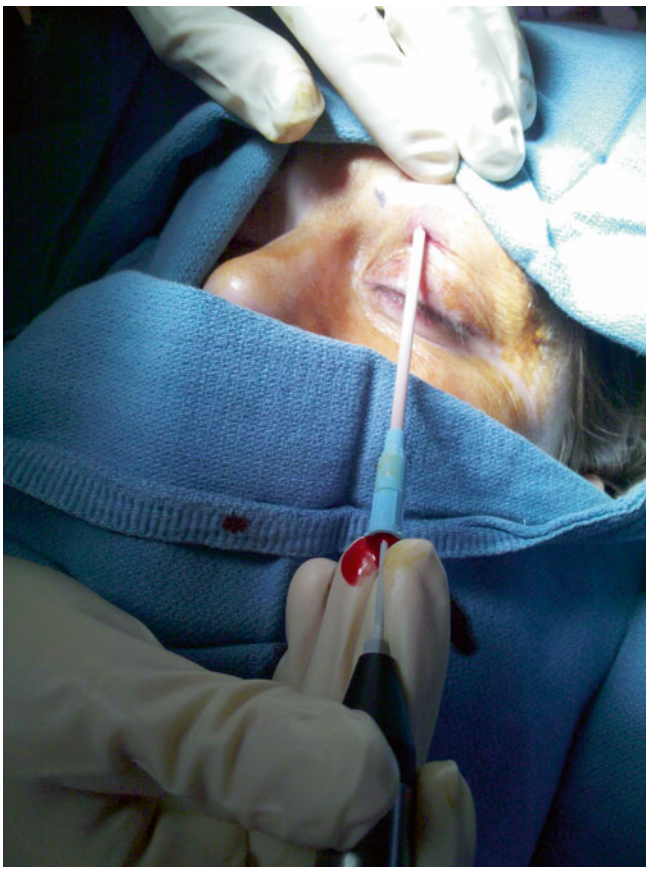

Fig. 14.10 Cryoneuroablation of the supraorbital (supratrochlear) nerve (Image courtesy of Andrea Trescot, MD)

Radiofrequency Lesioning

Although there are potential concerns regarding neuritis and neuromas when performing conventional *radiofrequency lesioning*, Weyker and colleagues [26] reported long-term (>7 months) relief from conventional radiofrequency lesioning (RF) of the supraorbital nerve for hemicrania continua, after diagnostic supraorbital injections gave temporary relief.

Chemical Neurolysis

Chemical neurolytic techniques involve the use of *alcohol*, *phenol*, and *botulinum toxin*. Chemical neurolysis with alcohol or phenol has been used to cause a neural destruction of the supraorbital nerve to promote longer-lasting effects compared to local anesthetic and steroid injection [1, 27, 28]. Wilkinson [27] described 60 injections of 6 % phenol in glycerol onto the peripheral trigeminal branches (including the supraorbital nerve) to treat 18 patients with tic douloureux; 80 % noted total or marked relief for a median of 9 months (though 30 % had relief for 2 years). Extreme caution should be used, however, because of the significant risk of skin sloughing and neuritis. Beyer noted that, after injection of the SON with absolute alcohol, there was "often an extreme, alarming swelling of the upper lid" [1], which would resolve after several days without sequelae.

Botulinum toxin use with frontal migraine headaches has shown to significantly help headaches, likely secondary to muscular entrapments of the SON [29, 30]. Risks include blepharoptosis and paresthesias [29], and the beneficial effects usually last only a few months.

Peripheral Nerve Stimulation

Neuromodulation in the form of peripheral nerve stimulation is another potentially effective treatment for SN. A stimulator lead is tunneled subcutaneously (Figs. 14.11 and 14.12) and connected to a subcutaneous generator. A paper published by Asensio-Samper and colleagues showed that stimulation of the supraorbital nerve provided good analgesic

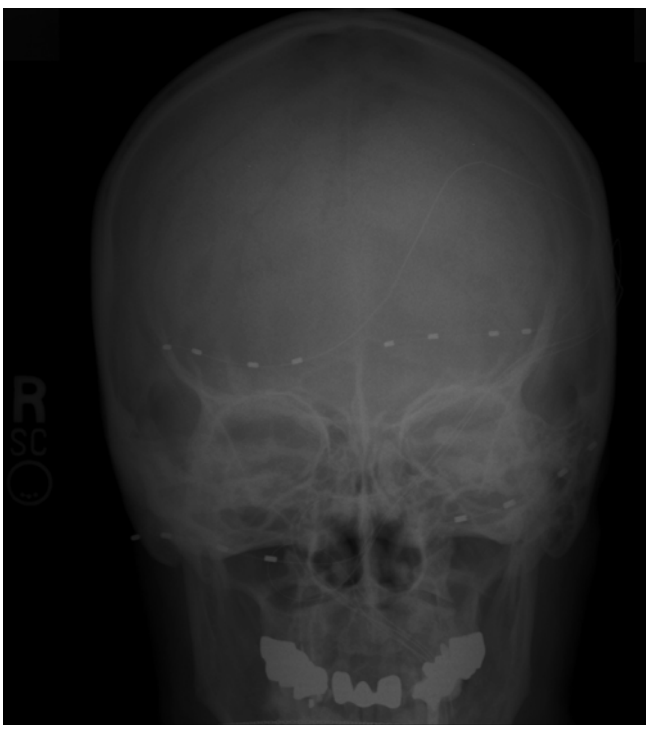

Fig. 14.11 Fluoroscopic image of the supraorbital and occipital stimulator trial (Image courtesy of Rafael Justiz, MD)

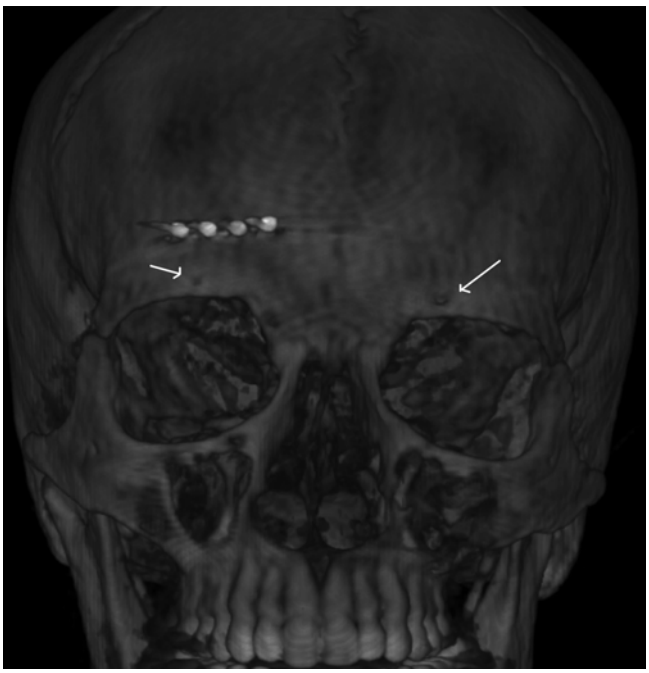

Fig. 14.12 3D image of the supraorbital peripheral nerve stimulator. Note *arrows*, which identify enclosed supraorbital foramina (Image courtesy of Andrea Trescot, MD)

control in a patient with posttraumatic SN, refractory to medical treatment [31]. Amin et al. [32] described 16 patients who responded temporarily to supraorbital nerve blocks, all underwent 5–7 day peripheral nerve stimulator trials. Of these, ten patients underwent permanent implantation and were monitored for 30 weeks; headache pain scores decreased, and opioid consumption was reduced by half. In a study of 60 chronic headache patients [33] with peripheral nerve stimulators for up to 42 months, Verrills and colleagues noted that medications were reduced in 83 % of the patients, and there were ten surgical revisions but no long-term complications. *Chronic intractable migraines* [34], *cluster headaches* [35], *postherpetic neuralgia* [36], and t*rigeminal autonomic cephalalgia* [37] have also been effectively treated with supraorbital/supratrochlear nerve peripheral stimulation.

Surgery

Surgical decompression of the SON has also shown to be efficacious. Guyuron et al. reported that corrugator resection leads to improvement in symptoms in over 80 % of patients, with complete resolution in 57 %, compared to a sham surgical group, which had only 57 % of patients improving with only 3.8 % complete elimination [38]. In 1999, Sjaastad et al. [39] reported results in five cases of SON decompression; the immediate improvement was "good," and after a mean observation time of more than 6 years, an improvement of 50–100 % was observed (mean, about 85 %). In the two patients with the longest postoperative observation time (approximately 8 years), pain had not recurred. Poggi et al. [40] reported on the surgical decompression of several nerves, including the supraorbital nerve, on 18 consecutive patients with migraines who received temporary relief from botulinum toxin injections. Three of the 18 patients (17 %) had complete relief, while 50 % (9 of 18) had at least 75 % relief.

In a more recent study, Chepla and colleagues [41] noted that surgical decompression was effective in relieving pain associated with SON entrapment in 92 % of patients. However, only two-thirds showed complete resolution of symptoms, with the remaining having recurrence.

Complications

Complications with this procedure are rare, but the scalp is very vascular, and there can be bleeding at the site; this can be easily remedied by compression over the injection site. Alopecia can occur if the steroid injection is performed in the eyebrow. Large volume injections may anesthetize the *levator palpebrae muscle*, causing a temporary ptosis; during cryoneuroablation, it is important to check for unwanted motor stimulation because of the risk of freezing the nerve to the levator palpebrae.

Summary

The supraorbital nerve is an underappreciated cause of "migraines," most likely the etiology of headaches triggered by frowning or squinting and treated with frontalis botulinum toxin. The recognition of this etiology can result in the relief of potentially debilitating headaches.

References

1. Beyer TE. Idiopathic supraorbital neuralgia. Laryngoscope. 1949;59(11):1273–5.
2. Agrawal SM, Kambalimath DH. Trigeminal neuralgia involving supraorbital and infraorbital nerves. Natl J Maxillofac Surg. 2010; 1(2):179–82.
3. Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders: 2nd edition. Cephalalgia. 2004;24(Suppl 1):9–160.
4. O'Brien Jr JC. Swimmer's headache, or supraorbital neuralgia. Proc (Bayl Univ Med Cent). 2004;17(4):418–9.
5. Bastiaensen LAK. Supra-orbital neuralgia and its treatment. Neuro-Ophthalmology. 1996;16(4):225–7.
6. McGuire TP, Gomes PP, Clokie CM, Sandor GK. Fractures of the supraorbital rim: principles and management. Journal. 2006;72(6): 537–40.
7. Janiri L, Gallo G, Nicoletti W. Calcium deficiency and supraorbital headache: a clinical study of adult subjects. Cephalalgia. 1986;6(4): 211–8.

8. Gennaro S, Secci F, Fiaschi P, Merciadri P. Supraorbital nerve entrapment neuropathy in Hansen's disease. Neurol Sci. 2013; 34(12):2243–4.
9. Evans RW, Pareja JA. Expert opinion. Supraorbital neuralgia. Headache. 2009;49(2):278–81.
10. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
11. Guerrero AL, Herrero-Velazquez S, Penas ML, Mulero P, Pedraza MI, Cortijo E, et al. Peripheral nerve blocks: a therapeutic alternative for hemicrania continua. Cephalalgia. 2012;32(6):505–8.
12. Sjaastad O, Petersen HC, Bakketeig LS. Supraorbital neuralgia. Vaga study of headache epidemiology. Cephalalgia. 2005;25(4): 296–304.
13. Andersen NB, Bovim G, Sjaastad O. The frontotemporal peripheral nerves. Topographic variations of the supraorbital, supratrochlear and auriculotemporal nerves and their possible clinical significance. Surg Radiol Anat. 2001;23(2):97–104.
14. Janis JE, Ghavami A, Lemmon JA, Leedy JE, Guyuron B. The anatomy of the corrugator supercilii muscle: part II. Supraorbital nerve branching patterns. Plast Reconstr Surg. 2008;121(1):233–40.
15. Fallucco M, Janis JE, Hagan RR. The anatomical morphology of the supraorbital notch: clinical relevance to the surgical treatment of migraine headaches. Plast Reconstr Surg. 2012;130(6):1227–33.
16. Klein DS, Schmidt Jr RE. Chronic headache resulting from postoperative supraorbital neuralgia. Anesth Analg. 1991;73(4):490–1.
17. Yangüela J, Pareja J, Lopez N, Sáchez del Ríos M. Trochleitis and migraine headache. Neurology. 2002;58:802–5.
18. Sharma RR, Pawar SJ, Lad SD, Netalkar AS, Musa MM. Frontal intraosseous cryptic hemangioma presenting with supraorbital neuralgia. Clin Neurol Neurosurg. 1999;101(3):215–9.
19. Guerrero AL, Herrero-Velazquez S, Penas ML, Mulero P, Pedraza MI, Cortijo E, et al. Peripheral nerve blocks: a therapeutic alternative for hemicrania continua. Cephalalgia. 2012;32(6):505–8.
20. Tijssen C, Schoemaker K, Visser L. Supraorbital neuralgia caused by nerve entrapment visualized on ultrasonography. Headache. 2013;53(2):376–7.
21. Ettl A, Kramer J, Daxer A, Koornneef L. High resolution magnetic resonance imaging of neurovascular orbital anatomy. Ophthalmology. 1997;104(5):869–77.
22. Matharu MS, Goadsby PJ. Persistence of attacks of cluster headache after trigeminal nerve root section. Brain J Neurol. 2002;125(Pt 5):976–84.
23. Waldman SD. Supraorbital nerve block. In: Waldman SD, editor. Atlas of pain management injection techniques. 3rd ed. Philadelphia: Saunders Elsevier; 2013. p. 4–5.
24. Caputi CA, Firetto V. Therapeutic blockade of greater occipital and supraorbital nerves in migraine patients. Headache. 1997;37(3): 174–9.
25. Spinner D, Kirschner JS. Accuracy of ultrasound-guided superficial trigeminal nerve blocks using methylene blue in cadavers. Pain Med. 2012;13(11):1469–73.
26. Weyker P, Webb C, Mathew L. Radiofrequency ablation of the supra-orbital nerve in the treatment algorithm of hemicrania continua. Pain Physician. 2012;15(5):E719–24.
27. Wilkinson HA. Trigeminal nerve peripheral branch phenol/glycerol injections for tic douloureux. J Neurosurg. 1999;90(5):828–32.
28. Ramamurthy S, Walsh NE, Schoenfeld LS, Hoffman J. Evaluation of neurolytic blocks using phenol and cryogenic block in the management of chronic pain. J Pain Symptom Manag. 1989;4(2): 72–5.
29. Persaud R, Garas G, Silva S, Stamatoglou C, Chatrath P, Patel K. An evidence-based review of botulinum toxin (botox) applications in non-cosmetic head and neck conditions. JRSM Short Rep. 2013;4(2):10.
30. Trescot AM. Headache management in an interventional pain practice. Pain Physician. 2000;3(2):197–200.
31. Asensio-Samper JM, Villanueva VL, Perez AV, Lopez MD, Monsalve V, Moliner S, et al. Peripheral neurostimulation in supraorbital neuralgia refractory to conventional therapy. Pain Pract. 2008;8(2):120–4.
32. Amin S, Buvanendran A, Park KS, Kroin JS, Moric M. Peripheral nerve stimulator for the treatment of supraorbital neuralgia: a retrospective case series. Cephalalgia. 2008;28(4):355–9.
33. Verrills P, Rose R, Mitchell B, Vivian D, Barnard A. Peripheral nerve field stimulation for chronic headache: 60 cases and long-term follow-up. Neuromodulation. 2014;17:54–9.
34. Hann S, Sharan A. Dual occipital and supraorbital nerve stimulation for chronic migraine: a single-center experience, review of literature, and surgical considerations. Neurosurg Focus. 2013; 35(3):E9.
35. Narouze SN, Kapural L. Supraorbital nerve electric stimulation for the treatment of intractable chronic cluster headache: a case report. Headache. 2007;47(7):1100–2.
36. Johnson MD, Burchiel KJ. Peripheral stimulation for treatment of trigeminal postherpetic neuralgia and trigeminal posttraumatic neuropathic pain: a pilot study. Neurosurgery. 2004;55(1):135–41.
37. Vaisman J, Markley H, Ordia J, Deer T. The treatment of medically intractable trigeminal autonomic cephalalgia with supraorbital/supratrochlear stimulation: a retrospective case series. Neuromodulation. 2012;15(4):374–80.
38. Guyuron B, Reed D, Kriegler JS, Davis J, Pashmini N, Amini S. A placebo-controlled surgical trial of the treatment of migraine headaches. Plast Reconstr Surg. 2009;124(2):461–8.
39. Sjaastad O, Stolt-Nielsen A, Pareja JA, Fredriksen TA, Vincent M. Supraorbital neuralgia. On the clinical manifestations and a possible therapeutic approach. Headache. 1999;39(3):204–12.
40. Poggi JT, Grizzell BE, Helmer SD. Confirmation of surgical decompression to relieve migraine headaches. Plast Reconstr Surg. 2008;122(1):115–22.
41. Chepla KJ, Oh E, Guyuron B. Clinical outcomes following supraorbital foraminotomy for treatment of frontal migraine headache. Plast Reconstr Surg. 2012;129(4):656e–62.

Auriculotemporal Nerve Entrapment

15

Andrea M. Trescot and Esther Rawner

Introduction

Temple headaches may be due to entrapment of the auriculotemporal nerve (ATN), a third-division branch of the trigeminal nerve. Other areas of pain when this nerve is entrapped may include pain in the auricular area and the temporomandibular joint. This is a common headache site (visualize all the headache patients rubbing or pressing their temples for relief) shared with migraine headaches, and it may often be misdiagnosed as an intracranial migraine. There are two distinct clinical presentations of ATN nerve dysfunction and a variety of names, including *auriculotemporal neuralgia*, *auriculotemporal syndrome*, *Frey syndrome*, *Baillarger syndrome*, *Dupuy syndrome*, *salivosudoriparous syndrome*, and *gustatory sweating syndrome*.

Clinical Presentation

Entrapment of the ATN is probably the most common of the trigeminal headaches. Calandre et al. [1] evaluated specific areas in the scalp that could be manually palpated to elicit a headache; 42.6 % of these "trigger points" were found in the temple region and 33.4 % in the suboccipital region. However, ATN entrapment is rarely diagnosed and represented only 0.4 % of the referrals to a tertiary headache outpatient clinic [2].

There are two main presentations of irritation of this nerve: *auriculotemporal neuralgia* (ATn) and the more rare *auriculotemporal syndrome* (ATS), both of which can have overlapping presentations, as well as multiple etiologies (Table 15.1). ATn is not specifically defined by the International Headache Society's International Classification of Headache Disorders (ICHD II); under this classification scheme, it is best described within the category of "Other terminal branch neuralgias" of the trigeminal nerve. According to this classification, "Injury or entrapment of peripheral branches of the trigeminal nerve may give rise to pain referred to the area innervated by the branch affected, there is tenderness over the affected nerve, and pain is abolished by local anesthetic blockade or ablation of the nerve" [11].

ATn typically presents as paroxysmal attacks of pain in the preauricular and temple regions as well as the retro-orbital region and the teeth [12]. The patient with ATn headaches due to ATN entrapment will often awaken at 3–4 a.m. with a headache in the auricular area that radiates into the temple (Fig. 15.1) and the retro-orbital as well as the occipital region. The headache may be unilateral [13] or bilateral and may have associated ear, parotid, and jaw pain or numbness [2]. The headache is often throbbing in nature, possibly due to its proximity to the *temporal artery*. There is often increased pain with talking, chewing, and menses [14]. The intensity of the pain is moderate to severe, and the quality is usually constant, though at times there may be paroxysms of sharp, jabbing, lancinating pain or a throbbing, pounding pain. The pain may be triggered by trauma to the jaw or temple, as well as by surgery, exacerbated by palpation over

Table 15.1 Occupation/exercise/trauma history relevant to auriculotemporal nerve entrapment

Chewing/clenching	TMJ pathology, trauma, and surgery [3]
Trauma to the jaw	Mandibular fracture [4]
Parotid surgery or infection [5]	TMJ synovial cyst [6]
Tumor invasion [7]	Trigeminal neuralgia [8]
Cutaneous surgery of the cheek [9]	Dental anesthesia [10]

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Fig. 15.1 Patient's description of pain from auriculotemporal neuralgia (Image courtesy of Andrea Trescot, MD)

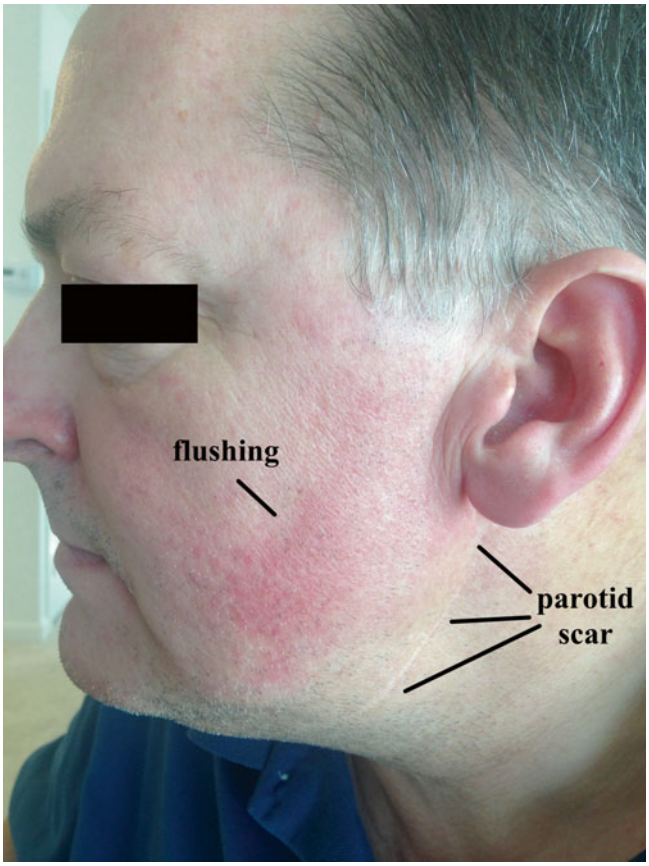

Fig. 15.2 Frey syndrome. Note the well-healed parotid scar and the facial flushing with eating (Image courtesy of Andrea Trescot, MD)

the preauricular area or by chewing, and can be associated with severe nausea and vomiting. The pain characteristics and accompanying nausea and vomiting meet the International Headache Society criteria [11] for a "migraine," though there is no specific listing for ATn. The early morning headache appears to be related to bruxism or nocturnal jaw clenching. Because there is a physical connection with the *facial nerve* (see *Anatomy* section), there can also be pain in the muscles of facial expression with ATN entrapment.

In 2005, Speciali and Gonçalves [2] evaluated six patients with ATn proven by diagnostic nerve blocks (see *Injection techniques* section). All were females with unilateral pain, suffering with symptoms from 1 month to 20 years, and all of the patients had pain with palpation of the preauricular area just above the tragus. Pain was primarily around the ear, radiating to the head of the mandible and temple in all of the patients. One patient had knifelike pain triggered by gustatory stimuli (see *Frey Syndrome* below). In four patients, the pain radiated to the occipital region, but there was no relief from occipital nerve blocks (see Chap. 17). There were complaints of facial tingling or paresthesia in four patients, but notably, *temporomandibular joint* (TMJ) pathology was absent in all of the patients. All of the patients had complete or near-complete relief with ATN injections.

Murayama et al. [12] described a patient with left ear pain and ipsilateral maxillary second molar pain. The dental exam was normal, and the tooth and ear pain resolved with a local anesthetic and steroid injection of the ATN, with remission still present 6 months later.

The *auriculotemporal syndrome*, also known as *Frey syndrome*, was described by Frey in 1923 [15] as a constellation of symptoms including unilateral hyperhidrosis and flushing of the cheek and ear that occurs when eating or drinking anything that stimulates the parotid gland to produce saliva (Fig. 15.2). Pain is uncommon, but there may be sensory changes in the ATN distribution, and there may be a concomitant trigeminal neuralgia. Also known as *Baillarger syndrome*, *Dupuy syndrome*, *salivosudoriparous syndrome*, and *gustatory sweating syndrome*, these symptoms usually occur 2–13 months after surgery, open trauma, or infection of the parotid gland, though it has also been described after dislocation of the mandibular condyle [16]. The proposed cause is an improper regeneration of the sympathetic and parasympathetic nerves of the parotid gland, and the syndrome occurs in about 5 % of surgical patients, even with careful technique. The severity of symptoms associated with Frey syndrome can range from mild to debilitating.

Gordon and Fiddian [5] reviewed the records of 71 patients who had parotid surgery; 17 patients had "noticeable" Frey syndrome. Fourteen patients had positive *Minors' test* (iodine/starch), with the *great auricular nerve* (Chap. 16) involved in six cases, the ATN in four cases, and both in two cases (with two inconclusive tests). Choi et al. [17] evaluated 59 patients after parotidectomy, using subjective symptoms, Minor's starch iodine test, and *thermography*, and found a "good" correlation between the three indicators. Pain triggered by taste stimuli can be a significant component of Frey syndrome; Scrivani et al. [18] described six patients with recurrent, episodic shocking facial pain triggered even by the smell of food, which occurred days to weeks after head and neck surgery.

Anatomy

The ATN is a branch of the third division (mandibular) of the trigeminal ganglion (Fig. 15.3). The *mandibular nerve* (see Chap. 24) exits the cranium through the *foramen ovale* (Table 15.2) and then immediately divides into an anterior and posterior division, providing sensation to the TMJ, tragus, external auditory canal, parotid, base of the auricle, and skin of the temporal region (Fig. 15.4). The anterior division travels between the roof of the *infratemporal fossa* and the *lateral pterygoid muscle* (LPM) and is composed of the *anterior deep temporal nerve*, the *posterior deep temporal nerve*, and the *masseteric nerves*. The posterior division consists of the *lingual nerve*, *inferior alveolar nerve*, and ATN, descending medially to the LPM [19]. Because the ATN has a long and tortuous course, it is at risk for irritation and entrapment. The ATN arises by two roots, which travel on either side of the *ascending middle meningeal artery* and then join together to form a short trunk. It leaves the *foramen ovale* and runs beneath the *pterygoideus externus* to the medial side of the neck of the mandible. It then turns upward with the superficial temporal artery, between the external ear and condyle of the mandible, under the parotid gland. After it leaves the gland, it ascends over the zygomatic arch, traveling in front of the *temporal mandibular joint* (providing the primary innervate of the joint as it goes by) to pierce the temporalis muscle.

The ATN then divides into five small branches:

- The nerve to the *external auditory meatus*, which innervates the skin of the meatus and the external tympanic membrane
- The *parotid branch nerves*
- The *anterior auricular nerve*, which innervates the base of the auricle) [2]
- The *articular branches*, which innervate the posterior part of the TMJ
- The *superficial temporal* nerve, which innervates the skin of the temporal region

The superficial temporal nerve communicates with the *facial nerve*, the *zygomaticotemporal nerve* (which is a branch of the *maxillary nerve*), and *zygomatic branches of the facial nerve*.

The ATN has been associated with migraine headaches. Chim et al. [20] dissected the ATN in 20 cadavers and found three potential compression points along the path of the nerve. Points 1 and 2 corresponded to fascial bands, with point 1 being 13.1 ± 5.9 mm anterior and 5 ± 7 mm superior to the superior point of the external auditory meatus and point 2 found at 11.9 ± 6 mm anterior and 17.2 ± 10.4 mm superior to the most anterosuperior portion of the *external auditory meatus*. Point 3

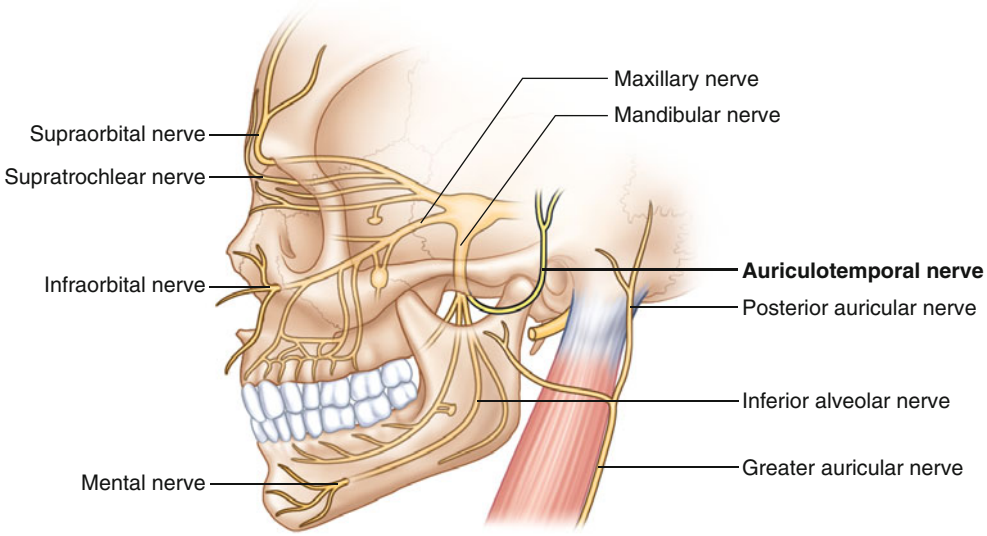

Fig. 15.3 Trigeminal anatomy (Image courtesy of Springer)

Table 15.2 Auriculotemporal nerve anatomy

Origin	Trigeminal ganglion 3rd division (III) – > foramen ovale – > mandibular nerve
General route	Anterior division – > anterior deep temporal nerve, posterior deep temporal nerve, masseteric nerve
	Posterior division – > lingular nerve, inferior alveolar nerve, auriculotemporal nerve (ATN)
	ATN leaves the foramen ovale, turns upward with the superficial temporal artery, under the parotid, in front of the TMJ, anterior to the tragus, and then passes through the temporalis muscle, lateral to the temporal artery; 5 branches
Sensory distribution	TMJ, tragus, external auditory canal, parotid, base of the auricle, skin of temporal region
Motor innervation	None
Anatomic variability	May connect with the temporal branch of the facial nerve, zygomaticotemporal nerve, the lesser occipital nerve, or greater occipital nerve
Other relevant structures	Lateral pterygoid muscle can be site of compression [18]; may be confused with the zygomaticotemporal nerve

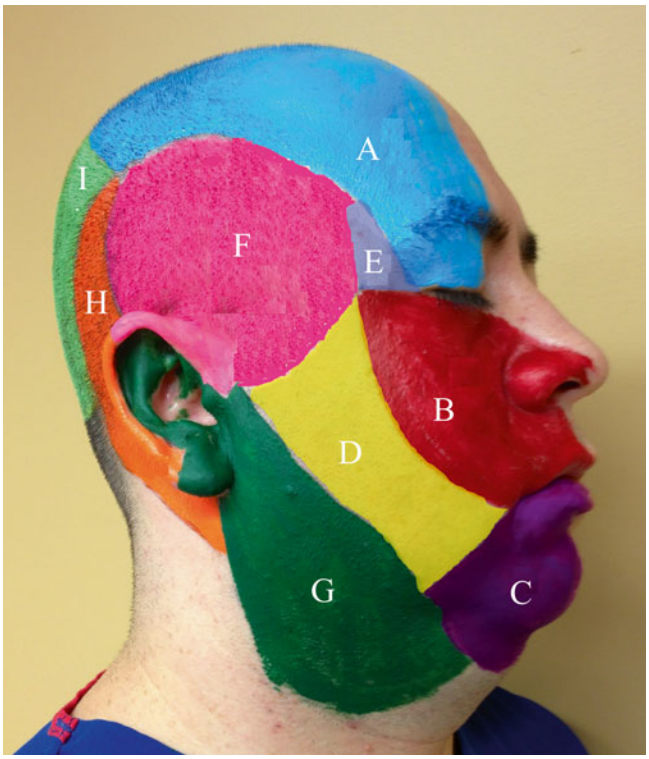

Fig. 15.4 Sensory areas of the trigeminal and cervical nerve branches: *A* supraorbital nerve, *B* infraorbital nerve, *C* mental nerve, *D* buccal nerve, *E* lacrimal nerve, *F* auriculotemporal nerve, *G* superficial cervical plexus, *H* posterior auricular nerve/great auricular nerve, *I* occipital nerve (Image courtesy of Terri Dallas-Prunskis, MD)

represented the interaction of the ATN and the superficial temporal artery, found in 80 % of the dissection. There were three types of interactions noted – the artery crossing over the nerve (62.5 %), the nerve crossing over the artery (18.8 %), or the artery wrapping helically around the nerve (18.8 %).

Andersen et al. [21] dissected ten heads and found the superficial ATN branch to be located between 8 and 20 mm anterior to the root of the ear helix. Only four of the cadavers had a single branch anterior to the tragus; the rest had multiple branches. The temporal artery was always found deep to the ATN, and in three dissections, the ATN connected with the temporal branch of the facial nerve. There was noted to be a connection between the ATN and *lesser occipital nerve* (Chap. 18) in two cases and between the ATN and the *greater occipital nerve* (Chap. 17) in four cases.

Janis et al. [22] dissected 50 cadaver temples and identified that the ATN was always found lateral to the artery in the upper preauricular temple. In another 50 cadavers, Janis et al. [23] dissected the periorbital and temporal regions, evaluating the path of the zygomaticotemporal branch of the ATN. In "exactly half" of the specimens, the nerve had no intramuscular path; in the other half of the cadavers, 11 had only a brief course through the muscle, but 14 had a long, tortuous path through the muscle, making entrapment likely.

Fernandes et al. [24] dissected 40 cadavers, focusing on the temporomandibular joint; they found significant variation in the relationship between the ATN and middle meningeal artery, with the ATN 10–13 mm inferior to the superior condyle and 1–2 mm posterior to the neck of the condyle. The branches to the facial nerve, usually two in number, pass forward from behind the neck of the mandible and join the facial nerve at the posterior border of the masseter muscle. The filaments to the otic ganglion are derived from the roots of the ATN close to their origin [25].

While these previous studies were done in cadavers, Totonchi et al. [26] endoscopically evaluated the zygomaticotemporal branch of the trigeminal nerve in 20 patients undergoing endoscopic forehead procedures. A prior pilot study on cadavers suggested that the *zygomaticotemporal nerve* emerges from the deep temporal fascia at an average location 17 mm lateral and 6 mm cephalad of the lateral palpebral commissure. This site is very close to the physical exam tenderness site described by Trescot [14] (see *Physical Exam* section). The tissues of the live patients were marked with dye at this same location, and the nerve was identified endoscopically. Three types of accessory branches were categorized: cephalad to the main branch, lateral to the main branch, and in the immediate vicinity of the main branch.

Entrapment

Entrapment of the posterior division of the mandibular nerve is not uncommon (see Chap. 24) [2], with one study showing entrapment in 6 % of 52 cadavers, primarily at the level of the LPM [19]. Another study of ten cadaver dissections showed myositis, local ischemia, and inflammation of the LPM in patients with premortem complaints of pain in an ATN distribution [27]. Spasm of the LPM or local changes within the muscle can entrap the nerve, causing numbness of the external ear and TMJ [19]. The ATN can also be entrapped between the medial and lateral pterygoid muscles, causing facial numbness, mandibular pain, or headache [28].

The anatomic relationships between the ATN and the muscles of mastication, the TMJ, and the surrounding vascular structures set up the potential for entrapment, so that ATn can play a role in TMJ pathology, headaches, and external ear pain. Komarnitki and colleagues [29] dissected the *infratemporal fossa* of 16 specimens and found one-, two-, three-, four-, and five-branch variants, each of which could be entrapped. Secretomotor fibers within the parotid gland may also be entrapped, leading to impairment of salivation (and therefore triggering Frey syndrome).

Janis et al. [22] proposed an entrapment of the ATN by the *superficial temporal artery*, finding a "direct relationship" between the ATN and the superficial temporal artery in 34 % of the cadavers studied, with the ATN wrapped helically around the superficial temporal nerve. Because of the ATN and facial nerve connection, entrapment of the nerve at this level can cause pain in the muscles of facial expression [19].

In addition, the temporal artery entraps the *zygomaticotemporal nerve* (a branch of the maxillary nerve that exits through the zygomaticotemporal foramen and innervates a small area of the forehead and temporal region), mimicking an ATN headache (Fig. 15.5) [30].

Chim et al. dissected 20 temples and identified several sites of entrapment. They found two consistent fascia bands in the preauricular area; the inferior band (located 13.1 ± 5.9 mm anterior and 5.0 ± 7.0 mm superior to the external auditory meatus) was present in all the specimens, while the superior band (11.9 ± 6.0 mm anterior and 17.2 ± 10.4 mm superior to the external auditory meatus) was present in 17 of 20 dissections. They also noted that in

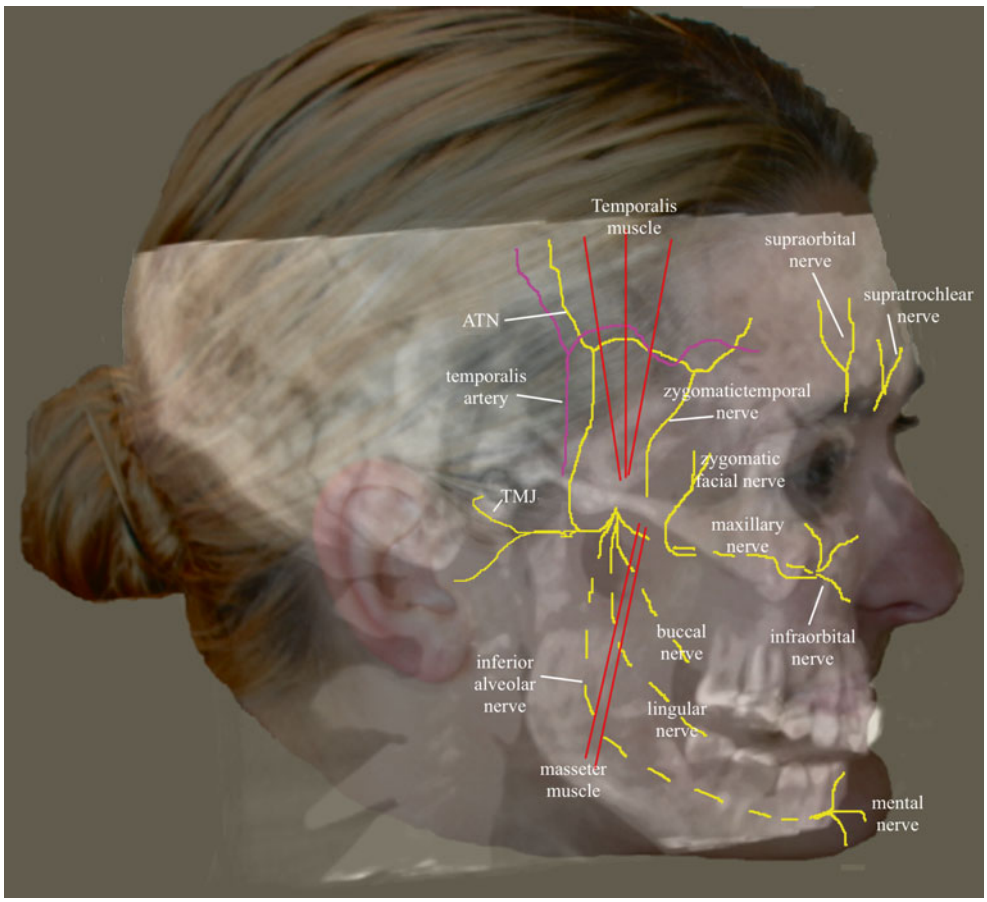

Fig. 15.5 Nerves of the face (Image created by Andrea Trescot, MD)

Fig. 15.6 Auriculotemporal nerve examination (Image courtesy of Andrea Trescot, MD)

16 of 20 dissections, the ATN passed over the superficial temporal artery, causing focal compression. In three of these cases, there was a helical intertwining of the ATN and artery.

Physical Exam

Since there are several sites of entrapment, the physical exam must be able to separate each area of pathology. Examination should include the TMJ (placing the examining fingers over the joint as the patient opens and closes their mouth), the masseter (rolling the examining thumb horizontally as the patient clenches their jaw), and the zygomatic branch of the facial nerve (placing the examining thumb at the anterior condyle of the mandible, just under the zygoma). The examiner should also palpate the temporalis muscle (looking for trigger point tenderness) and the superficial temporal artery (to evaluate for temporal arteritis).

The most clinically common site of entrapment for the ATN is in the temporal fossa; Trescot described finding the ATN by placing the index finger at the apex of an isosceles triangle created by resting (for the right-sided exam) the thumb on the tragus and the middle finger on the canthus (corner of the eye) (Fig. 15.6) (Video 15.1) [14].

Differential Diagnosis

The etiology of ATn is often elicited clinically. Although bruxism is common, other potential causes (see Table 15.1) must be considered. The differential diagnosis is extensive and easily confused (see Table 15.3), leading to misdiagnosis. The various tests for ATN entrapment are listed in Table 15.4.

Table 15.3 Differential diagnosis of temple and face pain

	Potential distinguishing features
Cervicogenic headache	Neck pain, cervical facet tenderness, occipital nerve tenderness (see Chap. 17)
Hemicrania continua	Usually improves with carbamazepine and indomethacin
Myofascial pain	Palpable trigger points
TMJ dysfunction	Pain over the TMJ
Otitis	Otologic exam is abnormal
Tooth pain	Dental exam is abnormal
Atypical facial pain	Diagnosis of exclusion (everything else is negative)
Chronic migraine	No response to ATN injections
Trigeminal neuralgia	Trigger zones
Temporal arteritis	Systemically ill, elevated ESR

Table 15.4 Diagnostic tests for auriculotemporal nerve entrapment

	Potential distinguishing features
Physical exam	Tenderness over the isosceles triangle
Diagnostic injection	Resolution of pain after diagnostic injection at distal and occasionally proximal sites
Ultrasound	Nerve identified next to the temporal artery
MRI	May be identified in perineural spread of tumor between the facial nerve and V3, seen as enlargement or abnormal gadolinium enhancement of the nerves, or obliteration of the perineural fat pad [7]
Arteriography	Not visualized
X-ray	Not visualized
Electrodiagnostic studies	EMG of the lateral pterygoid muscle

Identification and Treatment of Contributing Factors

In 1934, Costen described a complex of symptoms that includes loss of hearing, tinnitus, dizziness, headache, and a burning sensation of the throat, tongue, and side of the nose; their anatomical and physiological causes are uncertain but were originally believed to be the result of temporomandibular joint pathology. He also proposed that a lack of posterior teeth leads to a "mandibular overclosure," which could entrap the ATN [24]. Therefore, a dental evaluation for occlusive problems would be appropriate. The use of an anterior occlusion splint (which puts the front teeth together)

potentially prevents the temporalis spasm from bruxism that contributes to ATN entrapment. A low-dose, long-acting benzodiazepine such as clonazepam (0.25–0.5 mg at night) or muscle relaxant such as tizanidine (2–4 mg at night) has been effective in preventing the entrapment [14].

Injection Technique

Landmark-Guided Technique

Landmark-guided ATN injections are classically described below the TMJ in the posterior aspect of the mandibular condyle, just in front of the tragus. In 1970, Damarjian [13] described performing one to 14 consecutive diagnostic and therapeutic local anesthetic ATN injections (usually 3–4); 30 % had recurrence after 1–2 years. Trescot [14] described an injection technique using a more superior site, identifying an exquisitely tender area at the apex of an isosceles triangle with the base composed of lines connecting the tragus and the corner of the eye (Fig. 15.7). This is usually in close proximity to the artery. One cc of local anesthetic and depo-steroid are then injected in a cephalad direction, parallel to the path of the nerve (Fig. 15.8). The artery is usually slightly more posterior, so if the pulsations of the artery are not obvious, Trescot suggests placing the injection slightly anteriorly to avoid a hematoma (Video 15.2). Careful evaluation will confirm that the artery itself is not tender, therefore decreasing the likelihood of a missed diagnosis of temporal arteritis. Interestingly, a dissection in the temple suggested that this site of injection might be actually an injection of the *zygomaticotemporal branch of the maxillary nerve* (Fig. 15.9).

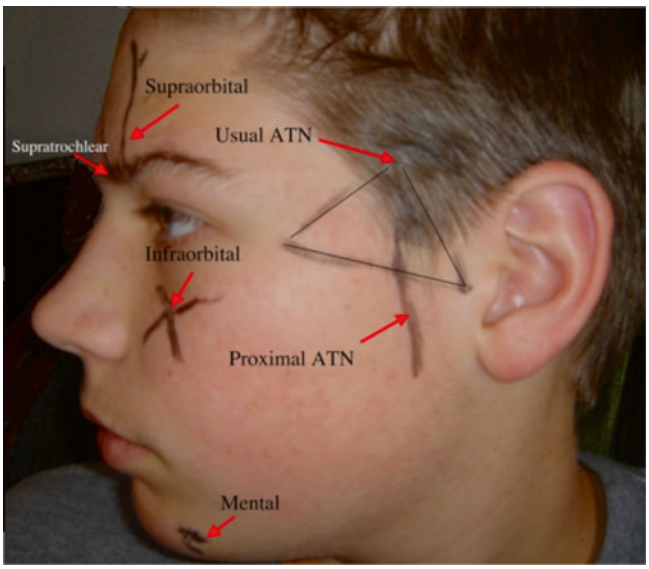

Fig. 15.7 Auriculotemporal site of injection (usual and proximal), including other sites of nerve injection (Image courtesy of Andrea Trescot, MD)

Fig. 15.8 Auriculotemporal injection (Image courtesy of Andrea Trescot, MD)

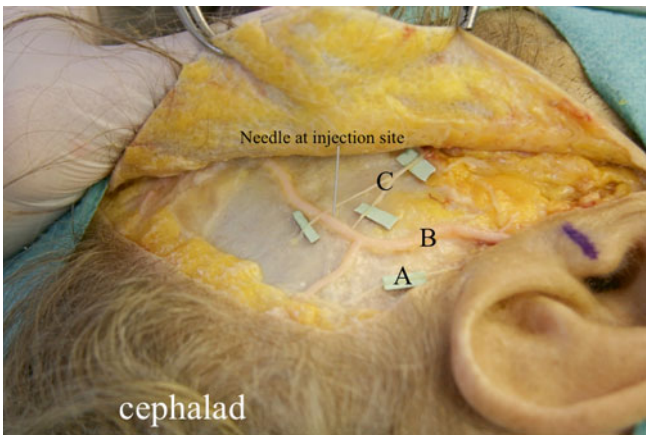

Fig. 15.9 Temple dissection; *A* auriculotemporal nerve, *B* temporal artery, *C* zygomaticotemporal branch of the maxillary nerve (Dissection by Gabor Balsa, MD, Semmelweis University, Laboratory for Applied and Clinical Anatomy, Budapest, Hungary; image courtesy of Andrea Trescot, MD)

The second injection site is more proximal and less common. It is located where the nerve passes anterior to the TMJ, just inferior to the zygoma (Fig. 15.7). This is close to the facial nerve, and the patient must be warned of the possibility of temporary facial weakness. For that reason, small volumes (less than 0.5 cc) and meticulous needle placement are important. There is occasionally a "double crush" syndrome, with the nerve trapped at both sites, and both would then need to be treated. Speciali and Gonçalves [2] described six patients with ATn; using 0.5 cc of 2 % lidocaine

with dexamethasone 2.5 mg (up to 1.5 cc), they injected the proximal ATN (below the TMJ at the posterior border of the mandibular condyle), just in front of the tragus, about 1 cm deep, with the needle 45° and directed to the nose. They had only one recurrence of pain after the injections, but they had a short follow-up (less than 1 year).

Fluoroscopy

There are no bony landmarks for fluoroscopy to be useful.

Ultrasound-Guided Technique

In 2007, Shankar and Brethauer [31] described a US technique for the ATN injection. The US probe is placed transversely just above the TMJ, and color Doppler is used to identify the superficial temporal artery; the nerve appears as a small hyperlucent bundle (Fig. 15.10a). The probe is then rotated to perform a longitudinal scan to track the nerve cephalad (Fig. 15.10b). They used a 25-g needle from an out-of-plane approach to deliver medication (2 cc of 1 % lidocaine and 10 mg of Depo-Medrol®) around the nerve.

Neurolytic/Surgical Technique

Cryoneuroablation

Cryoneuroablation has been an effective therapeutic technique for ATn. Trescot described the use of cryoneuroablation for the treatment of extracranial headache syndromes including ATN entrapment [14, 32]. The cryoprobe is placed parallel to the nerve and swept side to side to find the nerve (Fig. 15.11).

Radiofrequency Lesioning

Although there are potential concerns regarding neuritis and neuromas with conventional radiofrequency (RF) lesioning, there are case reports supporting the use of pulsed RF for peripheral headache pain generators [33].

Chemical Neurolysis

Chemical neurolytic techniques involve the use of alcohol, phenol, and botulinum toxin. Because of the proximity of the facial nerve, and the potential spread of medication, alcohol or phenol would not be recommended, except in special cases such as the treatment of cancer pain.

Botulinum toxin is commonly used in the temporalis muscle to treat parietotemporal migraines, which presumably results in decreased entrapment of the ATN. Totonchi et al. [26] stated that their study of the *zygomaticotemporal branch of the trigeminal nerve* was done for the express purpose of defining anatomy for botulinum injections.

Frey syndrome has not had many treatment options until recently. In 2007, Pomprasit and Chintrakam [34] described nine patients with Frey syndrome treated with an average of just under 11 units of botulinum type A intradermally over the affected skin. Five of the nine patients had complete resolution of the gustatory sweating, while the remaining four patients had a dramatic decrease in sweating, with both groups improving for 7–10 months without any side effects.

Peripheral Nerve Stimulation

Until recently, peripheral stimulation of the ATN has been limited by technical considerations. However, in 2010, Simopoulos et al. [35] described bilateral ATN stimulation, and in 2011, Deshpande and Wininger [36] described a technique of combined greater occipital (Chap. 17), lesser occipital (Chap. 18), and ATN stimulation (Fig. 15.12).

Surgery

Poggi et al. [37] reported on the surgical decompression of several nerves, including the zygomaticotemporal branch of the ATN, to treat migraines that responded temporarily to botulinum toxin injections. Three of the 18 patients (17 %) had complete relief, while 50 % (9 of 18) had at least 75 % relief. A 5-year randomized study of 69 migraine patients comparing surgery to botulinum toxin of "migraine trigger points" (including the ATN area) showed an 88 % improvement in headaches after surgical release [38]. Guyuron et al. [39] performed a prospective randomized trial of two different surgical techniques on 20 patients to address the entrapment of the zygomaticotemporal nerve. These patients, all of whom had bilateral temple headaches, were randomized to undergo a surgical decompression and temporal artery resection on one side compared with a nerve avulsion on the other side under endoscopy. The authors showed no statistical difference between the two techniques at 1 year, with both groups noting a decrease in migraines from 14 per month down to 2 per month, with no complications noted and allodynia or neuroma formation identified. However, the authors recommended decompression as the preferred first option.

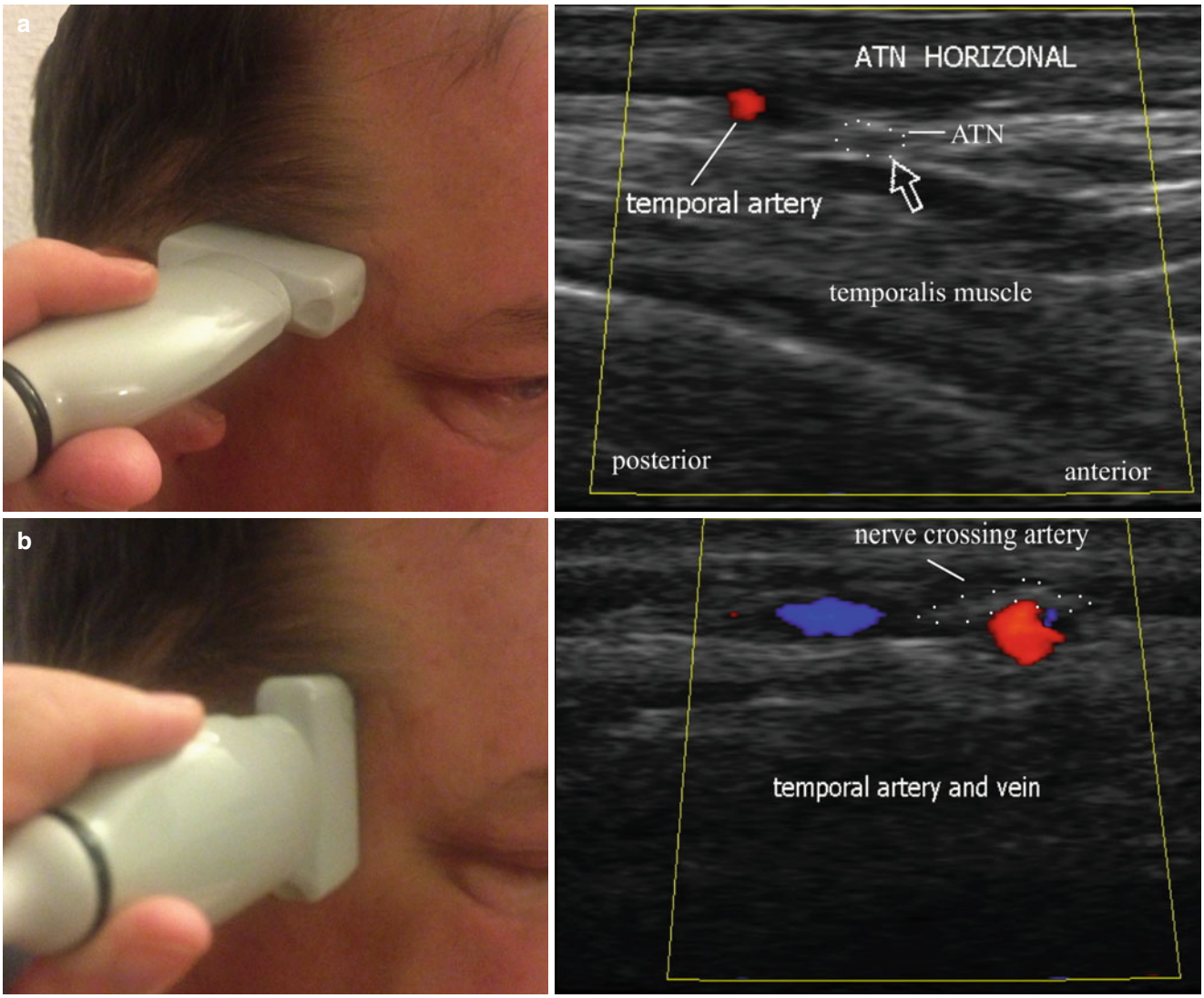

Fig. 15.10 Ultrasound evaluation of the auriculotemporal nerve. (**a**) Horizontal evaluation; (**b**) Vertical evaluation (Images courtesy of Andrea Trescot, MD)

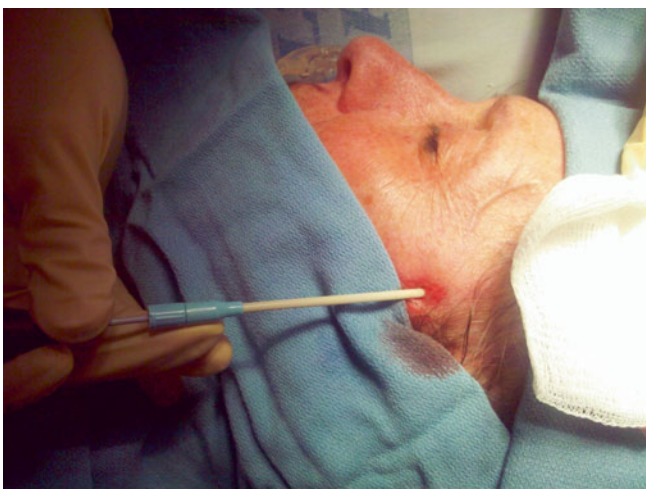

Fig. 15.11 Cryoneuroablation of the auriculotemporal nerve (Image courtesy of Andrea Trescot, MD)

Complications

Complications with this procedure are rare, but the scalp is very vascular, and there can be bleeding at the site; this can be easily remedied by compression over the injection site. Temporary *facial nerve palsy* is common when the injection is placed at the tragus (which is close to the path of the facial nerve), a more proximal injection site than recommended [2].

Summary

There at two very different syndromes associated with the auriculotemporal nerve, one associated with gustatory sweating and the other associated with headaches. This confusion has made it somewhat difficult to differentiate the two in the

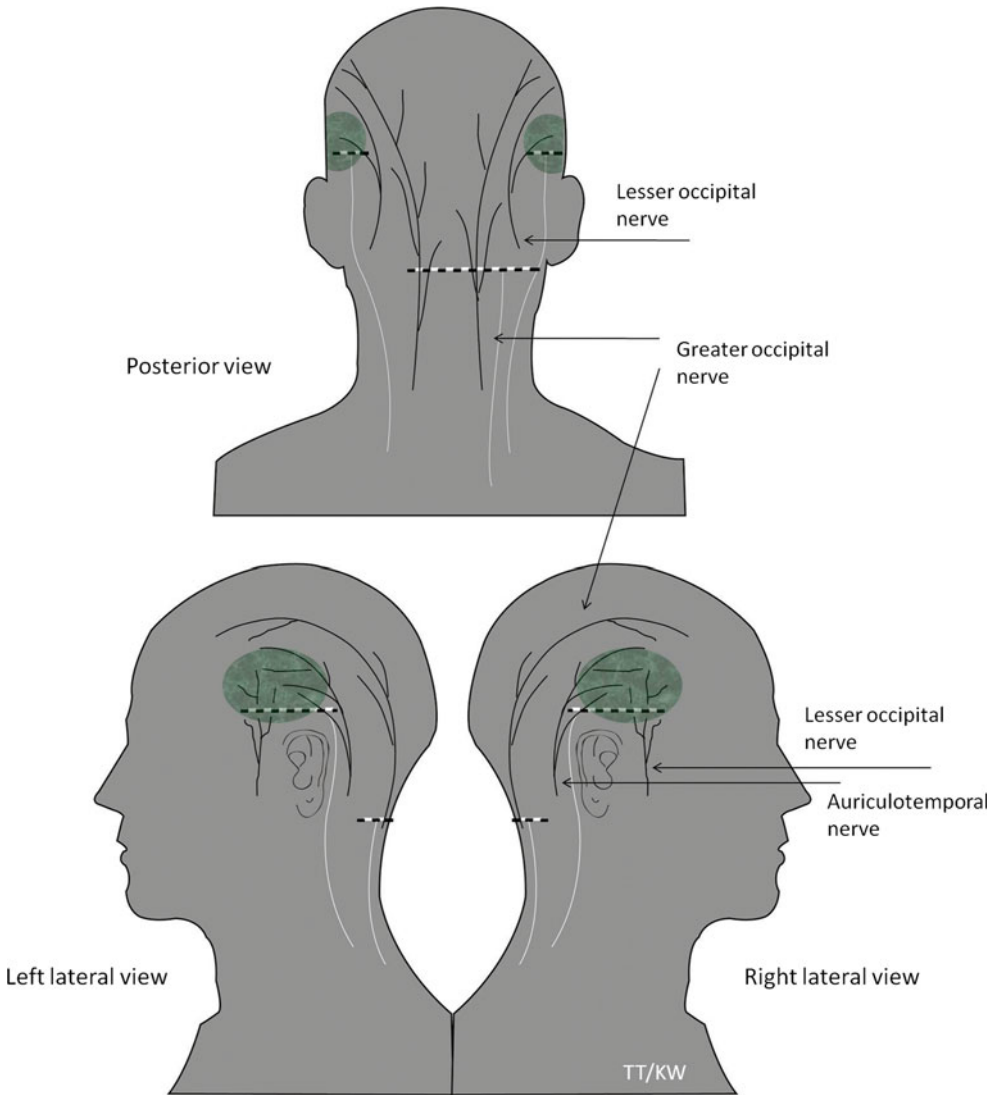

Fig. 15.12 Schematic drawing of the location of peripheral stimulator placement for the greater occipital, lesser occipital, and auriculotemporal nerves (From Deshpande and Wininger [36]. Reprinted with permission from the American Society of Interventional Pain Physicians)

literature. In addition, there may be a component of facial nerve entrapment as well, further complicating the literature. A headache patient who wakes up in the early morning with a headache should be evaluated for ATN entrapment.

References

1. Calandre EP, Hidalgo J, Garcia-Leiva JM, Rico-Villademoros F. Trigger point evaluation in migraine patients: an indication of peripheral sensitization linked to migraine predisposition? Eur J Neurol Off J Eur Fed Neurol Soc. 2006;13(3):244–9.
2. Speciali JG, Gonçalves DA. Auriculotemporal neuralgia. Curr Pain Head Rep. 2005;9:277–80.
3. Stuginski-Barbosa J, Murayama RA, Conti PC, Speciali JG. Refractory facial pain attributed to auriculotemporal neuralgia. J Headache Pain. 2012;13(5):415–7.
4. Martis C, Athanassiades S. Auriculotemporal syndrome (Frey's syndrome), secondary to fracture of the mandibular condyle. Plast Reconstr Surg. 1969;44(6):603–4.
5. Gordon AB, Fiddian RV. Frey's syndrome after parotid surgery. Am J Surg. 1976;132(1):54–8.
6. Ansari H, Robertson CE, Lane JI, Viozzi CF, Garza I. Auriculotemporal neuralgia secondary to TMJ synovial cyst: a rare presentation of a rare entity. Headache. 2013;53(10):1662–5.
7. Chan M, Dmytriw AA, Bartlett E, Yu E. Imaging of auriculotemporal nerve perineural spread. Ecancermedicalscience. 2013;7:374.
8. De Benedittis G. Auriculotemporal syndrome (Frey's syndrome) presenting as tic douloureux. Report of two cases. J Neurosurg. 1990;72(6):955–8.
9. Goldstein DS, Pechnik S, Moak J, Eldadah B. Painful sweating. Neurology. 2004;63(8):1471–5.
10. Ngeow WC, Chai WL. Numbness of the ear following inferior alveolar nerve block: the forgotten complication. Br Dent J. 2009;207(1):19–21.
11. Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders: 2nd edition. Cephalalgia. 2004;24(Suppl 1):9–160.
12. Murayama RA, Stuginski-Barbosa J, Moraes NP, Speciali JG. Toothache referred from auriculotemporal neuralgia: case report. Int Endod J. 2009;42(9):845–51.
13. Damarjian E. Auriculo-temporal neuralgia – an original diagnostic and therapeutic approach. R I Med J. 1970;53(2):100–1.

14. Trescot AM. Headache: is it a migraine? Think again. Technol Reg Anesth Pain Manag. 2005;9:68–72.
15. Frey L. Le syndrome du nerf auriculo-temporal. Rev Neurol (Paris). 1923;2:97–104.
16. Kamath RA, Bharani S, Prabhakar S. Frey's syndrome consequent to an unusual pattern of temporomandibular joint dislocation: case report with review of its incidence and etiology. Craniomaxillofac Trauma Reconstr. 2013;6(1):1–8.
17. Choi HG, Kwon SY, Won JY, Yoo SW, Lee MG, Kim SW, et al. Comparisons of three indicators for Frey's syndrome: subjective symptoms, minor's starch iodine test, and infrared thermography. Clin Exp Otorhinolaryngol. 2013;6(4):249–53.
18. Scrivani SJ, Keith DA, Kulich R, Mehta N, Maciewicz RJ. Posttraumatic gustatory neuralgia: a clinical model of trigeminal neuropathic pain. J Orofac Pain. 1998;12(4):287–92.
19. Loughner BA, Larkin LH, Mahan PE. Nerve entrapment in the lateral pterygoid muscle. Oral Surg Oral Med Oral Pathol. 1990;69(3):299–306.
20. Chim H, Okada HC, Brown MS, Alleyne B, Liu MT, Zwiebel S, et al. The auriculotemporal nerve in etiology of migraine headaches: compression points and anatomical variations. Plast Reconstr Surg. 2012;130(2):336–41.
21. Andersen NB, Bovim G, Sjaastad O. The frontotemporal peripheral nerves. Topographic variations of the supraorbital, supratrochlear and auriculotemporal nerves and their possible clinical significance. Surg Radiol Anat. 2001;23(2):97–104.
22. Janis JE, Hatef DA, Thakar H, Reece EM, McCluskey PD, Schaub TA, et al. The zygomaticotemporal branch of the trigeminal nerve: part II. Anatomic variations. Plast Reconstr Surg. 2010;126(2): 435–42.
23. Janis JE, Hatef DA, Thakar H, Reece EM, McCluskey PD, Schaub TA, et al. The zygomaticotemporal branch of the trigeminal nerve: part II. Anatomical variations. Plast Reconstr Surg. 2010;126(2):435–42.
24. Fernandes PR, de Vasconsellos HA, Okeson JP, Bastos RL, Maia ML. The anatomical relationship between the position of the auriculotemporal nerve and mandibular condyle. Cranio. 2003;21(3): 165–71.
25. Williams PL, Warwick R, editors. Gray's anatomy. 36th ed. Edinburgh: Churchill Livingstone; 1980.
26. Totonchi A, Pashmini N, Guyuron B. The zygomaticotemporal branch of the trigeminal nerve: an anatomical study. Plast Reconstr Surg. 2005;115(1):273–7.
27. Schmidt BL, Pogrel MA, Necoechea M, Kearns G. The distribution of the auriculotemporal nerve around the temporomandibular joint. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1998;86(2):165–8.
28. Anil A, Peker T, Turgut HB, Gulekon IN, Liman F. Variations in the anatomy of the inferior alveolar nerve. Br J Oral Maxillofac Surg. 2003;41(4):236–9.
29. Komarnitki I, Andrzejczak-Sobocinska A, Tomczyk J, Deszczynska K, Ciszek B. Clinical anatomy of the auriculotemporal nerve in the area of the infratemporal fossa. Folia Morphol (Warsz). 2012;71(3):187–93.
30. Kemp WJ, Tubbs RS, Cohen-Gadol AA. The innervation of the scalp: a comprehensive review including anatomy, pathology, and neurosurgical correlates. Surg Neurol Int. 2011;2:178.
31. Shanker H, Brethauer J. Ultrasound guided steroid injection for auriculo-temporal neuralgia (poster presentation). San Francisco: American Society of Anesthesiologists; 2007.
32. Trescot AM. Headache management in an interventional pain practice. Pain Phys. 2000;3(2):197–200.
33. Adams S. Cranial neuralgia and chronic post-traumatic headache with pulsed radio-frequency ablation of the left supraorbital, auriculotemporal and greater and lesser occipital nerves: a case report (poster presentation). Tampa: American Pain Society; 2014.
34. Pomprasit M, Chintrakarn C. Treatment of Frey's syndrome with botulinum toxin. J Med Assoc Thai Chotmaihet Thangphaet. 2007;90(11):2397–402.
35. Simopoulos T, Bajwa Z, Lantz G, Lee S, Burstein R. Implanted auriculotemporal nerve stimulator for the treatment of refractory chronic migraine. Headache. 2010;50(6):1064–9.
36. Deshpande KK, Wininger KL. Feasibility of combined epicranial temporal and occipital neurostimulation: treatment of a challenging case of headache. Pain Phys. 2011;14(1):37–44.
37. Poggi JT, Grizzell BE, Helmer SD. Confirmation of surgical decompression to relieve migraine headaches. Plast Reconstr Surg. 2008;122(1):115–22.
38. Guyuron B, Kriegler JS, Davis J, Amini SB. Five-year outcome of surgical treatment of migraine headaches. Plast Reconstr Surg. 2010;127(2):603–8.
39. Guyuron B, Harvey D, Reed D. A prospective randomized outcomes comparison of two temple migraine trigger site deactivation techniques. Plast Reconstr Surg. 2015;136(1):159–65.

Great Auricular/Posterior Auricular Nerve Entrapment

16

Leonard Benton and Andrea M. Trescot

Introduction

The *posterior auricular nerve* (PAN) is the terminal branch of the *great auricular nerve* (GAN) (also known as the *greater auricular nerve*, which is technically incorrect, since there is no "lesser auricular nerve") and provides innervation along the lower half of the ear and earlobe, the angle of the mandible, and the posterior auricular skin, as well as the side of the neck [1]. There is some confusion in names, since the posterior auricular nerve is sometimes described as arising from the facial nerve at the level of the *stylomastoid foramen*, running cephalad in front of the mastoid, and joining with the posterior branch of the GAN. It has several areas for entrapment and can also be a source of neuritis as a side effect from various types of surgeries including cranial, facial, and those requiring supporting of the neck and head along the base of the skull. It has been recognized that trauma to the PAN or GAN from various surgeries can cause chronic pain, dysesthesia, and/or anesthesia to the affected region. The surgical literature illuminates surgical attempts at recognizing the potential problem and tailoring positioning and surgical practices to reduce the risk. Each of these problems can cause a similar pattern of pain and dysesthesias.

Clinical Presentation (Table 16.1)

Depending on the etiology, several clinical presentations of PAN entrapment can occur. Trescot described fullness in the ear, decreased hearing, tinnitus, and vertigo as possible symptoms (Fig. 16.1), described as "*cryptogenic ear pain,*" mimicking an ear infection [10]. Parietal pain, dysesthesia, and posterior occipital and parietal headaches have also been described as a result of trauma at the mastoid process, entrapment of the posterior auricular nerve by the sternocleidomastoid muscle (SCM), or scar formation from mastoid infections or trauma [10]. This can occur during flexion/extension injuries, especially if the head was turned at impact [10], or with surgical positioning [6]. However, after resection during surgery, hypoesthesia and anesthesia are the symptoms most often mentioned [1–4, 6, 8, 9, 12–14].

Physical findings can include allodynia, hyperpathia, or hypoesthesia. Given that the PAN is sensory only, no muscle weakness should be noted, and electrodiagnostic studies would be expected to be normal.

Interestingly, Brown et al. [1] described that a third of their patients developed gustatory sweating (which is usually associated with auriculotemporal nerve pathology – see Chap. 15) after parotidectomy surgery, with or without PAN-sparing techniques. Several authors have described the hyperesthesia noted with PAN resection during surgery, seen

Table 16.1 Occupation/exercise/trauma history relevant to posterior/great auricular nerve entrapment

Infection	Mastoid or parotid injections
Trauma	Blow to mastoid region
Surgery	Parotidectomy [1–4]; face-lift [5]
Surgical positioning	Beach-chair position for shoulder surgery [6, 7]
Neurosurgical trauma	Craniotomy [8]; endolymphatic shunt [9]
Flexion/extension injuries	[10]
Trigeminal neuralgia	Tic douloureux [11]

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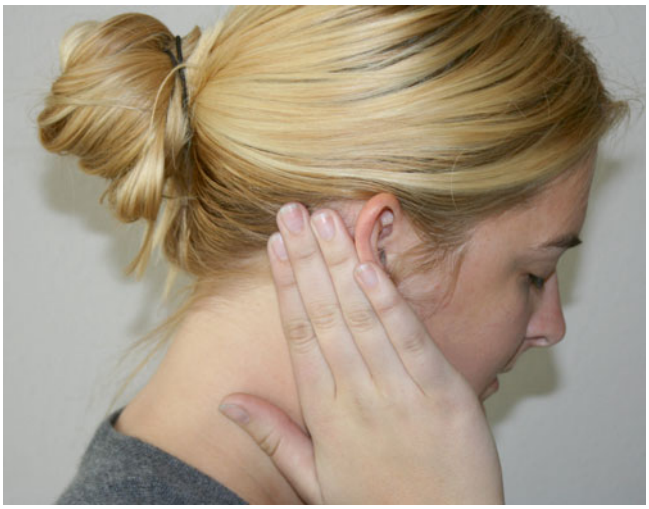

Fig. 16.1 Pattern of pain from posterior auricular neuralgia/great auricular neuralgia (Image courtesy of Andrea Trescot, MD)

in up to one-third of postoperative patients [1]. In those patients who were hypoesthetic after surgery, sensory loss at the angle of the mandible seems to be much better tolerated than sensory loss at the ear [1].

Anatomy (Table 16.2)

The posterior auricular region of the scalp has multiple contributing sensory nerves. The greatest contribution is from the GAN and PAN, which are the focus of this chapter. There is debate as to the use of landmarks to determine points of possible entrapment, given the variability of the various nerves that comprise all or part of the region's innervation [13, 15–18].

The GAN is the largest ascending branch of the *superficial cervical plexus* (Figs. 16.2 and 16.3), providing sensation to the skin over the parotid gland, external ear, angle of the jaw, and posterior auricular region. It arises from ventral branches of spinal nerves C1, C2, C3, and C4 (but primarily C3), branches of which also make up the *lesser occipital* (Chap. 18) and *suprascapular* (Chap. 28) nerves and emerge from behind the posterior border of the *sternocleidomastoid muscle* (SCM) at *Erb's point* (Figs. 16.2 and 16.3), puncturing the deep fascia just lateral to the *lesser occipital nerve*. It climbs the SCM (Fig. 16.4) either in the anterior or posterior surface of the muscle [15] and platysma to the parotid gland and supplies innervation of both sides of the ear (other than the tragus and the concha), the skin over the mastoid, and most of the skin over the parotid.

A study by Tubbs et al. [19] postulated that the mastoid branch of the GAN could be injured by *posterior cranial fossa surgeries* and is also potentially a cause of "*trigeminal neuralgia.*" Lefkowitz et al. [5] dissected 16 heads and identified a consistent relationship between the GAN and the SCM, finding the GAN at its most superficial location one-third the distance from either the mastoid process or the external auditory canal to the clavicle. The GAN can also be found by ultrasound approximately 1 cm superior and lateral to the external jugular vein [20].

Table 16.2 Anatomy of the posterior auricular nerve/great auricular nerve

Origin	C1, C2, C3, and C4 form the superficial cervical plexus
General route	Cervical foramen at C2 and C3 to superficial cervical plexus to posterior border of the SCM
	Anterior branch to parotid region
	Posterior branch to mastoid region
Sensory distribution	Anterior branch – the skin over parotid
	Posterior branch – the skin over mastoid, concha, ear lobe
Motor innervation	None
Anatomic variability	Connections with facial, lesser occipital, and suprascapular nerves
Other relevant structures	PAN may arise from the facial nerve at the stylomastoid foramen

At the inferior pole of the parotid, the GAN branches into an anterior and posterior branch. The anterior branch pierces the parotid and supplies the skin over the parotid gland and the angle of the jaw [21]. This branch is almost always sacrificed with a parotidectomy, and it is at risk during certain face-lift procedures [21]. The posterior branch, also known as the *posterior auricular nerve* (PAN), runs along the posterior border of the SCM, positioned superficially and immediately posterior to the mastoid. The posterior branch supplies the skin over the mastoid process and the angle of the jaw. It also supplies the lateral surface of the concha and ear lobe (Fig. 16.5) [1, 22]. Other innervations to this region include communication with the *lesser occipital nerve* (posteriorly) (Fig. 16.6) and the *transverse cutaneous nerve of the neck* (anteriorly), both of which also arise from C2 and C3 [1], as well as the *auricular branch of the vagus nerve* (*nerve of Arnold)* and the *posterior auricular branch of the facial nerve* [23]. Sand and Becser [16] dissected 17 GANs in 10 cadavers, and they were able to identify the PAN in 13 of the 17 dissections.

The PAN has alternatively been described as arising from the *facial nerve* close to the stylomastoid foramen and running cephalad to the mastoid. Smith et al. [17] dissected 11 hemi-faces; they found that the PAN arose from the facial nerve trunk 1.6–11.1 mm from the stylomastoid foramen, either as a single branch (45.4 %) or from a common trunk that divided into two (36.4 %) or three branches (18.2 %),

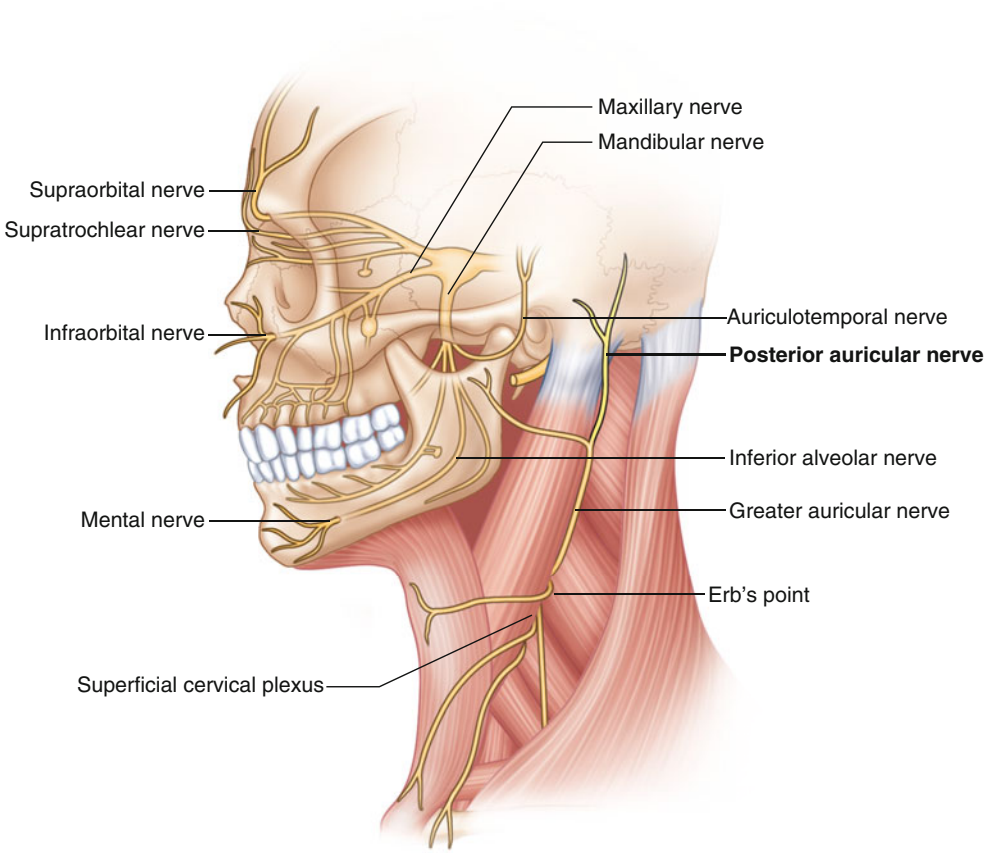

Fig. 16.2 Posterior auricular and great auricular nerve anatomy, lateral view (Image by Springer)

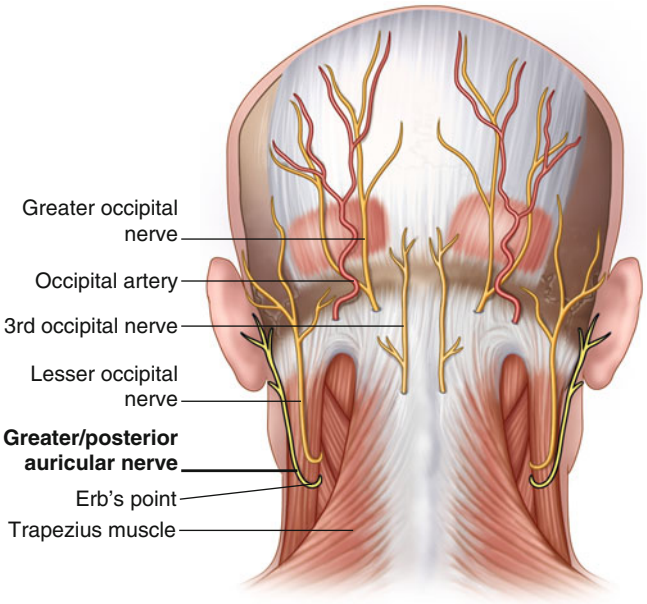

Fig. 16.3 Posterior auricular and great auricular nerve anatomy, posterior view (Image by Springer)

with the other branches passing into the parotid gland. The PAN continued deep (63 %), or lateral to the mastoid process (9.1 %), or through the tissue of the parotid gland (27.3 %).

Becser et al. [15] dissected ten cadavers and found that the GAN can ascend either on the anterior or posterior surface of the SCM. Tubbs et al. [19] found that the GAN can have a mastoid branch that, on average, lies 9 cm lateral to the inion and 1 cm superior to the mastoid tip. Smith et al. [17] evaluated the PAN (they used the terminology PAN for the nerve from the facial nerve trunk) in 11 hemi-faces; the PAN arose as a single branch in 45.4 %, from a common trunk that divided into two branches in 36.4 % or three branches in 18.2 %, with the other branches passing into the parotid gland. Convinced that they have identified a "safe, reliable, and surgically relevant" technique of avoiding GAN injury, Murphy et al. [24] identified the external jugular vein (EJV), the platysma, and the GAN and found the distance between the EJV and GAN was consistently 1.17 cm.

Liaqat and colleagues [25] reviewed 40 patients undergoing third molar extractions; 26 of the 40 patients required GAN injections in addition to the mandibular (inferior alveolar) nerve for analgesia at the angle of the jaw for the extraction.

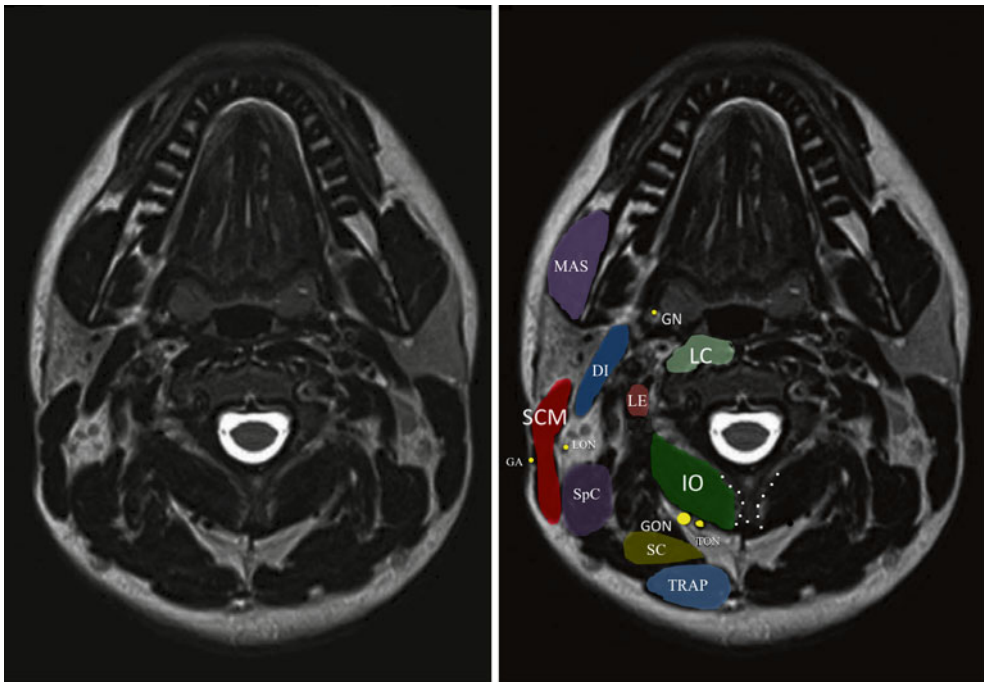

Fig. 16.4 MRI anatomy of the upper cervical region. *DI* digastric muscle, *IO* inferior oblique muscle, *LC* longus colli muscle, *LE* levator scapulae muscle, *MAS* masseter muscle, *SC* semispinalis cervicis muscle, *SCM* sternocleidomastoid muscle, *SpC* splenius capitis muscle, *TRAP* trapezius muscle, *GA* great auricular nerve, *GON* greater occipital nerve, *GN* glossopharyngeal nerve, *LON* lesser occipital nerve, *TON* third occipital nerve. Note the bifid spinous process (Image courtesy of Andrea Trescot, MD)

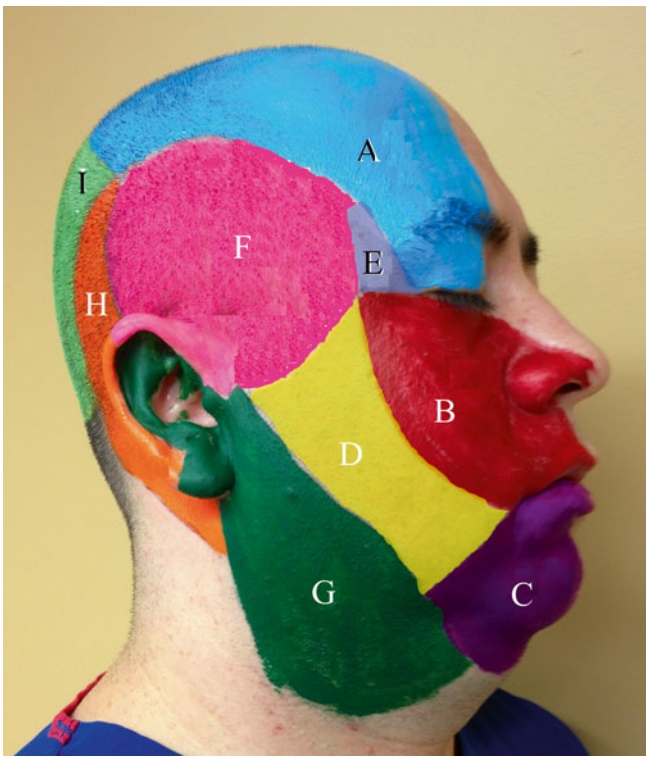

Fig 16.5 Sensory areas of the trigeminal and cervical nerve branches: *A* supraorbital nerve, *B* infraorbital nerve, *C* mental nerve, *D* buccal nerve, *E* lacrimal nerve, *F* auriculotemporal nerve, *G* superficial cervical plexus, *H* posterior auricular nerve/great auricular nerve, *I* occipital nerve (Image courtesy of Terri Dallas-Prunskis, MD)

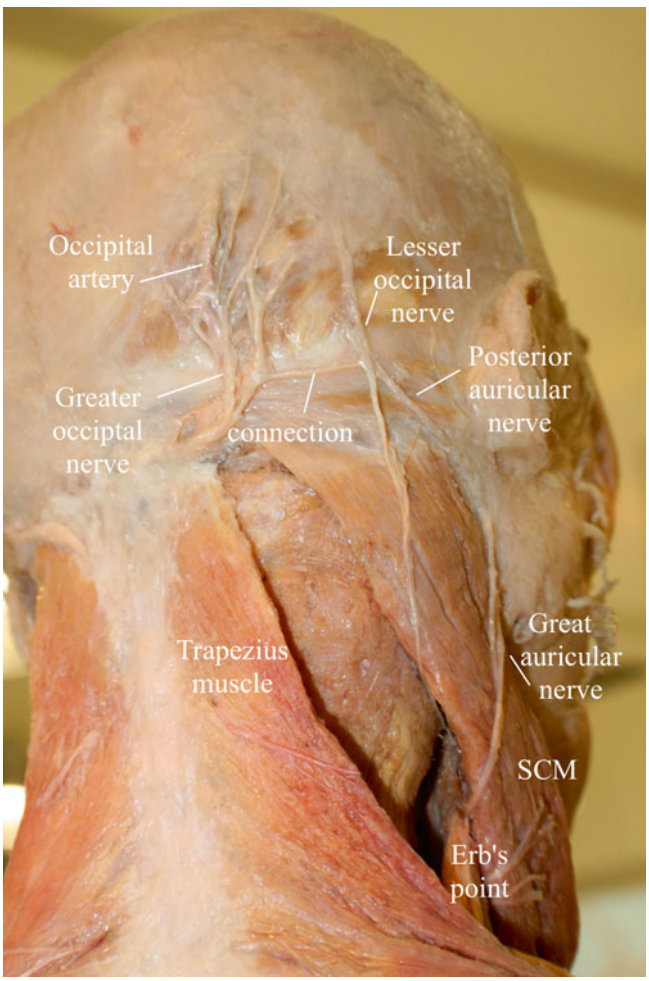

Fig. 16.6 Anatomy of the lateral head, modified from an image from *Bodies*, *The Exhibition*, with permission. Note the connection between the posterior auricular nerve and the lesser occipital nerve, as well as the connection of the great and lesser occipital nerves (Image courtesy of Andrea Trescot, MD)

Entrapment

Entrapment of the PAN can occur along the posterior border of the SCM (Figs. 16.2 and 16.3), in the posterior and inferior extrinsic ligaments, extrinsic auricular musculature, or by scar tissue. More proximal entrapment can occur along the route of the GAN, including the C2–C3 anterior nerve roots, the fascia, the SCM, the superficial cervical plexus, or the platysma, and proximal entrapment would be part of the differential diagnosis if a diagnostic nerve block of the PAN is not effective at relieving symptoms. Ng and Page [6], as well as Park and Kim [7], noted GAN entrapment during shoulder surgery in the beach-chair position, which they attributed to the hard edge of the headrest used, though they also noted that traction, joint distention, and direct compression at other sites might be involved. Park and Kim suggested that the headrest and the ear should be carefully padded to avoid injury [7].

Physical Exam

The physical exam should begin with visualization to look for masses or surgical scars. A thorough neurological exam and full cervical spine exam including extension, flexion, rotation, distraction, etc., should be performed to rule out cervical disk or cervical spinal nerve etiology.

Symptoms of entrapment often can be reproduced by application of digital pressure along the distribution of the posterior auricular nerve, as depicted in Fig. 16.7 (Video 16.1). The head should be supported at the forehead by the non-examining hand or by having the patient rest their forehead on their hands. The palm of the examining hand is placed across the occiput such that the fingers are along the contralateral mastoid and occiput; the thumb is then placed along the mastoid of the affected side. With the thumb, feel for the vertical groove between the mastoid process and the occiput. Application of pressure with the thumb should provoke the patient's symptoms, indicating the general location of the entrapment.

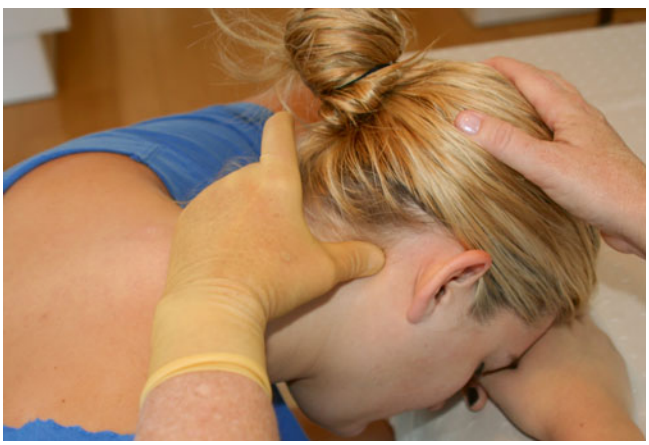

Fig. 16.7 Posterior auricular/great auricular nerve examination (Image courtesy of Andrea Trescot, MD)

Differential Diagnosis (Table 16.3)

The differential diagnosis should be determined from the history and physical exam. If a diagnostic nerve block in the region of the digital palpation of the exam did not provide relief, then one can consider examining and injecting more distally along the GAN. Other possibilities include entrapment along the distribution of the other posterior auricular innervations, including the *lesser occipital nerve*, the *auricular branch of the facial nerve*, and *auricular branch of the vagus* (*nerve of Arnold*) [26, 27]. For example, electrodiagnostic studies could suggest facial nerve etiology, given that the *facial nerve* innervates the *stylohyoid* and *posterior digastric muscles* before it enters the substance of the parotid gland [27].

Differential diagnosis also includes neuropraxia resulting from previous surgery [1, 3, 6, 8, 12, 28]. However, surgical resection usually causes hypoesthesia more often than hyperesthesia, which is usually not as distressing to a patient as pain [1]. Neuroma formation after surgery can cause painful symptoms and allodynia.

High cervical nerve root compression (C1–C4) has been described as causing similar symptoms of chronic otalgia and headaches (Fig. 16.8) [29]. Other symptoms can include pain, hyporeflexia, hypoesthesia, and muscle weakness in the distribution of the nerves, which may be seen on exam. *Otalgia* from cranial nerves (e.g., the *auricular branch of the vagus* or *nerve of Arnold*) [26], *glossopharyngeal neuralgia* (see Chap. 27), or inner ear pathology should also be ruled out [30].

Table 16.4 lists some of the diagnostic tests for the GAN/PAN. Electrodiagnostic and MRI may be beneficial in assessing the cause of the symptoms. Sand and Becser [16] evaluated 77 healthy volunteers and identified that the best site for diagnostic neurostimulation was on the SCM 6–7 cm from the external auditory meatus, noting that the amplitude of the sensory nerve action potential (SNAP) decreased significantly with age.

Table 16.3 Differential diagnosis of ear and parietal pain

	Potential distinguishing features
Inner ear pathology	ENT evaluation
C1 to C4 nerve root pathology	MRI, EMG evidence of radiculopathy
Lesser occipital nerve entrapment	Physical exam (see Chap. 18)
Facial nerve entrapment	EMG
Shingles/postherpetic neuralgia	History of lesions

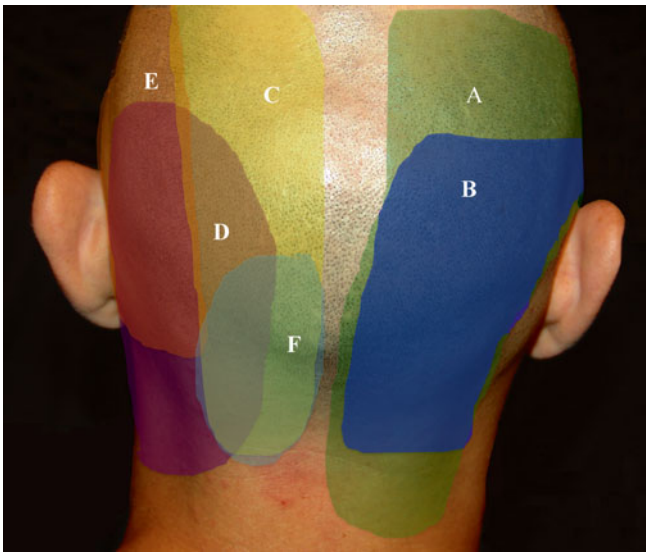

Fig. 16.8 Pattern of posterior cervical and occipital pain. *A* atlantoaxial joint, *B* atlantooccipital joint, *C* greater occipital nerve (GON), *D* posterior auricular nerve (PAN), *E* lesser occipital nerve (LON), *F* third occipital nerve (TON) (Image courtesy of Andrea Trescot, MD)

Fig. 16.9 Landmark-guided posterior auricular/great auricular nerve injection (Image courtesy of Andrea Trescot, MD)

Table 16.4 Diagnostic tests for posterior auricular nerve pathology

	Potential distinguishing features
Physical exam	Tenderness over the mastoid groove
Injection	Diagnostic for the nerve
Ultrasound	The GAN can be identified on the SCM [33]
MRI	Not usually visualized, but Ginsberg et al. [46] described visualization with both MRI and CT
Arteriography	Not visualized
X-rays	Not visualized
Electrodiagnostic studies	GAN conduction studies in normal volunteers showed the best stimulation at the posterior border of the SCM 6–7 cm from the external auditory meatus [16]

Identification and Treatment of Contributing Factors

Posterior auricular neuralgia is often seen weeks to years after blunt injury to the mastoid area. Trescot describes this neuralgia as being seen commonly in physically abused women; the left side is most often involved due to the preponderance of right-handed spouse abusers [10]. The clinical presentation consists of pain in the ear, along with a feeling of "fullness and tenderness." This syndrome is often misdiagnosed as a chronic ear infection [31, 32]. Spasm of the SCM can entrap the GAN and PAN, so flexion/extension injuries, especially if the head was turned at impact, can be associated with GAN/PAN entrapment. Trigger point treatment of the SCM, including myofascial release (see Chap. 5), trigger point injections, and botulinum toxin, can potentially relieve the GAN/PAN entrapment. Avoidance of the GAN/PAN at surgery can prevent subsequent pain and hypoesthesia.

Injection Technique

Landmark-Guided Technique

After location of the entrapment via physical examination, the first course of action is to give a diagnostic or therapeutic injection into the region identified. Aseptic technique should be used, including the use of a povodine, alcohol, or chlorhexidine cleaning solution, and sterile gloves. As depicted in Fig. 16.9 and Video 16.2, straddle the groove of the intended injection site with the index and middle fingers of the gloved, non-injecting hand. Using a 27-g needle directed caudal to cephalad, inject a very small volume (<1 cc) of local anesthetic (and deposteroid if the injection is to be therapeutic as well).

Fluoroscopy-Guided Technique

While the mastoid process is a bony landmark for a fluoroscopically guided injection, fluoroscopy provides no additional benefit over the landmark-guided technique, given the superficial nature of the injection. It also has the downside of ionizing radiation.

Ultrasound (US)-Guided Technique

If the assessment suggests that the entrapment is more proximal along the SCM, US guidance can be used. Several articles describing the US technique for the block of the GAN at the level of the SCM are available in the literature [33, 34]. While a literature search did not find descriptions for finding the PAN with US, distal branches of nerves can be traced using US.

To find the GAN by US, one would trace the nerve as it traverses the SCM as described by Thallaj et al. [33]. They describe having the patient:

> *…in supine position and head turned 45° to the contralateral side. A 6–13 MHz probe is placed on the skin in a transverse plane above the SCM at the level of the cricoid cartilage. After initial placement, the US probe has to be adjusted in a slightly oblique manner for clear identification of the GAN course…* (Fig. 16.10). *We identify the GAN deep to the posterior border of the SCM, on axial view, which appears as a solitary, hypoechoic rounded bobble-shaped structure with hyper-echoic border deep to the posterior border of the SCM with a diameter of 1.68±0.2 mm. When moving the US probe cephalad, the GAN winds around the lateral border of SCM to become anterior; at this site a 22G 50-mm cannula with a facet tip is introduced parallel to the long axis of the US probe (in-plane technique)* (Fig. 16.11) *and 2 mL bupivacaine 0.5% is injected*. The GAN can also be identified 1 cm lateral and superior to the external jugular vein [20].

Neurolytic/Surgical Technique

Cryoneuroablation

Cryoneuroablation has been used to provide prolonged neurolysis of various peripheral nerves when diagnostic and therapeutic blocks give good but short-term relief, as described by Trescot [31].

The PAN runs along the posterior border of the sternocleidomastoid superficially and immediately posterior to the mastoid. Precise localization is performed by the use of sensory and motor stimulation with the cryoprobe. Care must be taken to only anesthetize the superficial skin so as not to prevent localization to sensory stimulation (motor is not as important, given the lack of muscular innervation of the PAN). A 14-gauge intravenous catheter is used as the introducer for the 1.4-mm cryoprobe, taking care to protect the thin skin in this area [31].

Radiofrequency Lesioning

Radiofrequency lesioning and pulsed radiofrequency lesioning have been used to provide prolonged neurolysis for many different peripheral nerves [35–37], though treatment of the PAN/GAN has not been specifically described.

Alcohol/Phenol

Shaheen [38] described the injection of alcohol on the neuroma of the resected GAN to treat intractable hyperesthesia, while Wyburn-Mason [11] described the injection of alcohol onto the GAN to treat *tic douloureux* (*trigeminal neuralgia*).

Peripheral Nerve Stimulation

Peripheral nerve stimulation is a newer, more experimental treatment modality, which may be an option for prolonged relief if the other methods above fail [39–44]. Elahi and Reddy recently described US placement of a GAN peripheral nerve stimulation for a patient with a 4-year history of posttraumatic headaches and temporary relief from GAN injections (Fig. 16.12). These authors recommend the use of US to place the electrode to avoid injury to the spinal accessory nerve (see Chap. 26) and jugular vein.

Surgery

Surgery for possible sectioning of the sensory auricular branch of the facial nerve to treat recalcitrant otalgia has been described [45], although neuroma formation is a concern with ligation of peripheral nerves [9].

Complications

Complications of nerve injections include direct nerve trauma, medication effects, hematoma, and infections. Medication-related complications include steroid-related skin atrophy if the injection is too superficial [10]. Direct trauma to the nerve can result from needle or injection into the perineurium, causing nerve ischemia or disruption. The spinal accessory nerve (Chap. 26) has a very variable path in this part of the neck and can be injured by injections in the area. US permits visualization of the needle relative to the nerve sheath, decreasing this risk for injections more proximal along the GAN.

Due to the superficial nature of the PAN, cryoneuroablation should be used with caution, noting the very thin skin and the ease with which the probe could slide off the skull into the carotid sheath [10]. Radiofrequency lesioning and chemical neurolysis can have unwanted side

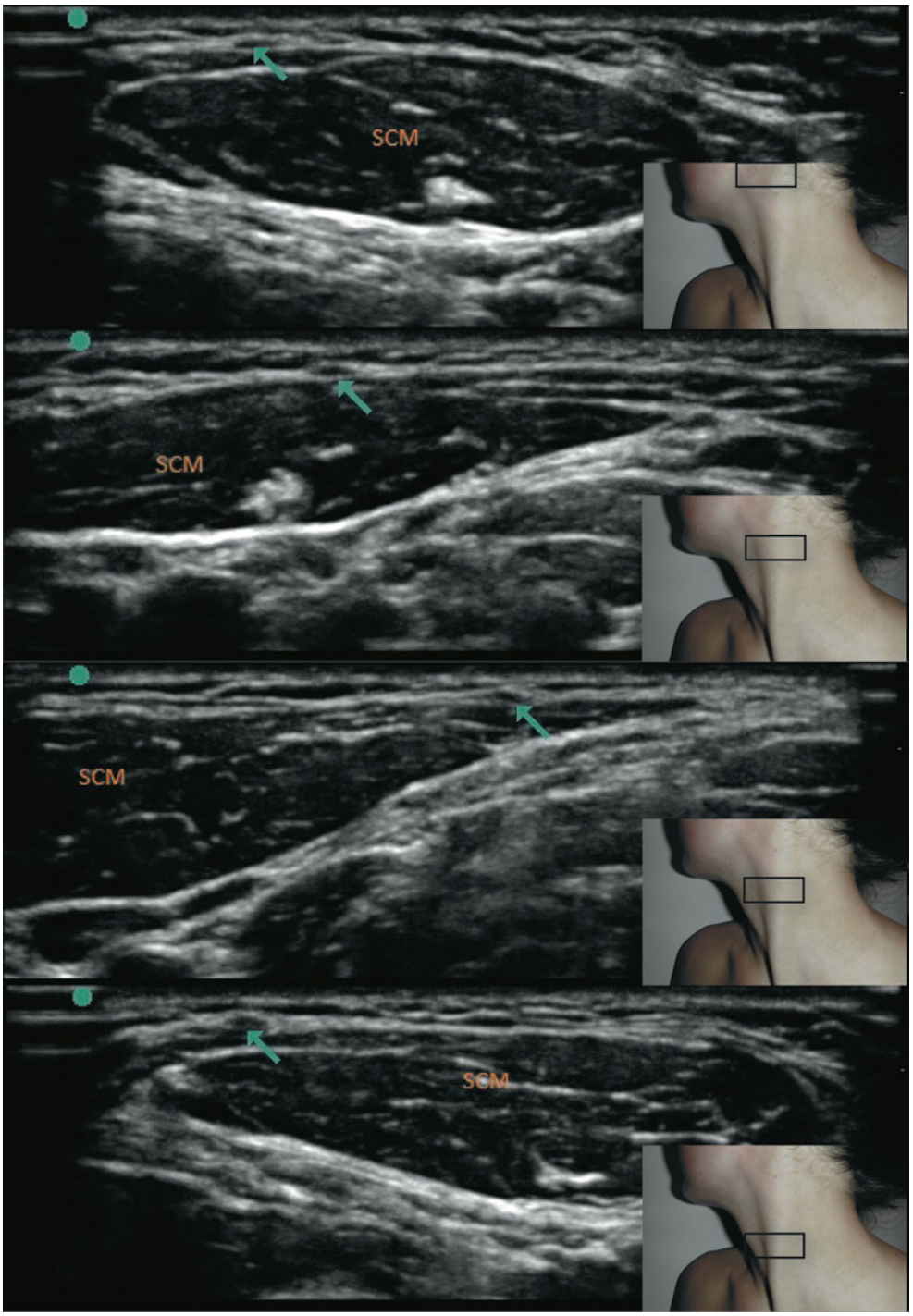

Fig. 16.10 Ultrasound examination of the posterior auricular/great auricular nerve. *SCM* sternocleidomastoid muscle. *Green arrow* = great auricular nerve (Images courtesy of Agnes Stogicza, MD)

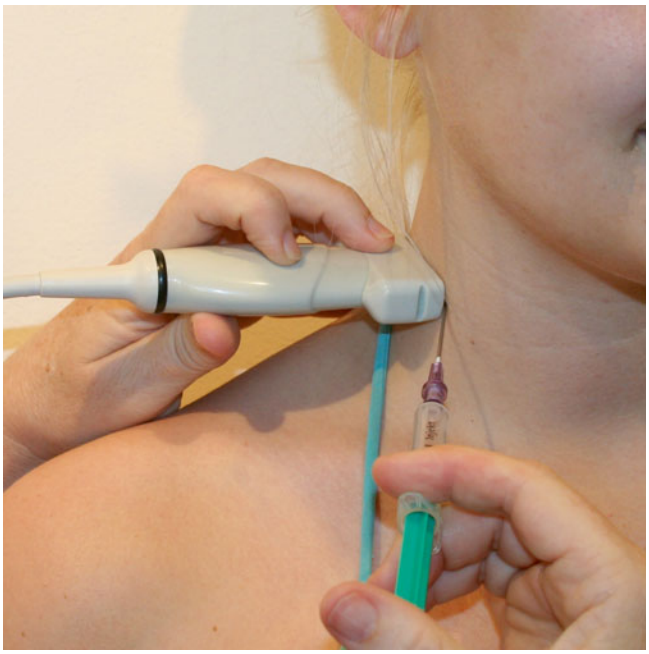

Fig. 16.11 Ultrasound injection of the great auricular nerve (Image courtesy of Andrea Trescot, MD)

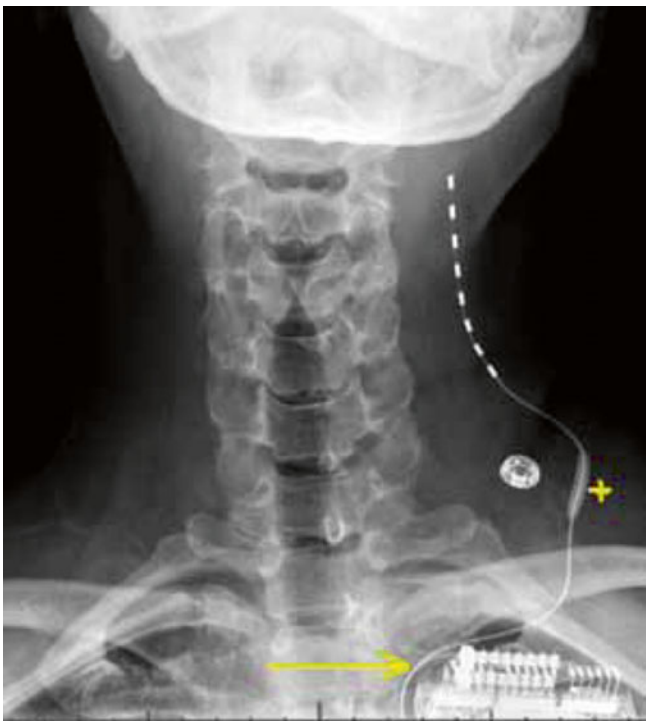

Fig. 16.12 X-ray image of a percutaneous peripheral nerve stimulator placed over the great auricular nerve (From Elahi and Reddy [20]. Reprinted with permission from the American Society of Interventional Pain Physicians)

effects of neuritis [35–37]. As with any injection or procedure, strict antiseptic techniques will limit the risk of infections.

Summary

The great auricular nerve and its branch, the posterior auricular nerve, can cause parietal headaches, potentially triggered by SCM spasm. Recognition of GAN/PAN entrapment can potentially avoid unnecessary and costly ENT and neurology workups.

References

1. Brown JS, Ord RA. Preserving the great auricular nerve in parotid surgery. Br J Oral Maxillofac Surg. 1989;27(6):459–66.
2. Vieira MB, Maia AF, Ribeiro JC. Randomized prospective study of the validity of the great auricular nerve preservation in parotidectomy. Arch Otolaryngol Head Neck Surg. 2002;128(10):1191–5.
3. Hui Y, Wong DS, Wong LY, Ho WK, Wei WI. A prospective controlled double-blind trial of great auricular nerve preservation at parotidectomy. Am J Surg. 2003;185(6):574–9.
4. Hu J, Ye W, Zheng J, Zhu H, Zhang Z. The feasibility and significance of preservation of the lobular branch of the great auricular nerve in parotidectomy. Int J Oral Maxillofac Surg. 2010;39(7):684–9.
5. Lefkowitz T, Hazani R, Chowdhry S, Elston J, Yaremchuk MJ, Wilhelmi BJ. Anatomical landmarks to avoid injury to the great auricular nerve during rhytidectomy. Aesthet Surg J. 2013;33(1):19–23.
6. Ng AK, Page RS. Greater auricular nerve neuropraxia with beach chair positioning during shoulder surgery. Int J Should Surg. 2010;4(2):48–50.
7. Park TS, Kim YS. Neuropraxia of the cutaneous nerve of the cervical plexus after shoulder arthroscopy. Arthroscopy. 2005;21(5):631.
8. de Gray LC, Matta BF. Acute and chronic pain following craniotomy: a review. Anaesthesia. 2005;60(7):693–704.
9. Vorobeichik L, Fallucco MA, Hagan RR. Chronic daily headaches secondary to greater auricular and lesser occipital neuromas following endolymphatic shunt surgery. BMJ Case Rep. 2012;2012.
10. Trescot AM. Headache management in an interventional pain practice. Pain Physician. 2000;3(2):197–200.
11. Wyburn-Mason R. The nature of tic douloureux: treatment by alcohol block or section of the great auricular nerve. Brit Med J. 1953;2:119–22.
12. Patel N, Har-El G, Rosenfeld R. Quality of life after great auricular nerve sacrifice during parotidectomy. Arch Otolaryngol Head Neck Surg. 2001;127(7):884–8.
13. Kemp WJ, Tubbs RS, Cohen-Gadol AA. The innervation of the scalp: a comprehensive review including anatomy, pathology, and neurosurgical correlates. Surg Neurol Int. 2011;2:178.
14. Di Francia R, Papon X, Mercier P. Anatomical study of the superficial cervical plexus: application to the prevention of the postoperative sensitive facial deficits. Morphologie. 2010;95(308):3–9.
15. Becser N, Bovim G, Sjaastad O. Extracranial nerves in the posterior part of the head. Anatomic variations and their possible clinical significance. Spine. 1998;23(13):1435–41.
16. Sand T, Becser N. Neurophysiological and anatomical variability of the greater auricular nerve. Acta Neurol Scand. 1998;98(5):333–9.
17. Smith OJ, Ross GL. Variations in the anatomy of the posterior auricular nerve and its potential as a landmark for identification of the facial nerve trunk: a cadaveric study. Anat Sci Int. 2012; 87(2):101–5.
18. Colbert SD, Davies J, Aldridge T, Brennan PA. Posterior auricular nerve found anterior to the cartilage of the external auditory meatus – a previously unreported variant. Br J Oral Maxillofac Surg. 2012;51(5):448–9.

19. Tubbs RS, Salter EG, Wellons JC, Blount JP, Oakes WJ. Landmarks for the identification of the cutaneous nerves of the occiput and nuchal regions. Clin Anat. 2007;20(3):235–8.
20. Elahi F, Reddy C. Neuromodulation of the great auricular nerve for persistent post-traumatic headache. Pain Physician. 2014;17(4):E531–6.
21. Izquierdo R, Parry SW, Boydell CL, Almand J. The great auricular nerve revisited: pertinent anatomy for SMAS-platysma rhytidectomy. Ann Plast Surg. 1991;27(1):44–8.
22. Peuker ET, Filler TJ. The nerve supply of the human auricle. Clin Anat. 2002;15(1):35–7.
23. Barry M, Bannister LH, Strandring SM. Nervous system. In: Bannister LH, Berry MM, Gray H, Williams PL, editors. Gray's Anatomy. 38th ed. New York, NY: Churchill Livingstone; 1995. p. 901–1937.
24. Murphy R, Dziegielewski P, O'Connell D, Seikaly H, Ansari K. The great auricular nerve: an anatomic and surgical study. J Otolaryngol. 2012;41 Suppl 1:S75–7.
25. Liaqat S, Ehsan A, Bukhari SGA. Efficacy of greater auricular nerve infiltration anesthesia in patients having less than optimal analgesia after conventional inferior alveolar nerve block for surgical removal of mandibular third molar. Pakistan Oral Dental J. 2011;31(2):252–4.
26. Blau JN. Ear pain referred by the vagus. BMJ. 1989;299(6715): 1569–70.
27. Monkhouse WS. The anatomy of the facial nerve. Ear Nose Throat J. 1990;69(10):677–83, 86–7.
28. Ryan WR, Fee Jr WE. Great auricular nerve morbidity after nerve sacrifice during parotidectomy. Arch Otolaryngol Head Neck Surg. 2006;132(6):642–9.
29. Kessler LA, Abla A. Syndrome of the cervical plexus caused by high cervical nerve root compression. Neurosurgery. 1991;28(4): 506–9.
30. Reiss M, Reiss G. Differential diagnosis of otalgia. Schmerz. 1999;13(6):392–7.
31. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
32. Klein DS, Edwards LW, Klein PW. Treatment of cryptogenic earache with posterior auricular nerve block. Albuquerque: AAPM; 1992.
33. Thallaj A, Marhofer P, Moriggl B, Delvi BM, Kettner SC, Almajed M. Great auricular nerve blockade using high resolution ultrasound: a volunteer study. Anaesthesia. 2010;65(8):836–40.
34. Thallaj A. Ultrasound guidance of uncommon nerve blocks. Saudi J Anaesth. 2011;5(4):392–4.
35. Teixeira MJ, Siqueira SR, Almeida GM. Percutaneous radiofrequency rhizotomy and neurovascular decompression of the trigeminal nerve for the treatment of facial pain. Arq Neuropsiquiatr. 2006;64(4):983–9.
36. Navani A, Mahajan G, Kreis P, Fishman SM. A case of pulsed radiofrequency lesioning for occipital neuralgia. Pain Med. 2006;7(5):453–6.
37. Kanpolat Y, Savas A, Bekar A, Berk C. Percutaneous controlled radiofrequency trigeminal rhizotomy for the treatment of idiopathic trigeminal neuralgia: 25-year experience with 1,600 patients. Neurosurgery. 2001;48(3):524–32; discussion 32–4.
38. Shaheen OH. Benign salivary tumours. In: Shaheen OH, editor. Problems in head and neck surgery. London: Balliere Tindall; 1984. p. 70–91.
39. Lenchig S, Cohen J, Patin D. A minimally invasive surgical technique for the treatment of posttraumatic trigeminal neuropathic pain with peripheral nerve stimulation. Pain Physician. 2012;15(5):E725–32.
40. Johnson MD, Burchiel KJ. Peripheral stimulation for treatment of trigeminal postherpetic neuralgia and trigeminal posttraumatic neuropathic pain: a pilot study. Neurosurgery. 2004;55(1):135–41; discussion 41–2.
41. Verrills P, Vivian D, Mitchell B, Barnard A. Peripheral nerve field stimulation for chronic pain: 100 cases and review of the literature. Pain Med. 2011;12(9):1395–405.
42. Mobbs RJ, Nair S, Blum P. Peripheral nerve stimulation for the treatment of chronic pain. J Clin Neurosci Off J Neurosurg Soc Aust. 2007;14(3):216–21; discussion 22–3.
43. Nashold Jr BS, Goldner JL, Mullen JB, Bright DS. Long-term pain control by direct peripheral-nerve stimulation. J Bone Joint Surg Am. 1982;64(1):1–10.
44. Huntoon MA, Burgher AH. Ultrasound-guided permanent implantation of peripheral nerve stimulation (PNS) system for neuropathic pain of the extremities: original cases and outcomes. Pain Med. 2009;10(8):1369–77.
45. Ulubil SA, Eshraghi AA, Telischi FF. Sectioning the sensory auricular branch of the facial nerve to treat recalcitrant otalgia. Otol Neurotol. 2009;30(4):522–4.
46. Ginsberg LE, Eicher SA. Great auricular nerve: anatomy and imaging in a case of perineural tumor spread. AJNR Am J Neuroradiol. 2000;21(3):568–71.

Greater Occipital Nerve Entrapment

17

Andrea M. Trescot, Esther Rawner, and David M. Irwin

Introduction

The greater occipital nerve (GON) entrapment commonly causes occipital and cervicogenic headaches and can sometimes cause headaches with characteristics similar to migraine headaches. The anatomic recognition of multiple occipital nerves (the *greater occipital*, the *lesser occipital*, and the *third occipital nerve*), each with different patterns of pain, neurologic origin, and treatment modalities, has been facilitated by the use of precision diagnostic injections. This chapter will focus specifically on the greater occipital nerve (GON); see Chap. 18 for the lesser occipital nerve (LON) and Chap. 19 for the third occipital nerve (TON). There are multiple names used for these nerve entrapments, including "*migraine,*" "*tension headache,*" "*cervicogenic headaches*" [1], and "*occipital myalgia-neuralgia syndrome*" [2]. Sometimes the term "*suboccipital injections*" has been used to describe greater occipital nerve injections, but for this book, the authors use "suboccipital" to mean the C1 nerve root or the high volume occipital decompression described in Chap. 20. Occipital neuralgia is also known as *C2 neuralgia* or *Arnold's neuralgia*.

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Clinical Presentation (Table 17.1)

Beuto et al. [15] described *occipital neuralgia* (ON) in 1821 as a sharp, lightning-bolt pain radiating from the occiput to the vertex. The medical terms *occipital neuralgia* and *cervicogenic headache* describe a syndrome of neck and head pain primarily referring to the occiput, as well as the temporal area, forehead, and retro-orbital areas, that may arise some distance away, in the upper cervical spine. Greater occipital neuralgia characteristically presents as paroxysmal shooting, stabbing pain from the suboccipital region to the vertex (Fig. 17.1). The cutaneous innervation of the posterior neck and occiput comes primarily from C1, C2, and C3, which provide overlapping dermatomes (Fig. 17.2). C2 covers the occiput, neck, and submental regions, while the C3 dermatome can span from the clavicle to the mandible to behind and over the ear, pinna, and the angle of the mandible [16]. Since the greater occipital nerve is made up of contributions from C1, C2, and C3, there can be a wide range of clinical presentations. As a subset of cervicogenic headaches (CGH), occipital neuralgia can cause pain and paresthesias to the posterior scalp; the periorbital, temporal, and mandibular regions; and the external ear and mastoid regions, as well as pain in the neck and shoulders. The first three cervical spinal nerve segments (C1–C3) that make up the occipital nerves share a relay station in the brainstem that continues into the upper cervical spinal cord with the trigeminal cell bodies (the *cervico-trigeminal complex*). The pain of occipital neuralgia and cervicogenic headaches can therefore be referred to structures innervated by the branches of the trigeminal nerve, namely, the forehead, temples, and eyes (Fig. 17.3).

There are three occipital nerves – the greater, the lesser, and the third occipital nerves (Fig. 17.4). Their patterns of pain are overlapping, and so the reader is encouraged to review Chaps. 18 and 19 as well.

Unilateral occipital neuralgia, perhaps because of the proximity of the occipital artery (see below), can present

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Table 17.1 Occupation/exercise/trauma history relevant to greater occipital nerve entrapment

Flexion/extension injuries	"Migraine" after motor vehicle collision [3]
	Postconcussive headache [4]
Manual labor	Levator scapula spasm causing cervical rotation and dysfunction [5]
	Excessive spasm of the cervical muscles
Improper ergonomics	"Desk job" with a computer [6] with head-forward positioning
Upper cervical surgery	Atlantoaxial screw fixation [7]
Compression	Anomalous vertebral artery [8]
	Schwannoma [9]
	Occipital nerve piecing through inferior oblique [10]
	Hypertrophy venous plexus at C2 [10]
Nerve injury	Herpes zoster [11]
Inflammation	C12 arthritis [10]
Myelitis	Cervical myelitis presenting as occipital neuralgia [12]
Cluster headache	"Suboccipital steroid injection" [13]
Postdural puncture headache	Persistent headache despite blood patch may result from occipital entrapment [14]

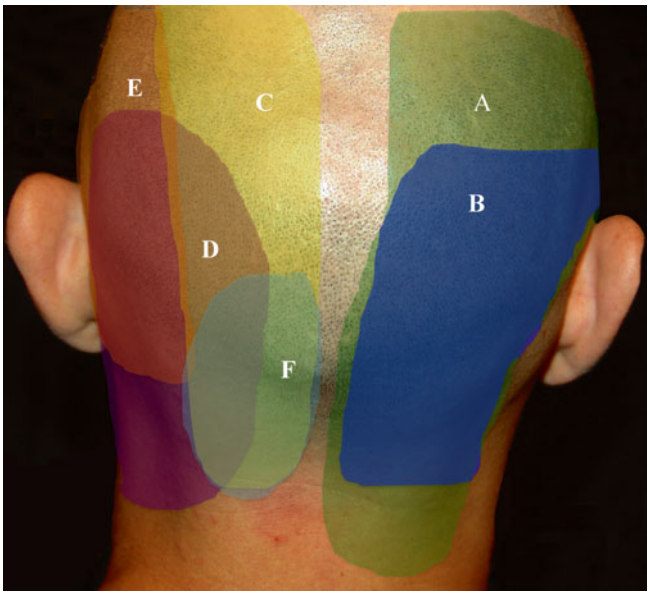

Fig. 17.2 Pattern of posterior cervical and occipital pain. *A* atlantoaxial joint, *B* atlantooccipital joint, *C* greater occipital nerve (GON), *D* posterior auricular nerve (PAN), *E* lesser occipital nerve (LON), *F* third occipital nerve (TON) (Image courtesy of Andrea Trescot, MD)

Fig. 17.1 Pattern of greater occipital pain (Image courtesy of Andrea Trescot, MD)

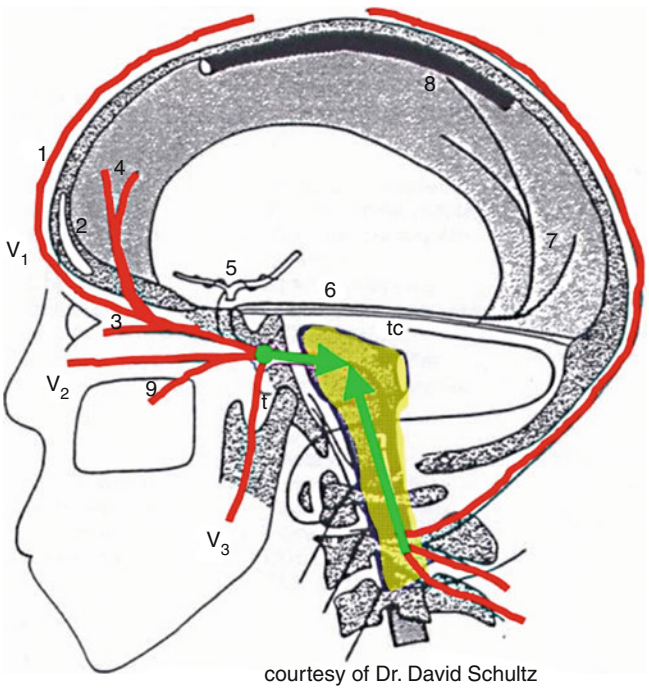

Fig. 17.3 Cervico-trigeminal relay, showing the relationship between the trigeminal and occipital/upper cervical nerve roots (Image courtesy of David Schultz, MD)

with throbbing, unilateral headaches associated with photophobia, phonophobia, and nausea, which will meet the International Headache Society (IHS) criteria for migraines [17]. There may be a history of occipital pain, often radiating to the face, specifically described as "behind my eye" and "like an ice pick." Occipital headaches may start as "tension headaches" in the upper cervical region but then center at the base of the skull and produce throbbing and unilateral or

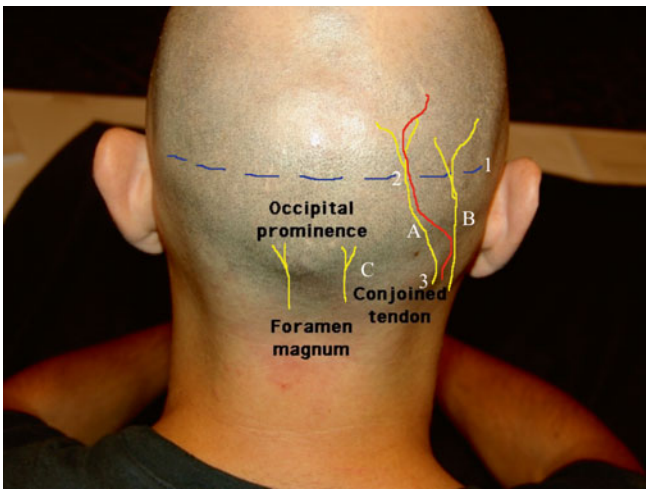

Fig. 17.4 Surface anatomy of the occipital nerves. *A* Greater occipital nerve, *B* Lesser occipital nerve, *C* 3rd occipital nerve, *1* Occipital ridge, *2* Injection at occipital ridge (classic approach), *3* Injection site for Trescot approach (Image courtesy of Andrea Trescot, MD)

bilateral pain, accompanied by nausea, photophobia, and phonophobia. These symptoms meet the International Classification of Headache Disorders (ICHD) [18] criteria for migraine headache. Occipital neuralgia has a close relationship with *cluster headaches*, with several authors [19–21] describing the use of occipital blocks to treat cluster headaches. *Hemicrania continua* [22] and "*transformed migraines*" [23] have also responded to occipital nerve blocks.

Anatomy (Table 17.2)

The largest of the three occipital nerves, the GON arises from the posterior ramus of C2 that runs inferiorly between the arch of C1 (atlas) and the lamina of C2 (axis) (Fig. 17.5), lateral to the lateral atlantoaxial (AA) joint, deep to the inferior oblique muscle (Fig. 17.6). The GON then curves up over the inferior oblique, between the *inferior oblique* and the *semispinalis capitis* muscles (Fig. 17.7). A branch from C3 may join at this point, as the nerve ascends up the neck and over the dorsal surface of the rectus capitis to pierce the semispinalis capitis muscle, deep to the *trapezius muscle* (Fig. 17.8). The GON then exits the neck through a muscular sling formed by the aponeurosis of the *sternocleidomastoid muscle* (SCM) and trapezius muscle at their attachment on the occipital bone (the *conjoined tendon*) (Fig. 17.7), where it is joined laterally by the occipital artery (Fig. 17.8). At this point, the greater occipital nerve is immediately medial to the occipital artery and lateral to the *inion* or *occipital prominence*, lying in a palpable groove. This proximity to the artery may account for the throbbing sensation that often accompanies GON entrapment.

Table 17.2 Greater occipital nerve anatomy

Origin	Medial branch of the posterior ramus of C2
General route	Inferiorly between the axis and atlas, curves up over inferior oblique muscle, through the semispinalis capitis muscle, between the SCM and trapezius, and then up the back of the head to the vertex
Sensory distribution	Occiput to vertex, portions of the ear, and the parotid gland
Motor innervation	None
Anatomic variability	May join with the lesser occipital or the suboccipital nerve with rare connection to trigeminal V2 or V3 [24]
Other relevant structures	Travels with the occipital artery cephalad of the SCM/trapezius junction (conjoined tendon)

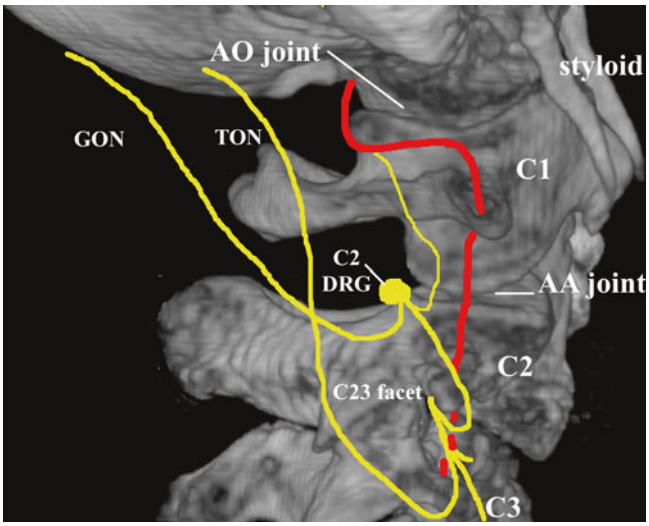

Fig. 17.5 Anatomy of the C2 and C3 nerve roots. *GON* greater occipital nerve, *TON* third occipital nerve, *C2 DRG* C2 dorsal root ganglion. Note the vertebral artery in *red* (Image courtesy of Andrea Trescot, MD)

The GON could therefore be considered to have three parts (Part 1, Part 2, and Part 3) as well as two bends (A1 and A2, where entrapments occur) [10] (Fig. 17.9). A cutaneous branch of the *suboccipital nerve* (the C1 dorsal ramus) will occasionally join the GON as it accompanies the occipital artery. The GON frequently connects with the lesser occipital nerve (see Chap. 18) (Fig. 17.10), which arises from the cervical plexus (formed by the upper four ventral cervical rami). The GON continues to ascend to innervate the skin along the posterior portion of the occiput to the vertex, portions of the ear, and parotid glands.

Shimizu et al. [25] looked at the suboccipital region of 24 cadaver heads; the occipital nerve was found to cross over the artery, with the nerve consistently indented by the artery but without histologic evidence of mechanical damage.

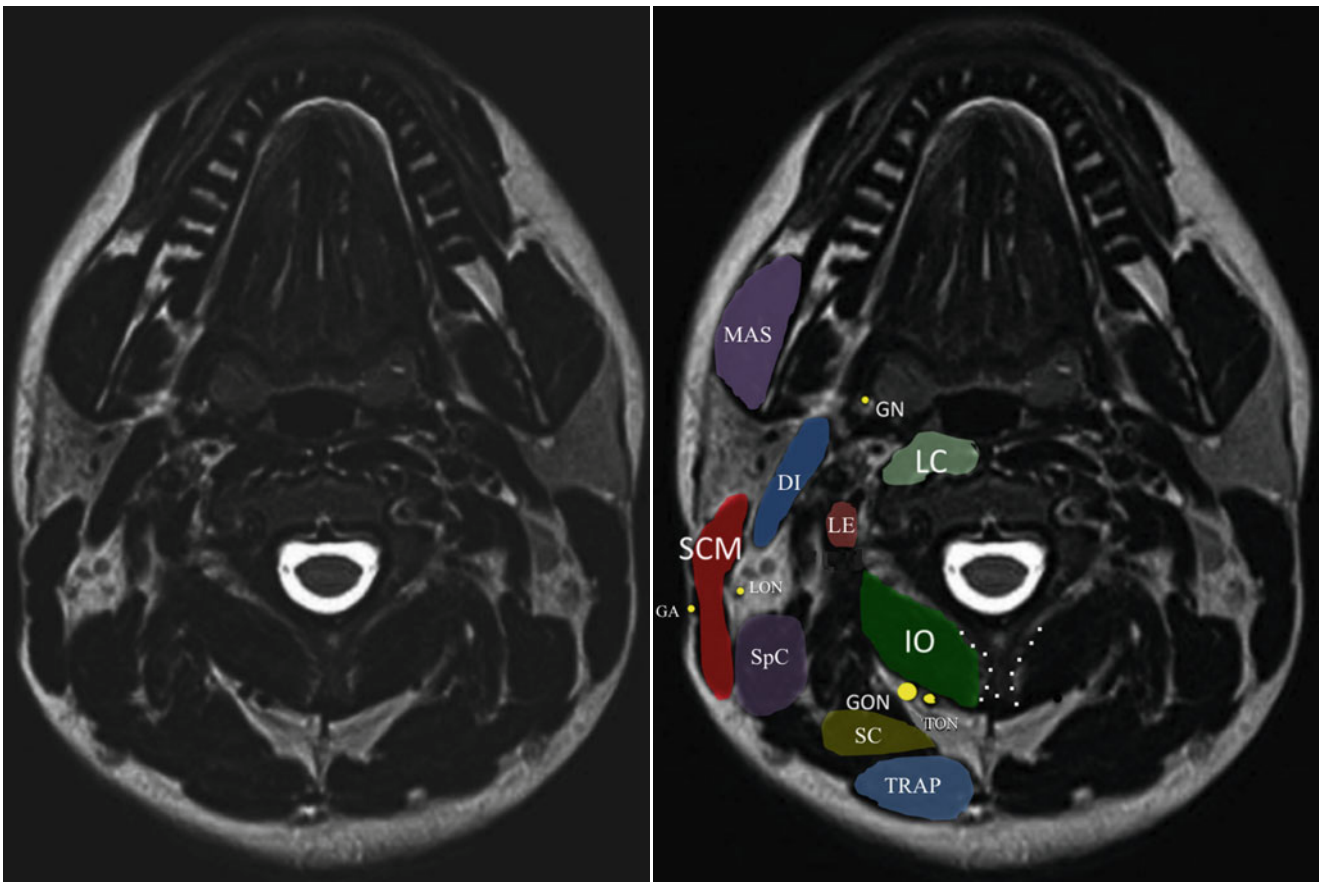

Fig. 17.6 MRI anatomy of the upper cervical region. *DI* digastric muscle, *IO* inferior oblique muscle, *LC* longus colli muscle, *LE* levator scapulae muscle, *MAS* masseter muscle, *SC* semispinalis cervicis muscle, *SCM* sternocleidomastoid muscle, *SpC* splenius capitis muscle, *TRAP* trapezius muscle, *GA* great auricular nerve, *GON* greater occipital nerve, *GN* glossopharyngeal nerve, *LON* lesser occipital nerve, *TON* third occipital nerve. Note the bifid spinous process (Image courtesy of Andrea Trescot, MD)

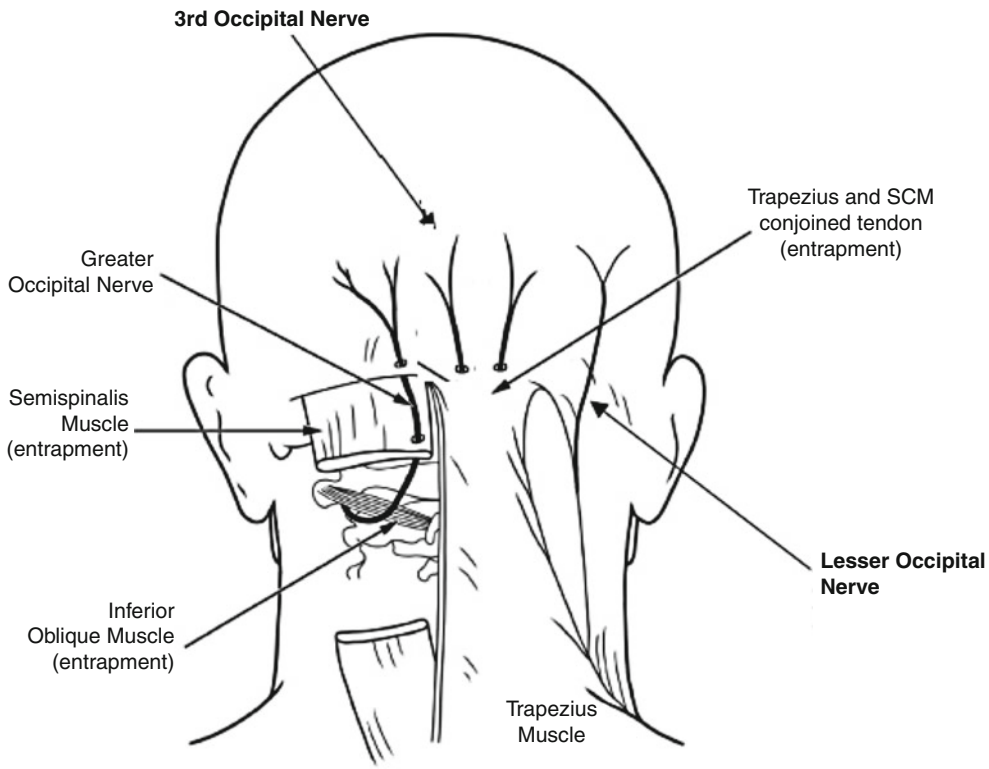

Fig. 17.7 Relationship of the occipital nerves with sites of entrapment (Image courtesy of Epimed International, with permission)

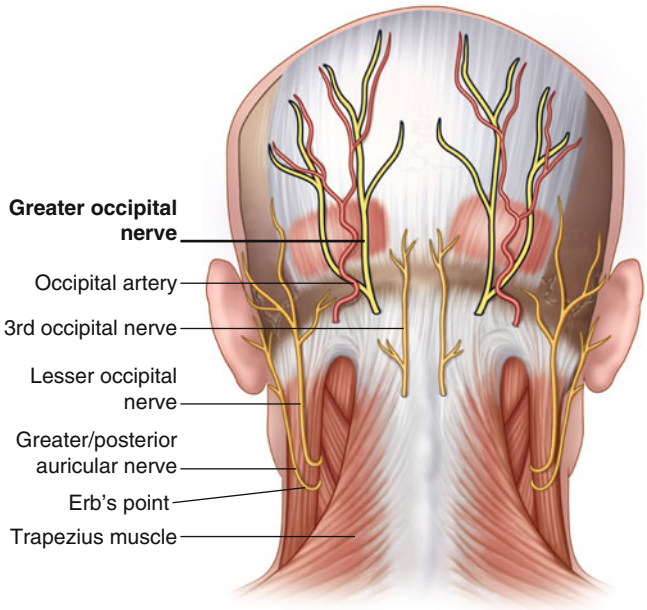

Fig. 17.8 Occipital anatomy (Image by Springer)

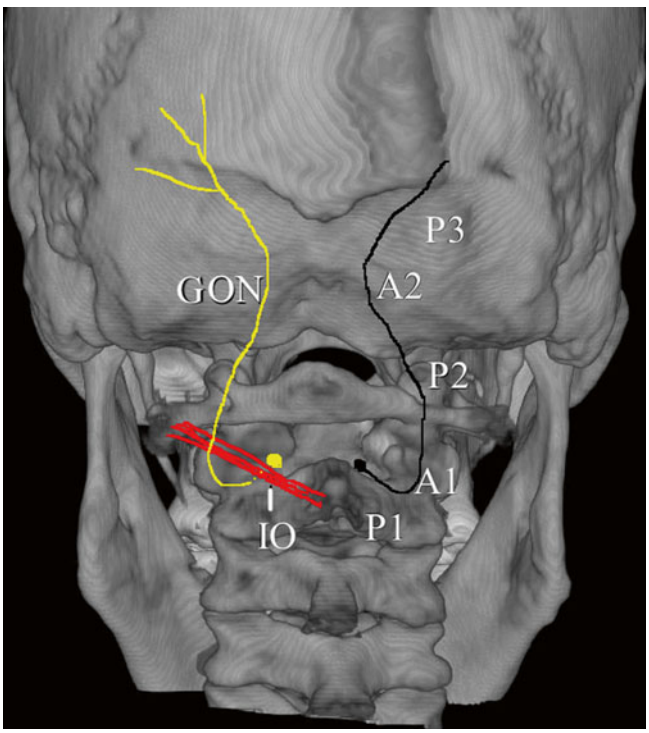

Fig. 17.9 The path of the occipital nerve (Modified from Gille [9]). *GON* greater occipital nerve, *IO* inferior oblique muscle, *P1* Part 1 of the occipital nerve, *P2* Part 2 of the occipital nerve, *P3* Part 3 of the occipital nerve, *A1* site of entrapment by the inferior oblique, *A2* site of entrapment by the trapezius muscle (Image courtesy of Andrea Trescot, MD)

Natsis el al. [26], based on the dissections of 40 cadavers (29 female and 11 males), noted that the course of the GON did not differ between males and females. There was a left/right

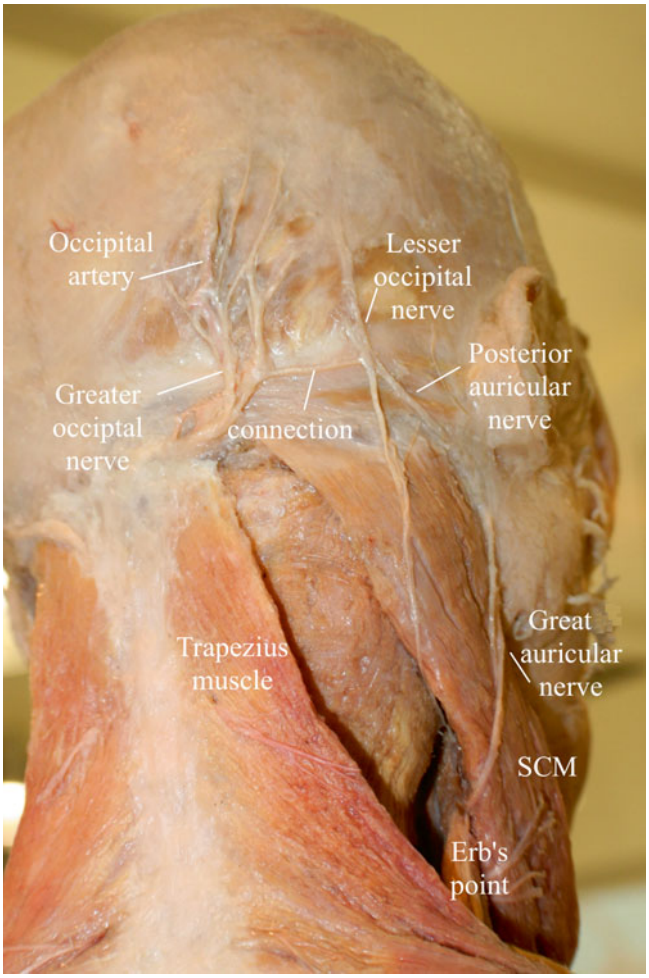

Fig. 17.10 Anatomy of the occipital region, modified from an image from *Bodies*, *The Exhibition*, with permission. Note the connection of the greater and lesser occipital nerves (Image courtesy of Andrea Trescot, MD)

difference noted in the site of the GON in the trapezius region but not the SCM. The nerve was noted to get wider as it moved toward the periphery, and, in three cases, the nerve split into two branches before piercing the trapezius. The GON and lesser occipital nerves were reunited at the level of the occiput in 80 % of the specimens.

In order to evaluate the most appropriate site for injections for headaches, Mosser et al. [27] dissected 14 cadaver heads and determined the location of the emergence of the occipital nerve from the semispinalis to be approximately 3 cm below the occipital protuberance and 1.5 cm lateral to the midline. Becser et al. [28] dissected ten cadavers and found that the GON ascended between 5 and 28 mm from the midline along the intermastoid line; 12 of the 20 GONs wrapped around the occipital artery. Loukas et al. [29] dissected 100 formalin-fixed adult cadavers and found the GON to be at a mean distance of 3.8 cm lateral to a vertical line through the external occipital protuberance and the spinous

processes of C2 through C7. It was also 41 % of the distance along the intermastoid line (a line connecting the medial aspects of the mastoid processes) and 22 % of the distance between the external occipital protuberance and the mastoid process.

Dash et al. [30] looked at 19 cadavers and noted that all of the GONs identified were found to pierce the semispinalis capitis. On the other hand, in the 20 cadavers that Bovim et al. [31] dissected (none of whom had a history of headaches), 10 specimens had the occipital nerve piercing the trapezius muscle on one side but not the other. They also identified 11 nerves with macroscopic signs of possible nerve compression from fibrotic tissue, with 6 compressions accompanied by a "kink" of more than 90°. Additionally, three of the 40 occipital nerves penetrated the inferior oblique. They also noted a marked variation of the amount of venous plexus at the level of C2; 50 % of the cases had the C2 nerve root surrounded by a marked vascular network. They noted a significant variation in the path of the occipital nerve, but they also remarked that, for many of the dissections, there was a significant difference noted within the two sides of the same individual.

Entrapment

There are several areas of entrapment of the occipital nerve: (1) where the greater occipital nerve emerges from the C2 dorsal root ganglia (DRG), between the atlas and axis (Fig. 17.11), (2) between the inferior oblique and the semispinalis capitis muscles (Fig. 17.7), (3) where the nerve pierces the semispinalis capitis (Fig. 17.7), and (4) where the nerve exits from the aponeurosis of the trapezius (Fig. 17.8). Because the GON pierces the nuchal fascia at the base of the skull, it is prone to trauma from flexion/tension injuries, which can lead to entrapment by a spasm of the trapezius muscle. Repetitive neck contractions secondary to work, recreational activities, and many other activities can cause entrapment and/or scarring of the GON. The trapezius or sternocleidomastoid muscles can cause entrapment secondary to myofascial spasms. Head-forward positions can entrap the GON at the level of the inferior oblique (Fig. 17.12), so posture is very important.

Medical conditions such as osteochondroma (benign tumors of the bone) [32], arterial compression of the C2 nerve root by a tortuous vertebral artery (Fig. 17.13), herpes zoster [33], osteoarthritis or rheumatoid arthritis (especially at C1–C2) [34, 35], or trauma such as blows to the back of the head or posterior fossa surgery and many others contribute to or are the root cause of entrapment or are the contributing factors causing pain. However, since the ganglion of this nerve interconnects with the trigeminal nerve in the brainstem

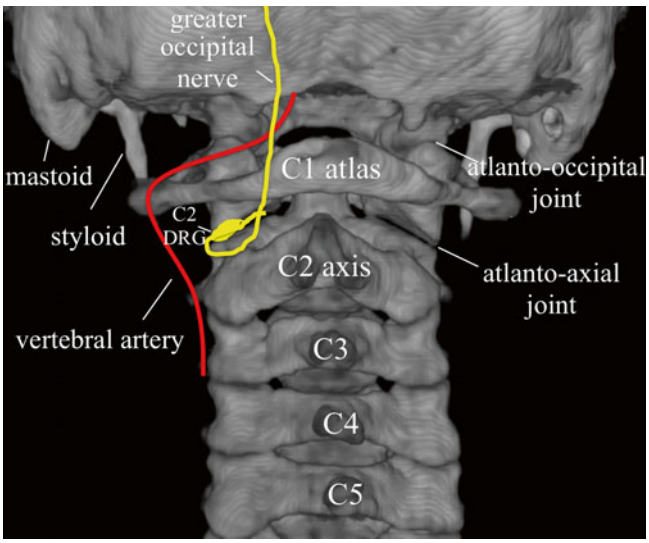

Fig. 17.11 Anatomy of the posterior cervical region, showing the relationship between the vertebral artery, the C2 dorsal root ganglion (DRG), and the greater occipital nerve (Image courtesy of Andrea Trescot, MD)

(Fig. 17.3), pain may be referred to any branch of the trigeminal nerve. The best way to diagnose an occipital nerve entrapment is by physical exam and injection.

Physical Exam

For the physical exam of the greater occipital nerve (Video 17.1), the patient should be positioned sitting with the neck slightly flexed, with the examiner supporting the forehead with the non-examining hand (Fig. 17.14a). With the examining hand, place the middle finger at the midline base of the head to identify the site of the foramen magnum (Fig. 17.14b). The index finger is placed at the conjoined tendon attachment (Fig. 17.14c). The thumb will then palpate the area just lateral to the conjoined tendon, approximately 3 cm inferior and 1.5 cm lateral to the occipital inion (Fig. 17.14d). If an area of paresthesia is created, reproducing the patient's pain and symptomatology, the GON is likely the source.

Differential Diagnosis (Table 17.3)

There is much confusion and controversy regarding the exact location and correct term for headaches that affect the occipital or suboccipital regions of the head with or without radiation to the frontal, temporal, or ocular areas. Headaches described as "*tension headaches*" do not often respond to occipital nerve injections [40]. *Hemicrania continua* (HC) is a headache that by definition needs to have a complete response to indomethacin, though Guerrero et al. [41]

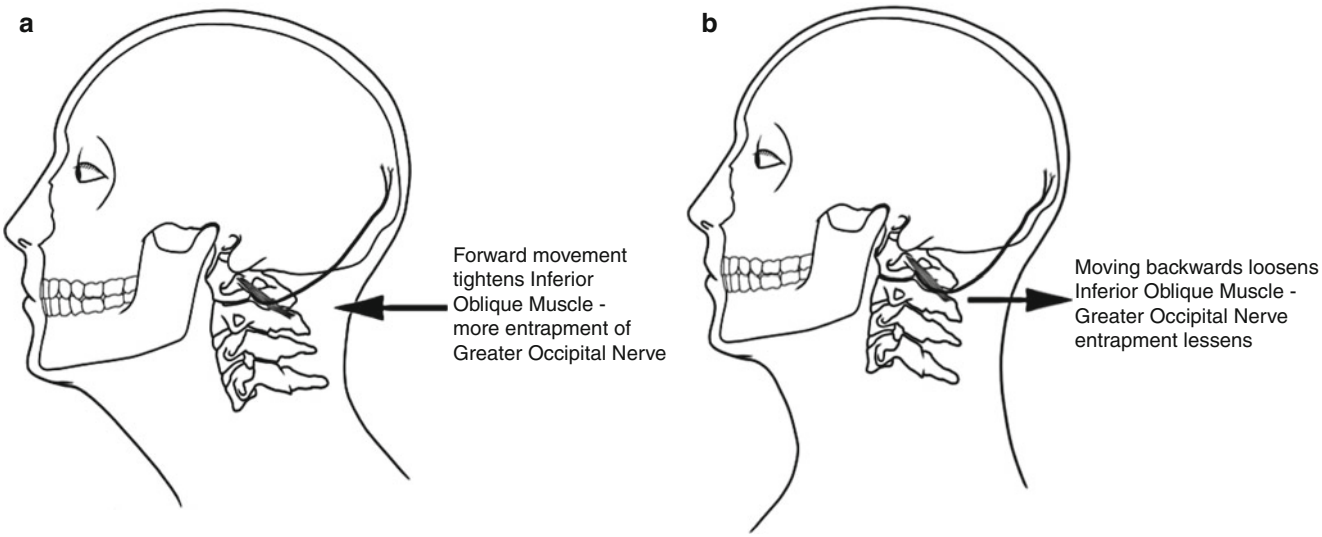

Fig. 17.12 Ergonomic issues of head position on occipital nerve entrapment. (**a**) head forward position causing entrapment of the greater occipital nerve; (**b**) posterior movement of the chin releasing the entrapment. (Images courtesy of Epimed International, with permission)

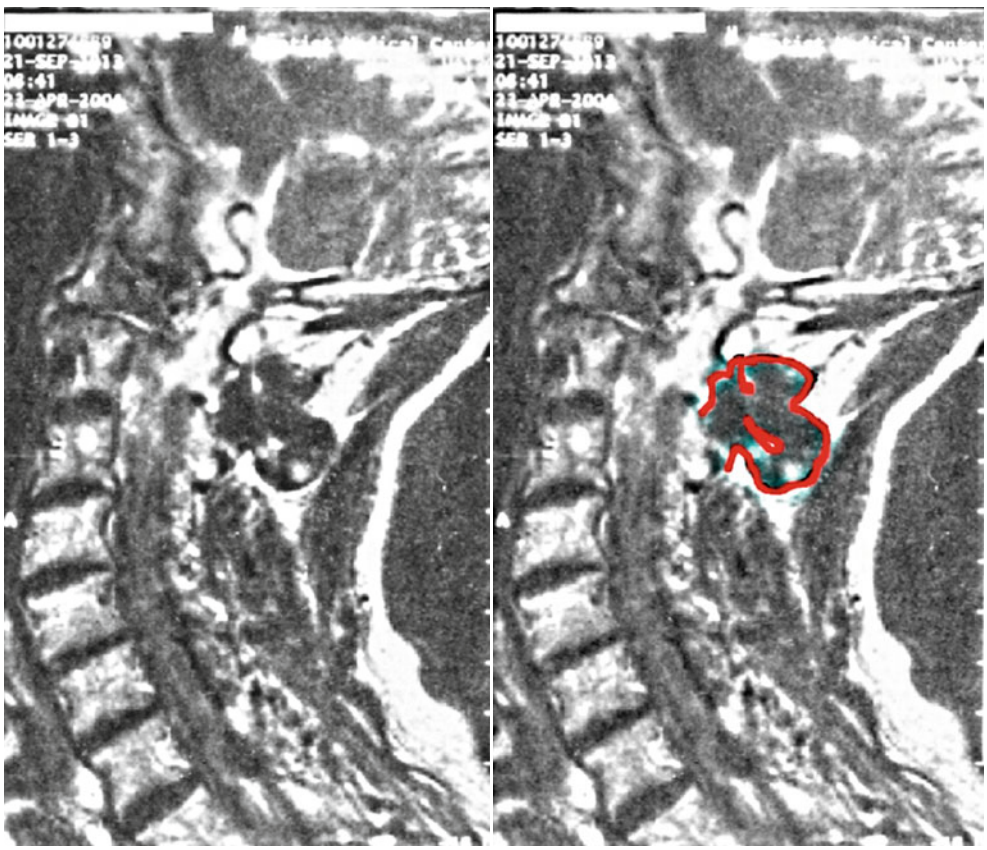

Fig. 17.13 MRI showing tortuous vertebral artery (Image courtesy of Andrea Trescot, MD)

described 14 HC patients who underwent supraorbital (see Chap. 14) and/or occipital nerve blocks due to indomethacin intolerance. Nine of these patients noted total or partial improvement for 2–10 months, and the authors recommend that HC patients be examined for possible injection therapy.

Occipital pain may be considered the result of referred pain from the posterior cervical muscles, the cervical ligaments, the C1 or C2 spinal nerve root, the C2/C3 zygapophysial joint, or the third occipital nerve [42]. It is also important to consider a vertebral dissection in patients with a "thunder-

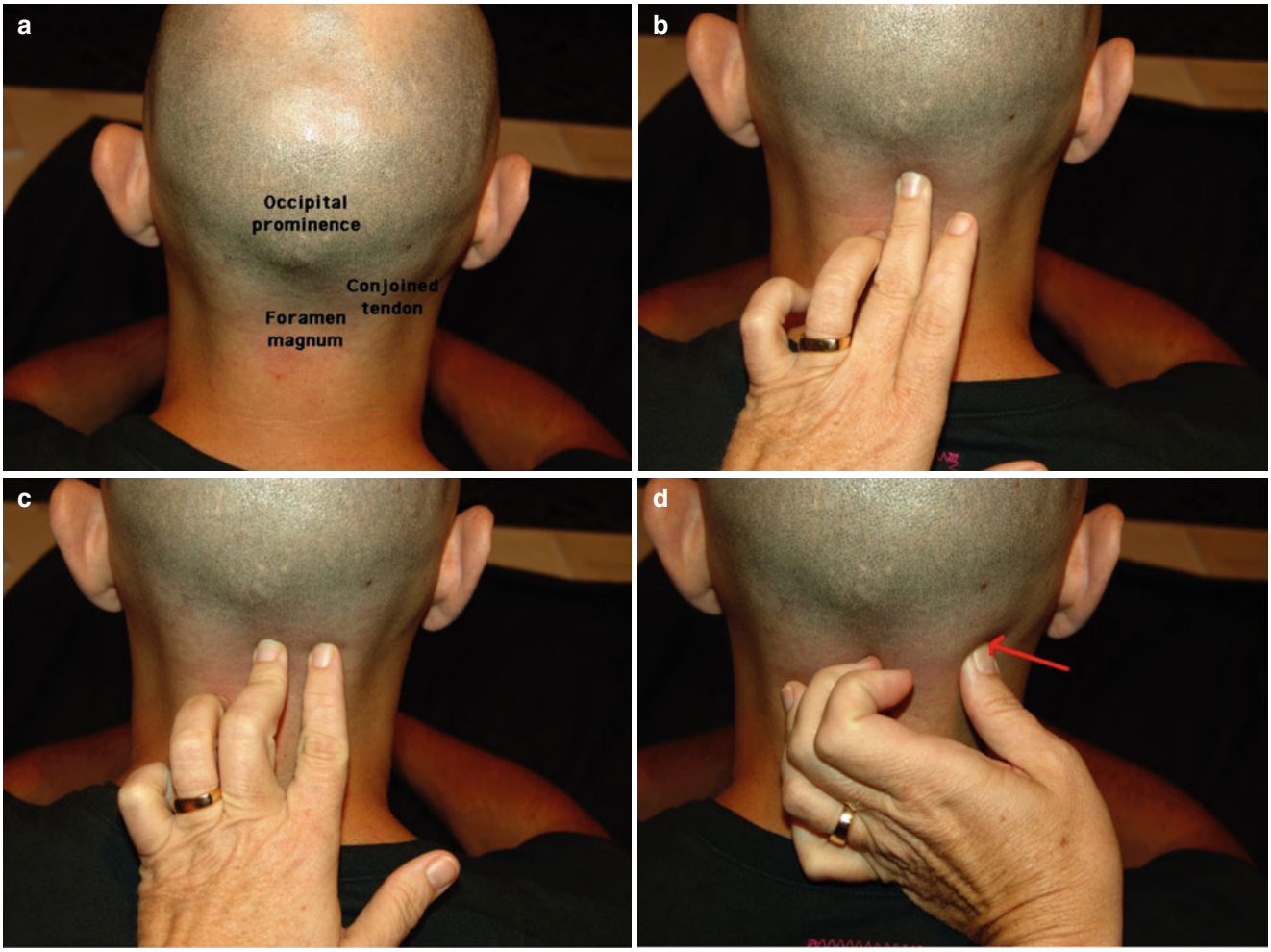

Fig. 17.14 Physical examination of the greater occipital nerve. (**a**) surface landmarks; (**b**) middle finger in region of the foramen magnum; (**c**) index finger on the conjoint tendon; (**d**) thumb palpating the greater occipital nerve (Images courtesy of Andrea Trescot, MD)

Table 17.3 Differential diagnosis of occipital pain

	Potential distinguishing features
Tumors of the posterior cranial fossa	Weight loss, visual changes, cranial nerve dysfunction
Neurosyphilis [36]	Cognitive dysfunction, ataxia, sensory disturbance
Zygapophysial joint dysfunction	Pain with neck extension or rotation
Temporal arteritis [37]	Fever, elevated ESR and CRP
Vertebral artery dissection/compression [38]	Horner's syndrome, vertigo, truncal ataxia
C2 myelitis [39]	Loss of function in C2 distribution
C2–C3 intervertebral disc dysfunction	Radiating pain from the neck into the shoulder
Atlantoaxial joint dysfunction [35]	Suboccipital pain, focal tenderness over the transverse process of C1, restricted head rotation with pain
Shingles [39]	Lesions, pain in dermatomal pattern

clap" occipital or supraorbital headache, especially if the patient complains of vertigo and truncal ataxia or increased pain after cervical manipulation or whiplash, though the onset of pain may also be slow (over days) and may start as neck pain rather than headache [43] (Tables 17.3 and 17.4).

Ultrasound can be useful to help to evaluate occipital nerve pathology. There are two main sites of ultrasound identification of the occipital nerve (Fig. 17.15). The patient is positioned prone with mild flexion to expose the suboccipital region. The first site is at the occipital ridge, and the probe is placed horizontally across the ridge (site 1 on Fig. 17.15a); the GON will be found medial to the occipital artery (Fig. 17.15b). The second site uses identification of the C2 spinous process as the landmark for identifying the inferior oblique in the long axis (site 2A on Fig. 17.15a) on which the inferior oblique muscle inserts, and then orienting the probe toward the distal inferior oblique attachment at the transverse process of C1 (site 2B on Fig. 17.15a) [44]. The

greater occipital nerve can then be identified as a hypoechoic round or oval structure within the fascial plane above the inferior oblique muscle (Fig. 17.15c). The third occipital nerve (Chap. 19) can be seen more medially. Cho and colleagues [45] measured the diameter of the occipital nerve at the inferior oblique muscle in symptomatic and asymptomatic patients and found a significant swelling of the nerve (a square area of 4.1 ± 2.6 mm^2) in the symptomatic patients compared to the asymptomatic patients (2.0 ± 0.7 mm^2).

Table 17.4 Diagnostic tests for greater occipital nerve entrapment

	Potential distinguishing features
Physical exam	Pain over the GON to palpation, pain radiates over the top of the head and to the ipsilateral eye
Diagnostic injection	GON block relieves the pain
Ultrasound	Swelling of the occipital nerve on US
MRI	May show tumor or space occupying lesion
Arteriography	Not useful
X-ray	May show C2 and C3 arthropathy
Electrodiagnostic studies	Not useful

Identification and Treatment of Contributing Factors (Table 17.5)

Poor posture can be due to poor vision, poor ergonomics, or ligament laxity. The need to peer forward because of poor vision, or poor ergonomics such as an inappropriately placed computer, will cause extension of the head on the neck, entrapping the occipital nerve at the inferior oblique (Fig. 17.12). The patient needs to be taught correct head position, and the ergonomic issues need to be addressed (e.g., computer location). Cervical ligament laxity causes a head-forward position to "take up the slack" of the ligament, and the subsequent trapezius spasm can entrap the occipital nerve; prolotherapy may relieve the myofascial entrapment.

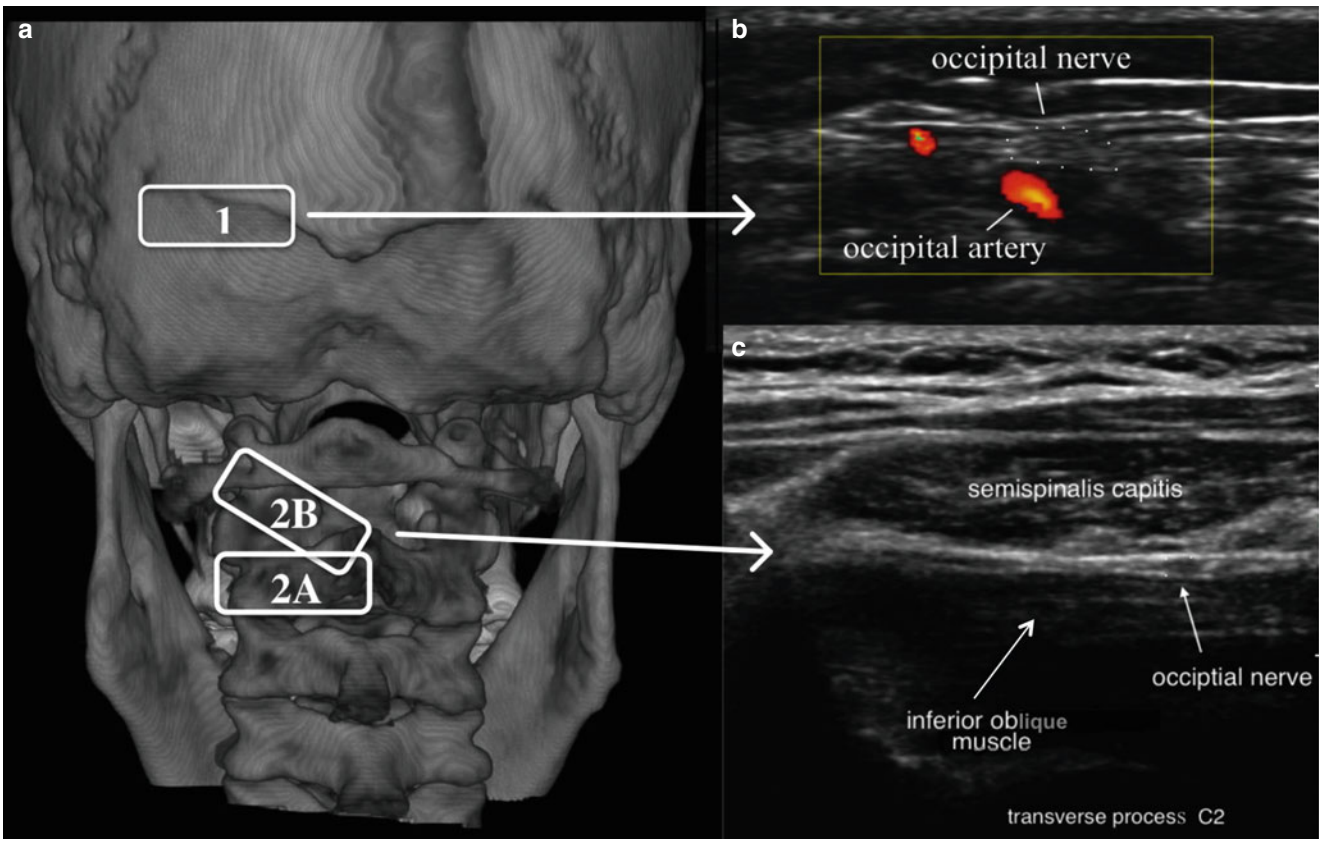

Fig. 17.15 Ultrasound identification of the greater occipital nerve. (**a**) Location of ultrasound transducer: *1* distal occipital nerve ultrasound site at nuchal ridge; *2* proximal occipital US approach with (2A) being the initial probe placement and (2B) being the final probe placement. (**b**) Distal US occipital nerve and artery. (**c**) Proximal US of greater occipital nerve (Images (**a**, **b**) courtesy of Andrea Trescot, MD; image (**c**) courtesy of Michael Sommer, MD)

Table 17.5 Contributing factors

Contributing factor	Etiology
Posture	Head-forward position entraps GON at inferior oblique
Cervical facet pathology	Irritation/entrapment of GON at nerve root level
Stress	Increased trapezius spasm leading to entrapment of GON
Ligament laxity	Results in head-forward position and trapezius spasm
Levator scapula spasm	Since the levator attaches on the cervical transverse process, spasm causes rotational dysfunction

Injection Technique

Landmark-Guided Technique

Greater occipital nerve blocks have been used to collaborate cervicogenic headache [46]. However, injection of local anesthetic into the suboccipital region in volumes greater than those required to anesthetize the greater occipital nerve is not a "greater occipital nerve block" but is best described as an "occipital block" or "*suboccipital* injection" (see Chap. 20), as it would not be selective for the greater occipital nerve [47].

The classic description of the occipital nerve block was for surgical anesthesia of the posterior scalp. Three to 5 cc of local anesthetic is injected subcutaneously across the superior nuchal ridge to develop a "wall" of local anesthetic across the nerve (site 1 on Fig. 17.4) [48]. This would usually effectively anesthetize all three nerves, but not at the site of the entrapment. In addition, because the galeal tissue is so adherent to the scalp at this level, large volumes of injectate could potentially create an entrapment of the nerves.

For the standard occipital nerve injection at the nuchal ridge (site 2 on Fig. 17.4), the inion and the occipital artery are the most useful landmarks for locating the greater occipital nerve, which lies immediately medial to the artery and lateral to the inion. According to Loukas et al. [29], "The location of GON for anesthesia or any other neurosurgical procedure has been established as one thumb's breadth lateral to the external occipital protuberance [inion] (2 cm laterally) and approximately at the base of the thumb nail (2 cm inferior)." A short, 1–1.5 inch, 25-gauge needle is inserted through the skin at the level of superior nuchal line, with the artery commonly being found at a point approximately one-third of the distance from the external occipital protuberance to the mastoid process on the superior nuchal line.

Bovim et al. [31] used the 2 cm lateral and 2 cm inferior location for their occipital nerve blocks, but note that this site (according to their dissections) "…is close to the nerve but not necessarily close to the point where it is prone to entrapment."

Fig. 17.16 Landmark-guided occipital nerve injection (Image courtesy of Andrea Trescot, MD)

Trescot [49] has described an alternate technique (Video 17.2), which places the injectate just below the trapezius attachment (the likely site of entrapment) (site 3 on Fig. 17.4). The patient is positioned sitting with the neck slightly flexed. As described in the *Physical Exam* section (Fig. 17.14), the injection site (in this case describing the right side) is identified by placing the middle finger of the left hand at the base of the skull (which identifies midline and avoids a cisternal injection). The index finger is then placed at the conjoined tendon attachment. The thumb will then identify the injection site for the GON at the base of the skull by creating a paresthesia with palpation. The needle (1.5 inch 27 g) is directed cephalad and slightly medially until contact is made with the bone (Fig. 17.16). Small volumes (less than 2 cc) of local anesthetic and steroid are then injected underneath the tendon where the nerve pierces the tendon attachment. Anthony [21] has proposed the use of a nerve stimulator for diagnostic nerve blocks to aid in localization of the nerve.

Trescot and Racz (personal communication) injected methylene blue into a fresh cadaver to compare Trescot's occipital nerve injection technique (see above) to a "*Stealth needle*" technique (see Chap. 20) to inject the occipital nerve (Fig. 17.17a). Dissection showed that 1 cc of dye using Trescot's approach stained the occipital nerve just below the trapezius attachment. However, the "Stealth needle" approach stained the whole path of the nerve (Fig. 17.17b).

Terzi et al. [50] evaluated occipital nerve blocks as a diagnostic tool in the evaluation of headaches. They studied 60 patients, with 20 patients in each group diagnosed with migraine without aura, tension-type headaches, or cervicogenic headaches. The groups were split into placebo (saline injections) or active treatment (1 cc of 2 % prilocaine) injections of the greater occipital nerve. The active agent GON injections reduced pain in the orbitofrontal and orbitonuchal areas in patients with cervicogenic headaches but not tension-type or migraine headaches. Leroux et al. [13] performed

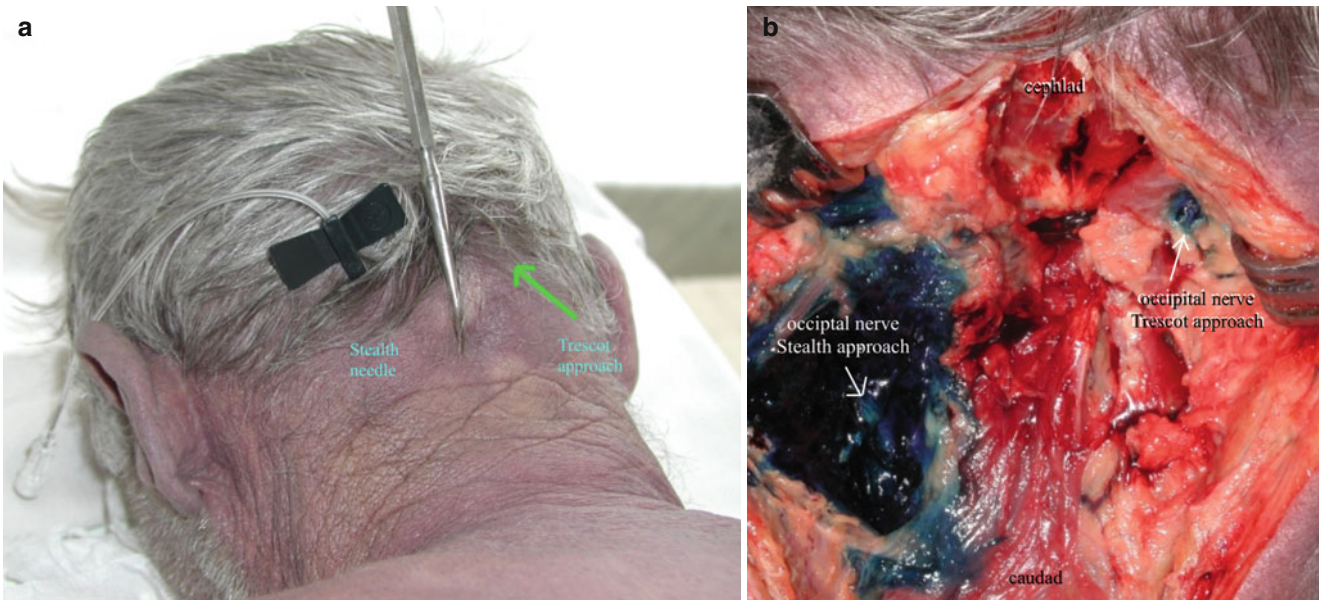

Fig. 17.17 Occipital dissection: (**a**) Occipital nerve injection sites prior to dissection; (**b**) methylene blue on the occipital nerves, comparing Stealth and Trescot techniques (Dissection by Umeshraya Pai, MD; image courtesy of Gabor Racz, MD)

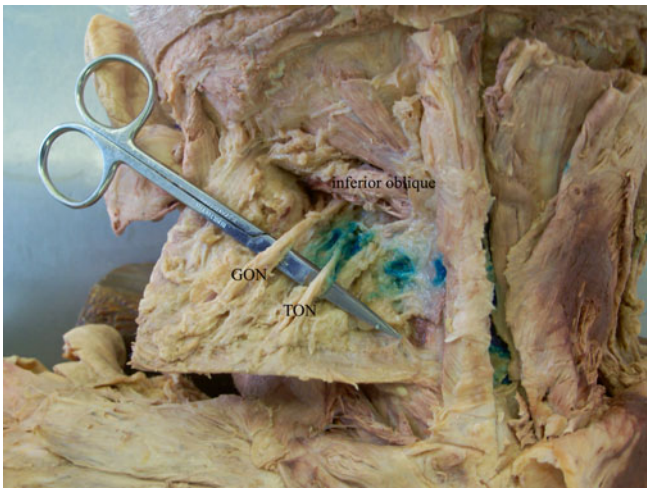

Fig. 17.18 Dissection of the suboccipital region after methylene blue dye injection of the third occipital nerve with the skin reflected inferiorly. *GON* greater occipital nerve, *TON* third occipital nerve (Image courtesy of Kyle Silva, DO and Sayed Wahezi, MD)

a prospective randomized, double-blind control study of blind "suboccipital steroid injections" with Cortivazol (a long acting corticosteroid) to treat cluster headaches, finding significant improvement in frequency and severity of the cluster headaches.

Silva et al. presented the results of small volume injections of methylene blue (0.3–0.5 cc) onto the third occipital nerve (TON) (see Chap. 19) in cadavers; dissections showed that with even just 0.3 cc of volume, 83.3 % of the cadavers had methylene blue coating the GON (Fig. 17.18), suggesting that injections of the TON usually affect the GON as well (and presumably the reverse is also true) [51].

Ultrasound-Guided Technique

There are several sites for injection of the GON under US. Shim et al. [52] used US-guided injections at the occipital ridge (site 1 on Fig. 17.15) to treat occipital headaches; they measured the distance from the "external occipital prominence" (the inion) to the occipital nerve and artery and then looked at the results of landmark-guided versus US-guided injections. They found a greater improvement in pain scores for the US patients at 4 weeks (blind 3.8 ± 0.3 vs. US 2.3 ± 0.2). Na and colleagues [53] did a similar study with 26 patients using flow Doppler versus blind injections at the nuchal ridge. All of the injections resulted in at least "partial" hypoesthesia; only 30.8 % of the blind injections had "complete" anesthesia, while 76.9 % of the Doppler-guided injections had "complete" anesthesia. Palamar et al. randomized 23 migraine patients to injections of 1.5 cc of local anesthetic (0.5 % bupivacaine) or saline under US guidance at the occipital ridge (Fig. 17.19); all actively treated patients noted a significant decrease in headache symptoms [54]. Each GON was found to be medial to the occipital artery.

Greher et al. [44] described an anatomic study on cadavers using ultrasound to compare the traditional injection site (site 1 on Fig. 17.15a) at the nuchal ridge with a more proximal technique (site 2 on Fig. 17.15a). They identified the bifid spinous process of C2 with the probe in a horizontal position (site 2A on Fig. 17.15a); the probe was then

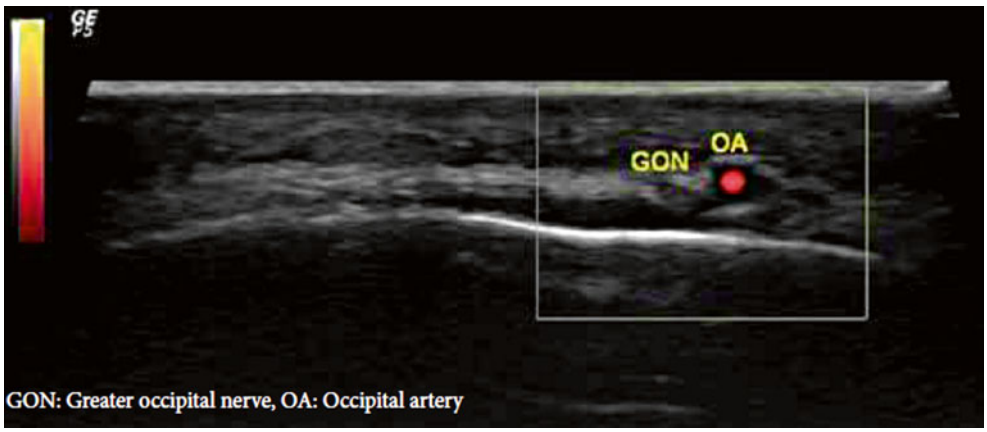

Fig. 17.19 Ultrasound image of the distal occipital nerve (From Palamar et al. [54]. Reprinted with permission from American Society of Interventional Pain Physicians)

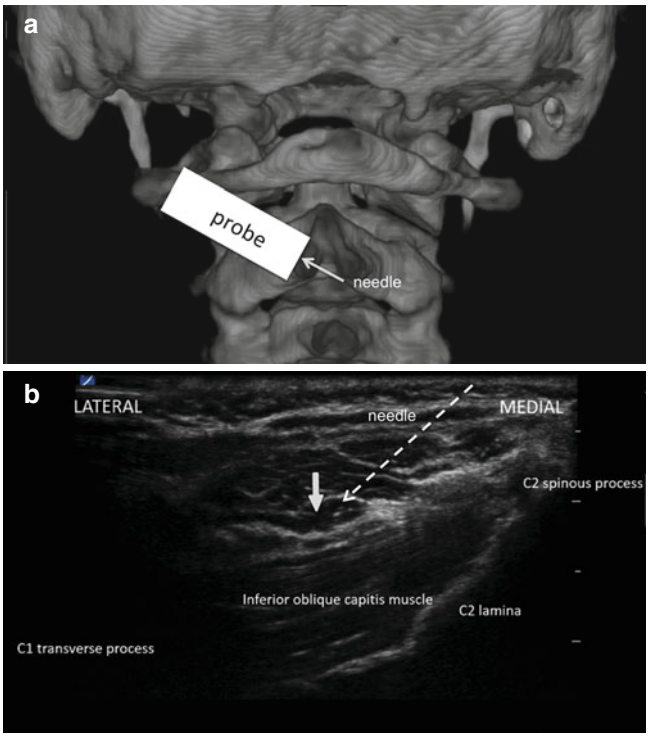

Fig. 17.20 Ultrasound injection of the proximal occipital nerve with simulated needle. (**a**) Location of US probe. (**b**) US image; solid arrow shows location of greater occipital nerve, broken arrow shows direction of needle (Image courtesy of Agnes Stogicza, MD)

rotated (site 2B on Fig. 17.15a) to visualize the inferior oblique muscle and the GON, which appears as a hypoechoic oval on top of the muscle (Fig. 17.15c). Using 0.1 cc injections of dye, they used ultrasound to identify the GON at both sites bilaterally in ten cadavers. They reported successful injection of the GON distally in 16 of 20 dissections, while all 20 of the proximal GON were stained with dye (i.e., successful). Figure 17.20 shows the technique of proximal occipital nerve injection under US guidance.

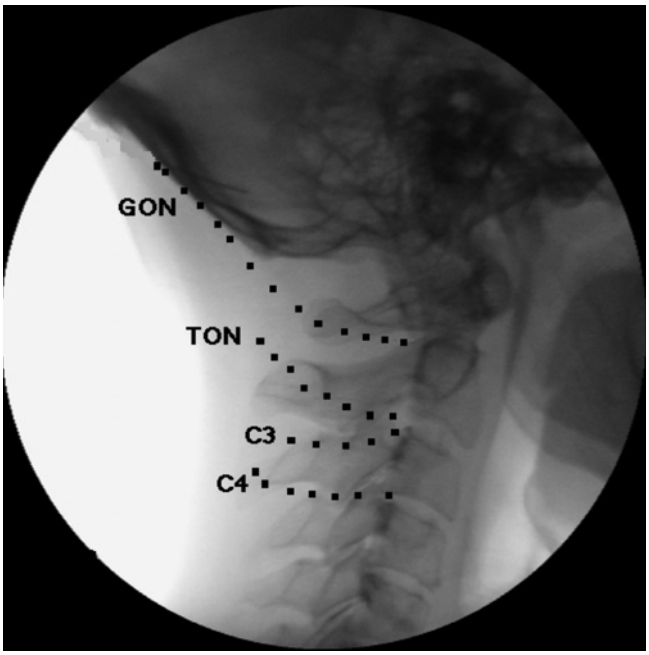

Fig. 17.21 Fluoroscopic location of the occipital nerves. *GON* greater occipital nerve, *TON* third occipital nerve (Image courtesy of Dr. Andrea Trescot)

Fluoroscopy-Guided Technique

The greater occipital nerve is not usually identified under fluoroscopy. However, all three occipital nerves have fluoroscopic landmarks (Fig. 17.21), and injection of the C2 dorsal ramus or the C2 nerve root is best done under fluoroscopy guidance (Fig. 17.22) or CT guidance (see below).

Stealth Technique

The technique of suboccipital decompression of the occipital nerve using a "Stealth needle" (Fig. 17.17) is discussed in Chap. 20.

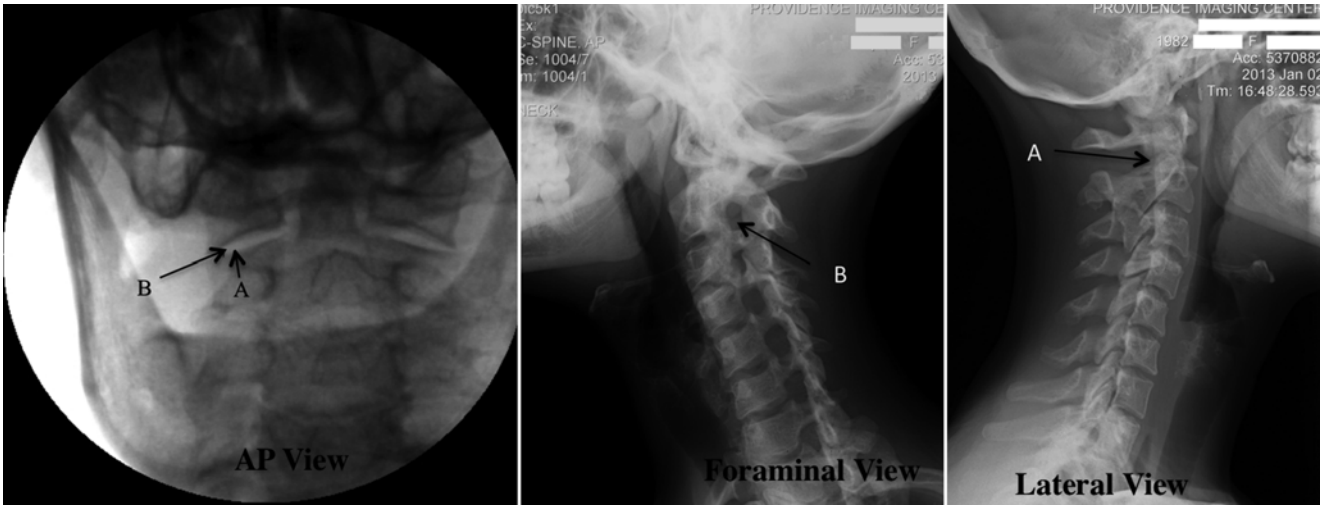

Fig. 17.22 AP, foraminal, and lateral fluoroscopic images of the C2 nerve root injection showing simulated needles. *A* posterior approach, *B* lateral approach (Image courtesy of Andrea Trescot, MD)

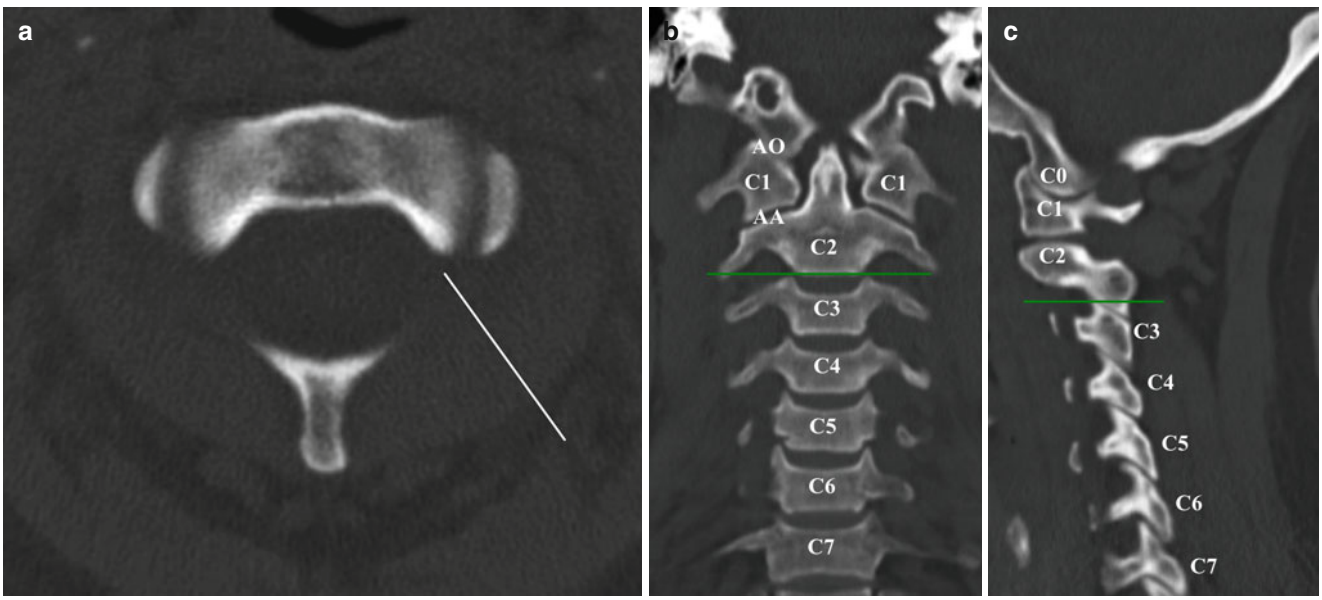

Fig. 17.23 CT-guided injection of the C2 nerve root. (**a**) Axial image at C2 with white line representing simulated needle; (**b**) coronal image and (**c**) sagittal image. *Green line* represents the level of the axial image (Image courtesy of Andrea Trescot, MD)

CT-Guided Technique

Kapoor et al. [55] treated 17 patients who underwent 32 occipital nerve blocks under CT guidance and followed them for at least 20 months. Needle placement for the C2 block was between posterior arches of C1 and C2, just behind the inferior lateral mass of C2 (Fig. 17.23). For the C3 injection, needle placement was at the lateral aspect of the C2–C3 foramen, just anterior to the base of C3 superior facet. If these blocks gave relief of the symptoms, the patients underwent intradural surgical sectioning of the upper cervical nerve roots (rhizotomy) [see below].

C2 Dorsal Root Ganglion Injections

Though not commonly performed, a *C2 dorsal root ganglion injection* (DRG) may be needed to trace the occipital nerve back to its origin. There are two approaches to the C2 DRG – posterior and lateral. For the posterior approach, the needle is advanced onto the inferior portion of the lateral aspect of the *atlantoaxial joint* in a posterior to anterior view (Fig. 17.24 arrow A). Once the needle touched the bone, the needle is redirected into the joint space by moving the needle hub caudally, taking care to avoid hitting the DRG. With the lateral approach, the patient is positioned

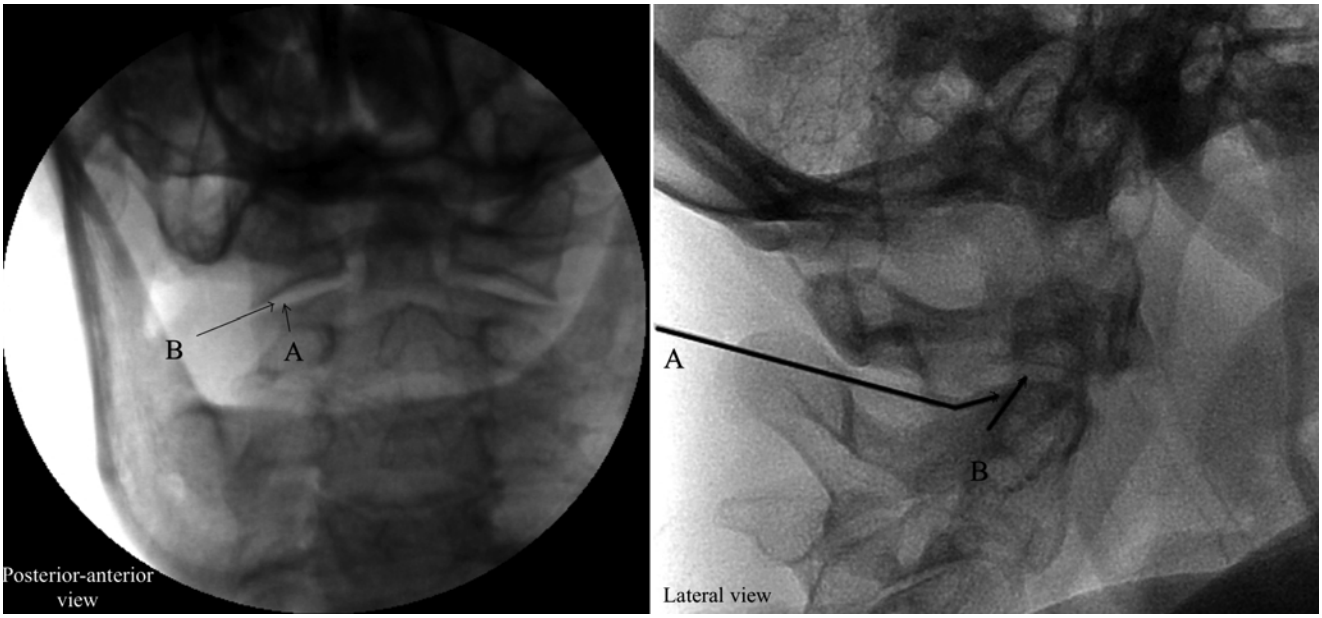

Fig. 17.24 Dorsal root ganglion (DRG) injection C2. *Arrow A* (on both the posterior-anterior and lateral views) represents the posterior approach to the DRG, while *arrow B* represents the lateral approach (Image courtesy of Andrea Trescot, MD)

supine with the C-arm in a lateral position; the needle is directed to the inferior lamina of C2 and then redirected slightly cephalad to enter the foramen (Fig. 17.24 arrow B).

Neurolytic Technique

Cryoneuroablation

If diagnostic injections of the occipital nerve have given temporary, but excellent, relief, the nerve can be a candidate for cryoneuroablation. Cryoneuroablation is conducted at the same site as the diagnostic injections for all three occipital nerves.

Trescot [56] described using landmarks for the landmark-directed placement of the cryoneuroablation probe to treat occipital neuralgia (Fig. 17.25); fluoroscopy confirmed the suboccipital location of the cryoprobe (Fig. 17.26). Kim et al. [24] reported on the results of 38 patients who underwent cryoneuroablation of the greater and lesser occipital nerves, noting an average of 70.5 % relief for 8.1 months.

Overbaugh and Compton described cryoneuroablation of the occipital nerve under ultrasound [57], using the nuchal ridge and occipital artery for landmarks. Color Doppler ultrasound was used to identify the occipital artery lateral to the occipital nerve, sensory stimulation reproduced the patient's usual distribution of headache pain, and an ice ball was seen on ultrasound surrounding the nerve.

Radiofrequency (RF)

Blume et al. described using up to 20 *radiofrequency* (RF) lesions along the base of the skull as well as the cervical facets and spinous process to treat cervicogenic headache [2].

Vanelderen and colleagues [58] studied *pulsed radiofrequency ablation* (*pulsed RF*) of the greater and/or lesser occipital nerve in 19 patients; after 6 months, 52.6 % noted "substantial" improvement in their headaches. Vanderhoek et al. [59] in 2013 used the linear US probe to identify the occipital nerve at the occipital ridge and then used pulsed RF (2 Hz/20 msec for 120 s at 42 °C times 2) to provide long-term relief.

Vu and Chhatre [60] described a case of bilateral greater occipital neuralgia treated with *cooled radiofrequency ablation* (*cooled RF*). They felt that cooled RF of the GON might be an alternative to pulsed and continuous RF to alleviate pain with less side effects and greater potential for long-term efficacy. However, both the conventional and the cooled RF carry a significant potential risk of *neuritis* and *anesthesia dolorosa*.

Alcohol/Phenol

As noted in Chap. 8, neurolytic injections of large myelinated nerves have been discouraged because of the risk of neuritis and neuromas. However, Weksler and colleagues [61] described using 4 % aqueous phenol, a peripheral nerve stimulator (PNS), and fluoroscopy to treat 42 patients with nonmalignant pains, including occipital neuralgia, and noted good relief in 35 patients without complications.

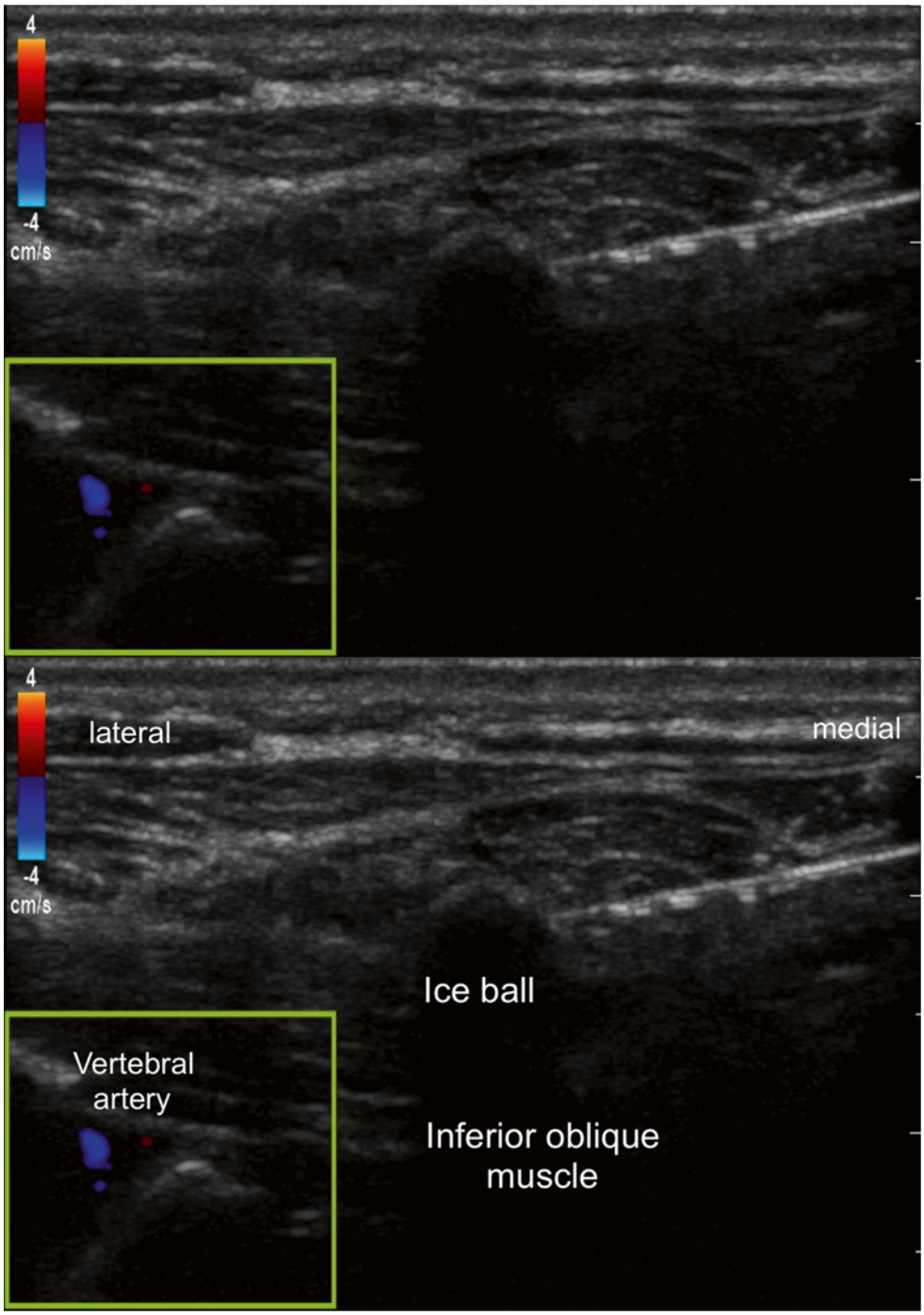

Fig. 17.25 Cryoneuroablation of the greater occipital nerve under ultrasound guidance (Image courtesy of Agnes Stogicza, MD)

Botulinum Neurotoxin

Botulinum toxin (*BTX*) has been used for several years to treat the trapezius spasm associated with occipital entrapment. In 2011, Linde and colleagues [62] performed a systematic review of BTX for secondary headaches, finding that 79 % of the papers reviewed showed only level 4 evidence. They concluded that BTX could not be recommended as an evidence-based treatment.

Neurostimulation Therapy

Spinal Cord Stimulation

Spinal cord stimulation (SCS) has been used for cervical radicular pain and cervicalgia, but it has not been commonly used for occipital neuralgia. However, Elahi and Reddy [63] described the use of a high cervical SCS for posttraumatic headaches (Fig. 17.27). The SCS electrode

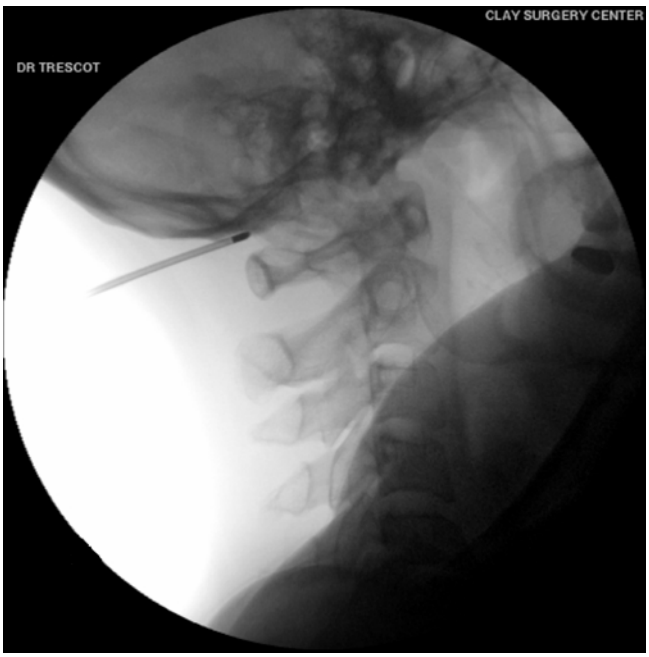

Fig. 17.26 Fluoroscopic imaging of placement of the cryoneuroablation probe (Image courtesy of Andrea Trescot, MD)

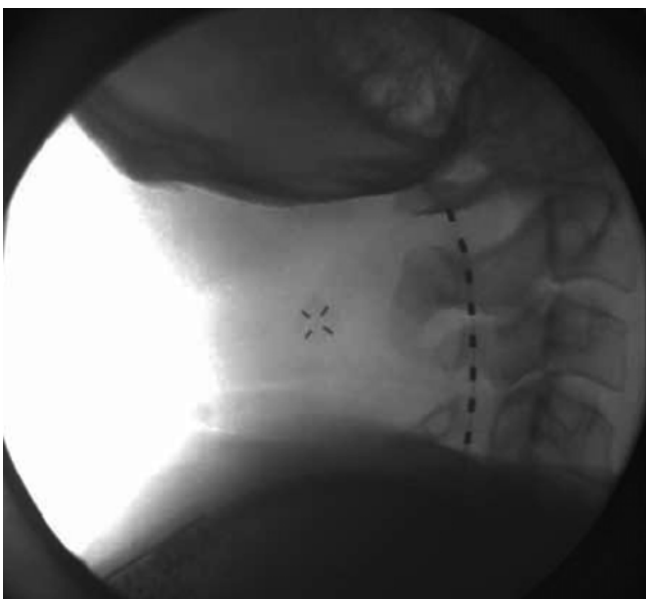

Fig. 17.27 Spinal cord stimulation for occipital neuralgia. Lateral image of spinal cord stimulator advanced to the high cervical region (From Elahi and Reddy [63]. Reprinted with permission from American Society of Interventional Pain Physicians)

can also be passed out the C2 foramen to provide stimulation of the dorsal root ganglion (Fig. 17.28).

Peripheral Nerve Stimulation

Occipital nerve stimulation was one of the first peripheral nerve stimulation techniques developed (see Chap. 9). The leads are placed percutaneously over the occipital nerve as a trial, and then, if the trial is successful, implanted subcutaneously (Fig. 17.29).

Current reports suggest that 60–80 % of patients with chronic headaches who have failed medication management will have relief from occipital stimulation [64–67].

Vallejo et al. [68] described the technique of subcutaneous placement of electrodes over the occipital ridge for chronic migraines. Johnstone and Sundaraj [69] described eight cases of using occipital nerve stimulation for occipital neuralgia, while Magis et al. [70] and Burns et al. [71] used occipital stimulators as part of a treatment program for drug-resistant *cluster headaches*. Occipital stimulation has been used in combination with other cranial stimulators, such as supraorbital (see Chap. 14) (Fig. 17.30) [5] and auriculotemporal (see Chap. 15) (Fig. 17.31) [5]; they have also been found to be useful for cluster headaches [72], trigeminal postherpetic neuralgia [73], and even weight loss [74].

Surgical Techniques

If the patient had good but only temporary relief from occipital nerve blocks, decompression of the nerve has been offered as a treatment. Li and colleagues [75] described 89 "micro-decompression" procedures on 76 patients. With a mean follow-up of 20 months, they noted "completely resolved" headaches in 68 patients (89.5 %), with 3 patients (3.9 %) noting recurrence. Recovery from the surgery took 1–6 months.

GON excision has also been described as a technique for treatment of persistent intractable ON. Ducic and colleagues [76] did a retrospective review of their GON excision cases; 71 of 108 patients responded to a follow-up survey, with 41 % of patients showing a 90 % or greater decrease in migraine headache index.

Jung et al. [77] described greater and lesser occipital neurectomy in a patient with persistent intractable occipital neuralgia. Kapoor et al. [55] described a surgical rhizotomy after positive response to CT-guided occipital nerve blocks. They used a midline cervical incision to bilaterally expose the ring of C1 and the lamina of C2, C3, and C4. After resection of the ring of C1 and the lamina of C2 and upper C3, the dura and arachnoid were opened and the cord with its exiting nerves was exposed. All dorsal nerve rootlets on the side of the pain for C1, C2, C3, and upper C4 were sectioned. Eleven of 17 patients (64 %) had complete relief of

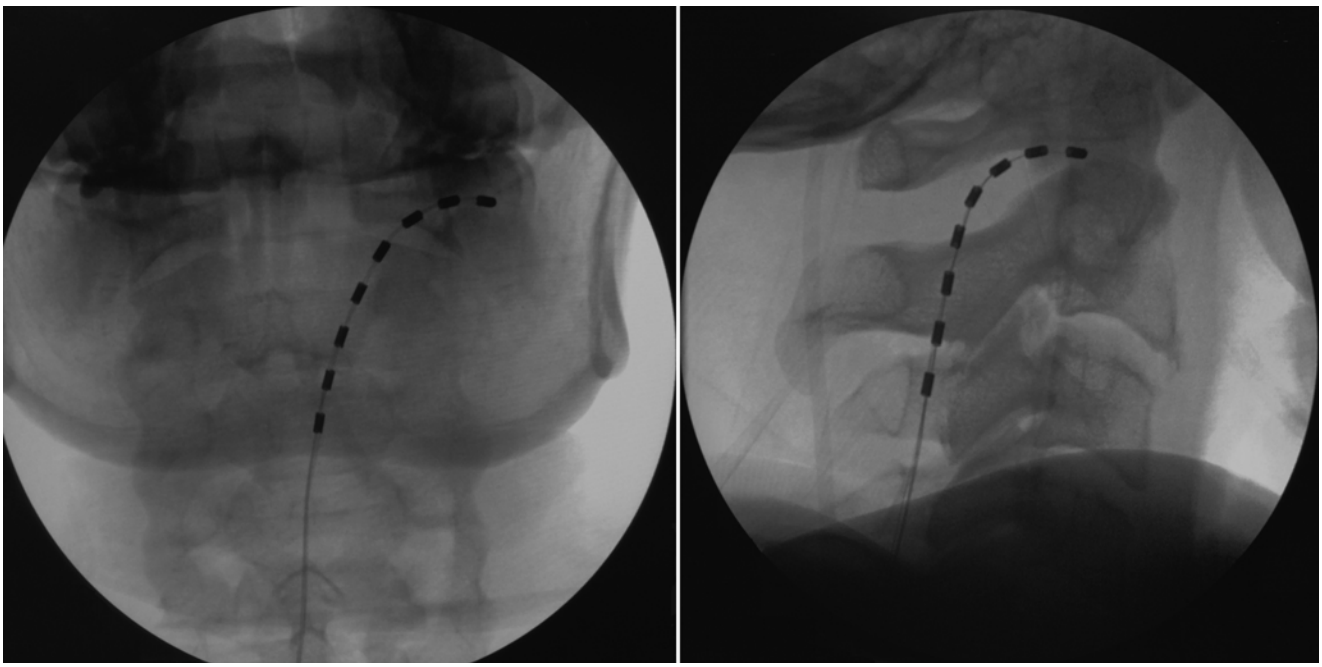

Fig. 17.28 C2 dorsal root ganglion (DRG) stimulation with an eight-contact array. AP image shows the first four electrodes out of the spinal canal. Lateral imaging shows the lead going from posterior to anterior at the upper portion of the C1–C2 foramen (Image courtesy of Mathew Rupert, MD)

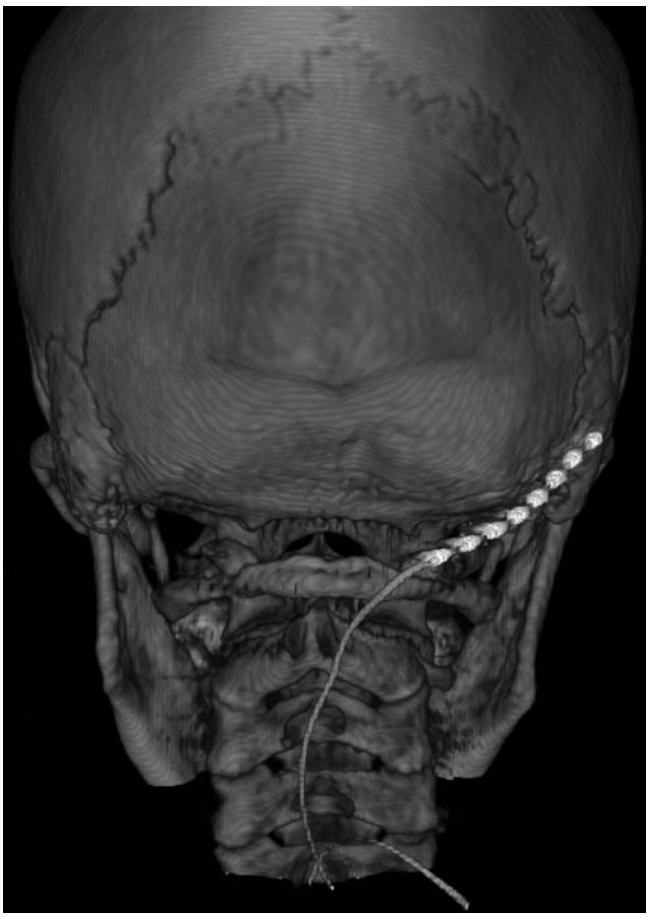

Fig. 17.29 3D image of subcutaneous occipital stimulator trial (Image courtesy of Andrea Trescot, MD)

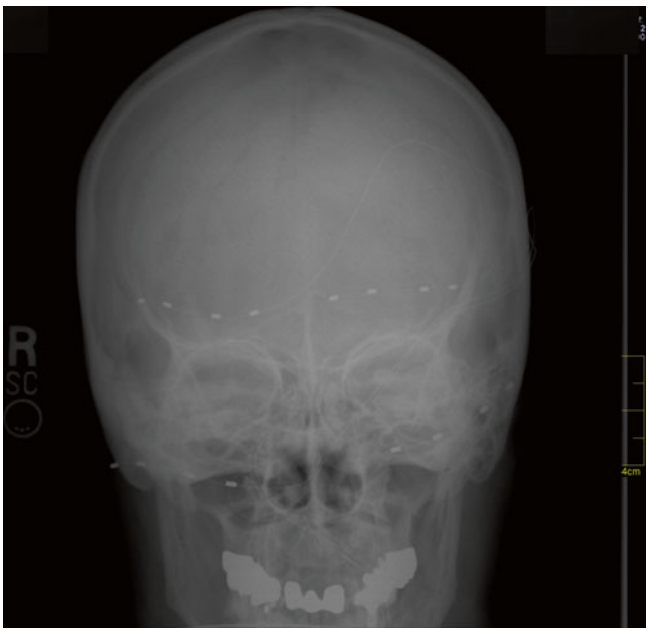

Fig. 17.30 Fluoroscopic image of bilateral supraorbital and occipital stimulator trial (Image courtesy of Rafael Justiz, MD)

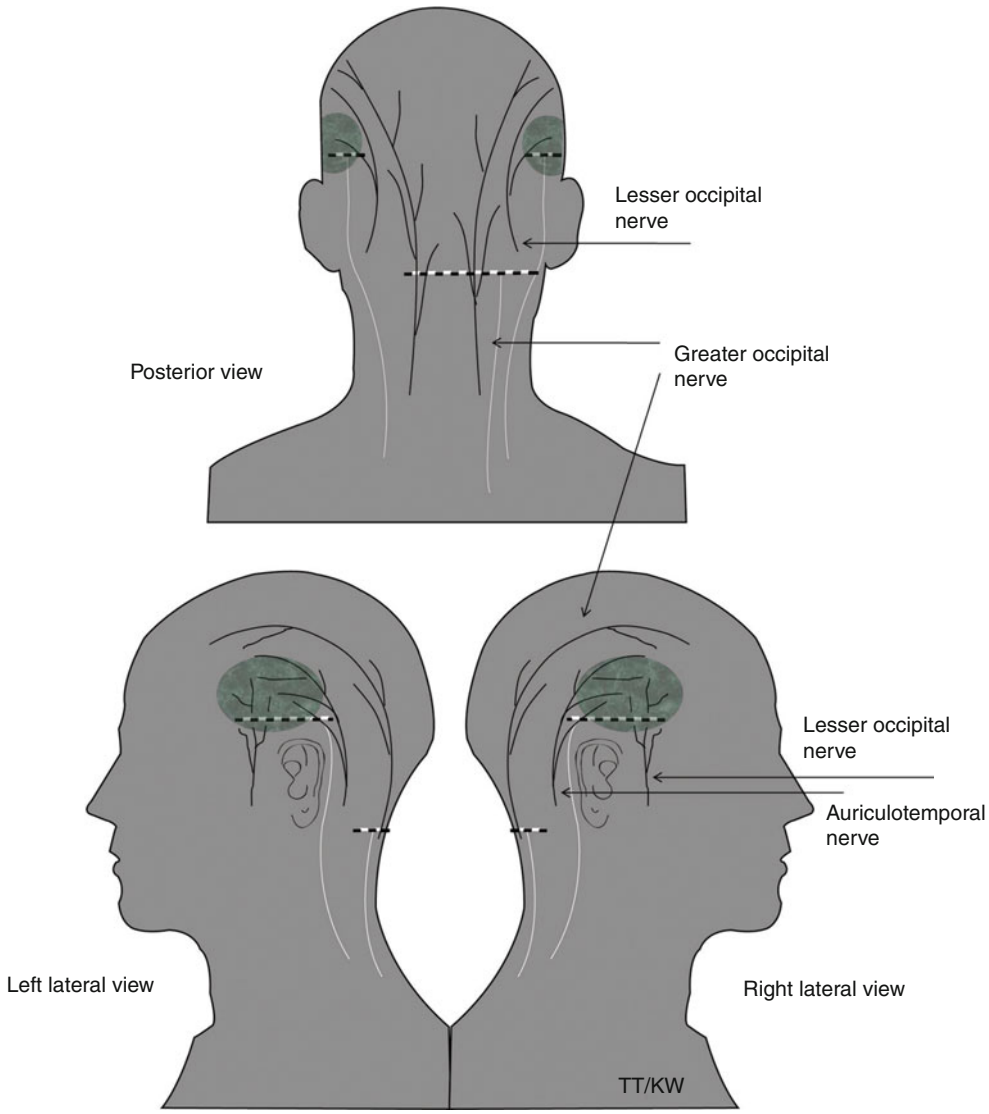

Fig. 17.31 Schematic drawing of the placement of occipital and auriculotemporal stimulators (From Deshpande and Wininger [5]. Reprinted with permission from American Society of Interventional Pain Physicians)

symptoms at follow-up (though the time frame was not described in the article).

In patients with pain on flexion, decompression of the occipital nerve at the inferior oblique may offer relief. Gille et al. [10] looked at ten patients retrospectively who underwent occipital decompression to the inferior edge of the inferior oblique; all patients decreased their analgesics, and their pain scores decreased from 80/100 to 20/100.

Ligation of the occipital artery has also been described. Chmielewski and colleagues [78] retrospectively looked at 170 chronic intractable migraine patients who underwent occipital nerve decompression; 55 patients had ligation of the occipital artery with occipital nerve decompression, while the remaining 115 patients served as controls with decompression alone. Interestingly, they found that the ligated patients did significantly worse.

Other Treatments

Regenerative injection therapy (RIT), or prolotherapy, has been used for at least 20 years for the treatment of cervical ligament laxity related to flexion/extension injuries and ergonomic issues; the laxity is felt to cause a forward head position, which causes trapezius spasm, entrapping the occipital nerve. Recently, platelet-rich plasma (PRP) and bone marrow/adipose harvested stem cells have been used to regenerate ligament pathology. These therapies have been used to treat pain of knees, elbows, and other joints; however, recently, Gaetani and colleagues [79] used adipose-derived stem cells to treat occipital neuralgia. They described the treatment of 24 patients with intractable occipital neuralgia; 19 patients (79.2 %) noted good relief, but there was recurrence of pain (albeit milder) in 7 patients (29.2 %) at 6 months.

Complications

Complications from occipital nerve blocks with local anesthetic and steroids or neurolytic procedures are exceedingly rare. However, serious complications with occipital interventions may occur and have been reported. Complications include those related to placement of the needle and those related to the administration of various drugs. Proximity of the needle to the vertebral artery, spinal cord, and nerve root creates risk for injury and makes precise and accurate needle placement extremely important.

These injections should not be attempted in patients who have undergone posterior cranial surgery because of the loss of protective skull coverage. Okuda et al. [80] described a patient who developed sudden unconsciousness during a lesser occipital nerve block. Apparently, unknown to the injector, the patient had undergone prior microvascular decompression for trigeminal neuralgia, with a subsequent craniotomy bony defect. The patient suddenly lost consciousness and stopped breathing, but her cardiopulmonary system was supported appropriately, and 2 h later, the patient was awake with no sequelae. The authors felt the patient had likely suffered a subarachnoid injection of local anesthetic.

Because of the use of corticosteroids, occipital nerve blocks may be associated with alopecia and cutaneous atrophy [81] or Cushing's syndrome with repeated injections [82]. Kinney et al. [83] described prolonged facial numbness after an occipital nerve block. Side effects related to the administration of steroids are generally attributed to the chemistry or to the pharmacology of the steroids [84].

Thus, complications may include dural puncture; spinal cord trauma; subdural injection; neural trauma; injection into the intervertebral foramen and intravertebral arteries; intravascular injection into veins, vertebral arteries, or occipital artery; steroid-induced alopecia [81]; infectious complications including epidural abscess and bacterial meningitis; and side effects related to the administration of steroids, local anesthetics, and other drugs. Vertebral artery and ventral ramus damage, along with a risk of embolus resulting in serious neurological sequelae with spinal cord damage and cerebral infarction are exceedingly rare, but are potential complications with occipital nerve blocks and other interventions. Dr. Gabor Racz described several cases of "locked-in syndrome" (paralysis without loss of consciousness) after occipital nerve injections with sharp needles (personal communication), prompting his development of the Stealth™ needle suboccipital approach (see Chap. 20).

Other minor complications include light-headedness, flushing, sweating, nausea, hypotension, syncope, pain at the injection site, and increased headaches.

Summary

The greater occipital nerve is a common source of headaches, often misdiagnosed as "migraines." Occipital neuralgia has several effective treatments that can provide significant relief from debilitating headaches.

References

1. Sjaastad O, Saunte C, Hovdahl H, Breivik H, Gronbaek E. "Cervicogenic" headache. An hypothesis. Cephalalgia. 1983;3(4):249–56.
2. Blume H, Atac M, Golnick J. Neurosurgical treatment of persistent occipital myalgia-neuralgia syndrome. In: Pfaffenrath V, Lundberg P-O, Sjaastad O, editors. Updating in headache. Berlin: Springer; 1985. p. 24–34.
3. Choi HJ, Choi SK, Lee SH, Lim YJ. Whiplash injury-induced atypical short-lasting unilateral neuralgiform headache with conjunctival injection and tearing syndrome treated by greater occipital nerve block. Clin J Pain. 2012;28(4):342–3.
4. Hecht JS. Occipital nerve blocks in postconcussive headaches: a retrospective review and report of ten patients. J Head Trauma Rehabil. 2004;19(1):58–71.
5. Deshpande KK, Wininger KL. Feasibility of combined epicranial temporal and occipital neurostimulation: treatment of a challenging case of headache. Pain Physician. 2011;14(1):37–44.
6. Borg-Stein J. Cervical myofascial pain and headache. Curr Pain Headache Rep. 2002;6(4):324–30.
7. Yeom JS, Buchowski JM, Kim HJ, Chang BS, Lee CK, Riew KD. Postoperative occipital neuralgia with and without C2 nerve root transection during atlantoaxial screw fixation: a post-hoc comparative outcome study of prospectively collected data. Spine J Off J N Am Spine Soc. 2013;13(7):786–95.
8. Lucchesi C, Puglioli M, Gori S. Occipital neuralgia: a symptomatic case caused by an abnormal left vertebral artery. Neurol Sci. 2013;34(2):243–5.
9. Garza I. Craniocervical junction schwannoma mimicking occipital neuralgia. Headache. 2007;47(8):1204–5.
10. Gille O, Lavignolle B, Vital JM. Surgical treatment of greater occipital neuralgia by neurolysis of the greater occipital nerve and sectioning of the inferior oblique muscle. Spine (Phila Pa 1976). 2004;29(7):828–32.
11. Feyzi Demir C, Akalin Y, Berilgen S. Occipital neuralgia following thoracic herpes zoster: case report. Agri. 2011;23(4):179–80.
12. Goicochea MT, Romero C, Leston JA. Occipital neuralgia with cervical myelitis. Cephalalgia. 2008;28(5):567–8.
13. Leroux E, Valade D, Taifas I, Vicaut E, Chagnon M, Roos C, et al. Suboccipital steroid injections for transitional treatment of patients with more than two cluster headache attacks per day: a randomised, double-blind, placebo-controlled trial. Lancet Neurol. 2011;10(10):891–7.
14. Akin Takmaz S, Unal Kantekin C, Kaymak C, Basar H. Treatment of post-dural puncture headache with bilateral greater occipital nerve block. Headache. 2010;50(5):869–72.
15. Beruto Y, Lentijo J, Ramus NM. Descodes de med y irugpract. Madrid. 1821;3:145–69.
16. Dubuisson D. Treatment of occipital neuralgia by partial posterior rhizotomy at C1-3. J Neurosurg. 1995;82(4):581–6.
17. Headache Classification Subcommittee of the International Headache Society. The international classification of headache disorders. 2nd edition. Cephalalgia. 2004;24 Suppl 1:9–160.

18. Headache Classification Committee of the International Headache Society (IHS). The international classification of headache disorders. 3rd edition (beta version). Cephalalgia. 2013;33(9): 629–808.
19. Scattoni L, Di Stani F, Villani V, Dugoni D, Mostardini C, Reale C, et al. Great occipital nerve blockade for cluster headache in the emergency department: case report. J Headache Pain. 2006; 7(2):98–100.
20. Peres MF, Stiles MA, Siow HC, Rozen TD, Young WB, Silberstein SD. Greater occipital nerve blockade for cluster headache. Cephalalgia. 2002;22(7):520–2.
21. Anthony M. Arrest of attacks of cluster headache by local steroid injections in the occipital myalgia-neuralgia syndrome. In: Pfaffenrath V, Lundberg PO, Sjaastad O, editors. Updating in headache. Berlin: Springer; 1985. p. 17–23.
22. Cortijo E, Guerrero AL, Herrero S, Mulero P, Munoz I, Pedraza MI, et al. Hemicrania continua in a headache clinic: referral source and diagnostic delay in a series of 22 patients. J Headache Pain. 2012;13(7):567–9.
23. Ashkenazi A, Matro R, Shaw JW, Abbas MA, Silberstein SD. Greater occipital nerve block using local anaesthetics alone or with triamcinolone for transformed migraine: a randomised comparative study. J Neurol Neurosurg Psychiatry. 2008;79(4): 415–7.
24. Kim CH, Hu W, Gao J, Dragan K, Whealton T, Julian C. Cryoablation for the treatment of occipital neuralgia. Pain Physician. 2015;18:E363–8.
25. Shimizu S, Oka H, Osawa S, Fukushima Y, Utsuki S, Tanaka R, et al. Can proximity of the occipital artery to the greater occipital nerve act as a cause of idiopathic greater occipital neuralgia? An anatomical and histological evaluation of the artery-nerve relationship. Plast Reconstr Surg. 2007;119(7):2029–34; discussion 35–6.
26. Natsis K, Baraliakos X, Appell HJ, Tsikaras P, Gigis I, Koebke J. The course of the greater occipital nerve in the suboccipital region: a proposal for setting landmarks for local anesthesia in patients with occipital neuralgia. Clin Anat. 2006;19(4):332–6.
27. Mosser SW, Guyuron B, Janis JE, Rohrich RJ. The anatomy of the greater occipital nerve: implications for the etiology of migraine headaches. Plast Reconstr Surg. 2004;113(2):693–7.
28. Becser N, Bovim G, Sjaastad O. Extracranial nerves in the posterior part of the head. Anatomic variations and their possible clinical significance. Spine. 1998;23(13):1435–41.
29. Loukas M, El-Sedfy A, Tubbs RS, Louis Jr RG, Wartmann CH, Curry B, et al. Identification of greater occipital nerve landmarks for the treatment of occipital neuralgia. Folia Morphol (Warsz). 2006;65(4):337–42.
30. Dash KS, Janis JE, Guyuron B. The lesser and third occipital nerves and migraine headaches. Plast Reconstr Surg. 2005;115(6):1752–8; discussion 9–60.
31. Bovim G, Bonamico L, Fredriksen TA, Lindboe CF, Stolt-Nielsen A, Sjaastad O. Topographic variations in the peripheral course of the greater occipital nerve. Autopsy study with clinical correlations. Spine. 1991;16(4):475–8.
32. Baer-Henney S, Tatagiba M, Samii M. Osteochondroma of the cervical spine causing occipital nerve neuralgia. Case report. Neurol Res. 2001;23(7):777–9.
33. Kihara T, Shimohama S. Occipital neuralgia evoked by facial herpes zoster infection. Headache. 2006;46(10):1590–1.
34. Star MJ, Curd JG, Thorne RP. Atlantoaxial lateral mass osteoarthritis. A frequently overlooked cause of severe occipitocervical pain. Spine (Phila Pa 1976). 1992;17(6 Suppl): S71–6.
35. Ehni G, Benner B. Occipital neuralgia and the C1-2 arthrosis syndrome. J Neurosurg. 1984;61(5):961–5.
36. Smith DL, Lucas LM, Kumar KL. Greater occipital neuralgia: an unusual presenting feature of neurosyphilis. Headache. 1987; 27(10):552–4.
37. Jundt JW, Mock D. Temporal arteritis with normal erythrocyte sedimentation rates presenting as occipital neuralgia. Arthritis Rheum. 1991;34(2):217–9.
38. Sharma RR, Parekh HC, Prabhu S, Gurusinghe NT, Bertolis G. Compression of the C-2 root by a rare anomalous ectatic vertebral artery. Case report. J Neurosurg. 1993;78(4):669–72.
39. Nikakis P, Koutsis G, Potagas C, Mandellos D, Sfagos C. Occipital neuralgia as an isolated symptom of C2 myelitis. Headache. 2006;46(8):1304–6.
40. Leinisch-Dahlke E, Jurgens T, Bogdahn U, Jakob W, May A. Greater occipital nerve block is ineffective in chronic tension type headache. Cephalalgia. 2005;25(9):704–8.
41. Guerrero AL, Herrero-Velazquez S, Penas ML, Mulero P, Pedraza MI, Cortijo E, et al. Peripheral nerve blocks: a therapeutic alternative for hemicrania continua. Cephalalgia. 2012;32(6): 505–8.
42. Bogduk N, Marsland A. On the concept of third occipital headache. J Neurol Neurosurg Psychiatry. 1986;49(7):775–80.
43. Hsu YC, Sung SF. Spontaneous vertebral artery dissection with thunderclap headache: a case report and review of the literature. Acta Neurol Taiwan. 2014;23(1):24–8.
44. Greher M, Moriggl B, Curatolo M, Kirchmair L, Eichenberger U. Sonographic visualization and ultrasound-guided blockade of the greater occipital nerve: a comparison of two selective techniques confirmed by anatomical dissection. Br J Anaesth. 2010; 104(5):637–42.
45. Cho JC, Haun DW, Kettner NW. Sonographic evaluation of the greater occipital nerve in unilateral occipital neuralgia. J Ultrasound Med. 2012;31(1):37–42.
46. Bovim G, Sand T. Cervicogenic headache, migraine without aura and tension-type headache. Diagnostic blockade of greater occipital and supra-orbital nerves. Pain. 1992;51(1):43–8.
47. Bogduk N. Role of anesthesiologic blockade in headache management. Curr Pain Headache Rep. 2004;8(5):399–403.
48. Brown D. Occipital block. In: Brown D, editor. Atlas of regional anesthesia. Philadelphia: Elsevier; 2006. p. 53–5.
49. Trescot AM. Headache management in an interventional pain practice. Pain Physician. 2000;3(2):197–200.
50. Terzi T, Karakurum B, Üçler S, İnan LE, Tulunay C. Greater occipital nerve blockade in migraine, tension-type headache and cervicogenic headache. J Headache Pain. 2002;3(3):137–41.
51. Silva K, Emmam M, Lederman A, Downie S, Wahezi S. Cervical third occipital nerve blocks concomitantly affect the greater occipital nerve: cadaveric study. NYNJSIPP annual meeting; Jersey City; 2014.
52. Shim JH, Ko SY, Bang MR, Jeon WJ, Cho SY, Yeom JH, et al. Ultrasound-guided greater occipital nerve block for patients with occipital headache and short term follow up. Kor J Anesthesiol. 2011;61(1):50–4.
53. Na SH, Kim TW, Oh SY, Kweon TD, Yoon KB, Yoon DM. Ultrasonic doppler flowmeter-guided occipital nerve block. Korean J Anesthesiol. 2010;59(6):394–7.
54. Palamar D, Uluduz D, Saip S, Erden G, Unalan H, Akarirmak U. Ultrasound-guided greater occipital nerve block: an efficient technique in chronic refractory migraine without aura? Pain Physician. 2015;18(2):153–62.
55. Kapoor V, Rothfus WE, Grahovac SZ, Amin Kassam SZ, Horowitz MB. Refractory occipital neuralgia: preoperative assessment with CT-guided nerve block prior to dorsal cervical rhizotomy. AJNR Am J Neuroradiol. 2003;24(10):2105–10.
56. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.

57. Overbaugh RH, Compton CH. Ultrasound guided cryoablation for treatment of occipital neuralgia (abstract). Reg Anes Pain Med. 2010;35(5):E1.
58. Vanelderen P, Rouwette T, De Vooght P, Puylaert M, Heylen R, Vissers K, et al. Pulsed radiofrequency for the treatment of occipital neuralgia: a prospective study with 6 months of follow-up. Reg Anesth Pain Med. 2010;35(2):148–51.
59. Vanderhoek MD, Hoang HT, Goff B. Ultrasound-guided greater occipital nerve blocks and pulsed radiofrequency ablation for diagnosis and treatment of occipital neuralgia. Anesthesiol Pain Med. 2013;3(2):256–9.
60. Vu T, Chhatre A. Cooled radiofrequency ablation for bilateral greater occipital neuralgia. Case Rep Neurol Med. 2014;2014:257373.
61. Weksler N, Klein M, Gurevitch B, Rozentsveig V, Rudich Z, Brill S, et al. Phenol neurolysis for severe chronic nonmalignant pain: is the old also obsolete? Pain Med. 2007;8(4):332–7.
62. Linde M, Hagen K, Stovner LJ. Botulinum toxin treatment of secondary headaches and cranial neuralgias: a review of evidence. Acta Neurol Scand Suppl. 2011;191:50–5.
63. Elahi F, Reddy C. High cervical epidural neurostimulation for posttraumatic headache management. Pain Physician. 2014;17(4):E537–41.
64. Saper JR, Dodick DW, Silberstein SD, McCarville S, Sun M, Goadsby PJ. Occipital nerve stimulation for the treatment of intractable chronic migraine headache: ONSTIM feasibility study. Cephalalgia. 2010;31(3):271–85.
65. Freeman JA, Trentman TL. Clinical utility of implantable neurostimulation devices in the treatment of chronic migraine. Med Devices (Auckland NZ). 2013;6:195–201.
66. Verrills P, Rose R, Mitchell B, Vivian D, Barnard A. Peripheral nerve field stimulation for chronic headache: 60 cases and long-term follow-up. Neuromodulation. 2014;17(1):54–9.
67. Hong J, Ball PA, Fanciullo GJ. Neurostimulation for neck pain and headache. Headache. 2014;54(3):430–44.
68. Vallejo R, Benyamin R, Kramer J. Neuromodulation of the occipital nerve in pain management. Tech Reg Anesth Pain Manag. 2006;10(1):12–5.
69. Johnstone CS, Sundaraj R. Occipital nerve stimulation for the treatment of occipital neuralgia-eight case studies. Neuromodulation. 2006;9(1):41–7.
70. Magis D, Allena M, Bolla M, De Pasqua V, Remacle JM, Schoenen J. Occipital nerve stimulation for drug-resistant chronic cluster headache: a prospective pilot study. Lancet Neurol. 2007;6(4):314–21.
71. Burns B, Watkins L, Goadsby PJ. Treatment of intractable chronic cluster headache by occipital nerve stimulation in 14 patients. Neurology. 2009;72(4):341–5.
72. Mueller O, Diener HC, Dammann P, Rabe K, Hagel V, Sure U, et al. Occipital nerve stimulation for intractable chronic cluster headache or migraine: a critical analysis of direct treatment costs and complications. Cephalalgia. 2013;33(16):1283–91.
73. Johnson MD, Burchiel KJ. Peripheral stimulation for treatment of trigeminal postherpetic neuralgia and trigeminal posttraumatic neuropathic pain: a pilot study. Neurosurgery. 2004;55(1):135–41; discussion 41–2.
74. Sobocki J, Herman RM, Fraczek M. Occipital C1-C2 neuromodulation decreases body mass and fat stores and modifies activity of the autonomic nervous system in morbidly obese patients – a pilot study. Obes Surg. 2013;23(5):693–7.
75. Li F, Ma Y, Zou J, Li Y, Wang B, Huang H, et al. Micro-surgical decompression for greater occipital neuralgia. Turk Neurosurg. 2012;22(4):427–9.
76. Ducic I, Felder 3rd JM, Khan N, Youn S. Greater occipital nerve excision for occipital neuralgia refractory to nerve decompression. Ann Plast Surg. 2014;72(2):184–7.
77. Jung SJ, Moon SK, Kim TY, Eom KS. A case of occipital neuralgia in the greater and lesser occipital nerves treated with neurectomy by using transcranial Doppler sonography: technical aspects. Korean J Pain. 2011;24(1):48–52.
78. Chmielewski L, Liu MT, Guyuron B. The role of occipital artery resection in the surgical treatment of occipital migraine headaches. Plast Reconstr Surg. 2013;131(3):351e–6.
79. Gaetani P, Klinger M, Levi D, Bussone G, Giannasi S, Caviggioli F, et al. Treatment of chronic headache of cervical origin with lipostructure: an observational study. Headache. 2013;53(3):507–13.
80. Okuda Y, Matsumoto T, Shinohara M, Kitajima T, Kim P. Sudden unconsciousness during a lesser occipital nerve block in a patient with the occipital bone defect. Eur J Anaesthesiol. 2001;18(12):829–32.
81. Shields KG, Levy MJ, Goadsby PJ. Alopecia and cutaneous atrophy after greater occipital nerve infiltration with corticosteroid. Neurology. 2004;63(11):2193–4.
82. Lavin PJ, Workman R. Cushing syndrome induced by serial occipital nerve blocks containing corticosteroids. Headache. 2001;41(9):902–4.
83. Kinney MA, Wilson JL, Carmichael SW, De Ruyter ML, Fulgham JR. Prolonged facial hypesthesia resulting from greater occipital nerve block. Clin Anat. 2003;16(4):362–5.
84. Manchikanti L. Pharmacology of neuraxial steroids. In: Manchikanti L, Singh V, editors. Interventional techniques in chronic spinal pain. Paducah: ASIPP Publishing; 2007. p. 167–84.

Lesser Occipital Nerve Entrapment

18

Esther Rawner, David M. Irwin, and Andrea M. Trescot

Introduction

There are several occipital nerves, each with different patterns of pain, neurologic origin, and treatment modalities. The use of precision diagnostic injections has facilitated the separation of these clinical presentations. This chapter will focus specifically on the lesser occipital nerve (LON); see Chap. 17 for the *greater occipital nerve* (GON) and Chap. 19 for the *third occipital nerve* (TON). There are multiple names used for these nerve entrapments, including "*migraine,*" "*tension headache,*" "*cervicogenic headaches*" [1], and "*occipital myalgia-neuralgia syndrome*" [2]. The LON is also called the *minor occipital nerve* or *nervus occipitalis minor*. There is much overlap of etiology and symptomology between the LON, the GON, and the TON.

Clinical Presentation (Table 18.1)

As a subset of cervicogenic headaches (CGH), lesser occipital neuralgia can cause pain and paresthesias to the posterior scalp; the periorbital, temporal, and mandibular regions; the external ear and mastoid regions; and the neck and shoulders (Fig. 18.1). The cutaneous innervation of the posterior neck and occiput comes primarily from C1, C2, and C3, which provide overlapping dermatomes. C2 covers the occiput, neck, and submental regions, while the C3 dermatome can span from the clavicle to the mandible to behind and over the ear, pinna, and the angle of the mandible (Figs. 18.2 and 18.3) [11].

The first three cervical spinal nerve segments (C1-C3) that make up the occipital nerves share a relay station in the brainstem that continues into the upper cervical spinal cord with the trigeminal cell bodies (the *cervico-trigeminal complex*). The pain of occipital neuralgia and cervicogenic headaches can therefore be referred to structures innervated by the branches of the trigeminal nerve, namely, the forehead, temples, and eyes (Fig. 18.4).

The LON originates from the posterior rami of C2 and C3 and has a pain pattern that overlaps with the GON; however, patients may describe radiation that is more lateral in the neck and posterior to the ear when the LON is the source of pathology. In addition, treatment of the GON for "migraine headaches" has resulted in recurrence of the headaches more laterally, leading to the diagnosis of lesser occipital neuralgia [12]. These headaches usually start as "tension headaches" in the upper cervical region and at the base of the skull, but

Table 18.1 Occupation/exercise/trauma history relevant to lesser occipital entrapment

Surgery	Surgical positioning – beach chair [3]
	Lateral suboccipital craniotomy [4]
	Platysma facial rejuvenation surgery [5]
	Shoulder arthroscopy [6]
	Endolymphatic shunt [7]
Flexion/extension injuries	"Migraine" or ear fullness after motor vehicle collision [8, 9]
Pregnancy	Swelling of subcutaneous tissue of the neck [10]
Manual labor	Excessive spasm in neck musculature
Myofascial spasm	Entrapment by SCM

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Fig. 18.1 Pattern of lesser occipital pain (Image courtesy of Andrea Trescot, MD)

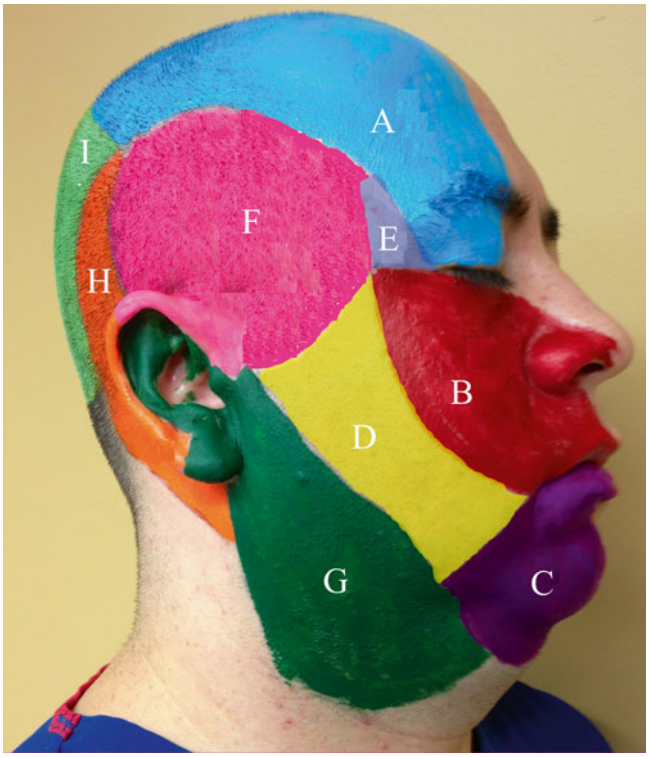

Fig. 18.3 Distribution of nerve sensation. *A* supraorbital nerve, *B* infraorbital nerve, *C* mental nerve, *D* buccal nerve, *E* lacrimal nerve, *F* auriculotemporal nerve, *G* superficial cervical plexus, *H* posterior auricular nerve/great auricular nerve, *I* lesser occipital nerve (Image courtesy of Terri Dallas-Prunskis, MD)

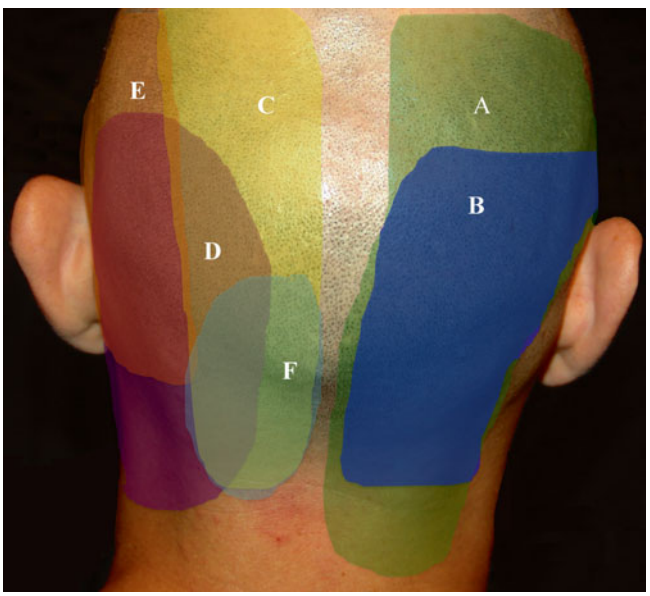

Fig. 18.2 Pattern of posterior cervical and occipital pain. *A* atlantoaxial joint, *B* atlantooccipital joint, *C* greater occipital nerve (GON), *D* posterior auricular nerve (PAN), *E* lesser occipital nerve (LON), *F* third occipital nerve (TON) (Image courtesy of Andrea Trescot, MD)

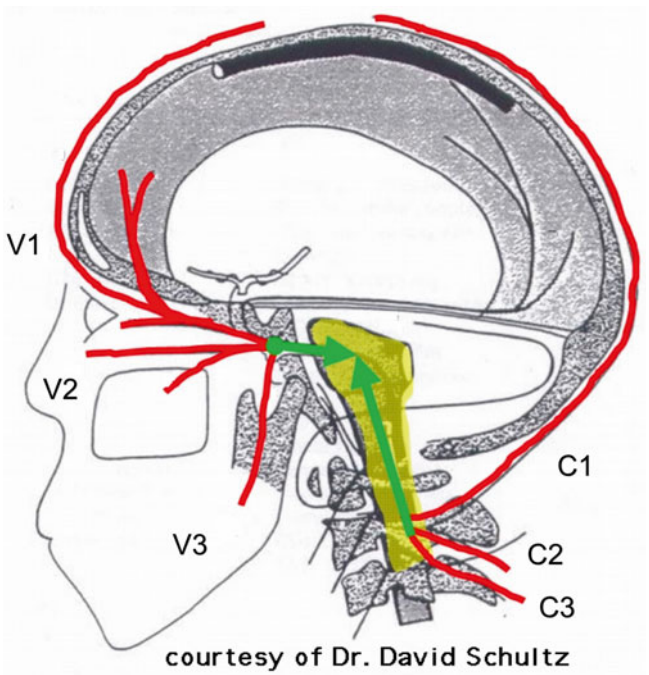

Fig. 18.4 Cervico-trigeminal relay, showing the relationship between the trigeminal nerves (V1, V2, and V3) and the occipital/upper cervical nerve roots, with the nerves connecting within the medulla. (Image courtesy of David Schultz, MD)

LON entrapment can produce throbbing, unilateral or bilateral pain, accompanied by nausea, photophobia, and phonophobia. These symptoms meet the International Classification of Headache Disorders (ICHD) [13] criteria for migraine headache.

Anatomy (Table 18.2)

Similar to the *greater occipital nerve* (Chap. 17), the LON is also a branch of C2. The LON is a purely sensory nerve that originates from the posterior ramus of the C2 (sometimes also from C3) (Fig. 18.5) and then travels as a superficial branch of the cervical plexus, piercing the *superficial cervical plexus* at *Erb's point*, ascending along the posterior border of the sternocleidomastoid muscle (SCM) close to the *spinal accessory nerve* (see Chap. 26) and then to the lateral portion of the skull (Fig. 18.6), exiting the neck lateral to the GON. As it approaches the base of the skull, it perforates the deep fascia and continues upward along the side of the head behind the ear, about 32–90 mm from the midline (Fig. 18.5) [14], supplying sensation to the skin of the mastoid and posterior skull (Fig. 18.2). The LON potentially communicates with the *greater occipital nerve (GON)* (Fig. 18.7) and the *posterior auricular nerve (PAN)/great auricular nerve (GAN)* (see Chap.16). The LON may arise directly from the GON, or it may be small and provide only a limited area of sensation over the neck [15].

Becser et al. [14] dissected ten cadavers and found that the LON was between 32 and 90 mm from the midline along the intermastoid line (Fig. 18.5). During a routine anatomic dissection, Madhavi and Holla [16] identified bilateral triplication of the lesser occipital nerve. The origin of one lesser occipital nerve was from a common origin with the *supraclavicular nerve* (C3/C4) from the cervical plexus. Another lesser occipital nerve ran through the trapezius and was closely related to the exit of the greater occipital nerve. The third ran in a standard path.

Pillay et al. [17] dissected 40 fetuses and identified a single LON in 70 % of the specimens, duplicate LONs in 26 %, and triplicate in 4 %. They also identified two patterns of assent to the occiput; the LON was found on the splenius capitis muscle in 85 % of the cases, but it was found on top of the SCM in 15 %. In 26 % of the specimens, the nerve split from the GAN, with one branch heading to the superior third of the ear and the other branch traveling up the SCM to innervate the mastoid region.

The GAN usually supplies the majority of sensation to the ear (Fig. 18.3); however, 5 of 19 cases dissected by Pantaloni and Sullivan [5] showed that the LON provided two-thirds or more of the sensation of the ear. In the majority of their spec-

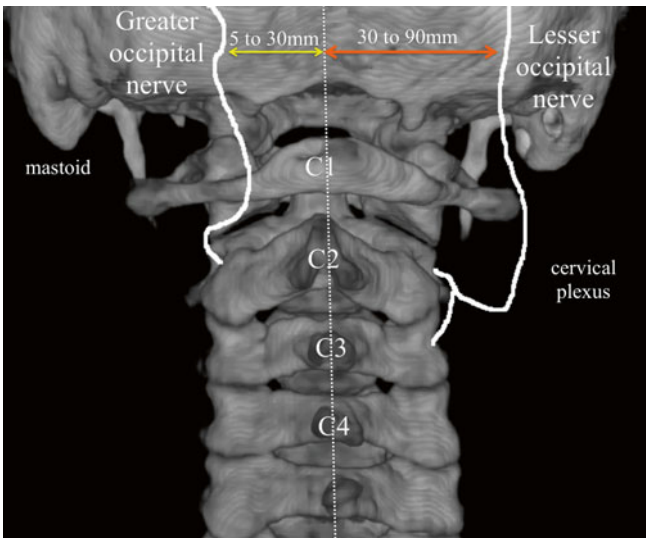

Fig. 18.5 Radiologic anatomy of the greater and lesser occipital nerves (Image courtesy of Andrea Trescot, MD)

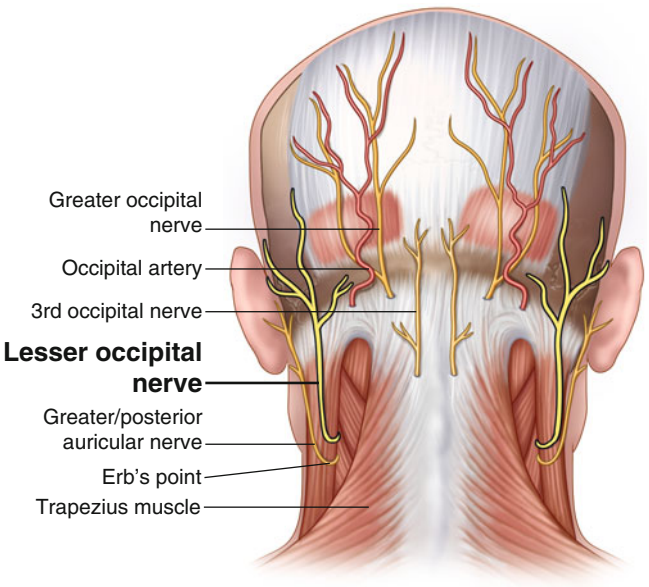

Fig. 18.6 Lesser occipital anatomy (Image courtesy of Springer)

Table 18.2 Lesser occipital nerve anatomy

Origin	Ventral ramus of C2 and sometimes C3
General route	Posterior border of SCM, lateral to the GON at about 2/3 of the way from the occipital protuberance
Sensory distribution	Behind the ear and posterolateral neck
Motor innervation	None
Anatomic variability	Can course along with the GON or physically connect
Other relevant structures	Lateral to occipital artery and superior to vertebral artery

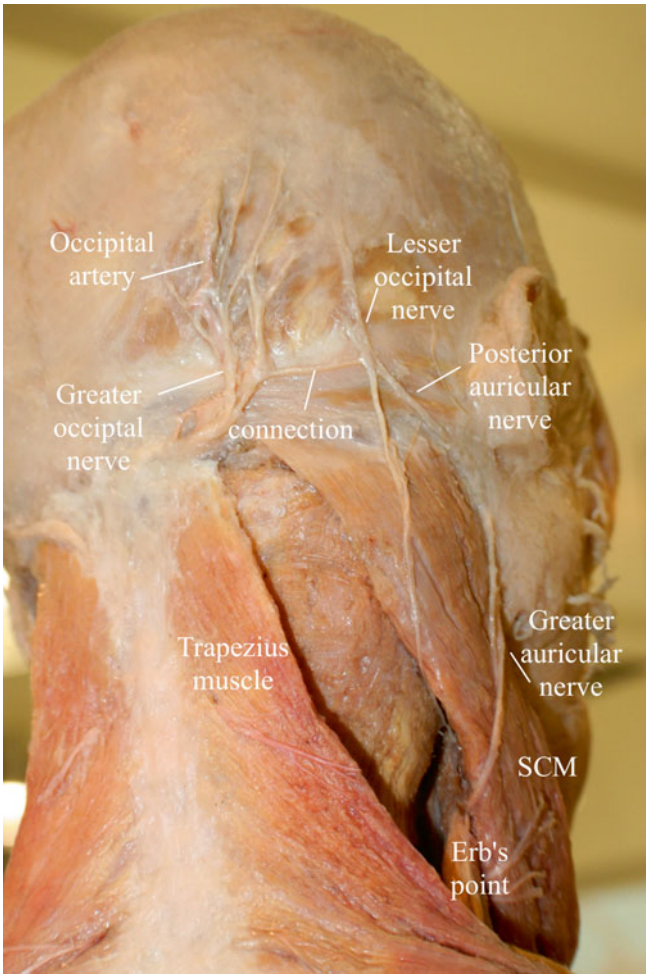

Fig. 18.7 Anatomy of the occipital region, modified from an image from *Bodies*, *The Exhibition*, with permission (Image courtesy of Andrea Trescot, MD)

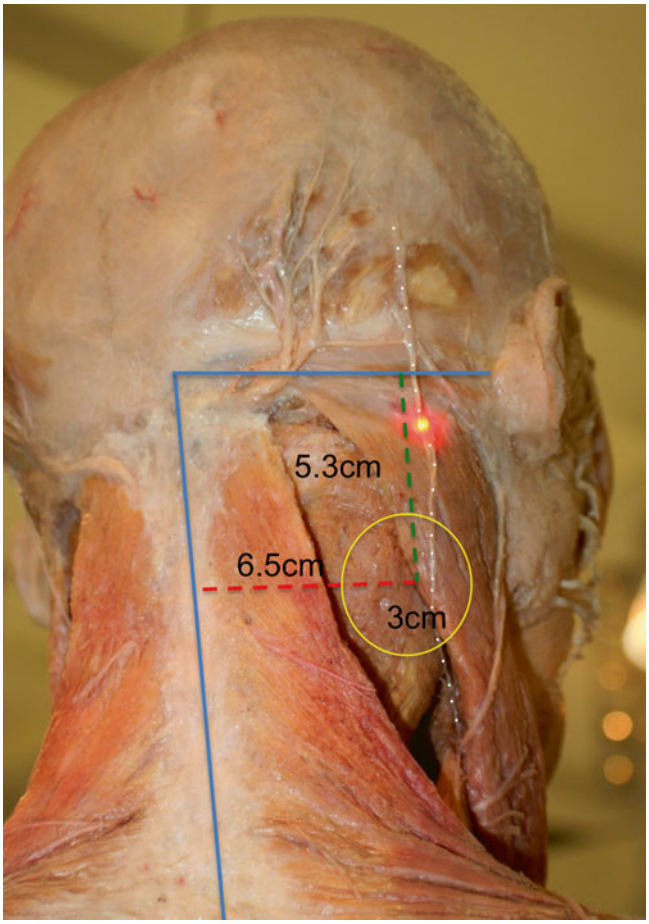

Fig. 18.8 Location of the lesser occipital nerve, according to Dash et al. [12]. *White dots* = lesser occipital nerve, with laser pointer light on the nerve at the occiput (Image created by Andrea Trescot, MD)

imens, the LON supplied the superior ear and mastoid. Though they noted a great deal of variability, the LON was found to consistently emerge from the posterior border of the SCM superior to the GAN (Fig. 18.7).

Dash et al. [12] dissected 30 lesser occipital nerves in 16 cadavers; they originally obtained 20 cadavers, but in 4 cadavers (7 nerves) the prosection was cut too high to find the lesser occipital nerves, and 3 nerves could not be found. Of the 30 lesser occipital nerves that were identified, the nerve consistently emerged posterior to the SCM in an area about 3 cm in diameter, centered about 6.5 cm from the midline and 5.3 cm below a line connecting the external auditory meatus (Fig. 18.8). Four of the nerves were found to pierce the SCM, though none pierced the trapezius muscle.

Entrapment

The C2-C3 facet and therefore the lesser occipital nerve are vulnerable to trauma from flexion-extension injuries [9], especially if the head was turned at impact. Repetitious neck contractions secondary to work, recreational activities, and many other activities can cause entrapment and/or scarring. The SCM can cause entrapment of the LON secondary to myofascial spasms (Fig. 18.9).

During procedures such as a lateral suboccipital craniotomy, the LON, which runs along the SCM into the caudal portion of the surgical field, can be found within 2–3 cm of the inferior border of the surgical incision [4, 7]. Although it is usually possible to mobilize the tissues to move the LON, sometimes a successful surgery cannot be done without sacrificing the nerve. Ng and Page [3] noted

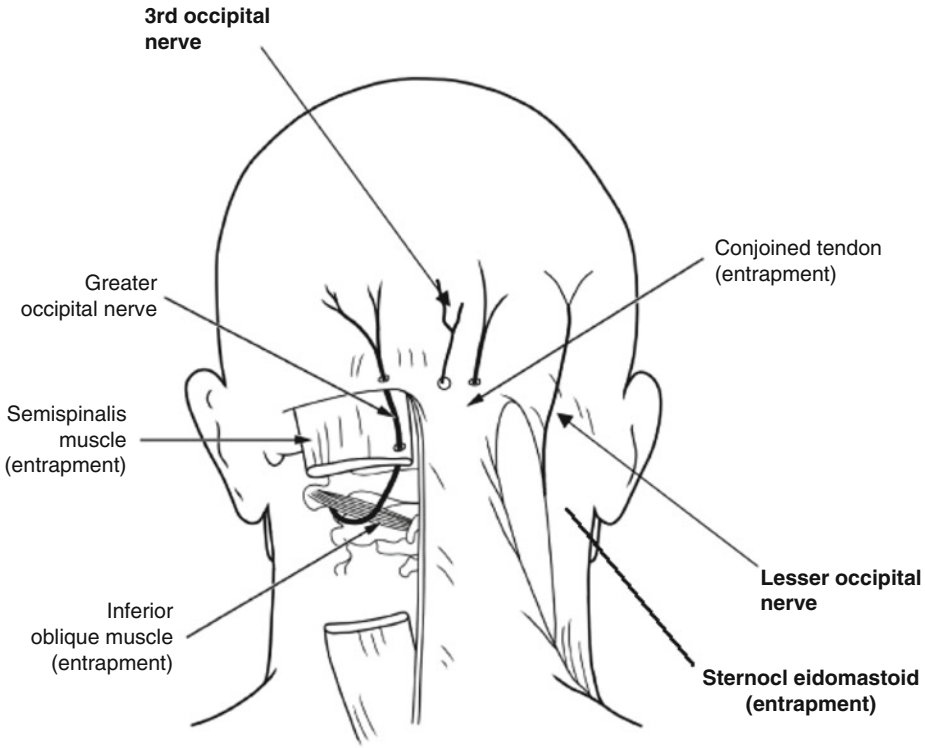

Fig. 18.9 Entrapment of the occipital nerves (Image courtesy of Epimed International®, with permission)

that the lesser occipital nerve and the GAN are at risk from the hard edge of the headrest used in beach chair positioning during shoulder surgery. Pantaloni and Sullivan [5] noted that, in the cases where the LON had a superficial course, it would be at risk of injury during a surgical postauricular flap. They also suggested that platysma suspension sutures should be placed in a vertical-oblique direction to avoid the LON.

LON and GON entrapment are often seen together, causing symptoms of migraines. Dash et al. [12] described 34 migraine patients who had temporary relief from botulinum toxin; 23 noted good relief from release of the GON, while the other 13 noted partial relief, as their headaches had shifted laterally, leading to evaluation of the LON.

Medical conditions such as *osteochondroma* (benign tumors of the bone), arterial compression of the C2 or C3 nerve root by a tortuous vertebral artery (Fig. 18.10), herpes zoster, osteoarthritis, and many others contribute to or are the root cause of entrapment or are the contributing factors causing pain. However, as the ganglion of this nerve interconnects with the trigeminal nerve in the brainstem, pain may be referred to any branch of the trigeminal nerve (Fig. 18.4). Any factor that causes a mechanical stress on the LON can be the source of a headache.

Physical Exam

For the physical exam of the lesser occipital nerve, the patient should be positioned sitting with the neck slightly flexed, supporting the forehead with the non-examining hand or the head resting on the patient's hands (Fig. 18.11) [18]. With the examining hand, place the middle finger at the midline base of the head to identify the site of the foramen magnum. The index finger is then placed at the conjoined tendon attachment. The lesser occipital nerve is commonly found at a point two-thirds of the distance from the external occipital protuberance to the mastoid on the superior nuchal line (Fig. 18.12) (Video 18.1) [19], along a palpable groove in the bone at the posterior edge of the mastoid.

Differential Diagnosis (Table 18.3)

Similar to the other occipital nerves, there is much confusion and controversy regarding the exact location and correct term for headaches that affect the occipital or suboccipital regions of the head with or without radiation to the frontal, temporal, or ocular areas. Headaches such as

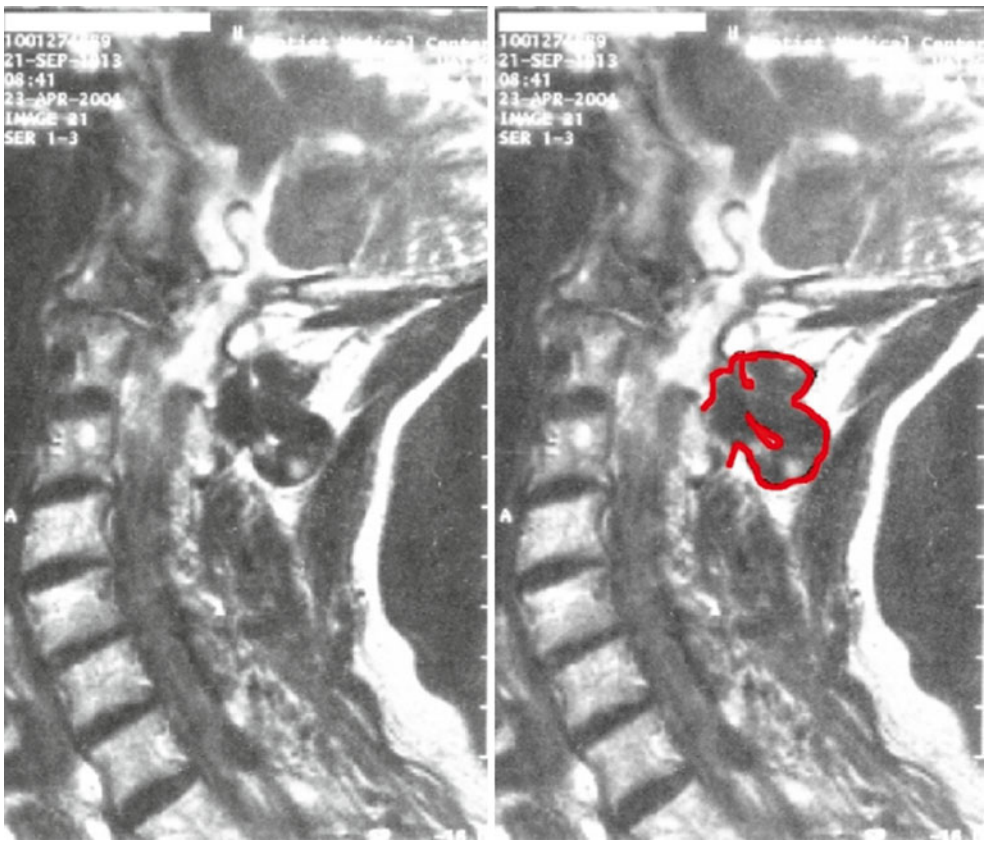

Fig. 18.10 Tortuous vertebral artery (Image courtesy of Andrea Trescot, MD)

Fig. 18.11 Physical examination of the lesser occipital nerve (Image courtesy of Andrea Trescot, MD)

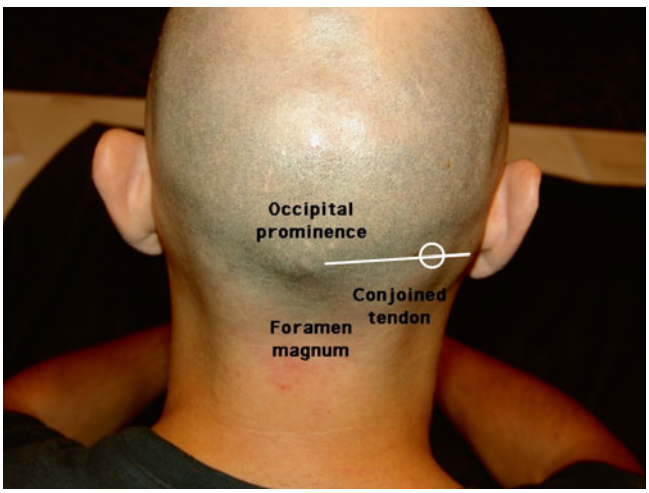

Fig. 18.12 Lesser occipital nerve location (Image courtesy of Andrea Trescot, MD)

migraines, tension-type headaches, and the *trigeminal autonomic cephalalgias* (*hemicranias* and *cluster headaches*) have overlapping features with the occipital neuralgias; therefore, trial injections may be beneficial to patients with these types of headaches, especially if they occurred after trauma or are not responding to the usual medication treatment.

Occipital pain may be considered the result of referred pain from the posterior cervical muscles, the cervical ligaments, the C1 or C2 spinal nerve root, the atlantooccipital joint, the atlantoaxial joint, the C2-C3 zygapophysial joint, the greater occipital nerve, the lesser occipital nerve, or the third occipital nerve (Fig. 18.2) [25]. Tables 18.4 and 18.5.

Table 18.3 Differential diagnosis of occipital pain

	Potential distinguishing features
Tumors of the posterior cranial fossa	Weight loss, visual changes, cranial nerve dysfunction
Neurosyphilis [20]	Cognitive dysfunction, ataxia, sensory disturbance
Zygapophysial joint dysfunction	Pain with neck extension or rotation
Temporal arteritis [21]	Fever, elevated ESR and CRP
Vertebral artery dissection/ compression [22]	Horner's syndrome
C2 myelitis [23]	Loss of function in C2 distribution
C2-C3 intervertebral disc dysfunction	Radiating pain from neck into the shoulder
Atlantoaxial joint dysfunction [24]	Suboccipital pain, focal tenderness over the transverse process of C1, restricted head rotation with pain

Table 18.4 Diagnostic tests for lesser occipital nerve

	Potential distinguishing features
Physical exam	Pain to palpation lateral to the occipital protuberance; pain radiates into the ear and lateral neck
Diagnostic injection	LON block relieves the pain
Ultrasound	Helpful aid to the nerve block
MRI	Radiculopathy, space-occupying lesion
Arteriography	No benefit
X-ray	May show C2 and C3 arthropathy
Electrodiagnostic studies	Not useful

Table 18.5 Contributing factors

Contributing factor	Etiology
Posture	Head forward position shortens the SCM, leading to LON entrapment
Cervical facet pathology	Irritation/entrapment of LON at nerve root level
Stress	Increased SCM spasm leading to entrapment of LON
Ligament laxity	Results in head forward position and SCM spasm
Levator scapula spasm	Since the levator attaches on the cervical transverse process, spasm causes rotational dysfunction
Posture	Cervical facet pathology
Stress	Ligament laxity

Identification and Treatment of Perpetuating Factors

Posture is a significant cause of occipital entrapment (Fig. 18.13), so the patient needs to be taught correct head position, and ergonomic issues must be addressed (e.g., computer location). Since laxity of the cervical ligaments results in spasm of the trapezius (which causes entrapment of the greater occipital nerve) and the SCM (which causes entrapment of the lesser occipital nerve), prolotherapy may relieve the myofascial entrapment.

Injection Technique

Landmark-Guided Injection

Injecting the lesser occipital nerve starts with the patient positioned sitting, with head supported on the patient's arms. With the non-injecting hand, grasp the parietal region and feel the lesser occipital groove with the thumb. Straddle the lesser occipital groove with the index and middle finger, and advance a 27-gauge needle from caudal to cephalad and onto the bone (Fig. 18.14) (Video 18.2). Inject 2 cc maximum volume of local anesthetic and deposteroid. Be careful not to inject superficially because of the risk of steroid-induced alopecia. The use of a nerve stimulator for diagnostic nerve blocks may be helpful.

Ultrasound-Guided Injection

Although there are many articles regarding US for the greater occipital nerve, there are no specific descriptions of US evaluations or injections of the lesser occipital nerve.

Fluoroscopy-Guided Injection

There are no specific fluoroscopic landmarks for the lesser occipital nerve. However, the technique of suboccipital decompression of the occipital nerves (see Chap. 20) uses fluoroscopy.

Neurolytic/Surgical Technique

Cryoneuroablation

If diagnostic injections of the occipital nerve have given temporary, but excellent, relief, the nerve can be a candidate for cryoneuroablation. Cryoneuroablation for all three occipital nerves is conducted at the same site as the diagnostic injections [26]. Kim et al. [27] reported on the results of 38 patients who underwent cryoneuroablation of the greater and lesser occipital nerves, noting an average of 70.5 % relief for 8.1 months.

Radiofrequency Lesioning (RF)

Although there are several articles on the use of RF lesioning of the GON, there has been very little written on the use of

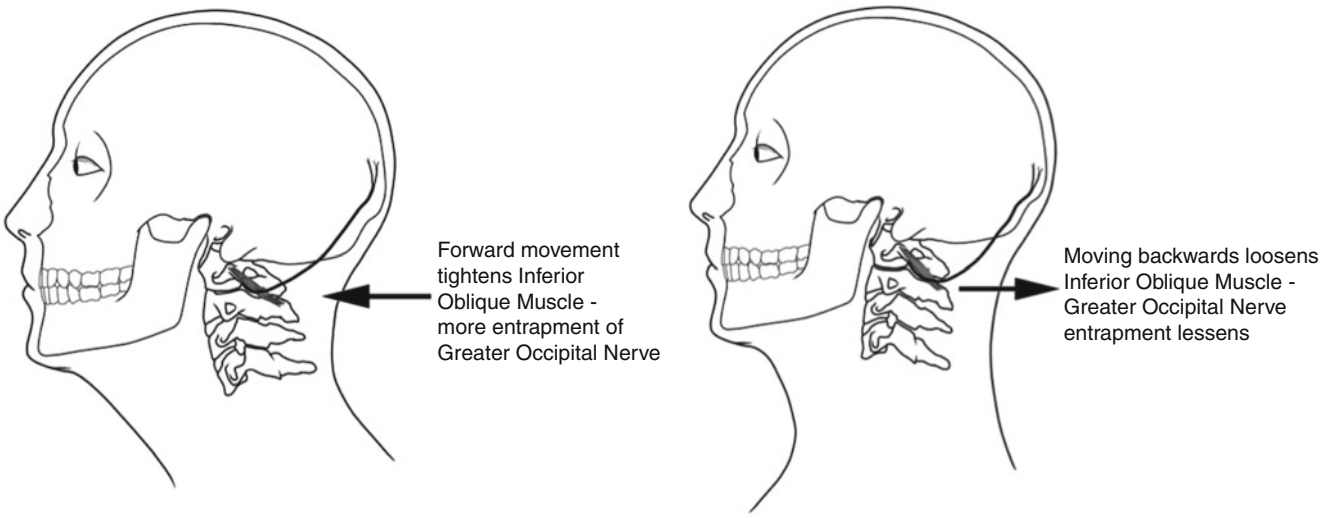

Fig. 18.13 (**a**, **b**) Ergonomic issues of head position on occipital nerve entrapment (Images courtesy of Epimed International®)

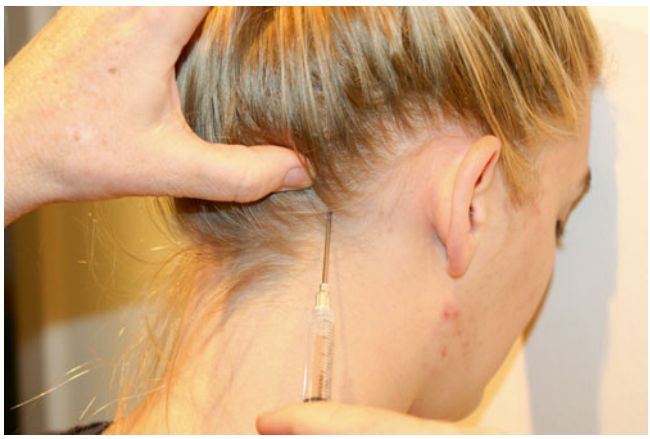

Fig. 18.14 Landmark-guided lesser occipital injection technique (Image courtesy of Andrea Trescot, MD)

this technique for the LON. Vanelderen and colleagues [28] studied pulsed radiofrequency ablation of the greater and/or lesser occipital nerve in 19 patients, using 42 °C for 4 min; after 6 months, 52.6 % noted "substantial" improvement in their headaches.

Neurolytic Injections

Because of the critical structures in this area, including the vertebral artery and spinal cord, injections of alcohol or phenol (because they can spread to unwanted regions) would not be recommended. However, Dash and colleagues [8] discussed the use of botulinum toxin in the treatment of lesser occipital nerve headaches, and they performed dissections to identify the most appropriate site of injection (see 3 "Anatomy" section).

Surgery

Most of the surgical release surgeries describe specifically the greater occipital nerve (see Chap. 17), but the lesser occipital nerve would be expected to be amenable to similar treatment. Jung et al. [29] described greater and lesser occipital neurectomy (with the nerves located by Doppler ultrasound) in a patient with persistent intractable occipital neuralgia.

Peripheral Nerve Stimulation

Occipital nerve stimulation was one of the first peripheral nerve stimulation techniques developed (see Chap. 9). The leads are trialed percutaneously, and then, if the trial is successful, implanted subcutaneously (Fig. 18.15). Although most reports describe its use for the greater occipital nerve (and do not specifically mention the lesser occipital nerve), Fig. 18.15 shows that the lead would cross the lesser occipital nerve, thereby stimulating it. Current reports suggest that 60–80 % of patients with chronic headaches who have failed medication management will have relief from occipital stimulation [30–33]. Occipital stimulation has been used in combination with other cranial stimulators (such as supraorbital) [34]; they have also been found to be useful for cluster headaches [35], trigeminal postherpetic neuralgia [36], and even weight loss [37].

Complications

Complications from occipital nerve blocks with local anesthetic and steroids or neurolytic procedures are exceedingly rare. However, serious complications with occipital interventions may occur and have been reported. Complications

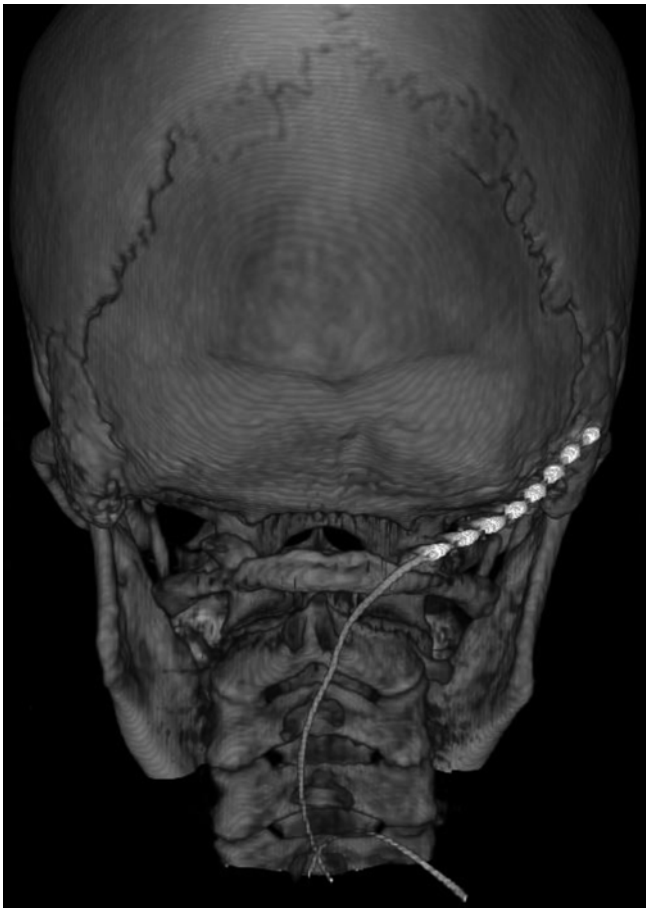

Fig. 18.15 3D image of percutaneous occipital stimulator trial (Image courtesy of Andrea Trescot, MD)

include those related to placement of the needle and those related to the administration of various drugs. Proximity of the needle to the vertebral artery, spinal cord, and nerve root creates risk for injury and makes precise and accurate needle placement extremely important.

These injections should not be attempted in patients who have undergone posterior cranial surgery because of the loss of protective skull coverage. Okuda et al. [38] described a patient who developed sudden unconsciousness during a lesser occipital nerve block. Apparently, unknown to the injector, the patient had undergone prior microvascular decompression for trigeminal neuralgia, with a subsequent craniotomy bony defect. The patient suddenly lost consciousness and stopped breathing, but her cardiopulmonary system was supported appropriately, and 2 h later, the patient was awake with no sequelae. The authors felt the patient had likely suffered a subarachnoid injection.

Because of the use of corticosteroids, occipital nerve blocks may be associated with alopecia and cutaneous atrophy [39] or Cushing's syndrome with repeated injections [40]. Kinney et al. [41] described prolonged facial numbness after an occipital nerve block. Side effects related to the administration of steroids are generally attributed to the chemistry or to the pharmacology of the steroids [42].

Thus, complications may include dural puncture; spinal cord trauma; subdural injection; neural trauma; injection into the intervertebral foramen and intervertebral arteries; intravascular injection into veins, vertebral arteries, or occipital artery; steroid-induced alopecia [39]; infectious complications including epidural abscess and bacterial meningitis; and side effects related to the administration of steroids, local anesthetics, and other drugs. Vertebral artery and ventral ramus damage, along with a risk of embolus resulting in serious neurological sequelae with spinal cord damage and cerebral infarction, are exceedingly rare, but are potential complications with occipital nerve blocks and other interventions. Other minor complications include lightheadedness, flushing, sweating, nausea, hypotension, syncope, pain at the injection site, and increased headaches.

Summary

There is much overlap of the etiology and symptomatology between the LON and the GON; their entrapments are often seen together, causing symptoms of "migraines," and it is usually related to trauma or bad posture due to work or recreational activities.

A good history and physical exam, along with diagnostic injection, can lead to correct diagnosis and treatment of the lesser occipital nerve pain.

References

1. Sjaastad O, Saunte C, Hovdahl H, Breivik H, Gronbaek E. "Cervicogenic" headache. An hypothesis. Cephalalgia. 1983;3(4):249–56.
2. Blume H, Atac M, Golnick J. Neurosurgical treatment of persistent occipital myalgia-neuralgia syndrome. In: Pfaffenrath V, Lundberg P-O, Sjaastad O, editors. Updating in headache. Berlin: Springer; 1985. p. 24–34.
3. Ng AK, Page RS. Greater auricular nerve neuropraxia with beach chair positioning during shoulder surgery. Int J Should Surg. 2010;4(2):48–50.
4. Kemp WJ, Tubbs RS, Cohen-Gadol AA. The innervation of the scalp: a comprehensive review including anatomy, pathology, and neurosurgical correlates. Surg Neurol Int. 2011;2:178.
5. Pantaloni M, Sullivan P. Relevance of the lesser occipital nerve in facial rejuvenation surgery. Plast Reconstr Surg. 2000;105(7):2594–9; discussion 600–3.
6. Tahir M, Corbett S. Lesser occipital nerve neurotmesis following shoulder arthroscopy. J Shoulder Elbow Surg. 2013;22(2):e4–6.
7. Vorobeichik L, Fallucco MA, Hagan RR. Chronic daily headaches secondary to greater auricular and lesser occipital neuromas following endolymphatic shunt surgery. BMJ Case Rep. 2012:on line.
8. Hecht JS. Occipital nerve blocks in postconcussive headaches: a retrospective review and report of ten patients. J Head Trauma Rehabil. 2004;19(1):58–71.

9. Lord SM, Barnsley L, Wallis BJ, Bogduk N. Chronic cervical zygapophysial joint pain after whiplash. A placebo-controlled prevalence study. Spine. 1996;21(15):1737–44.
10. Blumenfeld A, Ashkenazi A, Napchan U, Bender SD, Klein BC, Berliner R, et al. Expert consensus recommendations for the performance of peripheral nerve blocks for headaches – a narrative review. Headache. 2013;53(3):437–46.
11. Dubuisson D. Treatment of occipital neuralgia by partial posterior rhizotomy at C1–3. J Neurosurg. 1995;82(4):581–6.
12. Dash KS, Janis JE, Guyuron B. The lesser and third occipital nerves and migraine headaches. Plast Reconstr Surg. 2005;115(6): 1752–8.
13. The International Classification of Headache Disorders, 3rd edition (beta version). Cephalalgia. 2013;33(9):629–808.
14. Becser N, Bovim G, Sjaastad O. Extracranial nerves in the posterior part of the head. Anatomic variations and their possible clinical significance. Spine. 1998;23(13):1435–41.
15. Bergman RA, Afifi AK, Miyauchi R. Lesser occipital nerve 2014 [5/21/15]. Available from: http://www.anatomyatlases.org/AnatomicVariants/NervousSystem/Text/LesserOccipitalNerve.shtml.
16. Madhavi C, Holla SJ. Triplication of the lesser occipital nerve. Clin Anat. 2004;17(8):667–71.
17. Pillay P, Partab P, Lazarus L, Satyapai KS. The lesser occipital nerve in fetuses. Int J Morphol. 2012;30(1):140–4.
18. Trescot AM. Headache: is it a migraine? Think again. Technol Reg Anesthesiol Pain Manag. 2005;9:68–72.
19. Brown DL, Wong GY. Greater and lesser occipital nerve blocks. In: Waldman SD, editor. Pain management. 2. Philadelphia: Saunders Elsevier; 2007. p. 1140–51.
20. Smith DL, Lucas LM, Kumar KL. Greater occipital neuralgia: an unusual presenting feature of neurosyphilis. Headache. 1987; 27(10):552–4.
21. Jundt JW, Mock D. Temporal arteritis with normal erythrocyte sedimentation rates presenting as occipital neuralgia. Arthritis Rheum. 1991;34(2):217–9.
22. Sharma RR, Parekh HC, Prabhu S, Gurusinghe NT, Bertolis G. Compression of the C-2 root by a rare anomalous ectatic vertebral artery. Case report. J Neurosurg. 1993;78(4):669–72.
23. Nikakis P, Koutsis G, Potagas C, Mandellos D, Sfagos C. Occipital neuralgia as an isolated symptom of C2 myelitis. Headache. 2006;46(8):1304–6.
24. Ehni G, Benner B. Occipital neuralgia and the C1-2 arthrosis syndrome. J Neurosurg. 1984;61(5):961–5.
25. Bogduk N, Marsland A. On the concept of third occipital headache. J Neurol Neurosurg Psychiatry. 1986;49(7):775–80.
26. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
27. Kim CH, Hu W, Gao J, Dragan K, Whealton T, Julian C. Cryoablation for the treatment of occipital neuralgia. Pain Physician. 2015;18:E363–8.
28. Vanelderen P, Rouwette T, De Vooght P, Puylaert M, Heylen R, Vissers K, et al. Pulsed radiofrequency for the treatment of occipital neuralgia: a prospective study with 6 months of follow-up. Reg Anesth Pain Med. 2010;35(2):148–51.
29. Jung SJ, Moon SK, Kim TY, Eom KS. A case of occipital neuralgia in the greater and lesser occipital nerves treated with neurectomy by using transcranial Doppler sonography: technical aspects. Korean J Pain. 2011;24(1):48–52.
30. Saper JR, Dodick DW, Silberstein SD, McCarville S, Sun M, Goadsby PJ. Occipital nerve stimulation for the treatment of intractable chronic migraine headache: ONSTIM feasibility study. Cephalalgia. 2010;31(3):271–85.
31. Freeman JA, Trentman TL. Clinical utility of implantable neurostimulation devices in the treatment of chronic migraine. Medical Devices (Auckland, NZ). 2013;6:195–201.
32. Verrills P, Rose R, Mitchell B, Vivian D, Barnard A. Peripheral nerve field stimulation for chronic headache: 60 cases and long-term follow-up. Neuromodulation. 2014;17(1):54–9.
33. Hong J, Ball PA, Fanciullo GJ. Neurostimulation for neck pain and headache. Headache. 2014;54(3):430–44.
34. Deshpande KK, Wininger KL. Feasibility of combined epicranial temporal and occipital neurostimulation: treatment of a challenging case of headache. Pain Physician. 2011;14(1):37–44.
35. Mueller O, Diener HC, Dammann P, Rabe K, Hagel V, Sure U, et al. Occipital nerve stimulation for intractable chronic cluster headache or migraine: a critical analysis of direct treatment costs and complications. Cephalalgia. 2013;33(16):1283–91.
36. Johnson MD, Burchiel KJ. Peripheral stimulation for treatment of trigeminal postherpetic neuralgia and trigeminal posttraumatic neuropathic pain: a pilot study. Neurosurgery. 2004;55(1):135–41; discussion 41–2.
37. Sobocki J, Herman RM, Fraczek M. Occipital C1-C2 neuromodulation decreases body mass and fat stores and modifies activity of the autonomic nervous system in morbidly obese patients – a pilot study. Obes Surg. 2013;23(5):693–7.
38. Okuda Y, Matsumoto T, Shinohara M, Kitajima T, Kim P. Sudden unconsciousness during a lesser occipital nerve block in a patient with the occipital bone defect. Eur J Anaesthesiol. 2001; 18(12):829–32.
39. Shields KG, Levy MJ, Goadsby PJ. Alopecia and cutaneous atrophy after greater occipital nerve infiltration with corticosteroid. Neurology. 2004;63(11):2193–4.
40. Lavin PJ, Workman R. Cushing syndrome induced by serial occipital nerve blocks containing corticosteroids. Headache. 2001;41(9):902–4.
41. Kinney MA, Wilson JL, Carmichael SW, De Ruyter ML, Fulgham JR. Prolonged facial hypesthesia resulting from greater occipital nerve block. Clin Anat. 2003;16(4):362–5.
42. Manchikanti L. Pharmacology of neuraxial steroids. In: Manchikanti L, Singh V, editors. Interventional techniques in chronic spinal pain. Paducah: ASIPP Publishing; 2007. p. 167–84.

Third Occipital Nerve Entrapment

19

Esther Rawner and Andrea M. Trescot

Introduction

Anatomic recognition of multiple occipital nerves (the *greater occipital nerve*, the *lesser occipital nerve*, and the *third occipital nerve*), each with different patterns of pain, neurologic origin, and treatment modalities, has been facilitated by the use of precision diagnostic injections. The third occipital nerve (TON) is the least studied of all the occipital nerves, and TON blocks are usually done when greater occipital nerve (GON) and lesser occipital nerve (LON) injections fail to relieve pain. This chapter will focus specifically on the TON; see Chap. 17 for the GON and Chap. 18 for the LON.

Clinical Presentation (Table 19.1)

As a subset of *cervicogenic headaches* (CGH), occipital neuralgia can cause pain and paresthesias to the posterior scalp; the periorbital, temporal, and mandibular regions; the external ear and mastoid regions; and the neck and shoulders. Unilateral occipital neuralgia, due to the proximity of the occipital artery, can present with throbbing, unilateral headaches associated with photophobia, phonophobia, and nausea, which meet the International Headache Society (IHS) criteria for migraines [3].

There are multiple overlapping patterns of pain from the structures causing cervicogenic headaches, including the upper cervical facets, the greater occipital nerve (Chap. 17), the lesser occipital nerve (Chap. 18), and the posterior auricular nerve (Chap. 16) (Fig. 19.1).

Cervical facet joint pathology is one of the common causes of neck pain, and it is the origin of chronic neck pain in more than 50 % of patients after whiplash injuries [4]. However, cervical facet pathology can also cause occipital headaches, as evidenced by experimental facet injections on normal volunteers [5]. Among 100 patients with whiplash-related neck pain and headaches, 27 % were found to be due to third occipital neuralgia, while third occipital neuralgia was the cause of pain in 53 % of those patients with predominantly headache rather than neck pain [6].

Although the term "occipital neuralgia" is used primarily for pathology involving the greater occipital nerve, the clinical presentation of the greater occipital nerve (GON), lesser occipital nerve (LON), and third occipital nerve (TON) can have a great deal of overlap. However, the headache caused by the TON tends to be an occipital/suboccipital headache without radiation to the temples or vertex (Fig. 19.2).

The TON neuralgia was not recognized as a separate entity until 1986, when Bogduk [7] described the clinical presentation of TON pathology [8]. Initial studies of the TON used uncontrolled blocks; they reported complete occipital pain relief after blocking the third occipital nerves [9]. A study using controlled cervical facet injections collaborated the findings of the uncontrolled blocks [6] and also

Table 19.1 Occupation/exercise/trauma history relevant to third occipital nerve entrapment

Surgery	Posterior craniotomy [1]
Cervical facet radiofrequency	Neuralgia after C2-C3 facet denervation with subsequent third occipital neuritis [2]
Flexion/extension injuries	"Migraine" after motor vehicle collision
Manual labor	Hypertonicity in neck musculature
"Desk job" with a computer	Improper ergonomics

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provided estimates of the prevalence of headache and neck pain stemming from the C2-C3 zygapophysial joint (27 %). However, there were felt to be no clinical features by which pain from the TON can be diagnosed. The combination of an occipital headache and tenderness over the C2-C3 facet has an 85 % sensitivity, but collectively those clinical features have a positive likelihood ratio of only 2:1 when confirmed by diagnostic injections [6].

Fig. 19.1 Pattern of pain seen with third occipital neuralgia (Image courtesy of Andrea Trescot, MD)

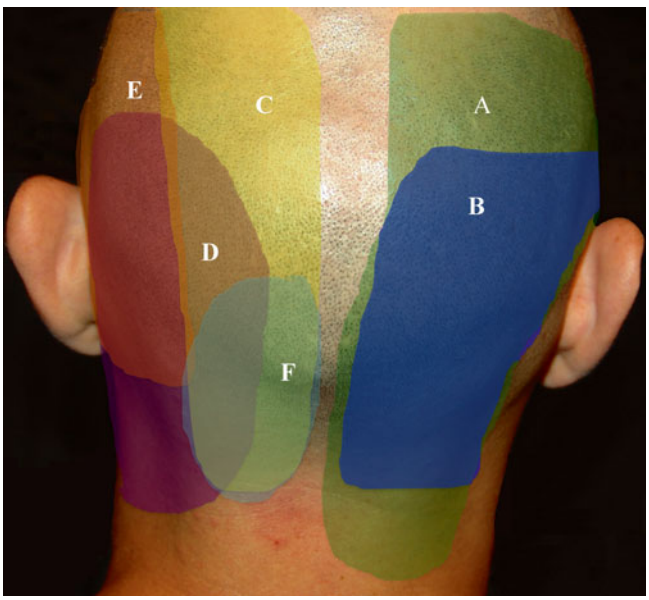

Fig. 19.2 Pattern of posterior cervical and occipital pain. *A* atlantoaxial joint, *B* atlantooccipital joint, *C* greater occipital nerve (GON), *D* posterior auricular nerve (PAN), *E* lesser occipital nerve (LON), *F* third occipital nerve (TON) (Image courtesy of Andrea Trescot, MD)

Anatomy (Table 19.2)

The greater, lesser, and third occipital nerves have distinct but often overlapping pain patterns. The third occipital nerve is the medial branch of the dorsal ramus of C2-C3 (Fig. 19.3). The innervation of the C2-C3 facet is different than the other, lower cervical facets. The deep branch of the medial dorsal ramus of C2-C3 innervates the C2-C3 facet; the TON is the superficial medial branch of that dorsal ramus, which crosses the lateral and posterior aspect of the C2-C3 zygapophysial joint (Figs. 19.3 and 19.4) [10], but it may run lower or higher than this plane. The C2-C3 facet, and thus the TON, appears vulnerable to trauma from acceleration-deceleration injuries of the neck [11]. After supplying the joint, the TON courses superiorly and medially, piercing the semispinalis capitis, splenius capitis, and trapezius muscle medial to the GON (Fig. 19.5), after which it joins the GON in sensory innervation to the suboccipital region [8]. Dash et al. [12] dissected 20 cadavers and found the TON to be 13.2 ± 5.3 mm from the midline and 62.0 ± 20.0 mm down from the line between the two external auditory canals.

Entrapment

Because the third occipital nerve pierces the nuchal fascia at the base of the skull (Fig. 19.5), it is prone to trauma from flexion/tension injuries. Dash et al. [12] felt that the TON is

Table 19.2 Third occipital nerve anatomy

Origin	Medial branch of the dorsal ramus of C2-C3
General route	Travels dorsomedial around the C2-C3 zygapophysial joint between the multifidus and the semispinalis capitis. Arises under and pierces the trapezius; it is located inferior and medial to the GON and communicates with the GON to supply the skin of the suboccipital region
Sensory distribution	C2-C3 zygapophysial joint, skin of the lower part of the back of the head
Motor innervation	Semispinalis capitis (in combination with the greater occipital nerve)
Anatomic variability	May be superiorly or inferiorly located within the superior articular process of C3
Other relevant structures	Trapezius muscle, splenius capitis

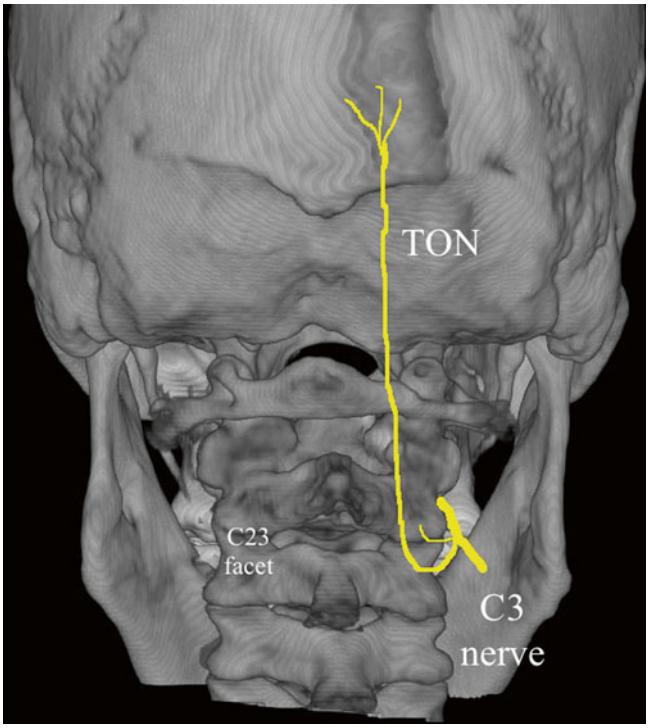

Fig. 19.3 Anatomy of the third occipital nerve (Image courtesy of Andrea Trescot, MD)

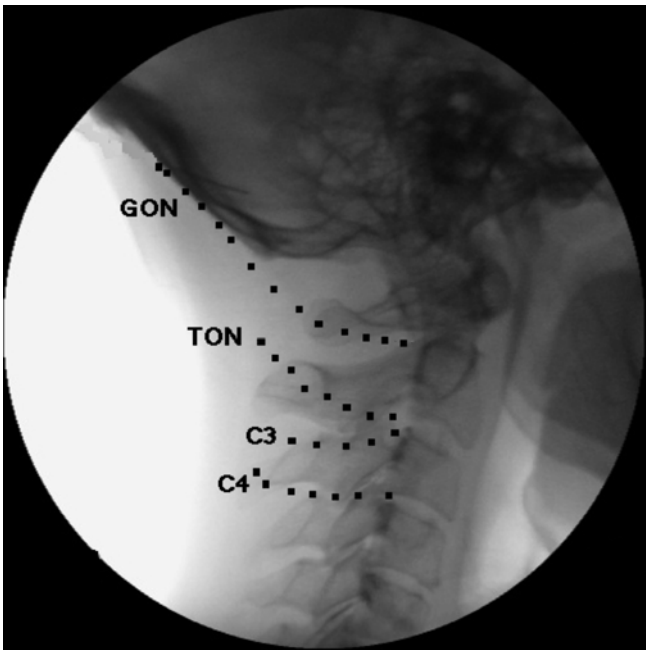

Fig. 19.4 Path of the occipital nerves – lateral fluoroscopy image. *GON* greater occipital nerve, *TON* third occipital nerve (Image courtesy of Andrea Trescot, MD)

primarily entrapped peripherally by the muscular structures. However, according to Bogduk and Marsland [7], the TON is most commonly injured due to C2-C3 facet arthropathy.

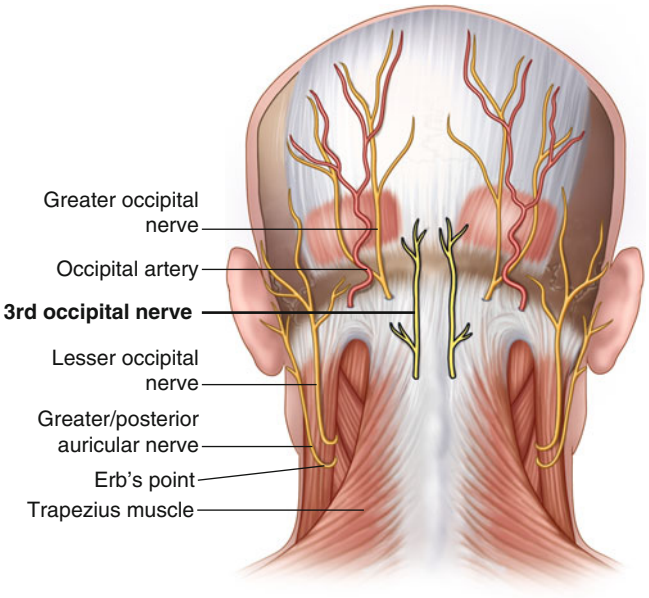

Fig. 19.5 Anatomy of the occiput and the third occipital nerve (Image by Springer)

They asserted that there are no clinical features by which pain from the C2-C3 zygapophysial joint can be diagnosed. They felt that TON pathology could be suspected if the patient has an occipital headache and tenderness maximally over the C2-C3 joint, but diagnostic blocks may be the only means of establishing the diagnosis for certain types of cervicogenic headaches originating from the third occipital nerve. Thus, there are two sites of entrapment and therefore two different injection sites (see below).

Surgery in the high posterior cervical region can also put the TON at risk, since midline retraction can irritate the nerve superficially in the soft tissues as well as deeper in the region close to the C2-C3 facet. After dissection of the posterior cervical nerves in 14 cadavers, Zhang et al. [13] concluded that a high degree of lateral retraction of the paravertebral muscles (PVM) during surgery could overstretch the medial branches, including C3 and the TON. Tubbs et al. [1] felt that tension on the TON by retractors could contribute to post-craniotomy headaches, and they recommended that surgeons occasionally loosen the midline retractors to release tension on the nerve.

Physical Exam

To examine the third occipital nerve peripherally, the surface landmarks are identified (Fig. 19.6). The patient should rest their head on their hands while the examiner places their thumbs together on the midline at the base of the skull and pushes laterally against the medial conjoint tendon (Fig. 19.7). Alternatively, the examiner places the middle fin-

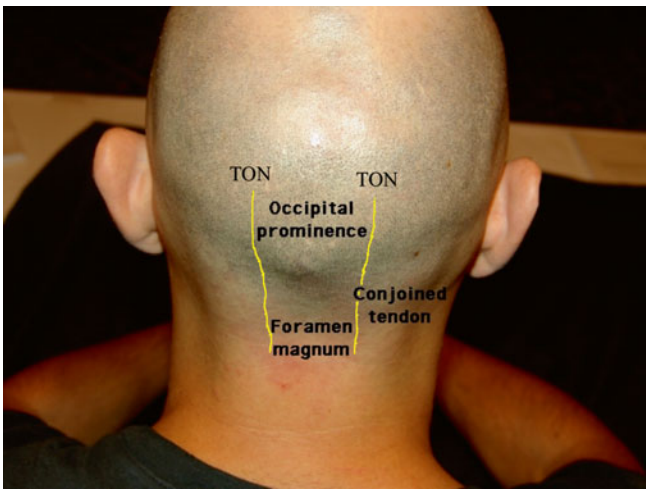

Fig. 19.6 Surface anatomy of the third occipital nerve (Image courtesy of Andrea Trescot, MD)

Fig. 19.7 Physical exam of the third occipital nerve – technique A with thumbs in the midline (Image courtesy of Andrea Trescot, MD)

ger over the foramen magnum (Fig. 19.8) and then places the index finger medial to the conjoined tendon (Fig. 19.9) (Video 19.1). Proximally, tenderness over the C2-C3 facet is the clue that the TON may be involved (Fig. 19.10).

Differential Diagnosis (Table 19.3)

Similar to the posterior head and neck pain of GON and LON entrapment, there is much confusion and controversy regarding the exact location and the correct term for headaches that affect the occipital or suboccipital regions of the head with or without radiation to the frontal, temporal, or ocular areas (see Table 19.3). Headaches described as "tension headaches" may respond to one of the occipital nerve injections [19]. Occipital pain may be considered the result of referred pain from the posterior cervical muscles, the cer-

Fig. 19.8 Physical exam of the third occipital nerve – technique B, identifying the foramen magnum with the middle finger (Image courtesy of Andrea Trescot, MD)

Fig. 19.9 Physical exam of the third occipital nerve – technique B, with the middle finger in the foramen magnum and the index finger placed medial to the conjoined tendon (Image courtesy of Andrea Trescot, MD)

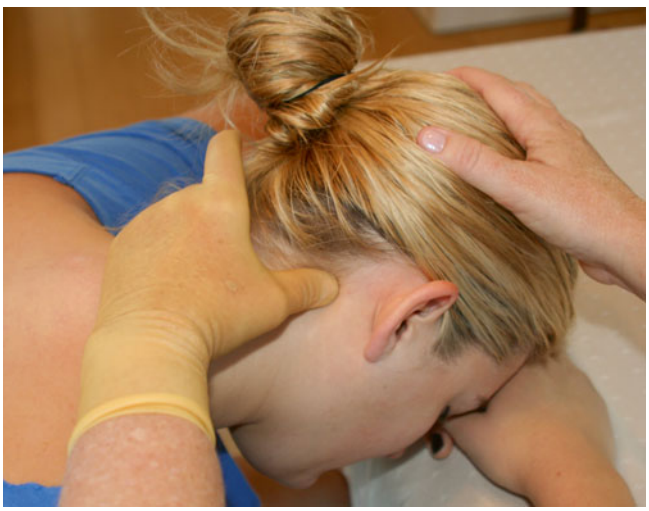

Fig. 19.10 Physical exam of the upper cervical facets (Image courtesy of Andrea Trescot, MD)

Table 19.3 Differential diagnosis of occipital pain

	Potential distinguishing features
Greater or lesser occipital nerve	Tenderness more laterally
Tumors of the posterior cranial fossa	Weight loss, visual changes, cranial nerve dysfunction
Neurosyphilis [14]	Cognitive dysfunction, ataxia, sensory disturbance, +RPR
Zygapophysial joint dysfunction	Pain with neck extension or rotation
Temporal arteritis [15]	Fever, elevated ESR and CRP
Vertebral artery dissection/ compression [16]	Horner's syndrome
C2 myelitis [17]	Loss of function in C2 distribution
C2-C3 intervertebral disk dysfunction	Radiating pain from the neck into the shoulder
Atlantoaxial joint dysfunction [18]	Suboccipital pain, focal tenderness over the transverse process of C1, restricted head rotation with pain

Table 19.4 Diagnostic tests for the third occipital nerve

	Potential distinguishing features
Physical exam	Tenderness medial to the conjoined tendon
Diagnostic injection	TON block relieves the pain
Ultrasound	Useful aid for nerve injection
MRI	Radiculopathy, space-occupying lesion
Arteriography	Not useful
Fluoroscopy	For localization of the zygapophysial joint
X-ray	Compressive radiculopathy
Electrodiagnostic studies	EMG may be useful if suspecting cervical radiculopathy

vical ligaments, the C1 or C2 spinal nerve root, the C2-C3 zygapophysial joint, or the third occipital nerve [7]. The diagnostic tests for the TON are listed in Table 19.4.

Although the history and physical exam may give clues to a TON entrapment, according to Bogduk [10], the diagnosis can only be made through diagnostic injections.

Identification and Treatment of Contributing Factors (Table 19.5)

Poor posture can be due to poor vision, poor ergonomics, or ligament laxity. The need to peer forward because of poor vision or poor ergonomics, such as an inappropriately placed computer, will cause extension of the head on the neck, entrapping the GON and TON at the inferior oblique. Cervical ligament laxity causes a head-forward position to "take up the slack" of the ligament; the subsequent trapezius spasm can entrap the GON and TON.

Table 19.5 Contributing factors for TON entrapment

Contributing factor	Etiology
Posture	Head-forward position entraps GON and TON at the inferior oblique
Cervical facet pathology	Irritation/entrapment of TON at nerve root level
Stress	Increased trapezius spasm leading to entrapment of GON and TON
Ligament laxity	Results in head-forward position and trapezius spasm
Levator scapula spasm	Since the levator attaches on the cervical transverse process, spasm causes rotational dysfunction

Injection Technique

TON blocks have diagnostic use in that they can pinpoint the source of pain in patients with headache for whom no other source or cause has been found. Among all three of the occipital nerve blocks, third occipital nerve blocks are the only validated diagnostic blocks for headache. TON injections have been the only blocks shown consistently to have a high yield of positive responses under controlled conditions.

Landmark-Guided Injections

For the TON landmark-guided injections, have the patient sit and support their head with their own hands. With the non-injecting hand, grasp the occiput and feel for the conjoint tendon. Direct a 27-gauge needle from caudal to cephalad, just medial to the conjoint tendon and onto the periosteum, taking care to avoid the foramen magnum (Fig. 19.11) (Video 19.2). Anthony [20] has proposed the use of a nerve stimulator for diagnostic nerve blocks to aid in localization of the nerve.

Fluoroscopic-Directed Injection

The fluoroscopic injection of the TON focuses on the C2-C3 facet. The patient can be positioned in the lateral position, with the needle directed to the center of the articular trapezoid (Fig. 19.12). Although there is a variable course of the TON, even 0.5 cc of local anesthetic at the center of the trapezoid should anesthetize the nerve. Alternatively, the patient is placed prone, and, after a cervical articular view is obtained, a needle is placed in a posterior approach in a parasagittal plane, tangential to the lateral margin of the articular pillar of C2-C3, at the "waist" of the articular pillar (Fig. 19.13). The advantage of the prone technique is that the radiofrequency lesioning technique (see below) is done in a similar manner.

Fig. 19.11 Landmark-guided injection of the third occipital nerve. The middle finger is midline over the foramen magnum; the index finger is on the conjoined tendon (Image courtesy of Andrea Trescot, MD)

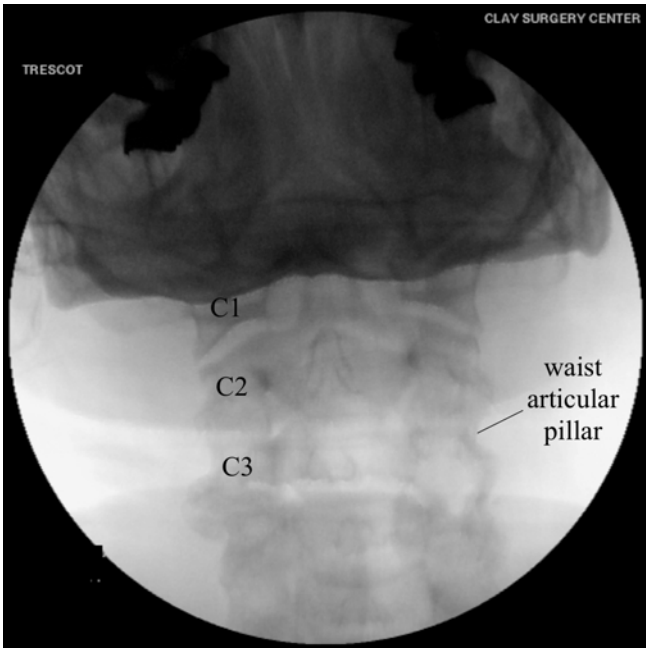

Fig. 19.13 Fluoroscopic landmarks for a third occipital nerve block, prone approach (Image courtesy of Andrea Trescot, MD)

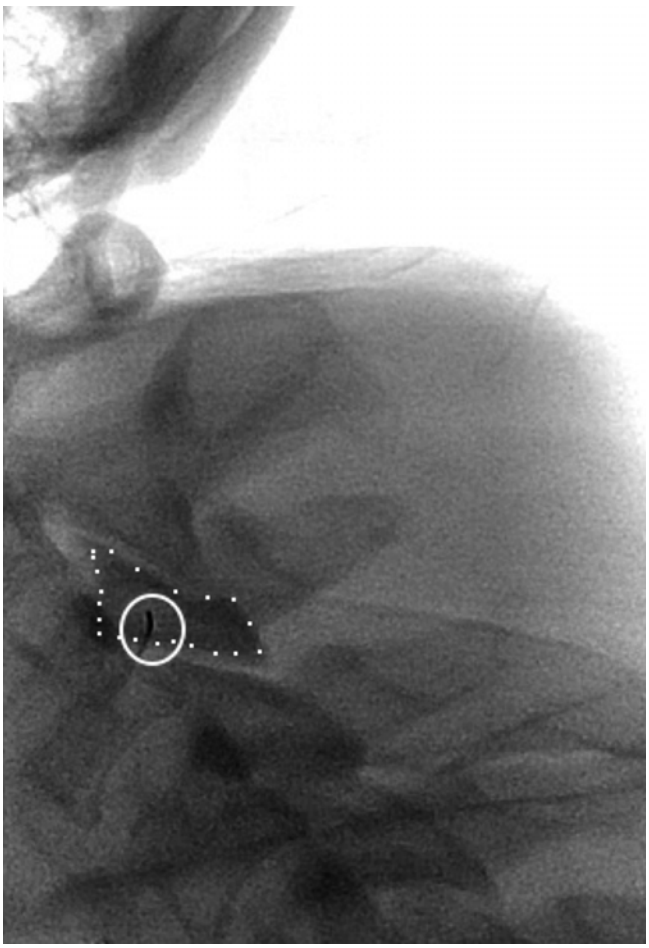

Fig. 19.12 Fluoroscopic injection of the third occipital nerve, lateral approach. *Dotted* area represents the articular trapezoid; the needle is identified by the *white circle* (Image courtesy of Andrea Trescot, MD)

Ultrasound-Directed Injection

The TON injection under US is performed with the patient prone or sitting with neck flexed and supported. The US transducer is placed transversely over the external occipital prominence and then moved caudally over the atlas (Fig. 19.14a). The C2 posterior elements are easily identified because the spinous process of C2 is always bifid; the probe is then moved laterally and rotated slightly (with the lateral portion of the probe rotated cephalad) (Fig. 19.14a), identifying the external oblique muscle and the GON crossing over the muscle (caudad to cephalad, lateral to medial), with the TON located medial to the GON (Fig. 19.14b, c) [21, 22].

Kim and colleagues [4] described a slightly different US technique. The patient is positioned in the lateral decubitus position with the side to be treated up (Fig. 19.15). The linear transducer is placed first vertically on the mastoid and then moved caudally to identify C2-C3. The TON is seen as a hypoechoic oval encircled by a hyperechoic "halo" of the C2-C3 facet (probably better described as a "sitting on a hyperechoic cup") at the superior portion of the joint (Fig. 19.16). The medial branch of C2-C3 is seen at the "waist" of the articular pillar inferiorly. The needle is advanced in-plane, cephalad to caudad under direct vision, with the final needle tip perpendicular to the nerve.

Siegenthaler et al. [23] reported a successful visualization of TON in 96 % of the attempted injections. Eichenberger et al. [22] reported that they could find the correct level of C2-C3 joint and were able to perform TON block on ultrasound guidance in 27 of 28 cases.

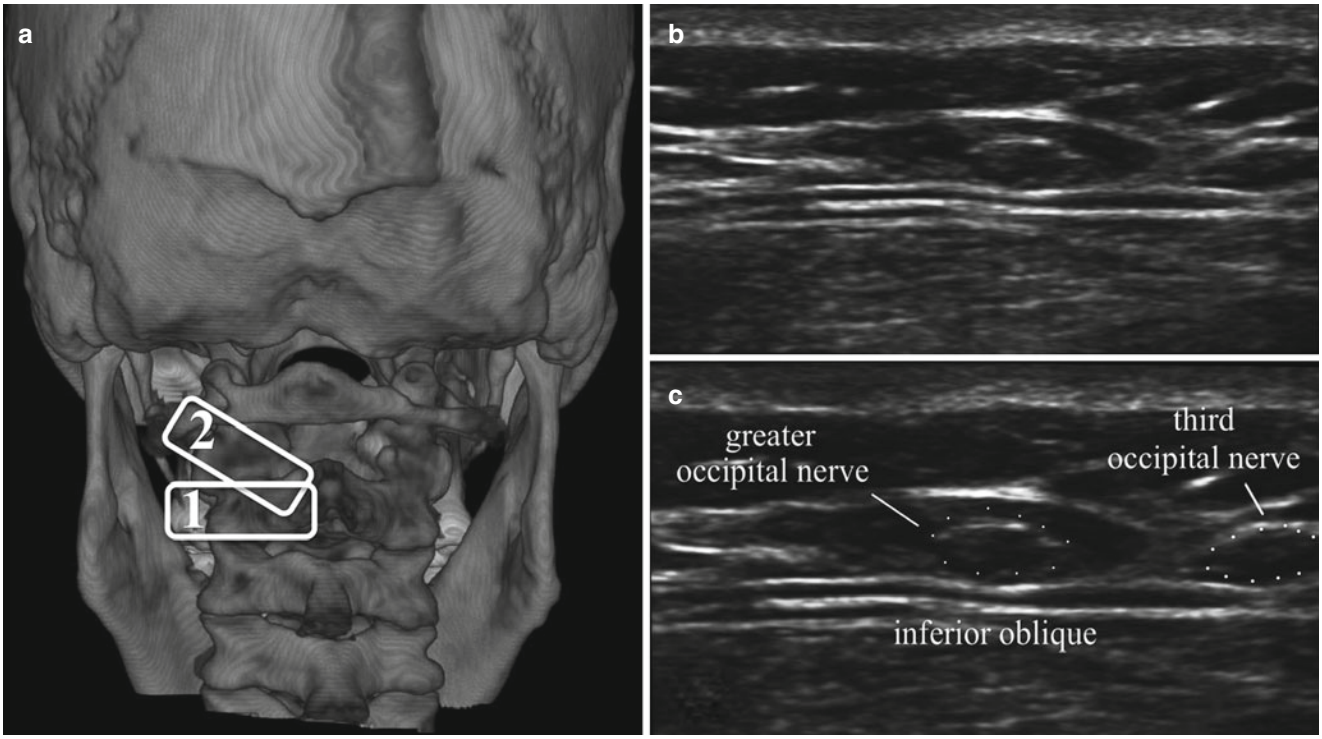

Fig. 19.14 US imaging of the third occipital nerve. (**a**) Location of ultrasound transducer: *1* = the initial probe placement; *2* = the final probe placement. (**b**) US image of the proximal third occipital nerve. (**c**) Labeled US image (Image courtesy of Andrea Trescot, MD, modified from Greher et al. [21])

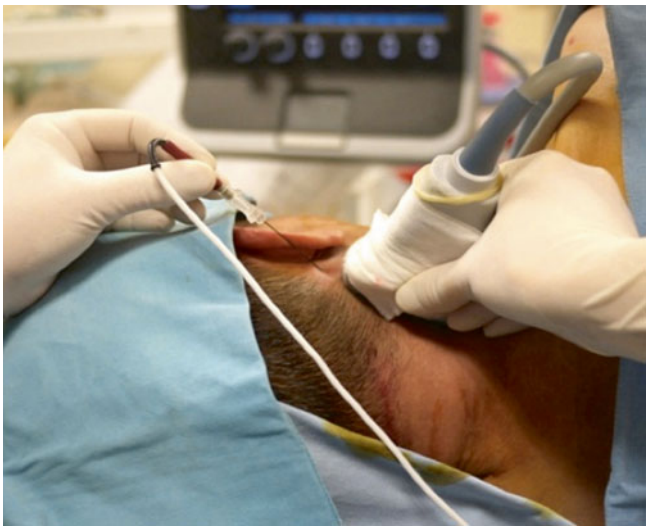

Fig. 19.15 Location of the US probe and needle – Kim technique [4] (Image from Kim et al. [4]; with permission)

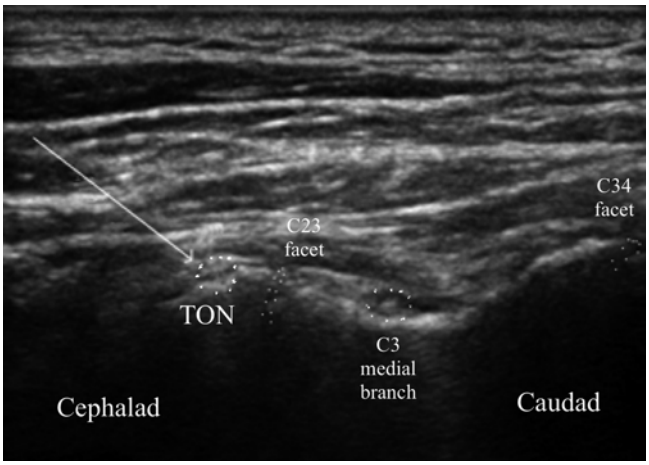

Fig. 19.16 US image of the third occipital nerve (TON) – Kim technique [4] (Image courtesy of Andrea Trescot, MD)

Finlayson et al. [24] performed a randomized trial with 40 patients coming for TON injections, comparing US and fluoroscopic injections of the TON. Ultrasound guidance was associated with significantly shorter performance time and fewer needle passes. Both techniques resulted in similar success rates (95–100 %) with hypoesthesia primarily in the suboccipital region. Under fluoroscopy, the contrast spread intra-articularly in 15 % of the patients, with vascular uptake in 10 %; there were no such findings under US visualization [but it is not possible to visualize inside the joint under US].

Silva et al. [25] presented the results of small-volume injections of methylene blue (0.3–0.5 cc) onto the third occipital nerve (TON) in cadavers; dissections showed that with even just 0.3 cc of volume, 83.3 % of the cadavers had

methylene blue coating the GON, suggesting that injections of the TON usually affect the GON as well (Fig. 19.17).

MRI-Directed Injections

Galliano and colleagues [26], in a letter to the editor, claimed that MRI could be used for the identification and injection of the TON (Fig. 19.18), but did not describe the technique.

Neurolytic/Surgical Technique

Cryoneuroablation

If diagnostic injections of the third occipital nerve have given excellent, but temporary, relief, the patient might be a candidate for cryoneuroablation. Cryoneuroablation for all three occipital nerves is conducted at the same site as the diagnostic injections. Although not well published, cryoneuroablation of the occipital nerves is one of the most common uses of the cryoneuroablation technique [27]. Cryoneuroablation for the TON can be performed at the base of the skull medial to the conjoined tendon (Fig. 19.19) or at the C2-C3 facet.

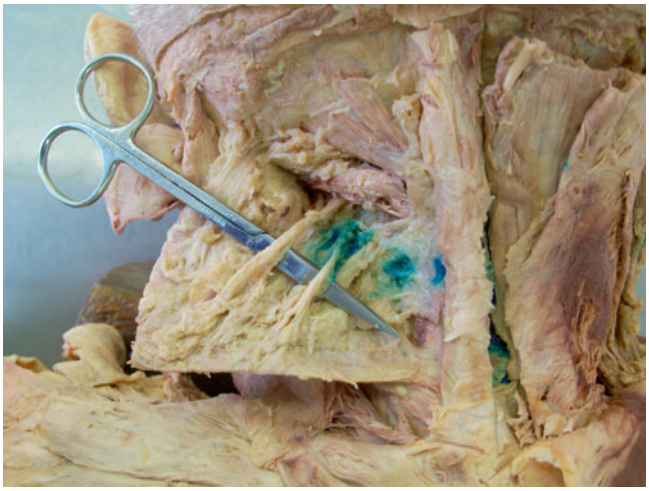

Fig. 19.17 Dissection of the greater occipital and third occipital nerves after methyl blue injection of the third occipital nerve (From Sayed Emal Wahezi from Silva et al. [25], reprinted with permission)

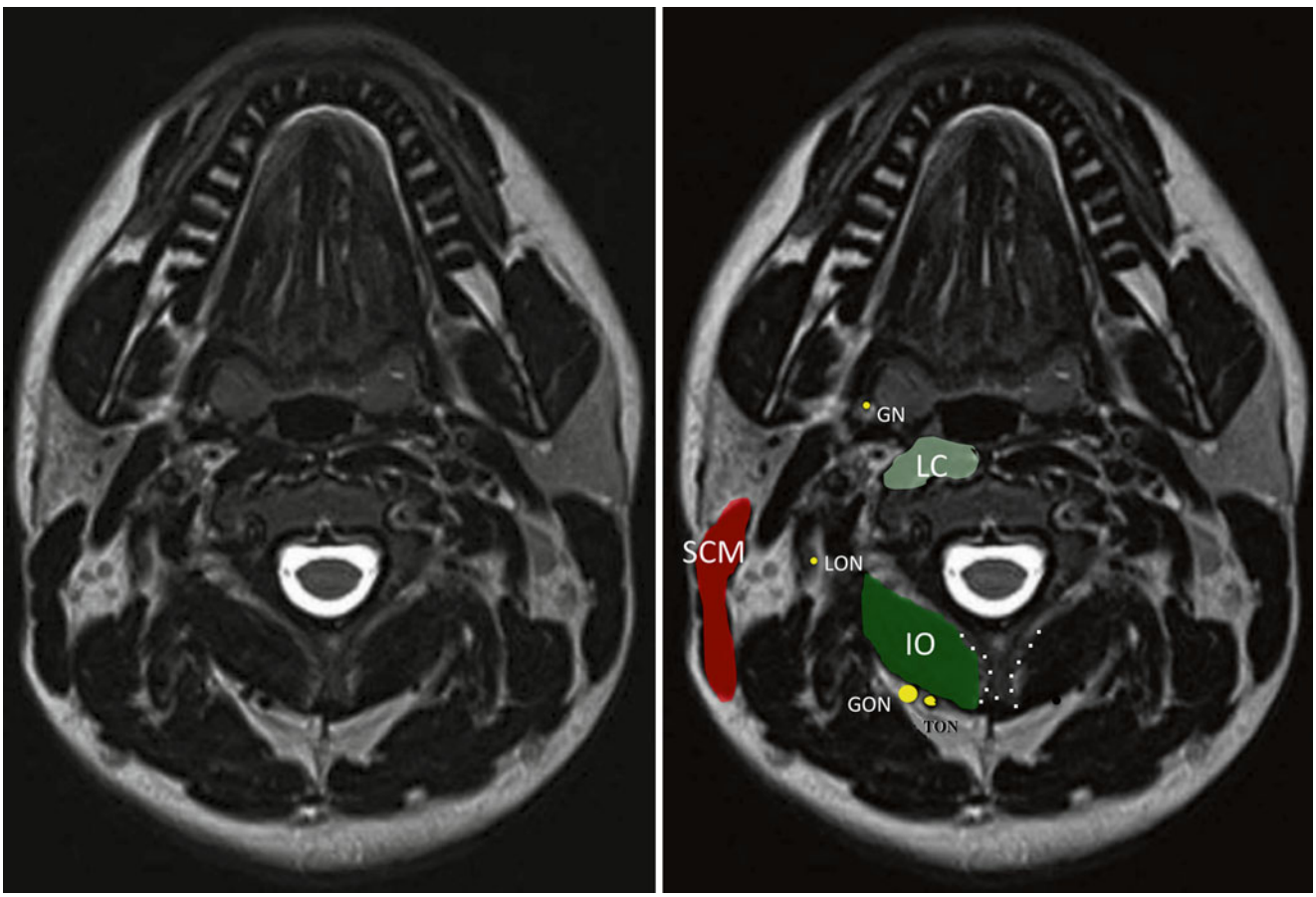

Fig. 19.18 MRI image showing the location of the third occipital nerve, noting the bifid spinous process (Image courtesy of Andrea Trescot, MD; adapted from Galliano et al. [25])

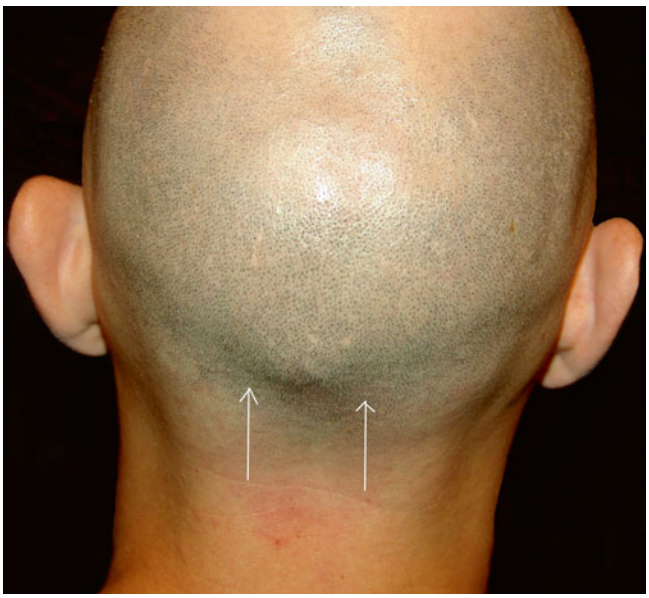

Fig. 19.19 *Arrows* show site of cryoneuroablation of the third occipital nerve (Image courtesy of Andrea Trescot, MD)

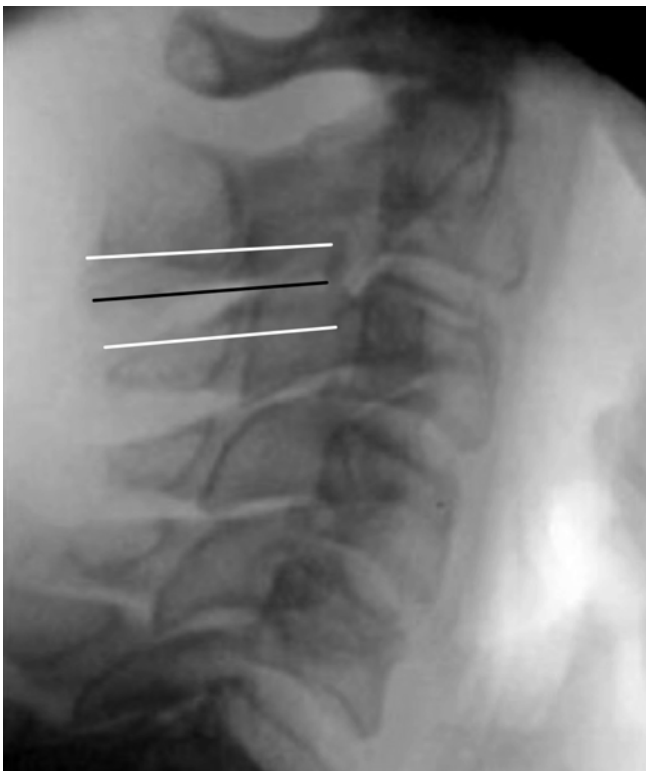

Fig. 19.20 Radiofrequency lesioning (articular view) of the third occipital nerve, with simulated needle placement above and below (Image courtesy of Andrea Trescot, MD)

Radiofrequency Lesioning

Continuous (or heat) radiofrequency lesioning (RFL) of the C2-C3 facet joint (and therefore the TON) has been described [28]. The TON most often crosses the C2-C3 joint opposite the center of the C3 superior articular process, but it may run lower or higher than this plane, thus requiring the needles to be placed opposite the middle of the joint and above and below the joint [29]. Since the electron density is greatest at the tip of the RF probe, in conventional RF, the electrode is placed parallel to the nerve [30]; because of the variability of the nerve location, additional lesions are performed caudad and cephalad. The patient is placed prone, and, after a cervical articular view is obtained, curved RF probes are placed in a posterior approach in a parasagittal plane, tangential to the lateral margin of the articular pillar of C2-C3, with lesions at several levels for complete denervation (Fig. 19.20).

Alternatively, the needle can be placed from a posterior approach; because of the high cervical location, an open-mouth approach view of the posterior C2-C3 facet for RF needle placement, as described by Park et al. [31], may be useful (Fig. 19.21).

There are case reports of good response to RFL, such as the report by Giblin et al. [32]; they described a patient with *trigeminal autonomic cephalgia* who underwent right TON diagnostic injections and then RFL, with "complete" relief. Hamer and Purath [33] reported on 40 refractory cervicogenic headache patients treated with RF of the C2 dorsal root ganglion and/or TON; they described that 35 % of the patients reported 100 % relief and 70 % reported 80 % or more relief. Perhaps more compelling, 92.5 % of the patients reported that they would undergo the procedure again.

Unfortunately, RFL carries a significant risk of neuroma formation or deafferentation pain (*anesthesia dolorosa*). Gaselka et al. [2] retrospectively reviewed charts on 64 patients who had undergone RFL at C2 and C3; 12 patients (19 %) identified new pain (burning or painful dysesthesias) postoperatively, consistent with TON neuralgia. Although some resolved spontaneously, one patient still had pain 1 year later.

Because of the lack of structural damage, pulsed RF (PRF) should have less of those neuropathic risks. In addition, it is technically more difficult to position the RF probe under US so that the probe is parallel to the nerve. With PRF, the optimal probe position is actually perpendicular to the nerve, a position that is much easier to place under US guidance. Kim and colleagues [4] described US placement of RF probes in two patients to perform PRF on the TON, positioned from a lateral approach with a vertical US probe orientation (see *US-guided injections*). Caution must be used, however, when performing RF (especially conventional RF) in the presence of a nearby spinal cord stimulator (SCS), because the electrical field could induce heating of the implanted SCS leads, causing damage to the SCS system as well as heat damage to unintended neural tissues [34].

Neurolytic Injections

Because of the critical structures in this area, including the vertebral artery and spinal cord, injections of alcohol or phenol would not be recommended, as they can spread to unwanted regions.

Botulinum Toxin

Botulinum toxin (type A and type B) has been used for headaches since the fortuitous observation by plastic surgeons that the use of the toxin on forehead wrinkles decreased the occurrence of "migraines" [35]. Its use for cervical dystonia is FDA approved, but off-label use for the muscle spasms associated with cervicogenic headaches has been reported [36]. However, the toxin has also been reported useful for severe occipital neuralgia [37]. Dash and colleagues [12] discussed the use of botulinum toxin in the treatment of third occipital nerve headaches, and they performed dissections to identify the most appropriate site (see 3 "*Anatomy*" section). According to the dissections, they recommended two injections of botulinum toxin along a 4 cm vertical line located 1.3 cm from midline, with one injection 1 cm above and the other 1 cm below a horizontal line connecting the most inferior part of the external auditory canal.

Occipital Nerve Stimulation

Vallejo et al. [38] described the technique of subcutaneous placement of electrodes over the occipital ridge for chronic migraines (Fig. 19.22). Johnstone and Sundaraj [39] described eight cases of using occipital nerve stimulation for occipital neuralgia, while Magiis et al. [40] and Burns et al. [41] used occipital stimulators as part of a treatment program for drug-resistant cluster headaches.

Surgical Treatment

Surgical decompression of the occipital nerves (primarily the greater occipital nerve) has been reported to "cure" headaches in 89 % of migraine patients [42]. However, it has been unclear what, if any, role there is for TON excision in the treatment of intractable migraines. Lee et al. [43] retrospectively compared the charts of 229 patients; the third occipital nerve "removed" group (111 patients) and the third occipital nerve "not removed" group (118 patients) were comparable in terms of age, gender, number of surgical sites, and were statistically well matched regarding preoperative headache characteristics. Comparing the patients who had the TON removed to the TON-not-removed group, migraine headache index reduction was 63 % vs. 64 %, suggesting a limited, if any, role.

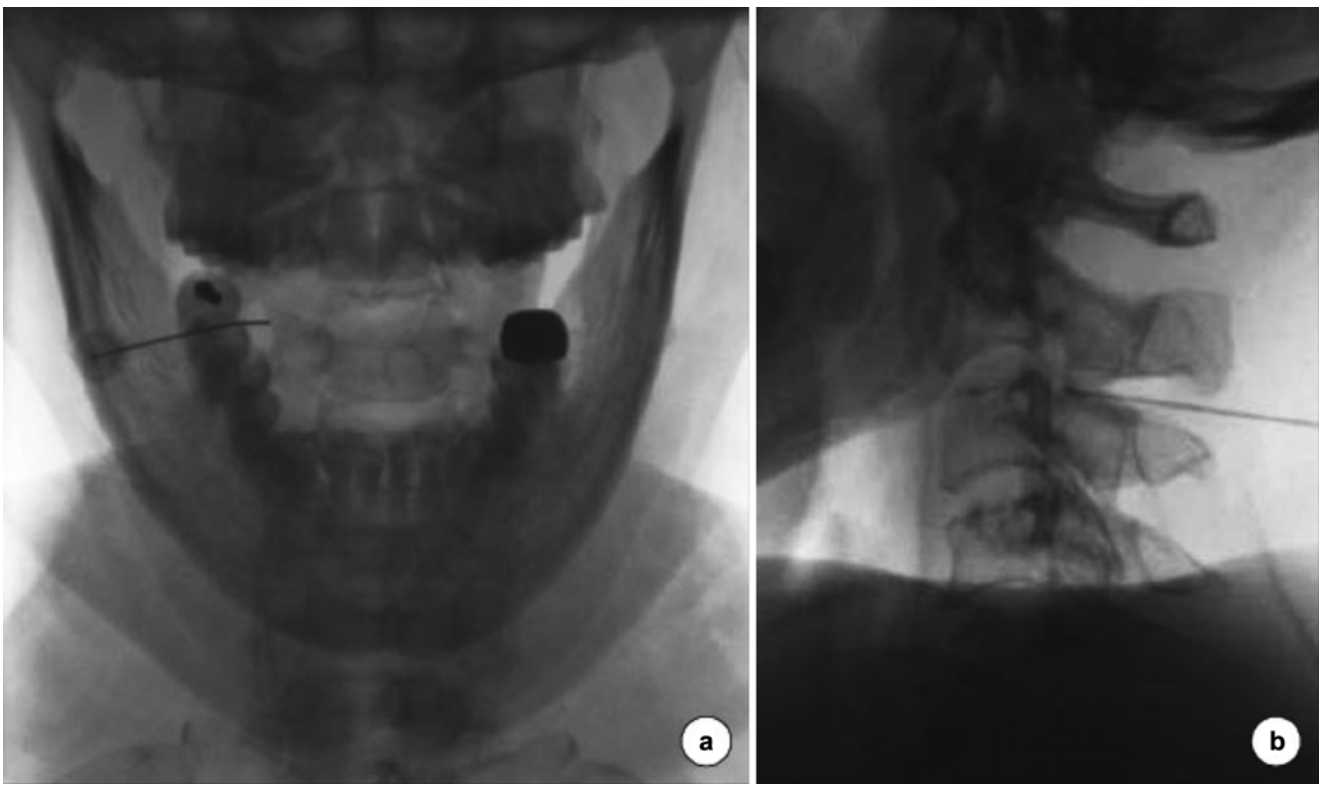

Fig. 19.21 Open-mouth radiofrequency lesioning; (**a**) AP view; (**b**) lateral view (Image from Park et al. [31]; with permission)

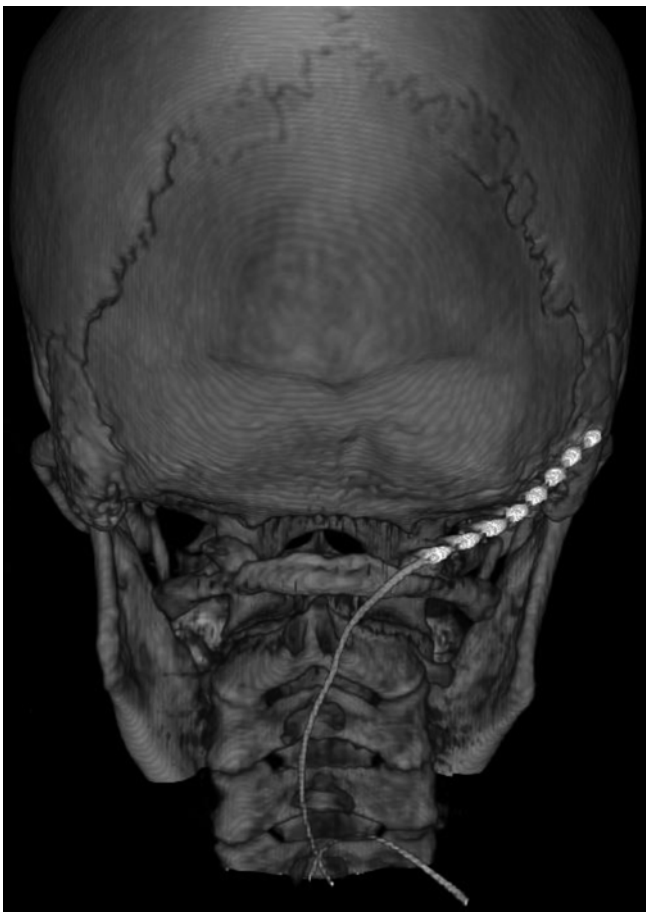

Fig. 19.22 Subcutaneous occipital nerve stimulation (Image courtesy of Andrea Trescot, MD)

Complications

Complications from occipital nerve blocks with local anesthetic and steroids or neurolytic procedures are exceedingly rare. However, serious complications with occipital interventions may occur and have been reported. Complications include those related to placement of the needle and those related to the administration of various drugs. Proximity of the needle to the vertebral artery, spinal cord, and nerve roots creates risk for injury and makes precise and accurate needle placement extremely important.

Complications from needle placement may include dural puncture; spinal cord trauma; subdural injection; neural trauma; injection into the intervertebral foramen and arteries; intravascular injection into veins, vertebral arteries, or occipital artery; infectious complications including epidural abscess and bacterial meningitis; and side effects related to the administration of steroids, local anesthetics, and other drugs. Vertebral artery damage, intrathecal local anesthetic injection [44], along with a risk of embolus resulting in serious neurological sequelae with spinal cord damage and cerebral infarction are exceedingly rare, but are potential complications with occipital nerve blocks and other upper cervical interventions [45].

Gazelka et al. [2] looked at the incidence of neuritis after C2-C3 and TON RF lesioning; of the 64 patients treated, 12 patients (19 %) were identified with "ablation-induced" neuritis, and 10 of those patients required treatment, despite the use of intraoperative steroids in eight of those ten patients (which is commonly used to reduce the risk of neuritis).

Summary

The TON is an under-recognized cause of occipital headaches, and it is the least studied of all the occipital nerves. A careful history and physical exam, as well as a high index of suspicion, will help the clinician recognize and treat TON entrapment appropriately.

References

1. Tubbs RS, Salter EG, Wellons JC, Blount JP, Oakes WJ. Landmarks for the identification of the cutaneous nerves of the occiput and nuchal regions. Clin Anat. 2007;20(3):235–8.
2. Gazelka HM, Knievel S, Mauck WD, Moeschler SM, Pingree MJ, Rho RH, et al. Incidence of neuropathic pain after radiofrequency denervation of the third occipital nerve. J Pain Res. 2014;7:195–8.
3. The International Classification of Headache Disorders: 2nd edition. Cephalalgia. 2004;24 Suppl 1:9–160.
4. Kim ED, Kim YH, Park CM, Kwak JA, Moon DE. Ultrasound-guided pulsed radiofrequency of the third occipital nerve. Korean J Pain. 2013;26(2):186–90.
5. Dwyer A, Aprill C, Bogduk N. Cervical zygapophyseal joint pain patterns. I: a study in normal volunteers. Spine (Phila Pa 1976). 1990;15(6):453–7.
6. Lord SM, Barnsley L, Wallis BJ, Bogduk N. Third occipital nerve headache: a prevalence study. J Neurol Neurosurg Psychiatry. 1994;57(10):1187–90.
7. Bogduk N, Marsland A. On the concept of third occipital headache. J Neurol Neurosurg Psychiatry. 1986;49(7):775–80.
8. Bogduk N. The clinical anatomy of the cervical dorsal rami. Spine (Phila Pa 1976). 1982;7(4):319–30.
9. Biondi DM. Cervicogenic headache: a review of diagnostic and treatment strategies. J Am Osteopath Assoc. 2005;105(4 Suppl 2):16S–22.
10. Bogduk N. 3rd occipital nerve. In: Bogduk N, editor. Practice guidelines for spinal diagnostic and treatment procedures. San Francisco: International Spine Intervention Society (ISIS); 2004.
11. Lord SM, Barnsley L, Wallis BJ, Bogduk N. Chronic cervical zygapophysial joint pain after whiplash. A placebo-controlled prevalence study. Spine. 1996;21(15):1737–44.
12. Dash KS, Janis JE, Guyuron B. The lesser and third occipital nerves and migraine headaches. Plast Reconstr Surg. 2005;115(6):1752–8; discussion 9-60.
13. Zhang J, Tsuzuki N, Hirabayashi S, Saiki K, Fujita K. Surgical anatomy of the nerves and muscles in the posterior cervical spine: a guide for avoiding inadvertent nerve injuries during the posterior approach. Spine. 2003;28(13):1379–84.
14. Smith DL, Lucas LM, Kumar KL. Greater occipital neuralgia: an unusual presenting feature of neurosyphilis. Headache. 1987;27(10):552–4.

15. Jundt JW, Mock D. Temporal arteritis with normal erythrocyte sedimentation rates presenting as occipital neuralgia. Arthritis Rheum. 1991;34(2):217–9.
16. Sharma RR, Parekh HC, Prabhu S, Gurusinghe NT, Bertolis G. Compression of the C-2 root by a rare anomalous ectatic vertebral artery. Case report. J Neurosurg. 1993;78(4):669–72.
17. Nikakis P, Koutsis G, Potagas C, Mandellos D, Sfagos C. Occipital neuralgia as an isolated symptom of C2 myelitis. Headache. 2006;46(8):1304–6.
18. Ehni G, Benner B. Occipital neuralgia and the C1-2 arthrosis syndrome. J Neurosurg. 1984;61(5):961–5.
19. Leinisch-Dahlke E, Jurgens T, Bogdahn U, Jakob W, May A. Greater occipital nerve block is ineffective in chronic tension type headache. Cephalalgia. 2005;25(9):704–8.
20. Anthony M. Arrest of attacks of cluster headache by local steroid injections in the occipital myalgia-neuralgia syndrome. In: Pfaffenrath V, Lundberg PO, Sjaastad O, editors. Updating in headache. Berlin: Springer; 1985. p. 17–23.
21. Greher M, Moriggl B, Curatolo M, Kirchmair L, Eichenberger U. Sonographic visualization and ultrasound-guided blockade of the greater occipital nerve: a comparison of two selective techniques confirmed by anatomical dissection. Br J Anaesth. 2010;104(5):637–42.
22. Eichenberger U, Greher M, Kapral S, Marhofer P, Wiest R, Remonda L, et al. Sonographic visualization and ultrasound-guided block of the third occipital nerve: prospective for a new method to diagnose C2–C3 zygapophysial joint pain. Anesthesiology. 2006;104(2):303–8.
23. Siegenthaler A, Schliessbach J, Curatolo M, Eichenberger U. Ultrasound anatomy of the nerves supplying the cervical zygapophyseal joints: an exploratory study. Reg Anesth Pain Med. 2011;36(6):606–10.
24. Finlayson RJ, Etheridge JP, Vieira L, Gupta G, de Tran QH. A randomized comparison between ultrasound- and fluoroscopy-guided third occipital nerve block. Reg Anesth Pain Med. 2013;38(3):212–7.
25. Silva K, Emmam M, Lederman A, Downie S, Wahezi S. Cervical third occipital nerve blocks concomitantly affect the greater occipital nerve: cadaveric study. NYNJSIPP annual meeting; Jersey City, 2014.
26. Galiano K, Obwegeser AA, Bodner G, Schatzer R, Schocke M, Gruber H. Ultrasound is not the only technique to visualize third occipital nerve blockade. Anesthesiology. 2006;105(4):858; author reply.
27. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
28. Govind J, King W, Bailey B, Bogduk N. Radiofrequency neurotomy for the treatment of occipital headache. J Neurol Neurosurg Psychiatry. 2003;74:88–93.
29. Bogduk N. Role of anesthesiologic blockade in headache management. Curr Pain Headache Rep. 2004;8(5):399–403.
30. Bogduk N. Pulsed radiofrequency. Pain Med. 2006;7(5):396–407.
31. Park JJ, Jung MA, Shim JC. Third occipital nerve radiofrequency neurotomy with radiologic open mouth view – a case report. Korean J Pain. 2006;19(2):244–8.
32. Giblin K, Newmark JL, Brenner GJ, Wainger BJ. Headache plus: trigeminal and autonomic features in a case of cervicogenic headache responsive to third occipital nerve radiofrequency ablation. Pain Med. 2014;15(3):473–8.
33. Hamer JF, Purath TA. Response of cervicogenic headaches and occipital neuralgia to radiofrequency ablation of the C2 dorsal root ganglion and/or third occipital nerve. Headache. 2014;54(3):500–10.
34. Jeon HY, Shin JW, Kim DH, Suh JH, Leem JG. Spinal cord stimulator malfunction caused by radiofrequency neuroablation -a case report. Korean J Anesthesiol. 2010;59(Suppl):S226–8.
35. Binder WJ, Brin MF, Blitzer A, Schoenrock LD, Pogoda JM. Botulinum toxin type A (BOTOX) for treatment of migraine headaches: an open-label study. Otolaryngol Head Neck Surg. 2000;123(6):669–76.
36. Kapural L, Stillman M, Kapural M, McIntyre P, Guirgius M, Mekhail N. Botulinum toxin occipital nerve block for the treatment of severe occipital neuralgia: a case series. Pain Pract. 2007;7(4):337–40.
37. Volcy M, Tepper SJ, Rapoport AM, Sheftell FD, Bigal ME. Botulinum toxin A for the treatment of greater occipital neuralgia and trigeminal neuralgia: a case report with pathophysiological considerations. Cephalalgia. 2006;26(3):336–40.
38. Vallejo R, Benyamin R, Kramer J. Neuromodulation of the occipital nerve in pain management. Technol Reg Anesthesiol Pain Manag. 2006;10(1):12–5.
39. Johnstone CS, Sundaraj R. Occipital nerve stimulation for the treatment of occipital neuralgia-eight case studies. Neuromodulation. 2006;9(1):41–7.
40. Magis D, Allena M, Bolla M, De Pasqua V, Remacle JM, Schoenen J. Occipital nerve stimulation for drug-resistant chronic cluster headache: a prospective pilot study. Lancet Neurol. 2007;6(4):314–21.
41. Burns B, Watkins L, Goadsby PJ. Treatment of intractable chronic cluster headache by occipital nerve stimulation in 14 patients. Neurology. 2009;72(4):341–5.
42. Li F, Ma Y, Zou J, Li Y, Wang B, Huang H, et al. Micro-surgical decompression for greater occipital neuralgia. Turk Neurosurg. 2012;22(4):427–9.
43. Lee M, Lineberry K, Reed D, Guyuron B. The role of the third occipital nerve in surgical treatment of occipital migraine headaches. J Plast Reconstr Aesthet Surg. 2013;66(10):1335–9.
44. Okuda Y, Matsumoto T, Shinohara M, Kitajima T, Kim P. Sudden unconsciousness during a lesser occipital nerve block in a patient with the occipital bone defect. Eur J Anaesthesiol. 2001;18(12):829–32.
45. Manchikanti L. Pharmacology of neuraxial steroids. In: Manchikanti L, Singh V, editors. Interventional techniques in chronic spinal pain. Paducah: ASIPP Publishing; 2007. p. 167–84.

Suboccipital Neuralgia and Decompression

20

Andrea M. Trescot, Esther Rawner, Matthew P. Rupert, Rafael Justiz, and Gabor Racz

Introduction

Although occipital nerve injections are fairly common and perceived as safe injections (see Chap. 17), there have been a series of devastating complications, including death, from these "easy injections." Some problems have arisen from the injections being directed too medially, injecting into the foramen magnum (Fig. 20.1), leading to a potential total spinal anesthetic. Other complications have included a "*locked-in syndrome*" or death, presumably due to injection of particulate steroids into the vertebral artery that is closely located to the site of injection (Fig. 20.2) and/or retrograde flow into the occipital artery (Fig. 20.3) with infarction of the medulla. However, local anesthetic can spread back along the nerve to the spinal cord, causing a total spinal; Selander and Sjöstrand [1] confirmed a longitudinal spread of local anesthetic along the injected nerve by injecting radioactive local anesthetic into rabbit nerves, noting that 20 % of the endoneurial injections reached the spinal cord.

For these reasons, a suboccipital approach to the occipital group of nerves was developed. The term "*suboccipital nerve block*" is sometimes used to describe a greater occipital nerve injection with an inferior to superior trajectory (see Chap. 17). However, the "suboccipital nerve" term is also sometimes used to describe the dorsal ramus of C1.

The technique of *suboccipital nerve decompression*, *suboccipital compartment injection*, *Stealth occipital decompression*, "*posterior branch of C1 injection*," or *Racz occipital decompression* is a high-volume injectate within the suboccipital triangle used to treat occipital neuralgia. The location of interest lies between the superficial occipital nerve and the dorsal root ganglion of C2.

Clinical Presentation (Table 20.1)

The clinical presentation for neuralgia of the *suboccipital nerve* is the same as for all of the occipital nerves (see Chaps. 17, 18, and 19). As a subset of *cervicogenic headaches* (CGH), *suboccipital neuralgia* can cause pain and paresthesias to the posterior scalp, the occipital region, and the suboccipital (upper cervical) region (Fig. 20.4). Because of connections with the other occipital nerves in this region (see Fig. 20.3), there is a great deal of overlap in pain patterns. Unilateral occipital neuralgia, due to the proximity of the occipital artery, can present with throbbing, unilateral headaches associated with photophobia, phonophobia, and nausea, which will meet criteria for migraine based on the International Classification of Headache Disorders (ICHD) presented by International Headache Society (IHS) [13].

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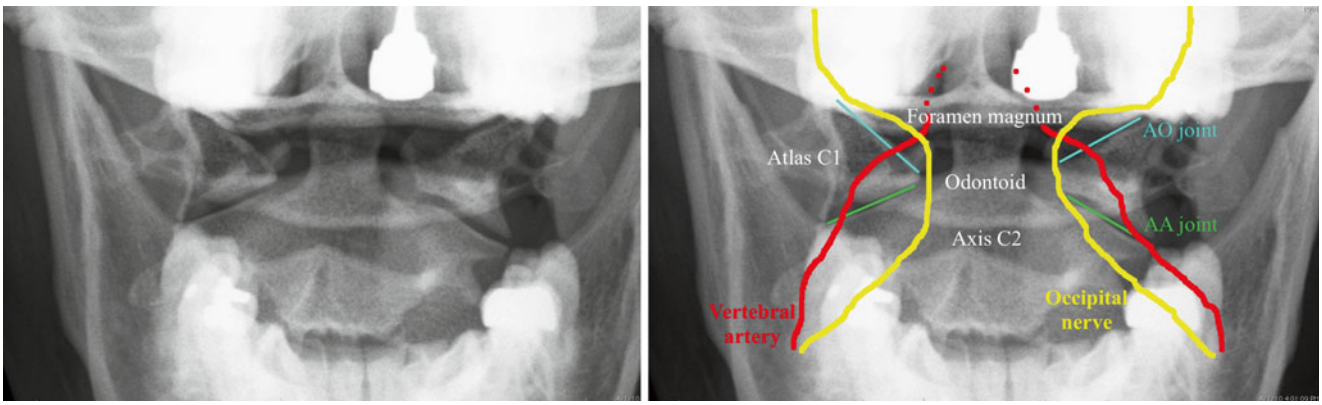

Fig 20.1 Anatomy of the suboccipital region (Image courtesy of Andrea Trescot, MD)

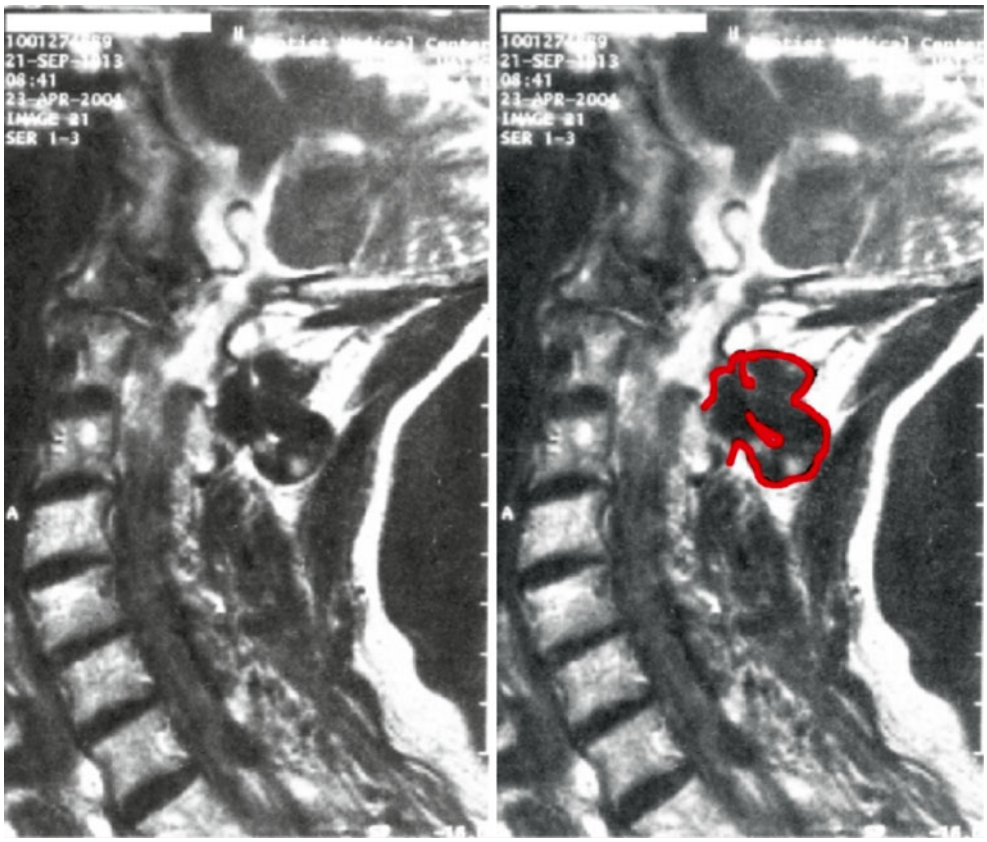

Fig. 20.2 MRI of a tortuous vertebral artery, outlined in *red* (Image courtesy of Andrea Trescot, MD)

Anatomy (Table 20.2)

The *suboccipital compartment* is composed of a triangle of bony articulations, ligaments, fibrous fatty tissue, and three different muscles: the *posterior rectus capitis major (RCM)* (also known as the *rectus capitis posterior major*), *inferior oblique capitis (IOC)* (also known as the *obliquus capitis inferior*), and *superior oblique capitis (SOC)* (also known as the *obliquus capitis superior*) (Fig. 20.5). The contents of the triangle are the *suboccipital nerve*, *the greater occipital nerve (GON)*, *the third occipital nerve (TON)*, and *vertebral artery*. The course of these nerves varies depending on the individual, and that course can be very torturous as the nerves enter and exit the suboccipital triangle. Preoperative

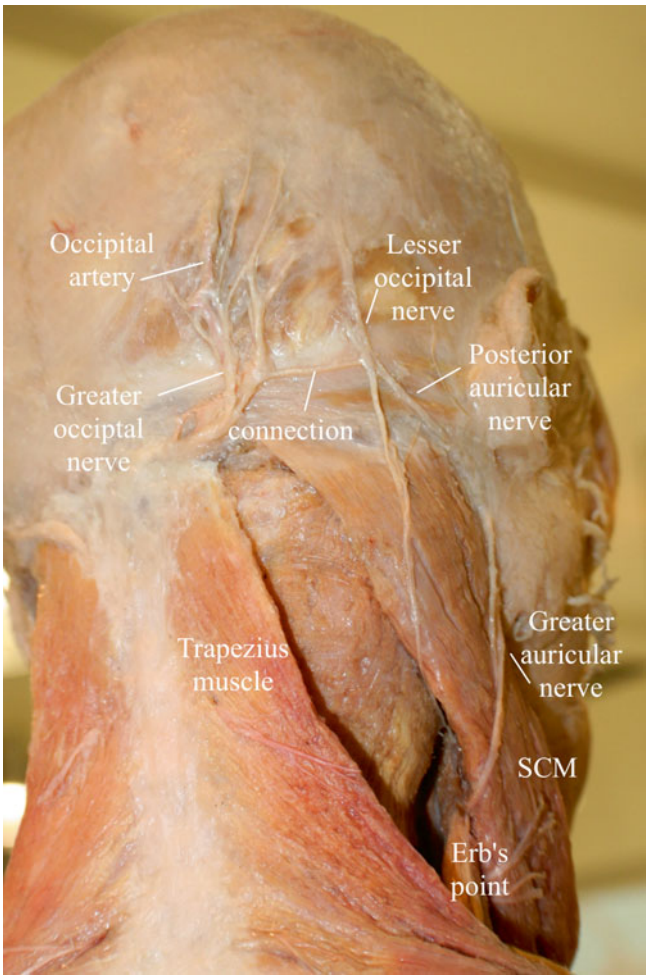

Fig. 20.3 Anatomy of the occipital region, modified from an image from *Bodies*, *The Exhibition*, with permission. Note the connection of the greater and lesser occipital nerves, as well as the intimate relationship between the occipital artery and nerve (Image courtesy of Andrea Trescot, MD)

Table 20.1 Occupation/exercise/trauma history relevant to suboccipital nerve entrapment

Flexion/extension injuries	"Migraine" after motor vehicle collision [2]
	Post concussive headache [3]
Improper ergonomics	"Desk job" with a computer [4] with head-forward positioning
Upper cervical surgery	Atlantoaxial screw fixation [5]
Compression	Anomalous vertebral artery [6]
	Schwannoma [7]
	Occipital nerve piecing through inferior oblique [8]
	Hypertrophic venous plexus at C2 [9]
Nerve injury	Herpes zoster [9]
Inflammation	C1/2 arthritis [8]
Myelitis	Cervical myelitis presenting as occipital neuralgia [10]
Cluster headache	"suboccipital steroid injection" [11]
Post-dural puncture headache	Persistent headache despite blood patch may result from occipital entrapment [12]

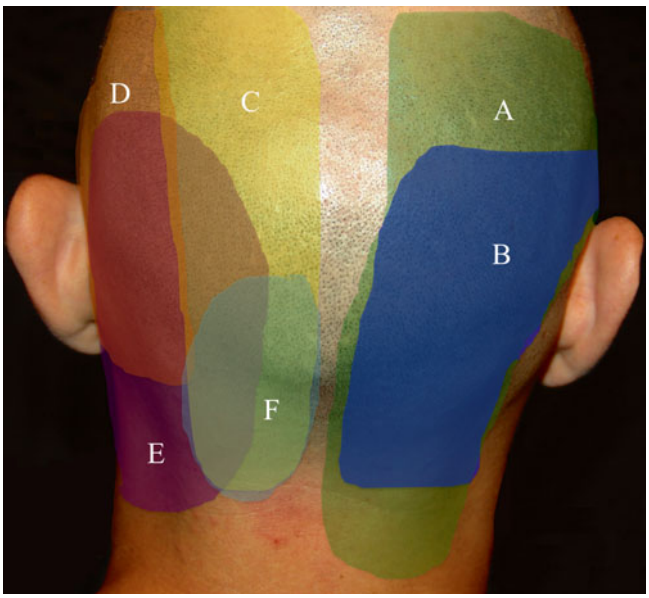

Fig. 20.4 Pattern of posterior cervical and occipital pain. *A* atlantoaxial joint, *B* atlantooccipital joint, *C* greater occipital nerve (GON), *D* posterior auricular nerve (PAN), *E* lesser occipital nerve (LON), *F* third occipital nerve (TON) (Image courtesy of Andrea Trescot, MD)

planning should include identification of the vertebral artery on axial and sagittal MRI (Fig. 20.2) to determine its relationship as it passes over the arch of C1 relative to the lateral masses of the facet joints.

The *suboccipital nerve* is the posterior branch of C1; this nerve exits posteriorly between the occiput and the posterior arch of the atlas. The suboccipital nerve is a motor nerve, which innervates five muscles: *semispinalis capitis muscle (SSC)*, *posterior rectus capitis minor (RCm)*, IOC, SOC, and RCM. Three of these muscles – the IOC, the SOC, and the RCM – make up the borders of the *suboccipital triangle* (Fig. 20.5). The RCM (the medial border of the triangle) originates on the C2 spinous process and inserts on the lateral occiput at the inferior nuchal line (generating extension and ipsilateral rotation). The IOC (the inferior border of the triangle) originates on the C2 spinous process and inserts on the C1 transverse process (providing rotation of C1 and therefore the head). The SOC (the lateral border of the triangle) originates on the C1 transverse process and inserts on the lateral aspect of the inferior nuchal line (generating head extension and ipsilateral flexion). Therefore, the muscles of

Table 20.2 Suboccipital nerve anatomy

Origin	Posterior branch of C1
General route	Exits between skull and C1 vertebrae and passes between the posterior arch of the atlas and vertebral artery into the suboccipital triangle
Sensory distribution	Atlantooccipital joint (Fig. 20.1); no cutaneous distribution
Motor innervation	Semispinalis capitis muscle (SSC), rectus capitis major (RCM), rectus capitis minor (RCm), superior oblique capitis (SOC), and inferior oblique capitis (IOC)
Anatomic variability	Very tortuous course of the nerve and artery
Other relevant structures	GON, TON (Fig. 20.3), C2-3 facet, vertebral artery, suboccipital triangle

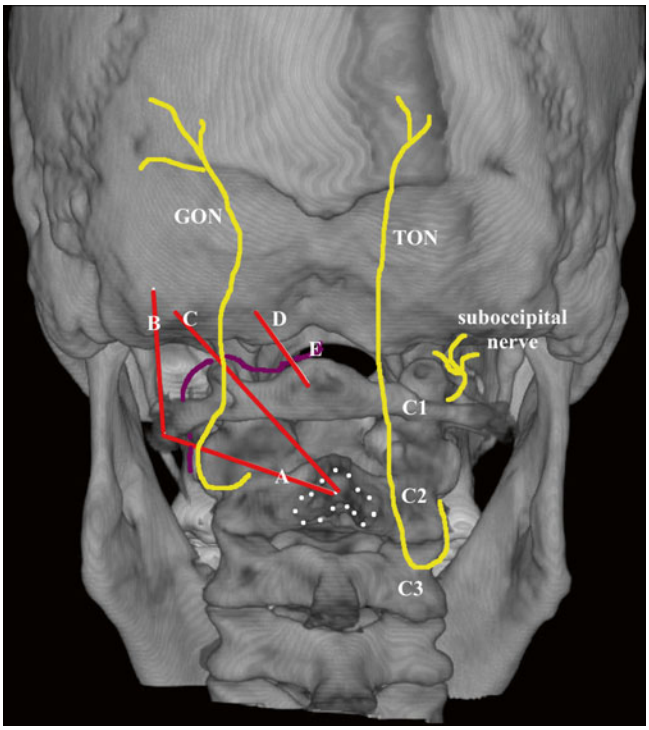

Fig. 20.5 Suboccipital contents and landmarks. *A* inferior oblique capitus muscle, *B* superior oblique capitus muscle, *C* posterior rectus capitus major muscle, *D* posterior rectus capitus minor muscle, *E* vertebral artery. Note the dotted outline of the bifid spinous process of C2. *Red* muscles, *yellow* nerves, *purple* arteries (Image courtesy of Andrea Trescot, MD)

the suboccipital triangle provide extension (RCM, RCm), rotation (RCM, IOC), and lateral bending or nodding of the head (SOC). These muscles, along with the SSC, the trapezius, and the nuchal fascia, all provide sites of possible nerve entrapment for the occipital group of nerves (see below). The IOC has a high density of Golgi organs and muscle spindles, which are important in cranial proprioception and balance.

The triangle is bordered anteriorly by the *posterior atlantooccipital membrane* and posteriorly by the SSC. The suboccipital nerve emerges dorsally through this anatomic triangle. The suboccipital nerve also has potential connections with the greater and lesser occipital nerves. An anterior root (the *McKenzie branch)* of C1 is often present and shares connections with the *spinal accessory nerve* (which provides motor innervation of the trapezius) [14]. The SSM is regionally innervated, which can include the dorsal rami of C1. The *vertebral artery* is in close proximity to the suboccipital nerve, and there is an extensive venous plexus above C1.

The GON runs along the inferior border of the IOC and then crosses over the IOC (Fig. 20.5) as it travels up to the back of the head. The GON then penetrates the SSC, the trapezius muscle, and the nuchal fascia as it travels superiorly (Fig. 20.6).

The *atlas* (C1) and *axis* (C2) are the only named vertebrae in the body. The *atlantooccipital joint* (AO) and *atlantoaxial joint* (AA) are common causes of suboccipital headaches (Fig. 20.4). Unfortunately, the vertebral artery runs quite close to these joints.

Entrapment

Entrapment of the occipital nerves occurs with spasm of the IOC, SSC, or trapezius muscles (themselves all potentially innervated by C1). The GON can therefore be entrapped by stenosis of the C2 foramen, compressed or entrapped by the ION, entrapped as it pierces the SSC, or entrapped by the trapezius muscle at its attachment to the occiput (Figs. 20.3, 20.6, and 20.7). The entrapment of the GON at the level of the IOC may be similar to piriformis entrapment of the sciatic nerve. In 1994, Rossi described a dissection down to the C1-C2 lamina, cutting the IOC to release the occipital nerve. He noted that the GON was flattened and pale; however, after surgical release, the nerve became round and pink, suggesting that there is a mechanical compression of the GON [15].

Chronic entrapment can lead to inflammation, which potentially causes adhesions, analogous to the adhesions seen in the epidural space after intervertebral disk leaking and disk herniations. The GON and TON nerves can become inflamed and then develop adhesions in the suboccipital region, leading to entrapment. There can also be mechanical entrapment caused by posture (Fig. 20.8).

Physical Exam

Unfortunately, the routine physical exam is often nonspecific, and more specific examination is necessary. With the patient in a supine position, place the fingers directly under

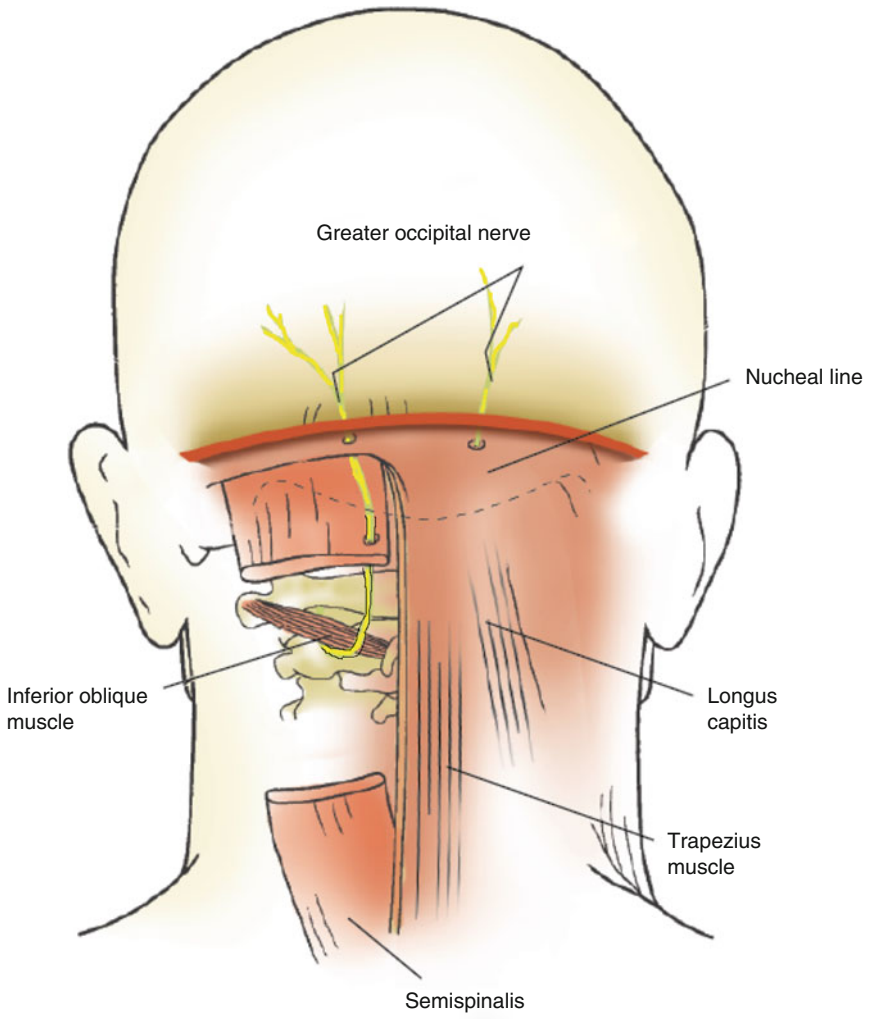

Fig. 20.6 Anatomy of the suboccipital region (Image courtesy of Epimed International®, with permission)

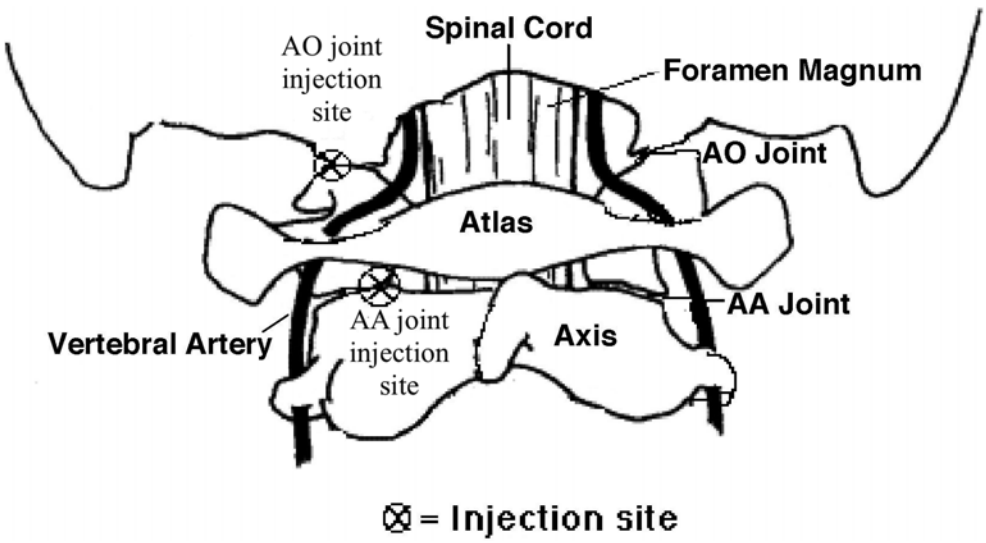

Fig. 20.7 Injection sites of the atlantoaxial (AA) joint and atlantooccipital (AO) joint (Image courtesy of Andrea Trescot, MD)

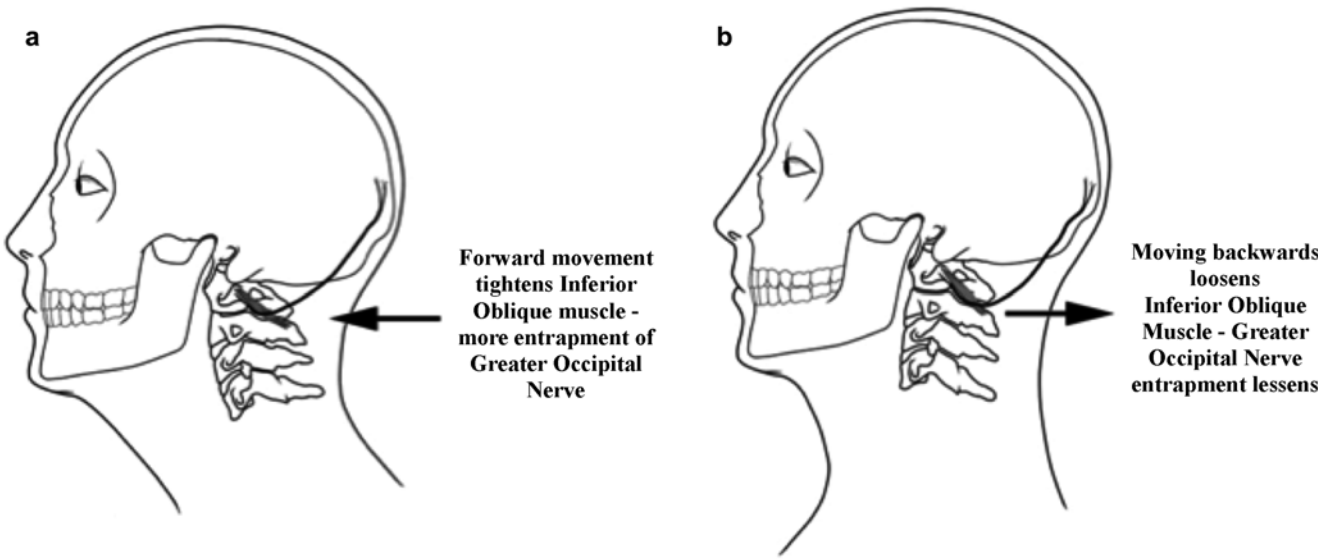

Fig. 20.8 (**a**, **b**) Ergonomic issues of head position on occipital nerve entrapment, as well as provocation maneuvers to evaluate occipital adhesions (Images courtesy of Epimed International®, with permission)

the base of the occiput while the patient actively tries to chin tuck and retract the head, similar to the provocative maneuvers seen in Fig. 20.8a. Suboccipital tenderness and pain with movement suggest occipital adhesions. The C1 vertebral body, which typically has small amounts of lateral translation with contralateral pressure, may be immobile and surrounded by tight musculature [16]. Patients will often visibly splint the head and upper cervical segments when ranging through cervical flexion and extension as well as head protraction and retraction. A *Tinel's sign* with referral along the GON is common. More specificity may be achieved through diagnostic injections.

Differential Diagnosis (Table 20.3)

Table 20.3 shows the differential diagnosis of suboccipital pain, which includes AA and AO joint dysfunction, third occipital neuralgia, and distal greater occipital neuralgia. Table 20.4 lists the diagnostic tests for proximal GON entrapment.

Identification and Treatment of Contributing Factors

Poor posture (Fig. 20.8) can contribute to suboccipital entrapment, so postural issues are important to address. Specific exercises to relax the suboccipital muscles can also be useful. Neuromuscular reeducation can be important before or after injection treatment.

Table 20.3 Differential diagnosis of suboccipital pain

	Potential distinguishing features
Atlantoaxial joint dysfunction	X-rays showing AA joint spondylosis, decreased rotation or pain with rotation
Atlantooccipital dysfunction	X-rays showing AO joint spondylosis, decreased lateral bending or pain with lateral bending
Third occipital neuralgia	Tenderness over C2-C3 facet
Greater occipital neuralgia	Tenderness lateral to the conjoined tendon

Table 20.4 Diagnostic tests for greater occipital nerve entrapment

	Potential distinguishing features
Physical exam	Pain over the suboccipital area to palpation, restricted head movement
Diagnostic injection	Suboccipital nerve block relieves the pain
Ultrasound	Useful aid to the nerve block
MRI	Identifies radiculopathy, space-occupying lesion, as well as AO, AA, or C2-C3 pathology
Arteriography	Not useful
X-ray	Shows AO, AA, or C2-C3 spondylosis
Electrodiagnostic studies	EMG useful to assess for cervical radiculopathy

Injection Technique

Because the vertebral artery is so close, injections of the AA and AO joints are associated with the potential risk of intra-arterial injection of local anesthetic (which could cause a seizure) or particulate steroid (which could act as an embolism) (Fig. 20.7).

Few interventionalists are therefore interested in AA or AO joint injections, and, as the C1 nerve travels over the posterior arch of the atlas, even fewer have been interested in direct injections of C1. In an effort to avoid the *"locked-in" syndrome* complication of injections into the vertebral or occipital artery, Dr. Gabor Racz developed a special needle and approach to the suboccipital space [17]. A curved, bullet-tipped needle with side port injection, attached tubing, and wings (Stealth™ *needle* (Fig. 20.9) is used to approach the suboccipital compartment from above.

The patient is positioned prone, with the chin tucked. The *inion* (*occipital prominence*) and *conjoined tendon* are identified by palpation; the bilateral entry sites should be just lateral to the conjoined tendon at the nuchal line, about 2.5 cm off midline (Fig. 20.10). The occipital hair is pulled cephalad, and the suboccipital area is prepped and draped. After making sure by palpation that the planned entry is not directly over the occipital artery, a small skin wheal of local anesthetic is injected via a 30 g needle followed by deeper infiltration of local anesthetic to the periosteum. Because the galea is so strong, an 18 g needle is used to create a small stab incision in the scalp and deep fascia to facilitate the placement of the blunt tipped Stealth™ needle.

The operator stands at the head of the bed (Fig. 20.11), and the Stealth™ needle is held by the wings like a butterfly needle (with the tip facing down) and advanced toward the feet (Fig. 20.12), parallel to the vertebral spine in the AP fluoroscopic view. The needle is advanced in AP and lateral views under fluoroscopic control into the suboccipital space, directing the needle toward the base of the lamina of C2 (Fig. 20.13), stopping 1 or 2 cm posterior to the C2 spinous process on lateral fluoroscopic view. A small amount of contrast is injected to confirm that the needle is deep to the SSC and not intravascular or intraneural. Contrast and approximately 10 cc of saline are then used to perform a volume decompression/adhesiolysis, freeing up the suboccipital and occipital nerves (Figs. 20.13, 20.14, 20.15, 20.16, and 20.17). This is followed by local anesthetic and steroid and repeated on the contralateral side if needed.

Justiz et al. [18] performed a retrospective study of 29 patients with confirmed occipital neuralgia. By measuring several variables, their study showed that the volume suboccipital decompression procedure was effective in reducing occipital headaches. In 58 % of patients, there was a significant pain relief on the numerical rating scale of greater than 50 % at the 6-month follow-up. At the 1-year follow-up, 34.5 % still had ongoing relief. Additionally, the study showed that patients had an increase in activities of daily living as well as a decrease in opioid use. This is in marked distinction to botulinum toxin injections, which provide a maximum of 3 months of relief from myofascial entrapment of the occipital nerve.

Lauretti et al. [19] compared "standard" occipital blocks (see Chap. 17) to what they described as a "GON subcompartmental technique" using volumes of 5, 10, or 15 cc, with the patients acting as their own control (i.e., only those patients who noted temporary relief from occipital nerve blocks, proving occipital involvement, were included in the study). Standard GON injections gave an average of 2 weeks of relief, compared to 24 weeks of relief from the decompression technique. Interestingly, there was no significant difference between the 5, 10, and 15 cc volumes. It is noted that they used sharp-tipped instead of blunt-tipped needles for this study.

Although primarily used for occipital nerve entrapment, the suboccipital approach has been used for AA and AO joint pathology, cervicogenic headaches, and cluster headaches [20, 21].

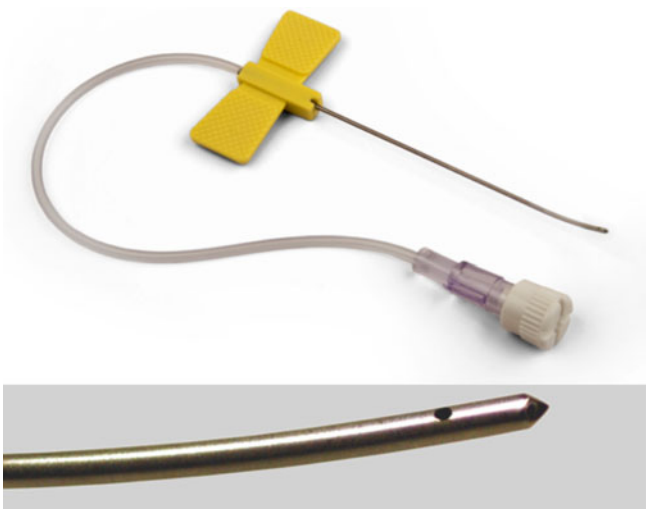

Fig. 20.9 The Stealth™ needle (Image courtesy of Epimed International®, with permission)

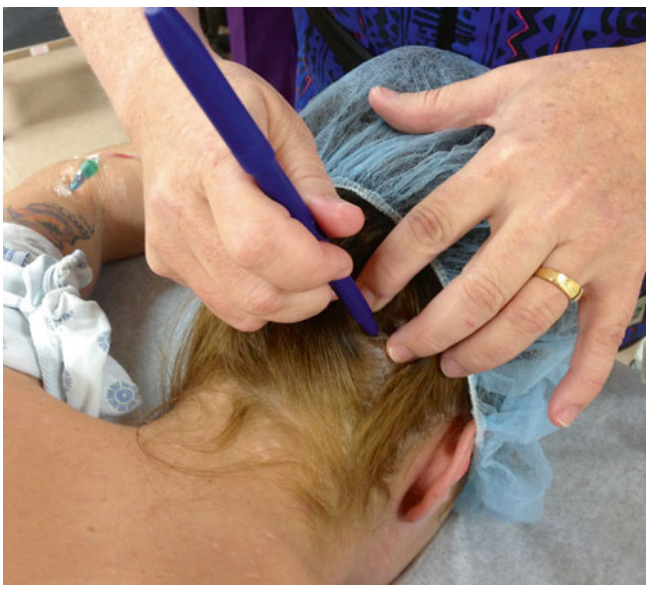

Fig. 20.10 Entry site for the suboccipital decompression needle (Image courtesy of Andrea Trescot, MD)

Fig. 20.11 Placement of the Stealth™ needle from the head of the bed, under fluoroscopic control (Image courtesy of Andrea Trescot, MD)

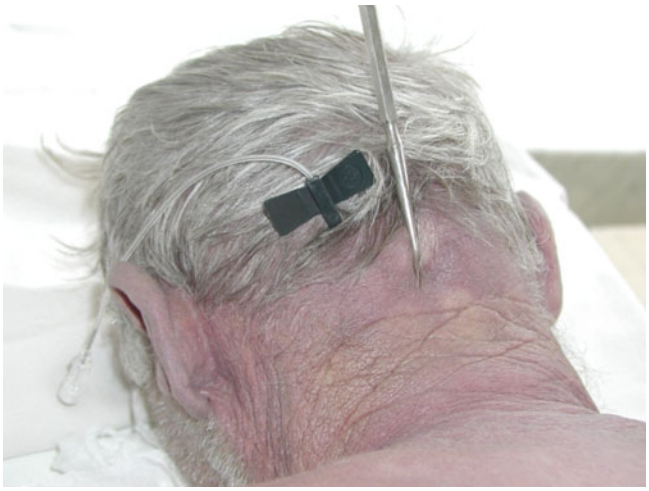

Fig. 20.12 Percutaneous placement of the Stealth™ needle in a cadaver (Image courtesy of Gabor Racz, MD)

Complications

To date, no complications from the suboccipital decompression technique have been reported, though there is a theoretical risk of local anesthetic toxicity, bleeding, or pain at the injection site. Standard occipital nerve blocks run the risk of "locked-in" syndrome, cerebellar infarcts, and respiratory arrest from injections placed too medially, from unrecognized vascular injections or from local anesthetic tracking longitudinally back along the nerve. Erdine et al. [15] described a case of "locked in phenomenon" after an occipital nerve block; the patient stopped breathing and stared with dilated pupils but "made a full and uneventful recovery" after 30 min of ventilation. Caution should be used when treating patients with a prior posterior craniotomy, since the surgery can cause the dura to detach from the cranium, resulting in potential dissection of medicine into the epidural space, which can lead to delayed respiratory arrest from a "total spinal anesthetic."

Summary

The suboccipital region contains a large number of structures associated with cervicogenic headaches, but this area has been relatively avoided as a treatment area because of real and perceived risks. Suboccipital decompression is a useful technique to treat occipital and suboccipital nerve entrapment. Because of the approach and the blunt-tipped needle, there should be a significantly decreased risk of vertebral or occipital artery or intraneural injection, while studies have shown a significant improvement in treatment outcome compared to standard occipital nerve blocks or botulinum toxin.

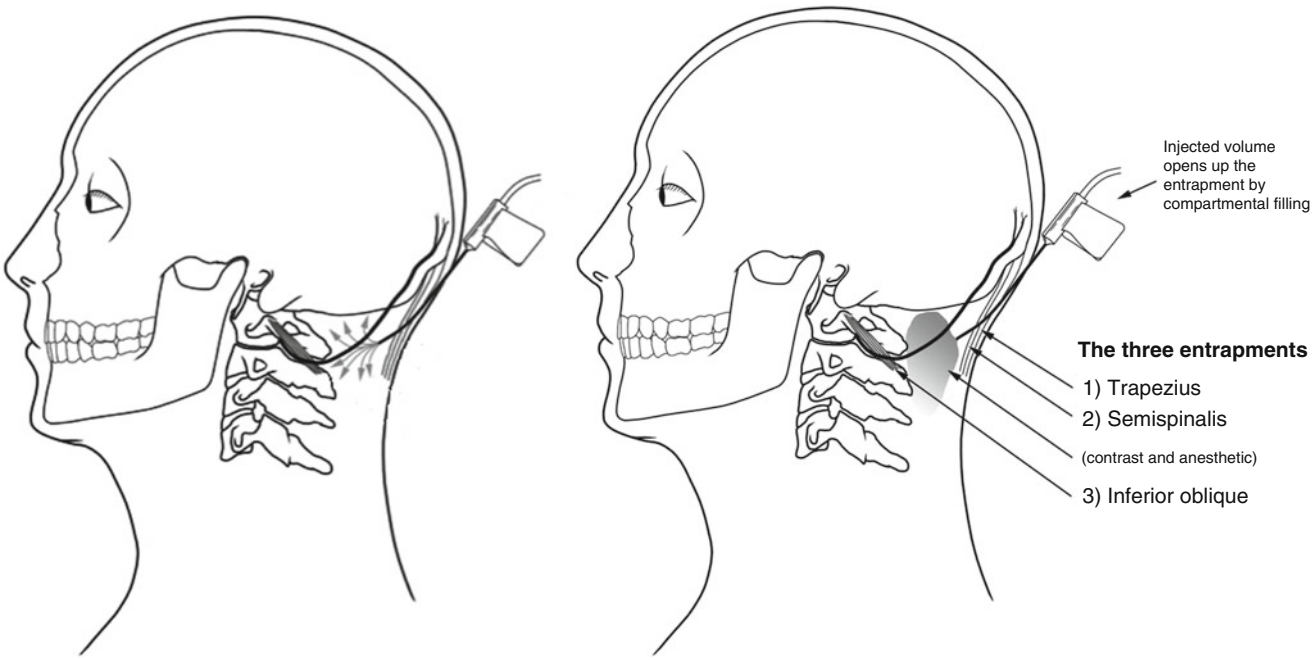

Fig. 20.13 (**a, b**) Diagram of the suboccipital decompression – Stealth™ needle approach (Image courtesy of Epimed International®, with permission)

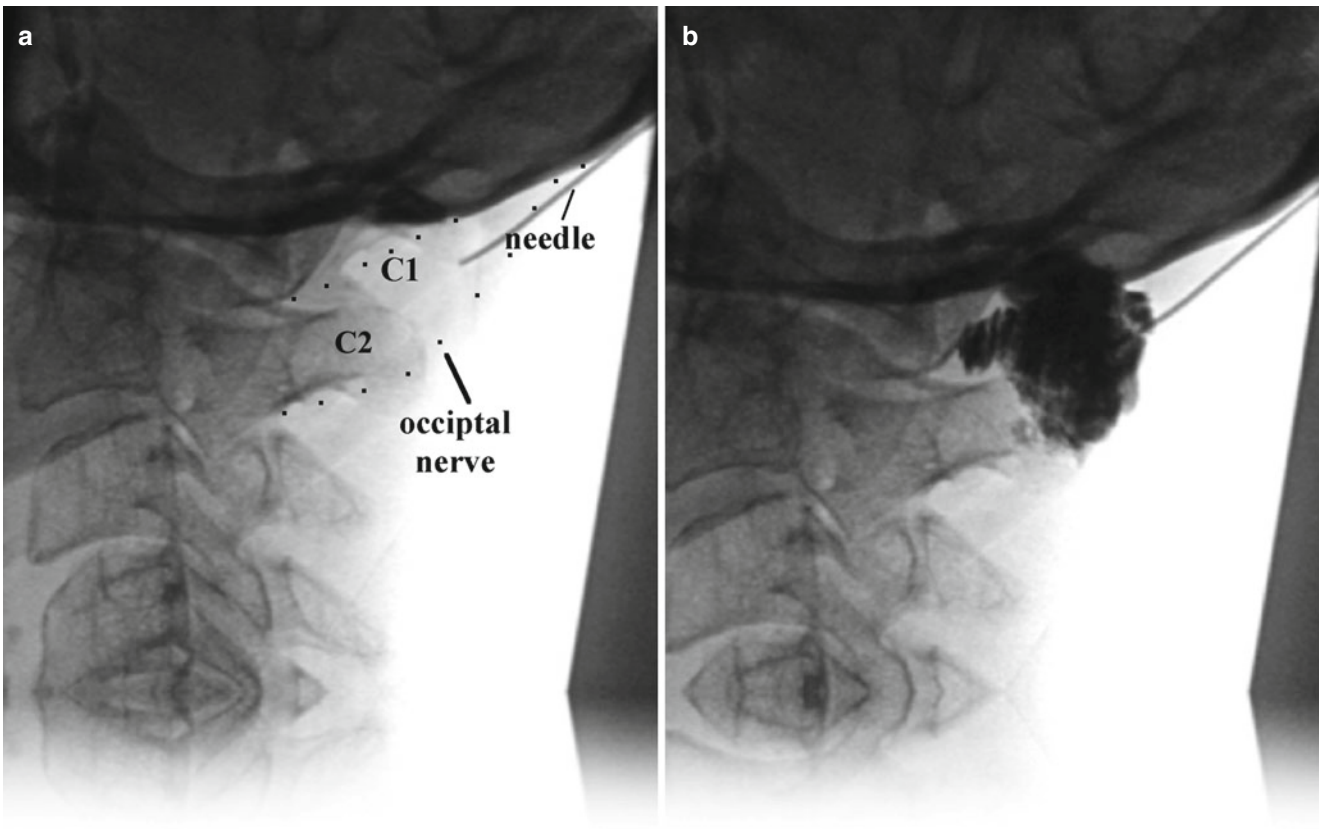

Fig. 20.14 (**a, b**) Fluoroscopic images of needle placement (**a**) and contrast injection (**b**) showing decompression/adhesiolysis of the suboccipital region (Image courtesy of Gabor Racz, MD)

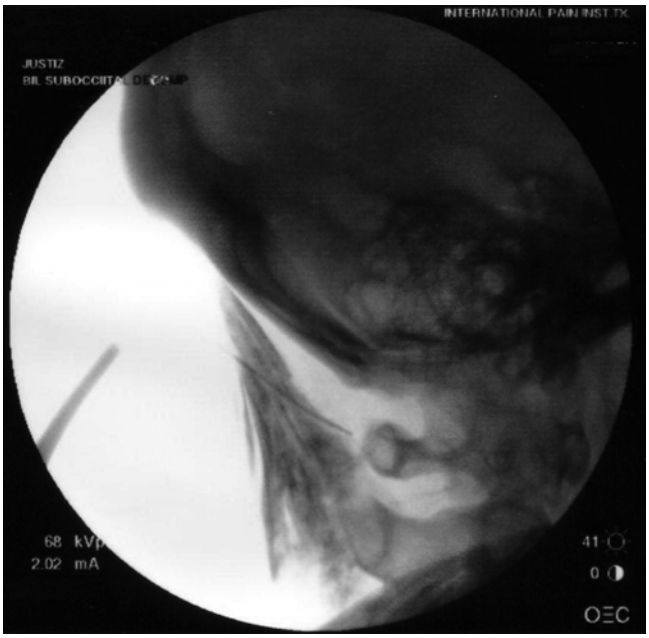

Fig. 20.15 Fluoroscopic image of contrast injection during Stealth™ injection (Image courtesy of Rafael Justiz, MD)

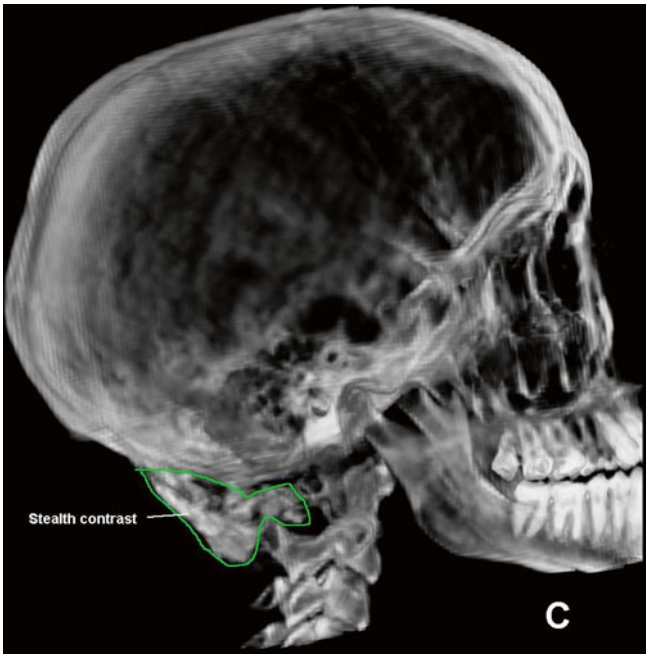

Fig. 20.17 3D image of suboccipital contrast from the Stealth™ decompression (Image courtesy of Andrea Trescot, MD)

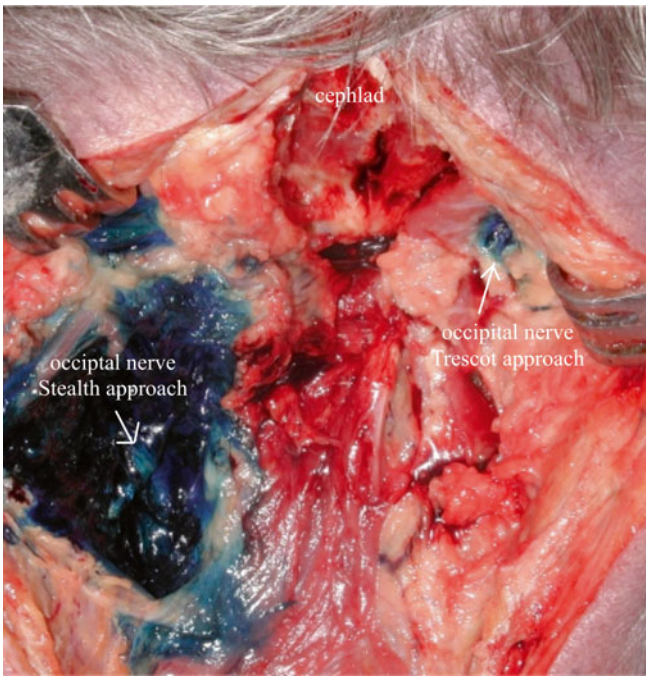

Fig. 20.16 Methylene blue suboccipital decompression dissection. On the *right* is the methylene *blue* dye on the occipital nerve from an occipital nerve block. On the *left* is the dye from the Stealth™ decompression bathing the occipital nerve (Image courtesy of Gabor Racz, MD)

References

1. Selander D, Sjöstrand J. Longitudinal spread of intraneurally injected local anesthetics. An experimental study of the initial neural distribution following intraneural injections. Acta Anaesthesiol Scand. 1978;22(6):622–34.
2. Choi HJ, Choi SK, Lee SH, Lim YJ. Whiplash injury-induced atypical short-lasting unilateral neuralgiform headache with conjunctival injection and tearing syndrome treated by greater occipital nerve block. Clin J Pain. 2012;28(4):342–3.
3. Hecht JS. Occipital nerve blocks in postconcussive headaches: a retrospective review and report of ten patients. J Head Trauma Rehabil. 2004;19(1):58–71.
4. Borg-Stein J. Cervical myofascial pain and headache. Curr Pain Headache Rep. 2002;6(4):324–30.
5. Yeom JS, Buchowski JM, Kim HJ, Chang BS, Lee CK, Riew KD. Postoperative occipital neuralgia with and without C2 nerve root transection during atlantoaxial screw fixation: a post-hoc comparative outcome study of prospectively collected data. Spine J Off J N Am Spine Soc. 2013;13(7):786–95.
6. Lucchesi C, Puglioli M, Gori S. Occipital neuralgia: a symptomatic case caused by an abnormal left vertebral artery. Neurol Sci. 2013;34(2):243–5.
7. Garza I. Craniocervical junction schwannoma mimicking occipital neuralgia. Headache. 2007;47(8):1204–5.
8. Gille O, Lavignolle B, Vital JM. Surgical treatment of greater occipital neuralgia by neurolysis of the greater occipital nerve and sectioning of the inferior oblique muscle. Spine (Phila Pa 1976). 2004;29(7):828–32.

9. Feyzi Demir C, Akalin Y, Berilgen S. Occipital neuralgia following thoracic herpes zoster: case report. Agri. 2011;23(4):179–80.
10. Goicochea MT, Romero C, Leston JA. Occipital neuralgia with cervical myelitis. Cephalalgia. 2008;28(5):567–8.
11. Leroux E, Valade D, Taifas I, Vicaut E, Chagnon M, Roos C, et al. Suboccipital steroid injections for transitional treatment of patients with more than two cluster headache attacks per day: a randomised, double-blind, placebo-controlled trial. Lancet Neurol. 2011;10(10):891–7.
12. Akin Takmaz S, Unal Kantekin C, Kaymak C, Basar H. Treatment of post-dural puncture headache with bilateral greater occipital nerve block. Headache. 2010;50(5):869–72.
13. The International Classification of Headache Disorders, 3rd edition. Cephalalgia. 2013;33(9):629–808.
14. Saylam CY, Orhan M, Ikiz Z, Ucerler H, Zileli M. The incidence and anatomical features of the McKenzie branch: a cadaver study. Turk Neurosurg. 2009;19(1):42–4.
15. Erdine S, Racz GB, Noe CE. Somatic blocks of the head and neck. In: Raj PP, Lou L, Erdine S, Staats PS, Waldman SD, Racz GB, editors. Interventional pain management: image-guided procedures. Philadelphia: Saunders; 2008. p. 77–107.
16. Sizer Jr PS, Poorbaugh K, Phelps V. Whiplash associated disorders: pathomechanics, diagnosis, and management. Pain Pract. 2004;4(3):249–66.
17. Racz GB, Noe CE, Justiz R. A new technique for suboccipital compartment decompression. 5th World Congress of the World Institute of Pain; March 2009. New York; 2009.
18. Justiz R, Taylor V, Racz GB, Meta G. Suboccipital decompression: a retrospective analysis of a novel technique for the treatment of occipital neuralgia. 5th World Congress of the World Institute of Pain; March 2009. New York; 2009.
19. Lauretti GR, Correa SW, Mattos AL. Efficacy of the greater occipital nerve block for cervicogenic headache: Comparing classical and subcompartmental techniques. Pain Pract. 2015;15(7):654–61.
20. Ambrosini A, Vandenheede M, Rossi P, Aloj F, Sauli E, Pierelli F, et al. Suboccipital injection with a mixture of rapid- and long-acting steroids in cluster headache: a double-blind placebo-controlled study. Pain. 2005;118(1–2):92–6.
21. Rossi P, Di Lorenzo G, Faroni J, Sauli E. Seasonal, extratrigeminal, episodic paroxysmal hemicrania successfully treated with single suboccipital steroid injections. Eur J Neurol. 2005;12(11):903–6.

Part III

Facial and Cervical Nerve Entrapments

Agnes R. Stogicza

Introduction

The International Headache Society (IHS) offers a classification system, the International Classification of Headache Disorders 3rd edition (IHS-ICHD-3), to organize the wide variety of headaches and facial pain syndromes [1]. Major categories include (1) *primary headaches*, which encompass migraine headaches, tension-type headaches, and cluster headaches; (2) *secondary headaches*, which include headaches with a likely etiology, such as from trauma, brain tumors, or sinus infections; and (3) *cranial neuralgias, central headaches, primary facial pain, and* (the catch all) *other headaches*, which encompass all the head and face pains that do not fall into a separate category.

Cranial neuralgias that cause facial pain, and nerve entrapments that cause neck and shoulder pain syndromes, are the primary focus of this section. In the authors' opinion, some of the "idiopathic headaches" are often undiagnosed entrapped or damaged nerves. So, we decided to discuss separately in Part II those neuralgias that can potentially mimic a primary headache, while focusing on face and neck pain in this section.

Trigeminal neuralgia is estimated to affect 4/100,000 patients per year, compared to glossopharyngeal neuralgia, which affects 0.7/100,000 patients per year [2]. The incidence of other neuralgias and nerve entrapments causing facial and neck pain is unknown, due to difficulties in clinical diagnosis, difficulties in differentiation from other neuralgias, and lack of awareness of these syndromes.

The trigeminal nerve (and its branches) supplies sensation to the face, but too often it is forgotten that the nervus intermedius, glossopharyngeal, and vagus nerves and the upper cervical fibers also transmit pain sensation from the head, face, neck, and cervical viscera. The superficial cervical plexus, supraclavicular nerve, and suprascapular nerve partly provide cutaneous innervation to neck and shoulder, explaining the pain pattern caused by their damage. The spinal accessory nerve is a motor nerve, yet injury to it can cause significant biomechanical alteration in the neck and shoulder area, therefore causing pain.

Damage to these nerves by trauma, vascular compression, malignancy, infection, or any other form of injury-causing neuropathy can result in hyperexcitability and inappropriate firing of these nerves, therefore provoking constant or intermittent pain and dysfunction at the innervated area. Most of the time, the underlying provoking factor remains unidentified. Regardless of the etiology, unless the provoking agent can be recognized and eliminated, the treatment is the same in primary and secondary cases.

For each nerve, we will highlight the clinical presentation of the entrapment syndrome and describe the anatomy, with particular focus on the various sites of potential entrapment. We will then discuss their associated physical examination findings. Finally, we will outline the various treatment approaches with an emphasis on injection techniques.

References

1. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition (beta version). Cephalalgia. 2013;33(9):629–808.
2. Katusic S, Beard CM, Bergstralh E, Kurland LT. Incidence and clinical features of trigeminal neuralgia, Rochester, Minnesota, 1945–1984. Ann Neurol. 1990;27(1):89–95.

Supraorbital Nerve Entrapment: Face

21

Rafael Justiz

Introduction

Supraorbital neuralgia (SN) originates, as the name implies, in the supraorbital nerve (SON) or within the path of its fibers and presents with supra-, retro-, or periorbital pain, which often radiates to the top of the head. Supraorbital neuralgia may arise from different causes, and trauma is the most common. Compression of the nerve with tight-fitting goggles or a motorcycle helmet, sinus infections, and inflammation have also been described as causes. Entrapment of the nerve occurs at the *supraorbital notch (foramen)*, *corrugator muscle*, and other locations along the path of the supraorbital nerve fibers. Diagnosis requires presence of a triad: unilateral variable quality pain or paresthesias in the SON distribution, localized tenderness over the *supraorbital notch* (foramen), and response to local anesthetic blockade or ablation of the nerve. Elimination of the pain trigger, nerve blocks with local anesthetic and steroid, cryoneuroablation, neuromodulation, and surgical decompression are all valuable means in remedying supraorbital neuralgia pain.

Clinical Presentation (Table 21.1)

Supraorbital neuralgia (SN) presents as pain in the forehead (Fig. 21.1). SN pain has likely been a cause of headaches throughout the history of humankind, given both the location of the supraorbital notch and the common finding in the ancestral human skulls of blows to the forehead. One of the first descriptions of supraorbital neuralgia was in 1949 by T.E. Beyer, an otorhinolaryngologist from Denver, CO [23]. It is one of several trigeminal nerve entrapments that can cause facial pain and headaches (Fig. 21.2).

Epidemiology of supraorbital neuralgia is not well researched. In 2005, Sjaastad and colleagues reported on the palpatory examination of 1,828 of the inhabitants of Vågå,

Table 21.1 Occupation/exercise/trauma history relevant to supraorbital nerve entrapment

Trauma	Blunt blow to the face [1]
	Supraorbital area fracture [2]
	Iatrogenic after surgery or injection [1, 3]
Compression on the nerve	Swimming goggles [4], motorcycle helmet
	Orbicularis oculi spasm [5]
	Skin plaque [6]
	Hemangioma [7]
	Nerve enlargement (e.g. Hansen's disease) [8]
Foraminal stenosis	Paget's disease [9]
Infection and inflammation	Frontal sinus infection [10]
Neoplasia	Primary craniofacial tumors [11–13]
	Metastatic disease [11, 12]
Neuropathy	Amyloidosis [14]
	Nerve enlargement in Hansen's disease [8]
	HIV [15]
	Post-herpetic neuralgia [16]
	Painful posttraumatic trigeminal neuropathy [17]
	Trigeminal neuralgia [18]
	Trigeminal trophic syndrome [19]
Vascular	Vascular lesions of the orbit [20, 21]
Endocrine and metabolic	Water retention during menstrual cycle, increased salt intake [22]
	Low calcium [5]

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Norway, stating that 98 % of the individuals had a cause identified by palpation, while 1.3 % had a non-palpable supraorbital notch, with increased local tenderness noted in 106 people (5.4 %). Among the 12 patients that had a clinical presentation of SON neuralgia, 10 had a confirmed history of forehead trauma [24]. In another study, a female preponderance of 67 % with a mean age of onset at 51.6 years and mean headache duration of 5.9 years was reported in 18 patients with supraorbital pain. Five patients had a history of forehead trauma, although all but four patients had a normal neurological exam; all noted "absolute" relief from supraorbital nerve blocks [25].

Fig. 21.1 Pattern of pain from supraorbital neuralgia (Image courtesy of Andrea Trescot, MD)

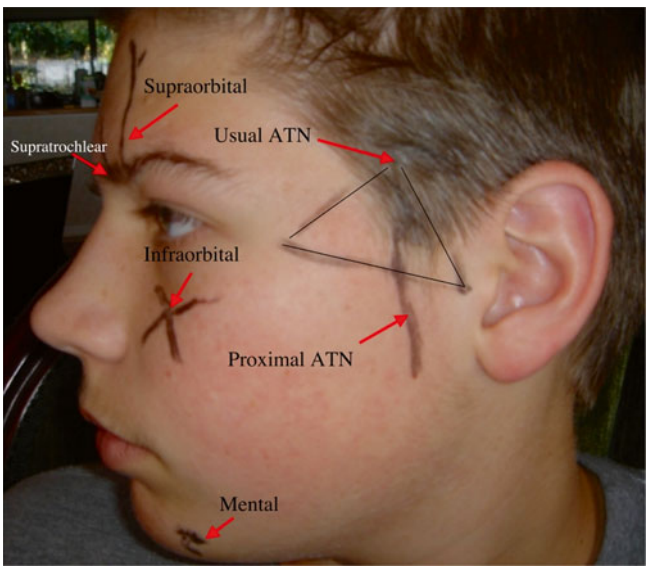

Fig. 21.2 Trigeminal entrapments causing headaches and face pain (Image courtesy of Andrea Trescot, MD)

Supraorbital neuralgia has multiple causes (Table 21.1) and may be confused with other conditions such as cluster or migraine-type headaches or sinusitis. The pain is different in quality in supraorbital, retro-orbital, or periorbital locations with variable radiation to the vertex of the skull, which may even be occipital along and next to the skull midline. These symptoms may worsen with palpation of the supraorbital area, but sharp, lancinating, shooting spontaneous pain is likely due to a different origin, possibly trigeminal neuralgia.

Supraorbital neuralgia may also act as a trigeminal "trigger zone" for *tic douloureux* (*trigeminal neuralgia*). The symptoms may progress with time, especially with active protracted perineural scarring. Pain in the forehead with squinting or frowning may be suggestive of entrapment within muscles participating in these movements. Pain increase may be provoked by direct pressure from tight swimming goggles [24] or helmets and by lowering the head (which leads to an increase in hydrostatic intravascular pressure and an increase in the diameter of supraorbital vessels compressing the nerve).

Increase in edema of the inflamed SON with self-entrapment within the narrow space of the supraorbital notch or supraorbital foramen due to the body fluid retention may be responsible for periodic worsening in symptoms for women in premenstrual period [26], in addition to excessive salt consumption and other conditions.

Anatomy (Table 21.2)

The SON is one of five peripheral branches of the ophthalmic division of the *trigeminal nerve* (CN V) (Fig. 21.3). The *trigeminal ganglion* gives rise to three divisions: *ophthalmic* (CN V1), *maxillary* (CN V2), and *mandibular* (CN V3) (Fig. 21.4). While the maxillary and mandibular divisions branch from the trigeminal ganglion downward, the ophthalmic division branches superomedially, continues below the oculomotor (CN III) and trochlear (CN IV) nerves within the lateral wall of the cavernous sinus, gives off the recurrent meningeal branch which provides sensation to the dura

Table 21.2 Supraorbital nerve anatomy

Origin	Trigeminal ganglion
General route	V1 through the supraorbital fissure, through the supraorbital notch/foramen and corrugator muscle
Sensory distribution	Forehead, upper eyelid, nose, and anterior scalp
Motor innervation	None
Anatomic variability	Separate exits for the medial and lateral branches
Other relevant structures	Supratrochlear notch vs. foramen

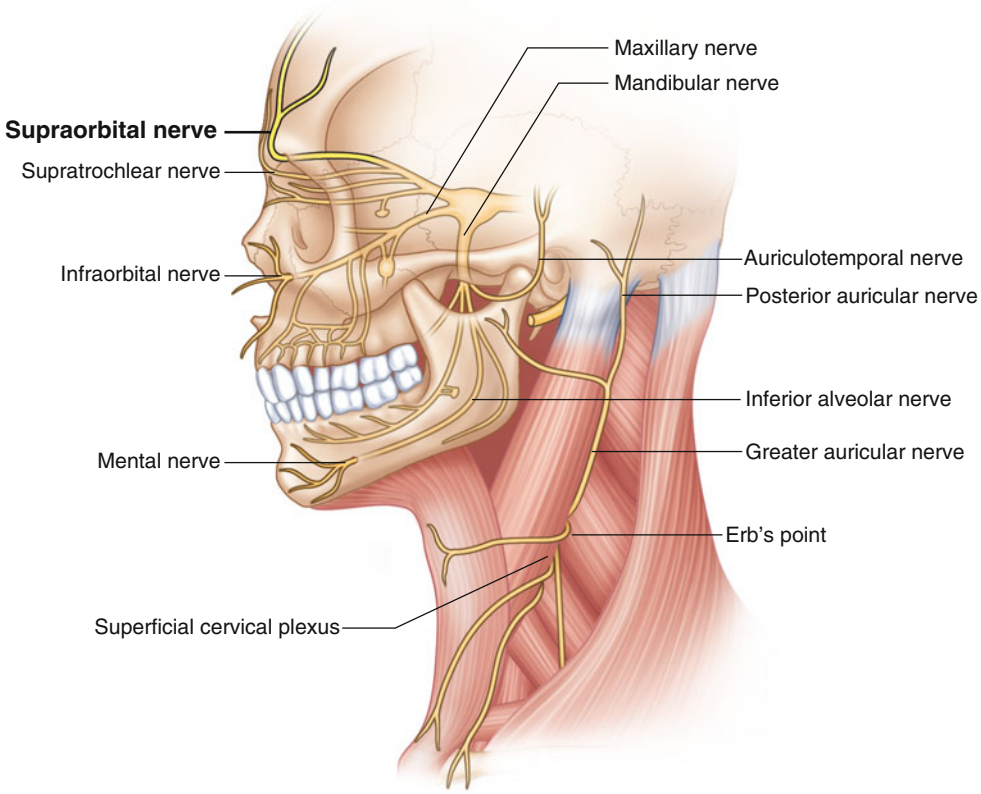

Fig. 21.3 Supraorbital anatomy (Image courtesy of Springer)

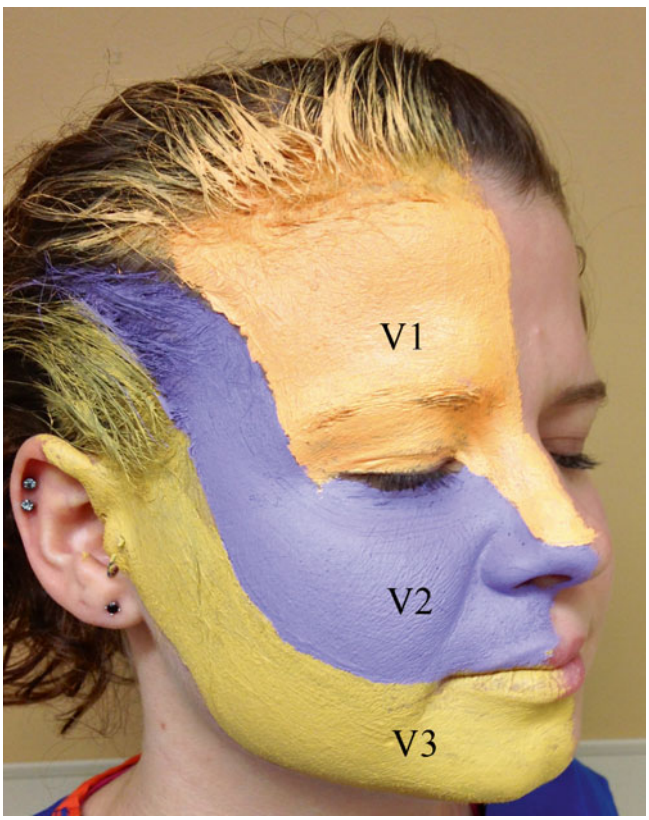

Fig. 21.4 Distribution of trigeminal nerve sensation. *V1* ophthalmic division/supraorbital nerve, *V2* maxillary division/maxillary nerve, *V3* mandibular division/mandibular nerve (Image courtesy of Terri Dallas-Prunskis, MD)

mater, and enters the orbit posteriorly via the *superior orbital fissure* (Table 21.2). There, the ophthalmic nerve divides into the *lacrimal nerve*, which leaves the orbit in the lower part of the lateral orbital margin (providing sensation to the lacrimal gland, conjunctiva, and upper eyelid skin near the lateral canthus), the *nasociliary nerve* (providing sensation to the mucosa of the anterior nasal septum and lateral wall; the skin over the side, ala, and the tip of the nose; conjunctiva near the inner canthus and the eyeball; and sympathetic fibers to dilator pupillae), and the *frontal nerve*. The frontal nerve then gives terminal branches – the *supraorbital nerve* (SON) and the *supratrochlear nerve* (STN) [27].

The SON exits the orbit via the *supraorbital notch* (*supraorbital foramen*) and divides into medial and lateral branches over the forehead. The medial branch continues superiorly up to the vertex of the skull, while the lateral branch moves superior laterally. Both branches supply sensory fibers to the forehead, upper eyelid, and anterior scalp, up to the lambdoidal suture (Fig. 21.5), and both are associated with arteries (often intimately entwined). Separate exits for the medial and lateral SON branches were observed by Anderson et al. in 29 % of the specimens (8 of 28) [28]. Janis et al. found that 73 % of the specimens examined had the SON passing through the corrugator muscle [29].

Fig. 21.5 Sensory areas of the trigeminal and cervical nerve branches: *A* supraorbital nerve, *B* infraorbital nerve, *C* mental nerve, *D* buccal nerve, *E* lacrimal nerve, *F* auriculotemporal nerve, *G* superficial cervical plexus, *H* posterior auricular nerve/great auricular nerve, *I* occipital nerve (Image courtesy of Terri Dallas-Prunskis, MD)

The STN exits via the supratrochlear notch and runs medially in a small groove at the junction of the ocular ridge and the nares, supplying sensation to the skin and soft tissues of the lower medial forehead, the upper eyelid and conjunctiva near the inner canthus, and the glabella (Fig. 21.6). The STN runs medially along the roof of the orbit, exiting between the supraorbital notch and the trochlea. An accompanying supratrochlear artery was observed by Janis et al. in 60 % of orbits (30/50); the STN shared a notch with the SON in 4 % (3/50) of cases; and in 84 %, the STN entered the corrugator muscle.

The supraorbital notch may be covered by a small ligament (creating a foramen), which can become thickened or calcified. Fallucco and colleagues called this ligament-covered foramen a "miniature carpal tunnel" [30]. In dissections done by Anderson et al., 71 % of nerves (20/28) exited through foramina with bony or connective tissue bridges instead of notches [28]. Fallucco et al. [30] dissected 50 SONs and found that 43 of the 50 (86 %) had fascial bands across the notch which were further classified into "simple" bands in 51.2 % (22/43), "partial bony" in 30.2 % (13/43), and "septums," which could be either horizontal or vertical. The horizontal and vertical type each presented in 9.3 % (4/43) of "septums." The authors noted a high degree of variability in the anatomic features and foramen diameters and suggested that this could account for unilateral migraine symptoms. In another study, 14.3 % of 866 dry skull samples had a smooth supraorbital rim without a notch or foramina [31].

Analysis of three-dimensional CT images in a group of Korean patients found a higher incidence of a single foramen (42.3 %) (compared to other races), and 39.5 % were noted to have a single notch. The mean SON diameter for the foramen was 2.34 ± 0.78 mm, while the mean diameter for the SON notch was 3.37 ± 1.71 mm. The mean SON foramina distance from the nasion was 22.1–32.3 mm (27.19 ± 4.03) and 16.1–30.8 mm (23.42 ± 2.45 mm) for the SON notch [32]. Interestingly, the horizontal SON branch connected with the temporal branch of the facial nerve (Fig. 21.6) in 8 of 18 (44 %) specimens sharing common epineurium, while having separate perineurium [33].

Entrapment

Entrapment may occur at any point on the SON path, though it is likely to be at the supraorbital notch (foramen). Another location of reported impingement is when the SON traverses through the orbicularis oculi, or the glabellar or corrugator muscles, with pain exacerbated by frowning and squinting. It was found that tight-fitting goggles [4], an anesthesia mask [3], and trauma from surgery or postoperative scarring in the frontal region [3] can all trigger SON pain. The SON

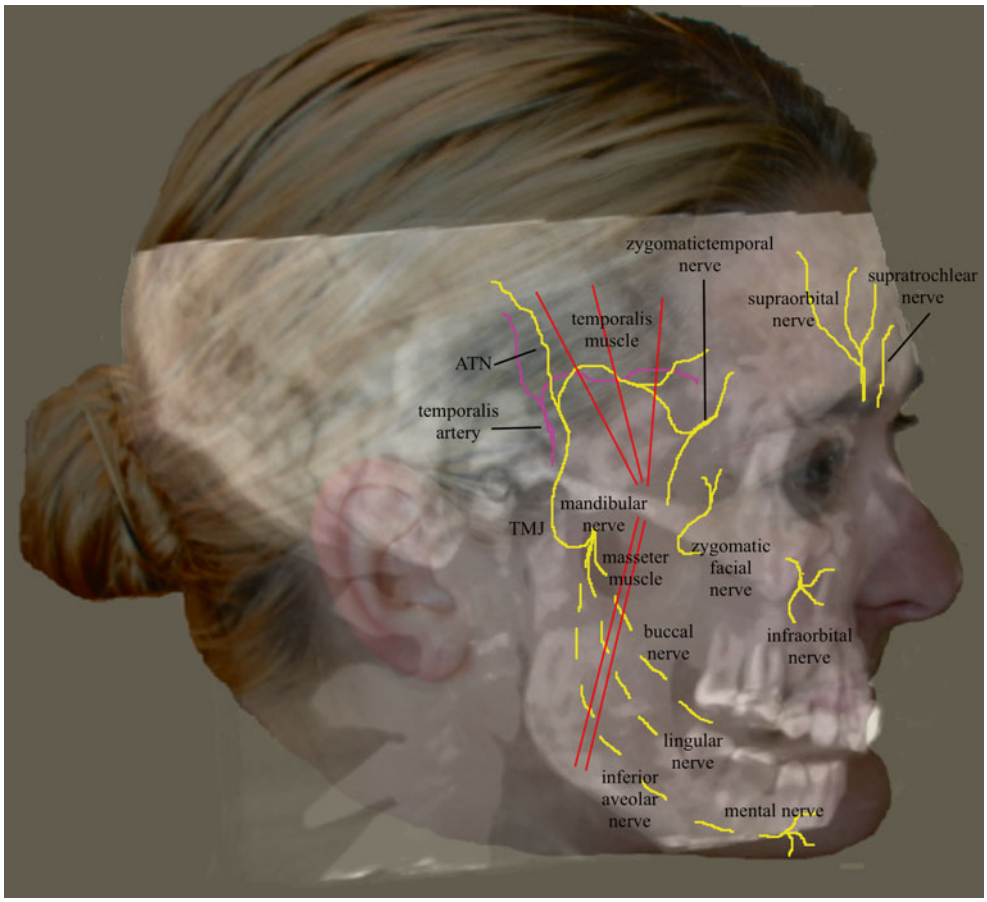

Fig. 21.6 Nerves of the face (Image courtesy of Andrea Trescot, MD)

entrapment may worsen with menses or increased salt intake, perhaps by causing swelling of the nerve in its canal, as well as with thickening or calcification of the supraorbital notch ligament.

Physical Exam

A thorough history should first be obtained to identify the etiology of the problem, accompanying symptoms, and possibility of previous trauma or other inciting events that could lead to supraorbital neuralgia. The SON runs superficially under the skin and may be traumatized even during a minor event, leaving the patient with no memory of the initial cause. Asking the patient to point exactly at the place of maximal pain may be helpful in better understanding the pain localization. There may be tenderness to palpation over the supraorbital notch (foramen) with possible radiation of symptoms, paresthesia, or dysesthesia along the nerve distribution on the affected side. With the head stabilized by the non-examining hand, palpation over the upper orbital rim while feeling for the supraorbital notch (foramen) may trigger localized paresthesias replicating the pain complaints (Video 21.1) (Fig. 21.7).

Fig. 21.7 Supraorbital examination (Image courtesy of Andrea Trescot, MD)

Differential Diagnosis (Table 21.3)

The etiology of supraorbital neuralgia is often diagnosed clinically. Although previous trauma is common, other potential causes (see Table 21.1) must be considered. The differential

Table 21.3 Differential diagnosis of supraorbital nerve entrapment

Condition	Potential distinguishing features
Hemicrania continua	History, distribution and pattern of pain, response to carbamazepine and indomethacin
Migraine headache	No response to supraorbital nerve blocks [22]
Shingles	Lesions in a V1 distribution, elevated herpes varicella-zoster IgM titers
Trigeminal neuralgia	Spontaneous, lancinating pain [24]
Sinus (frontal) infection	Fever, nasal purulent discharge, characteristic x-ray, MRI, or CT changes
Paget's disease	History, characteristic changes in the supraorbital foramen area on x-ray, MRI, or CT
Post-herpetic neuralgia	History of shingles
Trochleitis	CT shows soft tissue enhancement at trochlea [34]
Hemangioma or dilatation of the supraorbital artery	MRI, MRA, CTA, or arteriogram (gold standard) will show abnormal vascular structures [7]
Primary or metastatic neoplasia	History, characteristic x-ray, MRI, or CT changes
Temporal arteritis	History, age, high ESR, characteristic changes on bilateral temporal artery biopsy, MR, CT, or conventional angiography

Table 21.4 Diagnostic tests for supraorbital nerve entrapment

	Potential distinguishing features
Physical exam	Localized pain and/or sensory disturbance in the SON area
Diagnostic injection	Resolution of pain after diagnostic injection at supraorbital notch
Ultrasound	Evaluation of the soft tissue and bone conditions, location of the SON, supraorbital artery, supraorbital notch/foramen [35]
MRI	Evaluation of facial soft tissue, intraorbital and intracranial abnormalities, if suspected; contrast should be used when indicated; high resolution MRI can show the supraorbital nerve and artery [36]
Arteriography	Indicated for evaluation of intraorbital and intracranial vascular disorders
X-ray	May be used in cases of acute trauma when CT scanning is not available
Electrodiagnostic studies	Electromyographic (EMG) studies for blink reflex [37]

diagnosis is extensive and includes nerve compression by a neoplastic process invading the bone, neuropathy and neuralgia, infection, systemic inflammatory process, complications of cancer treatment (e.g., radiation fibrosis, chemotherapy-induced neuropathy), and others (see Table 21.3). Various tests for SON entrapment are listed in Table 21.4. Beyer felt that tenderness over the supraorbital and supratrochlear notch was "pathognomonic" for supraorbital neuralgia [23].

Identification and Treatment of Contributing Factors

Pain in the supraorbital nerve is an infrequent finding overall. Trauma is reported as the most common inciting event with entrapment likely being second, followed by other etiologies (see Tables 21.1 and 21.3). It is of interest that calcium and parathyroid hormone levels were significantly decreased, and phosphorus and beta-endorphin levels were increased in a group of patients with neuromuscular hyperexcitability, anxiety, dysautonomia, and supraorbital and oculofrontal headaches. This finding prompted Janiri et al. to suggest that for some patients the SON entrapment may be due to the orbicularis oculi tetany from low calcium [5].

Treatment of perpetuating factors for supraorbital neuralgia involves removing provoking stimuli, such as by using better-fitting goggles or helmets. Squinting can entrap the SON, so if poor vision or bright light sensitivity is the trigger, an optometrist evaluation or sunglasses may help. Frowning can also entrap the SON, so botulinum toxin (see below) can relieve the entrapment as well treat the perpetuating factor.

Injection Technique

Landmark-Guided Injections

The patient is placed in a supine or sitting position (with the head supported). The supraorbital notch is palpated, and the skin is cleaned and prepped in a sterile fashion. The supraorbital notch is typically located 2–2.5 cm lateral to the bridge of the nares. The non-injecting index and middle fingers are placed to straddle the notch, moving the skin cephalad to avoid injecting the eyebrow itself (Video 21.2) (Fig. 21.8). With the target identified, a 27 or 30 gauge 1-in. needle is inserted at an oblique angle to the skin until bony contact is made; this area is very tender, and the patient will often involuntarily withdraw. The needle is then slightly withdrawn and redirected medially and slightly cephalad. Care is

taken during needle insertion to not insert the needle tip into the supraorbital notch itself, as this will cause paresthesias along the distribution of the supraorbital nerve, and injection of the medication within the notch itself can cause increased entrapment. Following negative aspiration, a volume of 0.5–1 cc of local anesthetic with or without steroid solution is injected, though some authors recommend increasing the volume to 3–4 cc [38]. This procedure provides mostly short-term relief with pain recurrence and can be repeated multiple times. For a longer-lasting benefit, procedures such as cryoneurolysis or neuromodulation can be performed (see below). If indicated, the STN may also need to be treated with the same modalities.

Fig. 21.8 Landmark-guided supraorbital injection (Image courtesy of Andrea Trescot, MD)

Fluoroscopy-Guided Injections

Fluoroscopy can be used to visualize the supraorbital foramen. The C-arm should be placed in a cephalad rotation to identify the foramen (Fig. 21.9). Once identified, the procedure is then performed in the same fashion as landmark-guided technique with needle placement just medial to the supraorbital foramen.

Ultrasound-Guided Technique

Using ultrasound (US) to block the supraorbital nerve can be superior to the blind or fluoroscopic approaches. A linear probe is placed horizontally across the supraorbital notch to identify the supraorbital artery and nerve in the notch (Fig. 21.10). With direct visualization of the supraorbital foramen, a proper needle placement can be achieved while avoiding an inadvertent needle placement into the foramen. In a cadaveric study, Spinner and colleagues [39] demonstrated that, compared to conventional landmark-based techniques, US-guided blocks of the supraorbital nerve, infraorbital nerve (see Chap. 22), and mental nerve (see Chap. 25) had excellent accuracy rates of 100 % (18 of 18) for the in-plane approach and 94 % (17 of 18) for the

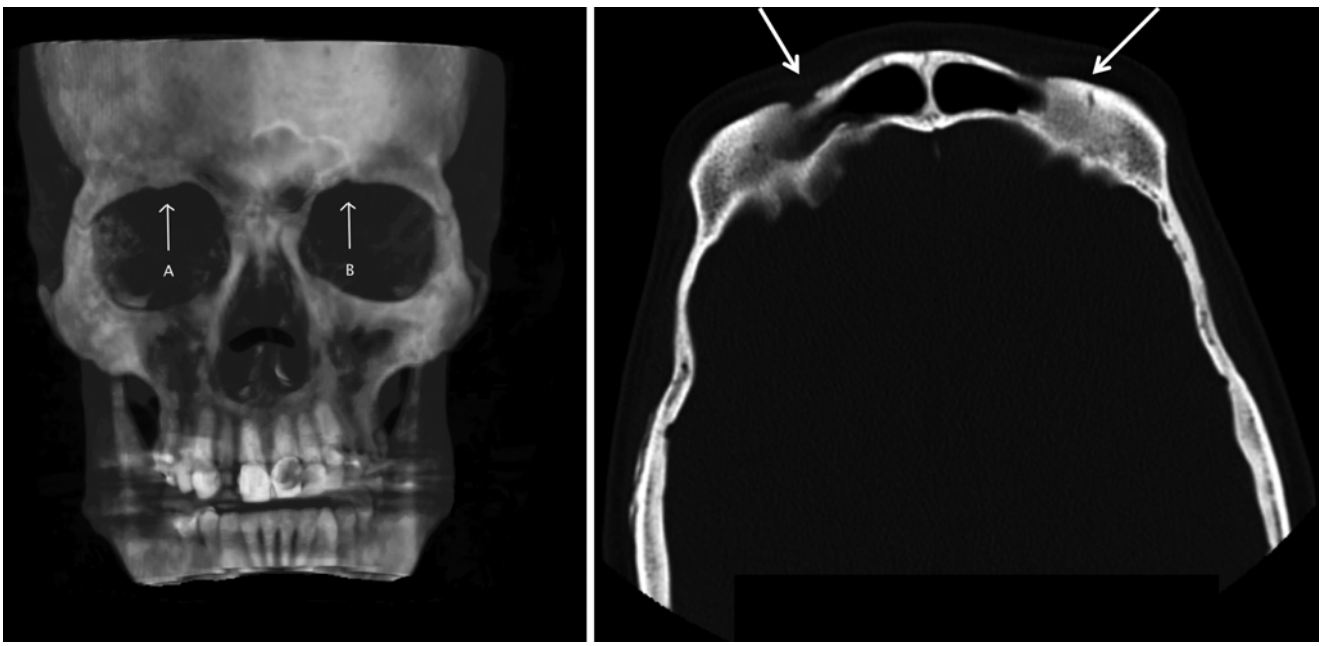

Fig. 21.9 Supraorbital notch, 3D skull. *Arrows* show supraorbital notch. *A* obliterated foramen, *B* supraorbital notch. (Image courtesy of Andrea Trescot, MD)

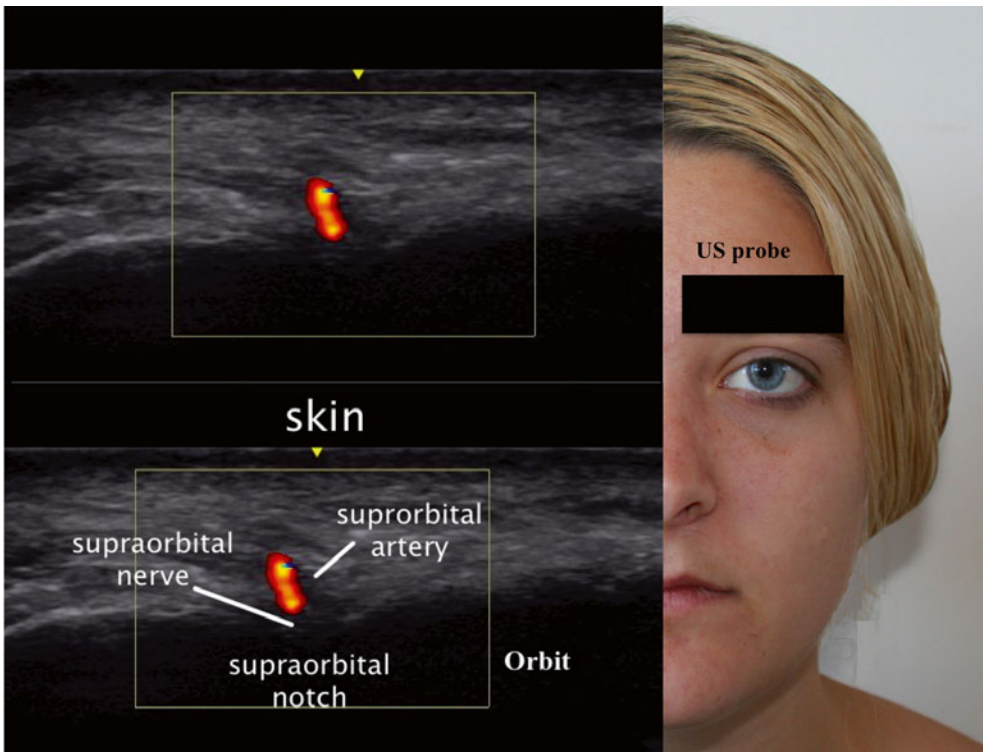

Fig. 21.10 Ultrasound supraorbital image (Image courtesy of David Spinner, MD, modified by Andrea Trescot, MD)

out-of-plane approach, with 97 % of injections considered accurate. The study concluded that US-guided injections had a higher degree of accuracy versus the standard techniques used today. The US technique utilizes the same steps as the conventional landmark-guided technique, but with better target visualization.

Neurolysis/Surgery

Cryoneuroablation

Cryoneuroablation has been an effective therapeutic technique for supraorbital neuralgia. Trescot described the use of cryoneuroablation for the treatment of craniofacial pain syndromes including supraorbital neuralgia [22]. Figure 21.11 depicts the use of cryoneuroablation in the treatment of the SON.

Radio-frequency Lesioning

Although there are potential concerns regarding neuritis and neuromas when performing conventional radio-frequency lesioning, Weyker and colleagues reported long-term relief for more than 7 months from conventional radio frequency of the SON in three patients with confirmative diagnostic supraorbital local anesthetic injections [40].

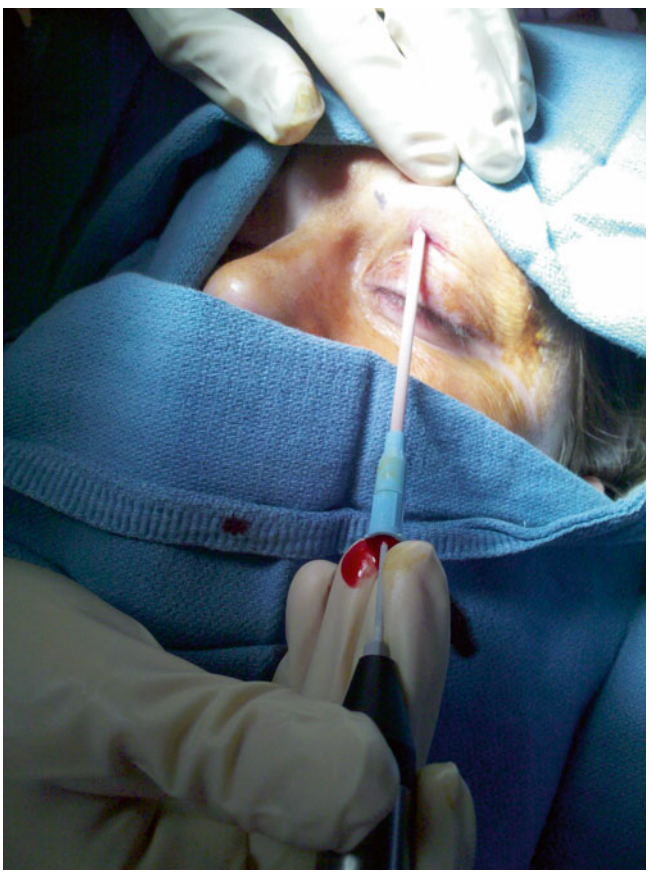

Fig. 21.11 Cryoneuroablation of supraorbital (supratrochlear) nerve (Image courtesy of Andrea Trescot, MD)

Chemical Neurolysis

Chemical neurolytic techniques involve the use of alcohol, phenol, and botulinum toxin. Chemical neurolysis with alcohol or phenol has been used to cause neural destruction of the supraorbital nerve in order to promote longer-lasting effects, compared to a local anesthetic and steroid injection [23, 41, 42]. Extreme caution should be used, however, because of the significant risk of skin sloughing and neuritis. Beyer noted that, after injection of the SON with absolute alcohol, there was "often an extreme, alarming swelling of the upper lid," which would resolve after several days without sequelae [23].

Botulinum toxin use with frontal migraine headaches has shown to significantly improve headaches for several months, likely secondary to relaxation of corrugator muscle in patients with intramuscular entrapments of the SON [43, 44]. Although not specifically reported, the same treatment should be effective for other supraorbital entrapments. Risks include paresthesias and transient blepharoptosis [43].

Peripheral Nerve Stimulation

Neuromodulation in the form of peripheral nerve stimulation is another potentially effective treatment for supraorbital neuralgia. A stimulator lead is tunneled subcutaneously (Figs. 21.12 and 21.13) and connected to a subcutaneous generator. A paper published by Asensio-Samper and colleagues showed that stimulation of the supraorbital nerve is able to provide good analgesic effect in a patient with post-traumatic, refractory supraorbital neuralgia [45]. Amin et al. [46] described 16 patients who responded temporarily to supraorbital nerve blocks; all underwent 5–7 day peripheral nerve stimulator trials. Of these, ten patients underwent permanent implantation and were monitored for 30 weeks; headache pain scores decreased, and opioid consumption was reduced in half. *Trigeminal autonomic cephalgia* [47], *post-herpetic neuralgia,* and *trigeminal neuralgia* [48] have also been effectively treated with supraorbital/supratrochlear nerve peripheral stimulation.

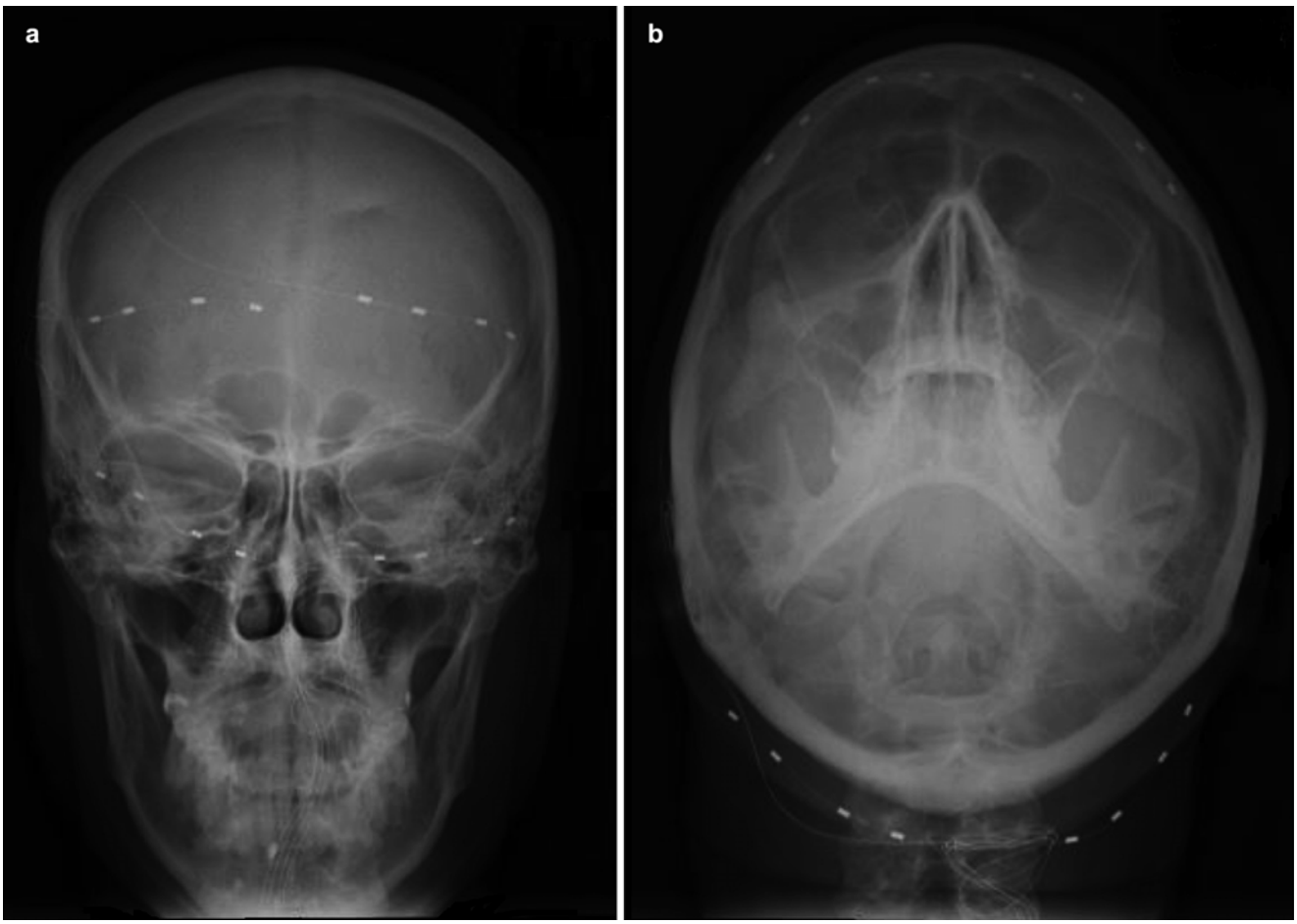

Fig. 21.12 Fluoroscopic image of supraorbital and occipital stimulator trial. *A* Frontal view showing the relative anatomy and proximity of leads to trajectory of the supraorbital and supratrochlear nerves and the occipital nerve group, *B* Waters view showing the peripheral lead placement in all four quadrants of the head (Image courtesy of Matthew Rupert, MD)

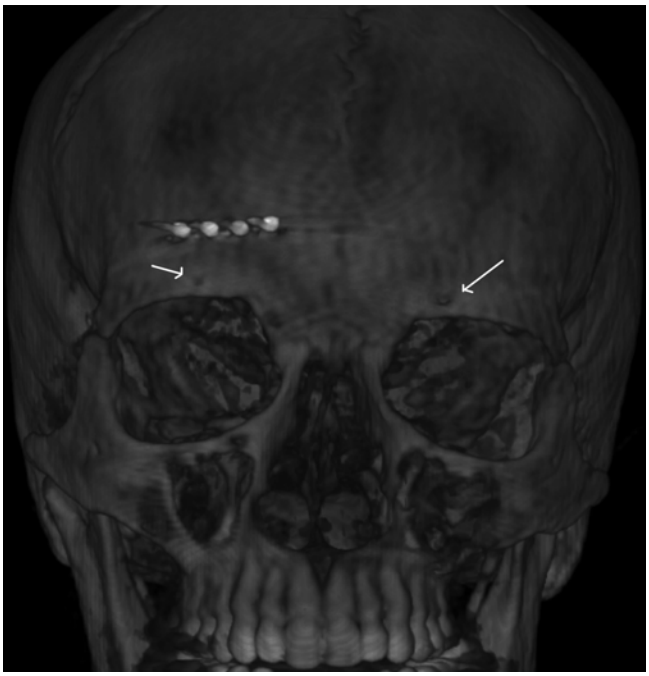

Fig. 21.13 3D image of supraorbital stimulator. Note *arrows*, which identify enclosed supraorbital foramen (Image courtesy of Andrea Trescot, MD)

Surgery

Surgical decompression of the SON has also shown to be promising. Guyuron et al. reported that corrugator resection leads to improvement in symptoms in over 80 % of patients (57 % complete resolution) compared to a sham surgical group, which had only 57 % of patients improving (3.8 % complete resolution) [49]. Sjaastad et al. reported results on five cases of SON decompression with "good" immediate improvement and improvement of 50–100 % (mean of about 85 %), after a mean observation time of more than 6 years [50]. In the two patients with the longest postoperative observation time of about 8 years, pain had not recurred. Poggi et al. [51] reported on the surgical decompression of several nerves, including the supraorbital nerve, on 18 consecutive patients with migraines who received temporary relief from botulinum toxin injections. Three of the 18 patients (17 %) had complete relief, while 50 % (9 of 18) had at least 75 % relief.

In a more recent study, Chepla and colleagues [52] noted that surgical decompression was effective in relieving pain associated with SON entrapment in 92 % of patients, with complete resolution of symptoms in 75 %.

The endoscopic approach has been found to be superior over transpalpebral for supraorbital and supratrochlear nerve decompression in reducing or eliminating frontal migraine headaches in 190 headache sufferers [53].

Ducic and Larson [1] described six consecutive patients with supraorbital neuralgia treated with nerve excision; five of the six patients noted at least 50 % improvement, and three had "complete" relief after a mean of 14 months.

Complications

Complications of SON neuralgia treatment may arise from damage to the local anatomical structures and systemic events. Prolonged compression over the injection site is commonly used to avoid bleeding due to high vascularity of this location. Alopecia can occur if the steroid injection is performed in the eyebrow. Large volume injections may anesthetize the levator palpebrae, causing a reversible, temporary ptosis without diplopia. Cryoneuroablation requires motor stimulation to avoid freezing the nerve to the levator palpebrae.

Trigeminal nerve branches, especially the periorbital, participate in the oculocardiac reflex with activation of the vagal nerve resulting in bradycardia [54, 55], other arrhythmias, and occasionally up to ten second asystole [56]. Because of this, interventional procedures in the trigeminal nerve area necessitate screening for the arrhythmia risk factors, monitoring vital signs, and having all the means and skills for cardiovascular complication treatment available.

Summary

Supraorbital neuralgia is an uncommon cause of face pain that presents with symptoms on the forehead. It has multiple causes and may be confused with other conditions like cluster or migraine-type headaches or sinusitis. A careful history and physical examination, as well as a diagnostic injection, will help the clinician to recognize and treat SON entrapments.

References

1. Ducic I, Larson EE. Posttraumatic headache: surgical management of supraorbital neuralgia. Plast Reconstr Surg. 2008;121(6):1943–8.
2. McGuire TP, Gomes PP, Clokie CM, Sandor GK. Fractures of the supraorbital rim: principles and management. Journal. 2006;72(6):537–40.
3. Klein DS, Schmidt Jr RE. Chronic headache resulting from postoperative supraorbital neuralgia. Anesth Analg. 1991;73(4):490–1.
4. O'Brien Jr JC. Swimmer's headache, or supraorbital neuralgia. Proc (Bayl Univ Med Cent). 2004;17(4):418–9.
5. Janiri L, Gallo G, Nicoletti W. Calcium deficiency and supraorbital headache: a clinical study of adult subjects. Cephalalgia. 1986;6(4):211–8.
6. Massone C, Clapasson A, Gennaro S, Nunzi E. Enlarging plaque on the face with enlarged supraorbital nerve. Dermatol Pract Concept. 2013;3(1):17–20.
7. Sharma RR, Pawar SJ, Lad SD, Netalkar AS, Musa MM. Frontal intraosseous cryptic hemangioma presenting with supraorbital neuralgia. Clin Neurol Neurosurg. 1999;101(3):215–9.

8. Gennaro S, Secci F, Fiaschi P, Merciadri P. Supraorbital nerve entrapment neuropathy in Hansen's disease. Neurol Sci. 2013;34(12):2243–4.
9. Poncelet A. The neurologic complications of Paget's disease. J Bone Miner Res. 1999;14 Suppl 2:88–91.
10. Talmi YP, Finkelstein Y, Wolf M, Ben-Shoshan Y, Kronenberg J. Coincidental supraorbital neuralgia and sinusitis. Am J Rhinol. 1999;13(6):463–8.
11. Benoliel R, Epstein J, Eliav E, Jurevic R, Elad S. Orofacial pain in cancer: part I – mechanisms. J Dent Res. 2007;86(6):491–505.
12. Sindou M, Leston J, Howeidy T, Decullier E, Chapuis F. Microvascular decompression for primary Trigeminal Neuralgia (typical or atypical). Long-term effectiveness on pain; prospective study with survival analysis in a consecutive series of 362 patients. Acta Neurochir (Wien). 2006;148(12):1235–45; discussion 45.
13. Dolenc VV. Frontotemporal epidural approach to trigeminal neurinomas. Acta Neurochir. 1994;130(1–4):55–65.
14. Yamazaki Y, Ochi K, Nakata Y, Dohi E, Eguchi K, Yamaguchi S, et al. Trigeminal neuropathy from perineural spread of an amyloidoma detected by blink reflex and thin-slice magnetic resonance imaging. Muscle Nerve. 2010;41(6):875–8.
15. Agbelusi GA, Wright AA, Danesi MA. Facial neuralgias: analysis of the different types seen at Lagos University Teaching Hospital, (Luth). Niger J Clin Pract. 2005;8(2):114–7.
16. Truini A, Haanpaa M, Zucchi R, Galeotti F, Iannetti GD, Romaniello A, et al. Laser-evoked potentials in post-herpetic neuralgia. Clin Neurophysiol. 2003;114(4):702–9.
17. Benoliel R, Zadik Y, Eliav E, Sharav Y. Peripheral painful traumatic trigeminal neuropathy: clinical features in 91 cases and proposal of novel diagnostic criteria. J Orofac Pain. 2012;26(1):49–58.
18. Agrawal SM, Kambalimath DH. Trigeminal neuralgia involving supraorbital and infraorbital nerves. Natl J Maxillofac Surg. 2010;1(2):179–82.
19. Garza I. The trigeminal trophic syndrome: an unusual cause of face pain, dysaesthesias, anaesthesia and skin/soft tissue lesions. Cephalalgia. 2008;28(9):980–5.
20. Warrier S, Prabhakaran VC, Valenzuela A, Sullivan TJ, Davis G, Selva D. Orbital arteriovenous malformations. Arch Ophthalmol. 2008;126(12):1669–75.
21. Arat YO, Mawad ME, Boniuk M. Orbital venous malformations: current multidisciplinary treatment approach. Arch Ophthalmol. 2004;122(8):1151–8.
22. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
23. Beyer TE. Idiopathic supraorbital neuralgia. Laryngoscope. 1949;59(11):1273–5.
24. Sjaastad O, Petersen HC, Bakketeig LS. Supraorbital neuralgia. Vaga study of headache epidemiology. Cephalalgia. 2005;25(4):296–304.
25. Caminero AB, Pareja JA. Supraorbital neuralgia: a clinical study. Cephalalgia. 2001;21(3):216–23.
26. White CP, Hitchcock CL, Vigna YM, Prior JC. Fluid retention over the menstrual cycle: 1-year data from the prospective ovulation cohort. Obstet Gynecol Int. 2011; on line.
27. Ellis H, Feldman SA, Harrop-Griffiths W. Anatomy for anaesthetists. 8th ed. Malden: Blackwell Science; 2004. p. 358.
28. Andersen NB, Bovim G, Sjaastad O. The frontotemporal peripheral nerves. Topographic variations of the supraorbital, supratrochlear and auriculotemporal nerves and their possible clinical significance. Surg Radiol Anat. 2001;23(2):97–104.
29. Janis JE, Hatef DA, Hagan R, Schaub T, Liu JH, Thakar H, et al. Anatomy of the supratrochlear nerve: implications for the surgical treatment of migraine headaches. Plast Reconstr Surg. 2013;131(4):743–50.
30. Fallucco M, Janis JE, Hagan RR. The anatomical morphology of the supraorbital notch: clinical relevance to the surgical treatment of migraine headaches. Plast Reconstr Surg. 2012;130(6):1227–33.
31. Tomaszewska A, Kwiatkowska B, Jankauskas R. The localization of the supraorbital notch or foramen is crucial for headache and supraorbital neuralgia avoiding and treatment. Anat Rec Adv Integr Anat Evol Biol. 2012;295(9):1494–503.
32. Woo SW, Lee HJ, Nahm FS, Lee PB, Choi EJ. Anatomic characteristics of supraorbital foramina in Korean using three-dimensional model. Korean J Pain. 2013;26(2):130–4.
33. Hwang K, Hwang JH, Cho HJ, Kim DJ, Chung IH. Horizontal branch of the supraorbital nerve and temporal branch of the facial nerve. J Craniofac Surg. 2005;16(4):647–9; discussion 50.
34. Yangüela J, Pareja J, Lopez N, Sáchez del Ríos M. Trochleitis and migraine headache. Neurology. 2002;58:802–5.
35. Tijssen C, Schoemaker K, Visser L. Supraorbital neuralgia caused by nerve entrapment visualized on ultrasonography. Headache. 2013;53(2):376–7.
36. Ettl A, Kramer J, Daxer A, Koornneef L. High resolution magnetic resonance imaging of neurovascular orbital anatomy. Ophthalmology. 1997;104(5):869–77.
37. Matharu MS, Goadsby PJ. Persistence of attacks of cluster headache after trigeminal nerve root section. Brain J Neurol. 2002;125(Pt 5):976–84.
38. Waldman SD. Supraorbital nerve block. In: Waldman SD, editor. Atlas of pain management injection techniques. 3rd ed. Philadelphia: Saunders Elsevier; 2013. p. 4–5.
39. Spinner D, Kirschner JS. Accuracy of ultrasound-guided superficial trigeminal nerve blocks using methylene blue in cadavers. Pain Med. 2012;13(11):1469–73.
40. Weyker P, Webb C, Mathew L. Radiofrequency ablation of the supra-orbital nerve in the treatment algorithm of hemicrania continua. Pain Physician. 2012;15(5):E719–24.
41. Wilkinson HA. Trigeminal nerve peripheral branch phenol/glycerol injections for tic douloureux. J Neurosurg. 1999;90(5):828–32.
42. Ramamurthy S, Walsh NE, Schoenfeld LS, Hoffman J. Evaluation of neurolytic blocks using phenol and cryogenic block in the management of chronic pain. J Pain Symptom Manag. 1989;4(2):72–5.
43. Persaud R, Garas G, Silva S, Stamatoglou C, Chatrath P, Patel K. An evidence-based review of botulinum toxin (Botox) applications in non-cosmetic head and neck conditions. JRSM Short Rep. 2013;4(2):10.
44. Trescot AM. Headache management in an interventional pain practice. Pain Physician. 2000;3(2):197–200.
45. Asensio-Samper JM, Villanueva VL, Perez AV, Lopez MD, Monsalve V, Moliner S, et al. Peripheral neurostimulation in supraorbital neuralgia refractory to conventional therapy. Pain Pract. 2008;8(2):120–4.
46. Amin S, Buvanendran A, Park KS, Kroin JS, Moric M. Peripheral nerve stimulator for the treatment of supraorbital neuralgia: a retrospective case series. Cephalalgia. 2008;28(4):355–9.
47. Vaisman J, Markley H, Ordia J, Deer T. The treatment of medically intractable trigeminal autonomic cephalalgia with supraorbital/supratrochlear stimulation: a retrospective case series. Neuromodulation. 2012;15(4):374–80.
48. Johnson MD, Burchiel KJ. Peripheral stimulation for treatment of trigeminal postherpetic neuralgia and trigeminal posttraumatic neuropathic pain: a pilot study. Neurosurgery. 2004;55(1):135–41; discussion 41–2.
49. Guyuron B, Reed D, Kriegler JS, Davis J, Pashmini N, Amini S. A placebo-controlled surgical trial of the treatment of migraine headaches. Plast Reconstr Surg. 2009;124(2):461–8.
50. Sjaastad O, Stolt-Nielsen A, Pareja JA, Fredriksen TA, Vincent M. Supraorbital neuralgia. On the clinical manifestations and a possible therapeutic approach. Headache. 1999;39(3):204–12.

51. Poggi JT, Grizzell BE, Helmer SD. Confirmation of surgical decompression to relieve migraine headaches. Plast Reconstr Surg. 2008;122(1):115–22; discussion 23–4.
52. Chepla KJ, Oh E, Guyuron B. Clinical outcomes following supraorbital foraminotomy for treatment of frontal migraine headache. Plast Reconstr Surg. 2012;129(4):656e–62e.
53. Liu MT, Chim H, Guyuron B. Outcome comparison of endoscopic and transpalpebral decompression for treatment of frontal migraine headaches. Plast Reconstr Surg. 2012;129(5):1113–9.
54. Kosaka M, Asamura S, Kamiishi H. Oculocardiac reflex induced by zygomatic fracture; a case report. J Craniomaxillofac Surg. 2000;28(2):106–9.
55. Barnard NA, Bainton R. Bradycardia and the trigeminal nerve. J Craniomaxillofac Surg. 1990;18(8):359–60.
56. Gold RS, Pollard Z, Buchwald IP. Asystole due to the oculocardiac reflex during strabismus surgery: a report of two cases. Ann Ophthalmol. 1988;20(12):473–5, 7.

Infraorbital Nerve Entrapment

22

Rafael Justiz and Eugene D. Kaplan

Introduction

Infraorbital neuralgia (IN) is a nerve entrapment syndrome of the infraorbital nerve (ION) that is often secondary to trauma, inflammation, or infection. Patients will complain of unilateral cheek, upper teeth, or nasal area pain, described as sharp, electric-like, tingling pain over the distribution of the infraorbital nerve. These symptoms may be very similar to maxillary infection, and IN is commonly misdiagnosed as maxillary sinusitis. Diagnosis of IN is based on symptomatology and location of pain on physical examination. The ION is the peripheral terminal branch of the maxillary nerve and can become entrapped as the nerve exits the skull via the infraorbital foramen or anywhere along the path of the nerve. Treatment of the ION typically includes a nerve block at the site of entrapment, cryoneuroablation, neuromodulation, and surgical decompression.

Clinical Presentation (Table 22.1)

Infraorbital neuralgia (*IN*) can be caused by trauma to the zygoma or the teeth, as well as inflammation from *maxillary sinusitis* (Table 22.1). It is sometimes confused with a maxillary infection due to sinusitis, *periodontal disease*, or viral infection. Clinical findings include pain over the upper cheek radiating to upper teeth, nasal region, and lower eyelid, with or without tenderness to pressure over the *infraorbital foramen* but without fever, cough, or congestion (Fig. 22.1). Presentation is frequently unilateral but can occur bilaterally, with pain below the eye that occasionally will radiate distally along the infraorbital nerve to the upper teeth, nasal region, lower eyelid, and upper cheek.

The ION can be injured by zygomatic fractures, orbital fractures, and dental traumas. Symptoms may worsen with time if associated with scarring around the nerve. Smiling, laughing, or excessive tension on zygomatic muscles may exacerbate pain symptoms, as can fluid retention associated with pre-menses, excessive salt consumption, and stress. All these precipitating factors can lead to further compression of the ION [4].

Anatomy (Table 22.2)

The ION is one of three peripheral branches of the maxillary division (V2) of the trigeminal nerve (Fig. 22.2). The V2 nerve branches forward and slightly inferior until it exits the skull via the *foramen rotundum* (Table 22.2). The *maxillary nerve* is the branch of the trigeminal ganglia that supplies the zygoma/upper cheek portion of the face. As the nerve diverges distally, it supplies the upper cheek region as the ION. The nerve then continues across the sphenopalatine fossa where it enters the orbit via the inferior orbital fissure and courses along the floor of the orbit in the infraorbital groove. The nerve exits the eye socket through the *infraorbital foramen* under the eye (Fig. 22.3) where it forms its terminal branch

Table 22.1 Occupation/exercise/trauma history relevant to infraorbital nerve entrapment

Zygomatic fracture	76 % demonstrated sensory changes [1]
Sinusitis	
Dental trauma	
Trigeminal neuralgia	Supraorbital and infraorbital trigger zones [2]
Postherpetic neuralgia	V2 distribution [3]

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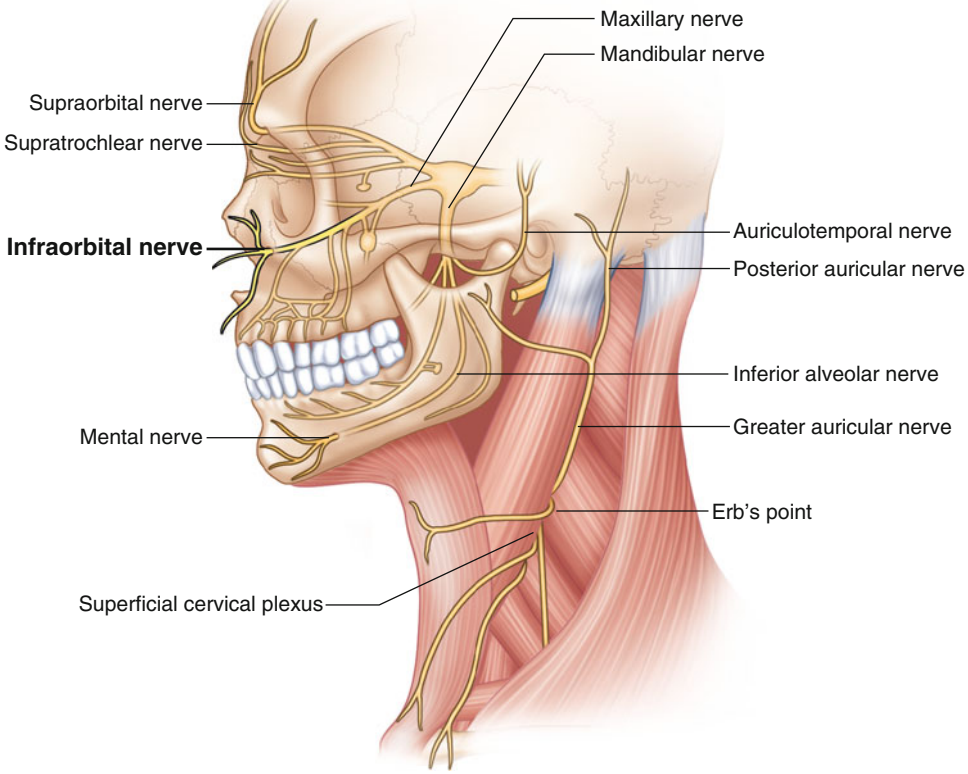
Fig. 22.1 Pattern of pain from infraorbital neuralgia (Image courtesy of Andrea Trescot, MD)

Table 22.2 Infraorbital nerve anatomy

Origin	Trigeminal ganglion V2
General route	Leaves the foramen rotundum as the maxillary nerve; travels across the sphenopalatine fossa, through the inferior orbital fissure, and then exits through the infraorbital foramen. Divides into four branches
Sensory distribution	Lateral nares, upper lip and lower eyelid, upper teeth, and gingiva
Motor innervation	None
Anatomic variability	Artery within the nerve bundle in almost 75 % of dissection [5]
Other relevant structures	Maxillary nerve, pterygoid plate, infraorbital artery

Fig. 22.2 Infraorbital anatomy (Image courtesy of Springer)



(infraorbital nerve). The ION further divides into four branches (Fig. 22.4) (the *inferior palpebral*, *internal nasal*, *external nasal*, and *superior labial* branches) and supplies sensation to the lower eyelid, lateral nares, upper lip, upper teeth, and gingiva (Fig. 22.5). The infraorbital artery is located within the nerve bundle in 73.8 % of the cases [5].

Smith and colleagues [6] measured the distance from the *zygomaticomaxillary suture* (ZMS) to the infraorbital foramen, finding that the foramen was located 23.8 ± 3.1 mm medial and 30.9 ± 3.8 mm inferior to the ZMS. Song et al. [7] measured the distance between the infraorbital foramen in 50 Korean cadavers, finding an average distance of 54.9 ± 3.4 mm. In 2011, a study of 80 dried human skulls showed that the location of the infraorbital foramen was about 6.5 mm inferior to the inferior orbital rim, about 25 mm from the midline, and about 43 mm below the

supraorbital foramen in the same vertical line [8] (Fig. 22.6). Interestingly, 40 % of the skulls showed a significant difference between the left and right sides of the same skull, with a greater difference in men than women.

Ference et al. [9] looked at 100 consecutive CT sinus studies to evaluate the route of the ION through the maxillary sinus (200 nerves evaluated) (Fig. 22.7). They found that

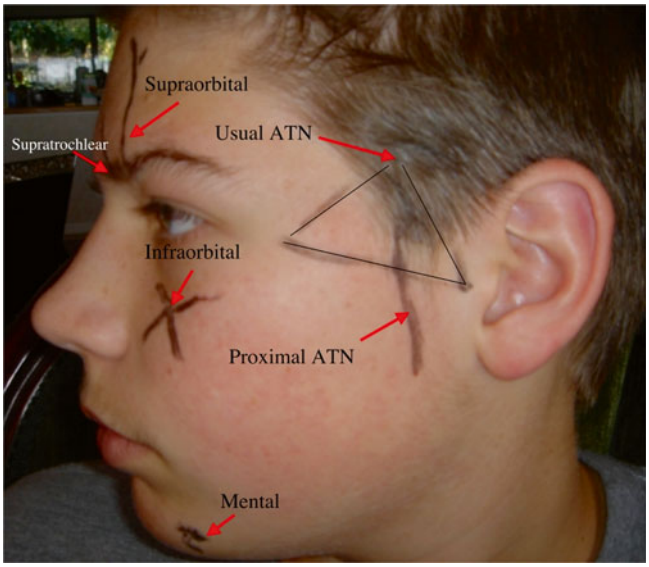


Fig. 22.3 Common nerve entrapments of the face (Image courtesy of Andrea Trescot, MD)

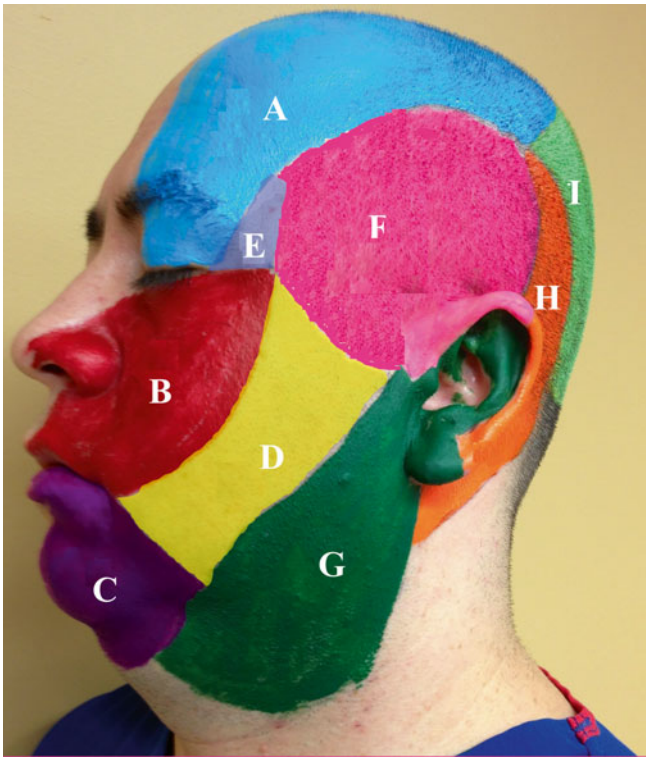


Fig. 22.5 Sensory areas of the trigeminal and cervical nerve branches: *A* supraorbital nerve, *B* infraorbital nerve, *C* mental nerve, *D* buccal nerve, *E* lacrimal nerve, *F* auriculotemporal nerve, *G* superficial cervical plexus, *H* posterior auricular nerve/great auricular nerve, *I* occipital nerve (Image courtesy of Terri Dallas-Prunskis, MD)

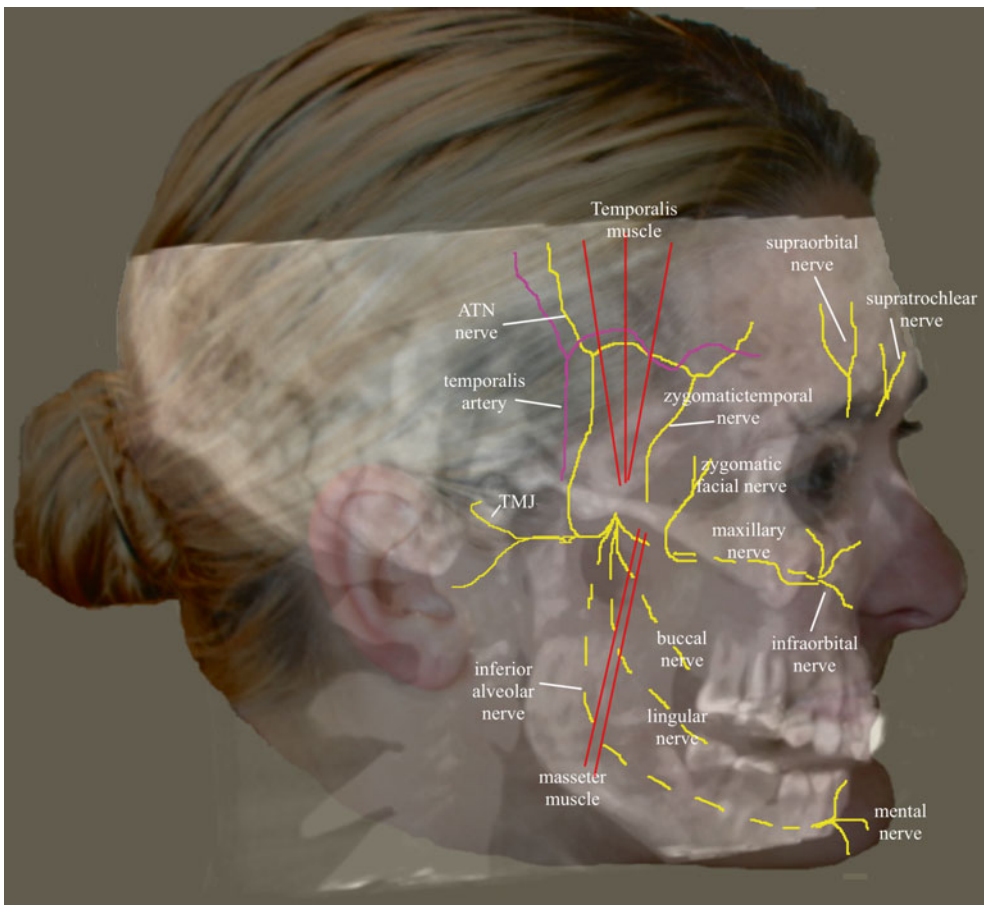


Fig. 22.4 Nerve anatomy related to headaches and face pain (Image courtesy of Andrea Trescot, MD)

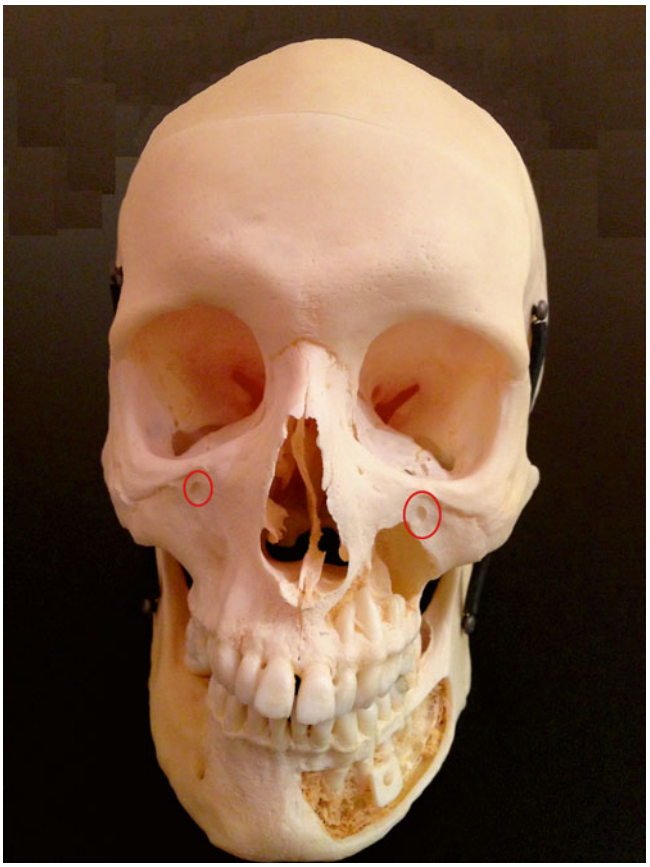


Fig. 22.6 Anatomical location of the infraorbital foramen (Image courtesy of Andrea Trescot, MD)

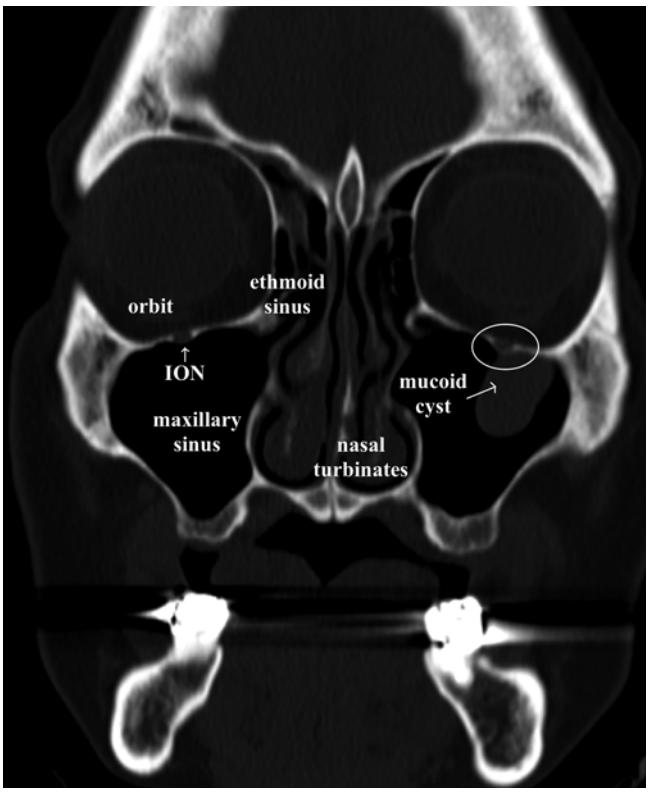


Fig. 22.7 Coronal CT image showing the location of the infraorbital nerve (*ION*) in relation to the maxillary sinus. Note the intra-sinus mucus cyst on the left, incorporating the infraorbital nerve (*circled*) (Image courtesy of Andrea Trescot, MD)

60.5 % of the IONs were entirely contained within the roof of the sinus, but descended below the roof in 27 % and into the lumen in 12.5 % (more commonly if there was an associated infraorbital ethmoid cell).

Entrapment

The infraorbital foramen is the site where the entrapment primarily occurs. Because the infraorbital artery travels within the body of the nerve in almost 75 % of dissections [5], anything that increases arterial blood flow or pressure (such as coughing, head-down position, or hypertension) could increase entrapment of the nerve within the foramen. Other entrapment sites include within the zygoma (after zygomatic fractures) [10], in the maxillary sinus [9], and at the maxillary nerve (see Chap. 23).

Physical Exam

History and physical examination should support the diagnosis of infraorbital neuralgia. A visual inspection and physical exam of the upper cheek, lateral nares, upper lip, lower eyelid, and infraorbital foramen should be performed. IN will present with tenderness to palpation over the infraorbital foramen with possible radiation of symptoms along the nerve distribution of the affected side. Support the head with the non-examining hand, and then palpate with the opposing hand over the zygoma, feeling for the infraorbital foramen (Fig. 22.8) (Video 22.1). Paresthesias should replicate the pain complaints.

Differential Diagnosis (Table 22.3)

The etiology of infraorbital neuralgia (IN) is discovered by a history and physical exam, with the most common cause being an entrapment syndrome (see Table 22.1) along the path of the nerve. The nerve entrapment can occur from multiple reasons, with facial trauma from fractures or blunt injuries being the most common causes. The differential diagnosis is primarily dental and sinus related and easily confused (Table 22.3). Other diagnostic differentials include are trigeminal neuralgia, facial nerve irritations, migraine headaches, sinus infection, acute herpes zoster outbreaks, postherpetic neuralgia, Paget's disease, gingival disease, and neoplastic process (e.g., invasion of the bone by tumor, nerve compression), or by complications of treatment (e.g., radiation fibrosis, chemotherapy-induced neuropathy). The diagnostic tests are found on Table 22.4.

Fig. 22.8 Infraorbital examination (Image courtesy of Andrea Trescot, MD)

Table 22.3 Differential diagnosis of zygomatic face pain

	Potential distinguishing features
Maxillary sinusitis	Fever, purulent drainage, elevated WBC
Periodontal disease	Dental exam shows periodontal disease
Trigeminal neuralgia	Lancinating pain with a trigger zone
Migraine headache	Unilateral, throbbing head pain, response to triptans, family history, aura, triggers present
Acute herpes zoster, especially prior to lesion outbreak	Easy to diagnose after lesions break out
Postherpetic neuralgia	History of shingles
Neoplasm	
Direct tumor compression	MRI/CT evidence of tumor
Complications of treatment	History of radiation/surgery/chemotherapy

Identification and Treatment of Contributing Factors

The most common cause of pain along the infraorbital nerve is an entrapment of that nerve. ION entrapment can occur from multiple reasons (see Tables 22.1 and 22.3), but trauma may be the most common inciting event.

Infraorbital neuralgia can be caused by maxillary sinus infection, and that treatment is often initiated with antibiotic therapy. Periodontal diseases can be treated with antibiotics or surgical therapy. Perimenstrual edema and excessive salt intake can be treated with periodic diuretics or salt-restricted diets.

Table 22.4 Diagnostic tests for infraorbital nerve entrapment

	Potential distinguishing features
Physical exam	Tenderness over the infraorbital foramen
Diagnostic injection	Percutaneous or intraoral injection
Ultrasound	Used for injection
MRI	High-resolution MRI can show the infraorbital nerve and artery [11]
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies	Not useful

Injection Technique

Landmark-Guided Technique

The patient is placed in a supine or sitting position, with the head being supported. The ION can be injected percutaneously or intraorally. To inject percutaneously, the infraorbital foramen is palpated with the index and middle finger of the examining hand. The skin is cleaned and prepped in a sterile fashion with alcohol. The infraorbital foramen will be situated 1.6 ± 2.7 mm lateral and 14.1 ± 2.8 mm superior to the ala of the nose [7]. With the target identified, a 25 or 27 gauge 1/2–1 inch needle is inserted at a 90° angle to the skin until bony contact is made (Fig. 22.9) (Video 22.2). The needle is then slightly withdrawn and redirected in a cephalad direction. Care is taken during needle insertion so as not to insert the needle tip into the infraorbital foramen, as this can precipitate trauma and ischemia of the infraorbital nerve. Injections can also be done intraorally, which avoids cosmetic issues (Fig. 22.10). Following negative aspiration, a volume of 0.5–1 ml of local anesthetic with or without steroid solution is injected. Some authors recommend using larger volumes of injectate, such as 3–4 ml [4]; however, Trescot [12] recommends smaller volumes to avoid the potential increase in entrapment from the injectate. This procedure can be repeated; if there is recurrence of pain, other longer-lasting procedures such as radiofrequency thermocoagulation, cryoneurolysis, or peripheral field stimulation can be performed (see below).

Fluoroscopic-Guided Technique

The use of fluoroscopy can be implemented for better visualization of the infraorbital foramen. The C-arm should be rotated in a cephalad direction until the infraorbital foramen is viewed (Fig. 22.11). Once the foramen is identified, the procedure is

Fig. 22.9 Infraorbital percutaneous injection (Image courtesy of Andrea Trescot, MD)

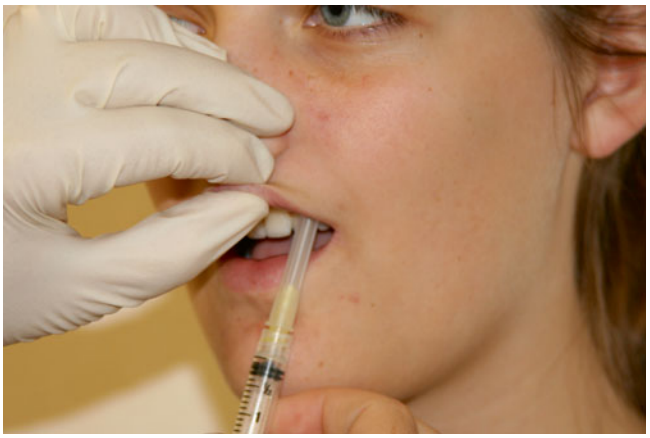


Fig. 22.10 Infraorbital intraoral injection (Image courtesy of Andrea Trescot, MD)

performed in the same fashion as the blind technique, with needle placement just inferior to the infraorbital foramen.

Ultrasound-Guided Technique

Using ultrasound (US) to block the infraorbital nerve can be beneficial over the blind or fluoroscopic approaches. With direct visualization of the infraorbital foramen, proper needle placement can be achieved, and inadvertent needle placement into the foramen can be avoided (Fig. 22.12). Spinner and Kirschner were able to show a 100 % success rate (6 of 6 mental nerve injections) on cadavers; they injected methylene blue under US guidance and then dissected the injected region to confirm location of the medication [13]. The US-guided technique would be performed the same way as the conventional blind technique, but with improved visualization of the target [3, 14, 15]. Iwese and colleagues [16] recently reported the use of a "hockey stick" probe and an in-plane approach to the infraorbital nerve.

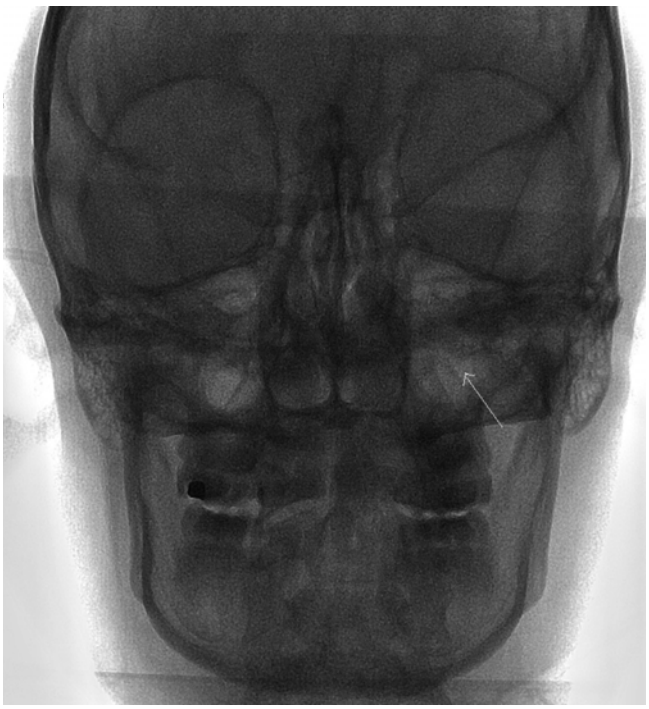


Fig. 22.11 Infraorbital fluoroscopy image. *Arrows* show infraorbital foramen (Image courtesy of Andrea Trescot, MD)

Neurolytic/Surgical Technique

Cryoneuroablation

Cryoneurolysis has been employed as an effective therapeutic technique for ION pain. If the low volume diagnostic injection has given good but only temporary relief, cryoneuroablation may give longer-term relief. The cryoprobe can be placed percutaneously or intraorally (Fig. 22.13), with landmark, US, or fluoroscopic guidance. Zakrzewska and Nally [17] used cryoneuroablation to treat 145 patients with paroxysmal trigeminal neuralgia, including the ION, and followed up these patients for 1 month to 6 years. The mean length of relief for cryoneuroablation of the ION was 20 months; all patients regained sensation within 2–3 months, long before the return of pain (if any). Trescot described the use of cryoanalgesia for the treatment of craniofacial pain, including ION [18].

Radiofrequency Lesioning

Conventional radiofrequency lesioning of the ION would not be recommended because of the high risk of neuritis. However, pulsed radiofrequency (PRF) has been described as a treatment for IN. Lim and colleagues [3] demonstrated the use of PRF of the infraorbital nerve with US guidance. The patient was diagnosed with facial herpes zoster in the left V2 distribution, and medication treatment was prescribed

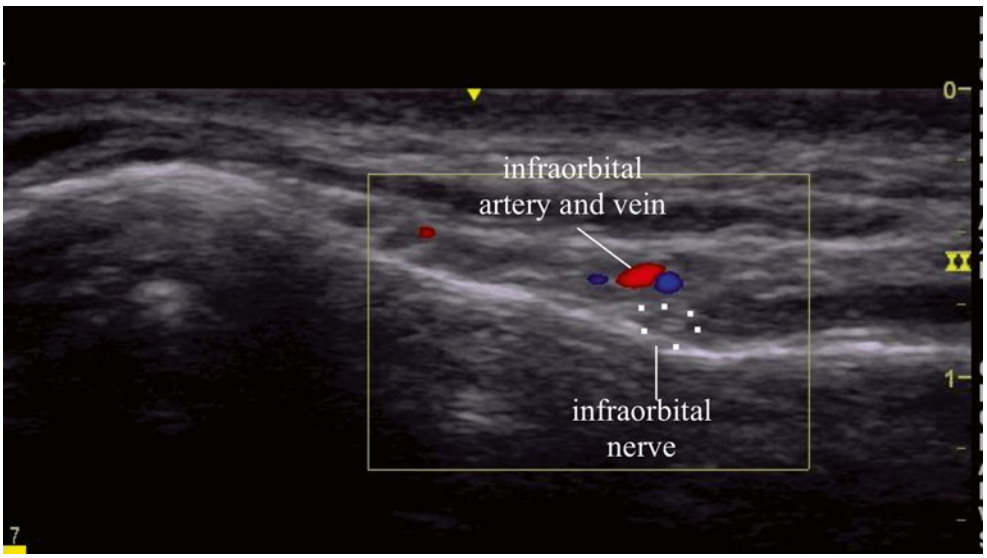


Fig. 22.12 Ultrasound infraorbital image (Image courtesy of David Spinner, MD)

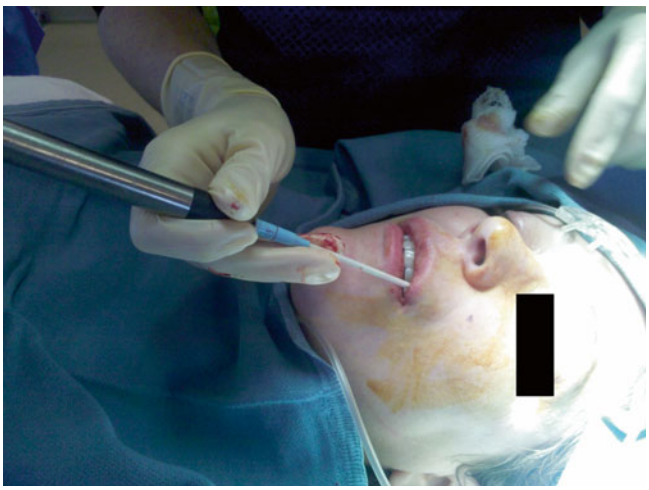


Fig. 22.13 Cryoneuroablation of the infraorbital nerve utilizing an intraoral approach (Image courtesy of Andrea Trescot, MD)

for 6 months with minimal effect. A diagnostic block of the left ION was performed under ultrasound guidance, with good short-term pain relief. This was followed by pulsed radiofrequency treatment on the left infraorbital nerve under ultrasound guidance. The patient experienced 6 months of relief after the procedure.

Chemical Neurolysis

Chemical neurolytic techniques involve the use of alcohol, phenol, and botulinum toxin. Chemical neurolysis with phenol causes a neural destruction of the ION to promote longer-lasting effects compared to local anesthetic and steroid injection [19]. Wilkinson [19] described 60 injections of 6 % phenol in glycerol onto the peripheral trigeminal branches (including the infraorbital nerve) to treat 18 patients with tic douloureux; 80 % noted total or marked relief for a median of 9 months, though 30 % had relief for 2 years. Botulinum toxin use with frontal-type migraine headaches and pain over the lateral nares has shown to significantly help headaches likely secondary to nerve entrapments of the ION [20].

Peripheral Nerve Stimulation

Neuromodulation in the form of peripheral nerve stimulation is another technique that has been used as an effective treatment for ION pain. A stimulator lead is tunneled subcutaneously and connected to a subcutaneous generator (see Chap. 9). A published case report by Lenchig and colleagues showed that peripheral nerve stimulation of the supraorbital and infraorbital nerves provided a 50 % decrease in pain with improved daily function in a patient with posttraumatic trigeminal neuropathic pain [21].

Surgical Techniques

Surgical avulsion of the infraorbital nerve has also shown to be effective. Agrawal and colleagues published a case report of a patient with supraorbital and infraorbital neuralgia that was refractory to medical treatment. The patient underwent avulsion of the supraorbital and infraorbital nerve with complete resolution of pain [22].

Complications

Complications from infraorbital injections are rare but can include bleeding at the site, which can be easily remedied by compression over the injection site. Other complications are worsening pain after injection, neuritis with neurolytic techniques, and bruising related to any subcutaneous bleeding from associated procedures. Despite injections through the "dirty" mouth, intraoral injections and neurolysis have not been associated with infections. This is perhaps not surprising given the large number of intraoral injections and procedures by dentists and oral surgeons without sterilization of the mouth.

Summary

Infraorbital neuralgia is a nerve entrapment syndrome that is usually secondary to trauma, inflammation, or infection that can lead to pain over the upper cheek radiating to upper teeth, nasal region, and lower eyelid. The etiology of ION is discovered, in most cases, by history and physical exam, along with the diagnostic block.

References

1. Fogaca WC, Fereirra MC, Dellon AL. Infraorbital nerve injury associated with zygoma fractures: documentation with neurosensory testing. Plast Reconstr Surg. 2004;113(3):834–8.
2. Agrawal SM, Kambalimath DH. Trigeminal neuralgia involving supraorbital and infraorbital nerves. Natl J Maxillofac Surg. 2010;1(2):179–82.
3. Lim SM, Park HL, Moon HY, Kang KH, Kang H, Baek CH, et al. Ultrasound-guided infraorbital nerve pulsed radiofrequency treatment for intractable postherpetic neuralgia – a case report. Korean J Pain. 2013;26(1):84–8.
4. Waldman SD. Infraorbital nerve block. In: Waldman SD, editor. Atlas of interventional pain management techniques. 3rd ed. Philadelphia: Saunders Elsevier; 2013. p. 70–2.
5. Hu KS, Kwak HH, Song WC, Kang HJ, Kim HC, Fontaine C, et al. Branching patterns of the infraorbital nerve and topography within the infraorbital space. J Craniofac Surg. 2006;17(6):1111–5.
6. Smith JD, Surek CC, Cortez EA. Localization of the supraorbital, infraorbital, and mental foramina using palpable, bony landmarks. Clin Anat. 2010 Epublished.
7. Song WC, Kim SH, Paik DJ, Han SH, Hu KS, Kim HJ, et al. Location of the infraorbital and mental foramen with reference to the soft-tissue landmarks. Plast Reconstr Surg. 2007;120(5):1343–7.
8. Chrcanovic BR, Abreu MH, Custodio AL. A morphometric analysis of supraorbital and infraorbital foramina relative to surgical landmarks. Surg Radiol Anat. 2011;33(4):329–35.
9. Ference EH, Smith SS, Conley D, Chandra RK. Surgical anatomy and variations of the infraorbital nerve. Laryngoscope. 2015; 125(6):1296–300.
10. Sakavicius D, Juodzbalys G, Kubilius R, Sabalys GP. Investigation of infraorbital nerve injury following zygomaticomaxillary complex fractures. J Oral Rehabil. 2008;35(12):903–16.
11. Ettl A, Kramer J, Daxer A, Koornneef L. High resolution magnetic resonance imaging of neurovascular orbital anatomy. Ophthalmology. 1997;104(5):869–77.
12. Trescot AM. Headache: is it a migraine? Think again. Tech Reg Anesth Pain Manag. 2005;9:68–72.
13. Spinner D, Kirschner JS. Accuracy of ultrasound-guided superficial trigeminal nerve blocks using methylene blue in cadavers. Pain Med. 2012;13(11):1469–73.
14. Tsui BC. Ultrasound imaging to localize foramina for superficial trigeminal nerve block. Can J Anaesth. 2009;56(9):704–6.
15. Michalek P, Donaldson W, McAleavey F, Johnston P, Kiska R. Ultrasound imaging of the infraorbital foramen and simulation of the ultrasound-guided infraorbital nerve block using a skull model. Surg Radiol Anat. 2013;35(4):319–22.
16. Iwase N, Fukui H, Yuunaiyama Y, Kaneko K, Ohseto K, Uchino H. The efficacy of ultrasound-guided infraorbital nerve block with hockey stick typed probe. Masui. 2013;62(10):1210–3.
17. Zakrzewska JM, Nally FF. The role of cryotherapy (cryoanalgesia) in the management of paroxysmal trigeminal neuralgia: a six year experience. Br J Oral Maxillofac Surg. 1988;26(1):18–25.
18. Trescot AM. Cryoanalgesia in interventional pain management. Pain Phys. 2003;6(3):345–60.
19. Wilkinson HA. Trigeminal nerve peripheral branch phenol/glycerol injections for tic douloureux. J Neurosurg. 1999;90(5):828–32.
20. Persaud R, Garas G, Silva S, Stamatoglou C, Chatrath P, Patel K. An evidence-based review of botulinum toxin (botox) applications in non-cosmetic head and neck conditions. JRSM Short Rep. 2013;4(2):10.
21. Lenchig S, Cohen J, Patin D. A minimally invasive surgical technique for the treatment of posttraumatic trigeminal neuropathic pain with peripheral nerve stimulation. Pain Phys. 2012; 15(5):E725–32.
22. Agrawal SM, Kambalimath DH. Peripheral neurectomy: a minimally invasive treatment for trigeminal neuralgia. A retrospective study. J Maxillofac Oral Surg. 2011;10(3):195–8.

Maxillary Nerve Entrapment

23

Ava Yoon and Vinay Puttanniah

Introduction

Patients with unilateral lancinating facial pain often carry the diagnosis of trigeminal neuralgia, inflammation of one of the three branches of *trigeminal nerve*. Damage and consequential inflammation of a nerve can be caused by physical entrapment of the nerve. The maxillary nerve (MN) is the second division of the trigeminal nerve and is also known as the V2 division of the *trigeminal ganglion*. It has several areas of entrapment, such as the foramen rotundum and infraorbital foramen, and proper diagnoses and treatments can help decrease symptoms of pain and improve quality of life for patients.

Clinical Presentation

There are many potential causes of maxillary nerve entrapment (Table 23.1), and there may be more than one cause in a given patient. Maxillary nerve entrapment can also present similarly to *infraorbital neuralgia*, since the MN can be affected by a narrow infraorbital foramen at its terminal branch, the *infraorbital nerve* (see Chap. 22).

Patients with maxillary nerve entrapment will complain of cheek pain and paresthesias (Fig. 23.1). The pain is located on the cheek and possibly upper lip regions, and it can radiate to the ipsilateral oral mucosa. The symptoms are often unilateral in location and intermittent in duration. The character of the pain is often described as "pins and needles, lancinating, electrical, and burning". Pain may increase with smiling, washing the face, or chewing, but often it occurs without warning. Many patients describe intense anxiety in anticipation of the next painful episode. For patients whose symptoms are due to a tumor involving the maxillary nerve, symptoms such as facial weakness can accompany the more common symptoms of facial pain and altered facial sensation [1].

Anatomy (Table 23.2)

The *trigeminal* (*gasserian*) *ganglion* in the foramen ovale consists of three branches: the *ophthalmic* (*V1*), the *maxillary* (*V2*), and the *mandibular* (*V3*), seen in Fig. 23.2. Anatomically, the mandibular branch is on the lateral part of the foramen ovale, while the maxillary and ophthalmic braches are more medial. The maxillary nerve (MN) is the continuation of the V2 branch, and it is a pure sensory nerve, supplying sensation to the middle one-third of the face, from the inferior portion of the nose, the upper lip, across the cheek, and into the temple (Fig. 23.3). Anterior to the *trigeminal ganglion*, the MN crosses the *cavernous sinus* anteriorly and inferiorly and then exits the skull through the *foramen rotundum*. The MN innervates the maxillary sinus, as well as the anterior upper teeth via the *anterior and middle superior alveolar nerves*. It then extends through the superior aspect of the *pterygopalatine fossa* and enters the orbit through the *inferior orbital fissure*. The terminal branch of the maxillary nerve is the *infraorbital nerve* (see Chap. 22), which exits the skull through the

Table 23.1 Possible etiologies that can lead to maxillary nerve entrapment

Trauma	Malar fracture
	Dental trauma
Tumor	Schwannoma
	Malignant peripheral nerve sheath tumor
Compression	Anomalous blood vessel
	Stenosis of rotundum foramen

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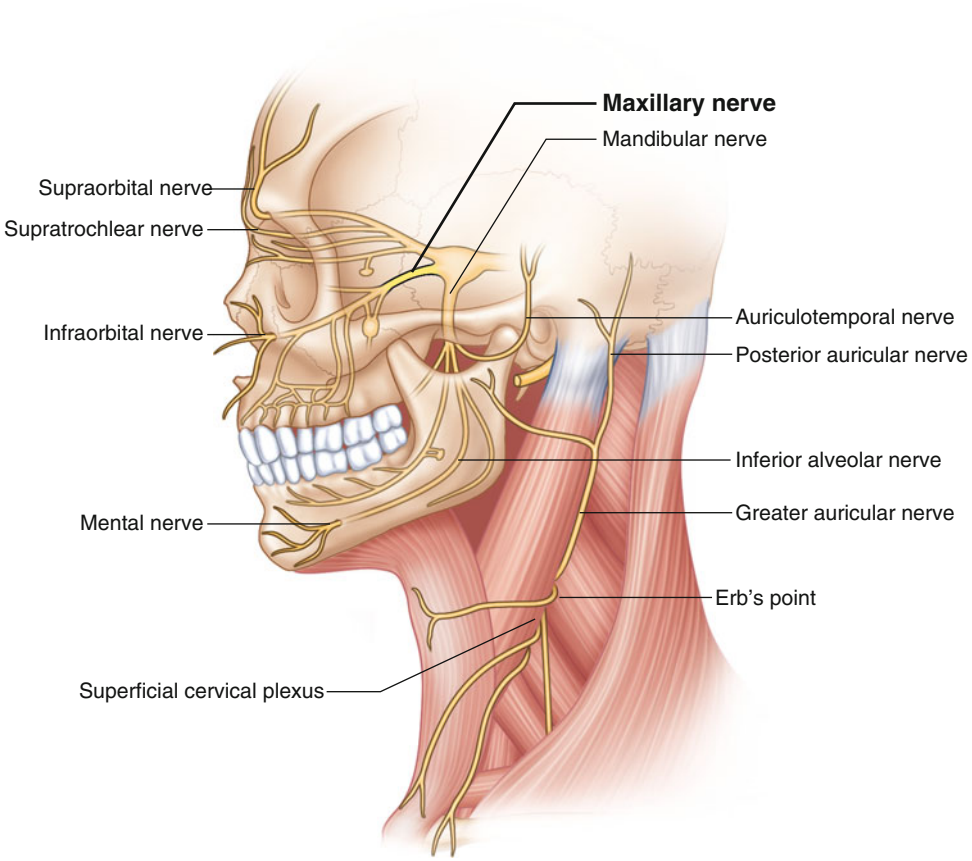
Fig. 23.1 Pattern of maxillary nerve pain (Image courtesy of Andrea Trescot, MD)

Table 23.2 Maxillary nerve anatomy

Origin	Trigeminal ganglion (gasserian ganglion), division 2 (V2)
General route	Maxillary nerve exits skull through foramen rotundum, then through superior pterygopalatine fossa. Exits through inferior orbital fissure, then the infraorbital foramen, ending as the infraorbital nerve
Sensory distribution	Cheek, from lower eyelid to upper lip
Motor innervation	None
Anatomic variability	Foramen rotundum significantly more narrow on the right than the left [2]
Other relevant structures	Pterygomaxillary fissure, pterygopalatine fossa, lateral pterygoid muscle, maxillary artery

Fig. 23.2 Anatomy of the trigeminal nerve and cervical nerves (Image courtesy of Springer)



infraorbital foramen (Fig. 23.3) to innervate the skin and the underlying mucosa from the lower eyelid to the upper lip. While the MN is in the pterygopalatine fossa, it is connected to the *pterygopalatine ganglion*, through which it gives the branches to the nasal cavity, pharynx, and palate. The *zygomaticotemporal* branch of the MN supplies the lateral portion of the face and temple (Fig. 23.4).

Entrapment

The maxillary nerve can be entrapped as it crosses through the *foramen rotundum* [2] and as it exits the *infraorbital foramen* as the infraorbital nerve (Fig. 23.3) (see Chap. 22), resulting in sensory changes and pain in the sensory distribution of the nerve, as described in Table 23.2. Dental trauma

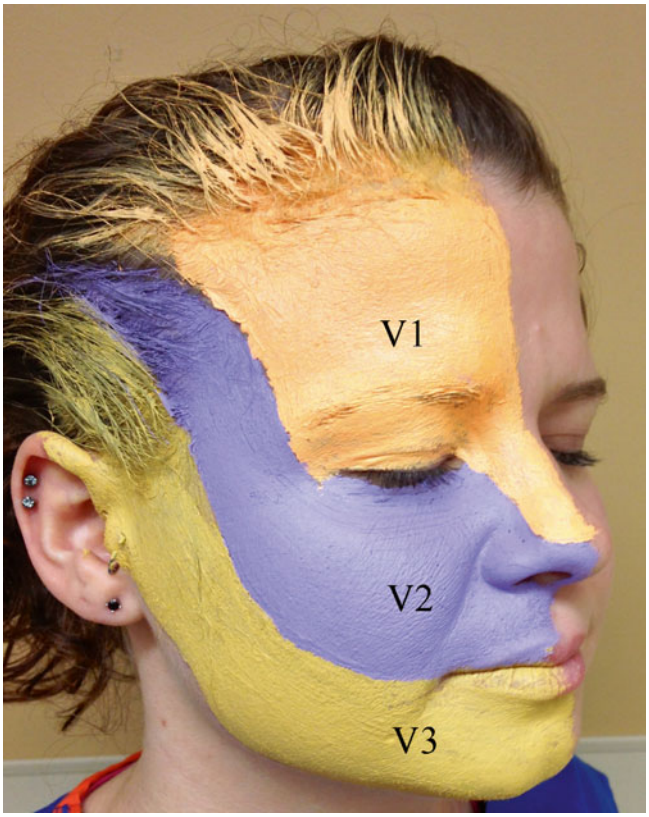


Fig. 23.3 Distribution of trigeminal nerve sensation. *V1* ophthalmic division/supraorbital nerve, *V2* maxillary division/maxillary nerve, *V3* mandibular division/mandibular nerve (Image courtesy of Terri Dallas-Prunskis, MD)

and malar fractures may contribute to maxillary nerve entrapment. The maxillary nerve can also be compressed by fibrous dysplasia, tumors such as schwannoma, or an anomalous blood vessel [3].

Trigeminal neuralgia or *tic douloureux* (also known as *prosopalgia* or *Fothergill's disease*) is a neuropathic pain of the face, usually in a V2 or V3 distribution, characterized by attacks or paroxysms of severe facial pain, often lasting only a few seconds or minutes. Vascular compression of the dorsal root of the trigeminal nerve by an aberrant loop of blood vessels is currently accepted as the most common cause of trigeminal neuralgia. The right side of the face is affected by trigeminal neuralgia twice as often as the left side, but there are no anatomical reasons for the blood vessel loop to be present more frequently on the right side of the cranial fossa. Additionally, vascular compression in asymptomatic patients and in trigeminal neuralgia patients without an aberrant blood vessel loop has been reported, thereby arguing against the idea that vascular compression is solely responsible for trigeminal neuralgia. Anatomical and radiological studies have shown that the foramen rotundum (the exit of the maxillary nerve) on the right side of the human cranium is significantly narrower than on the left side [2]. Based on demographic and epidemiological data of trigeminal neuralgia patients, and on anatomical findings in the foramens, entrapment of the maxillary nerve when it crosses the rotundum foramen was concluded by Neto et al. [2] to be a primary cause of trigeminal neuralgia and accounts for the higher incidence of trigeminal neuralgia on the right side. In a study performed by Burchiel et al. [4], 36 patients out of 42 with trigeminal neuralgia were found to have anatomical distortions of the nerve by an artery, vein, bony prominence, or a combination of factors. In a literature review on malignant peripheral nerve sheath tumors of the trigeminal nerve performed by Schmidt et al. [1], 36 patients with the tumor were reviewed. The average age of onset was 44.6 years. The tumors were seen more commonly in male patients (77.1 %), and the gasserian ganglion was involved in 36.1 % of the cases. Of the cases in which the nerve distribution was specified ($n=25$), the mandibular branch was most commonly involved (72.0 %), followed by the maxillary branch (60.0 %) and the ophthalmic branch (32.0 %), with 44.0 % of patients exhibiting involvement of two or more branches.

Physical Exam

A thorough examination of the teeth, jaw, and sinuses is performed to exclude other causes of pain, such as infection. The neurological exam includes an assessment of which nerve is involved based on the location of pain. Involvement of the maxillary nerve produces pain along the upper lip, teeth and gum, the side of the nose, the part of the cheek under the eye, and the lower eyelid. If the individual is examined during an episode of pain, involuntary twitching of the facial muscles along the affected nerve branch may be seen. To locate the maxillary nerve, identify the coronoid notch by palpating just below the zygoma while having the patient open and close the mouth slightly (Fig. 23.5). Applying pressure at this point should not induce pain, as the nerve is deep to the coronoid notch. However, there may be tenderness to palpation with intraoral palpation of the pterygoid plate (Fig. 23.6).

Differential Diagnoses (Table 23.3)

There are many causes of maxillary nerve entrapment (Table 23.2) and a fairly long list of differential diagnoses (Table 23.3), but a careful history and physical exam will help distinguish these from maxillary nerve entrapment. *Trigeminal neuralgia* can be caused by maxillary nerve entrapment, making these two diagnoses difficult to distinguish. *Myofascial pain* involving the *masseter*, *temporalis*, *zygomaticus*, and *levator labii superioris* muscles can be identified by direct palpation of these muscle groups and is

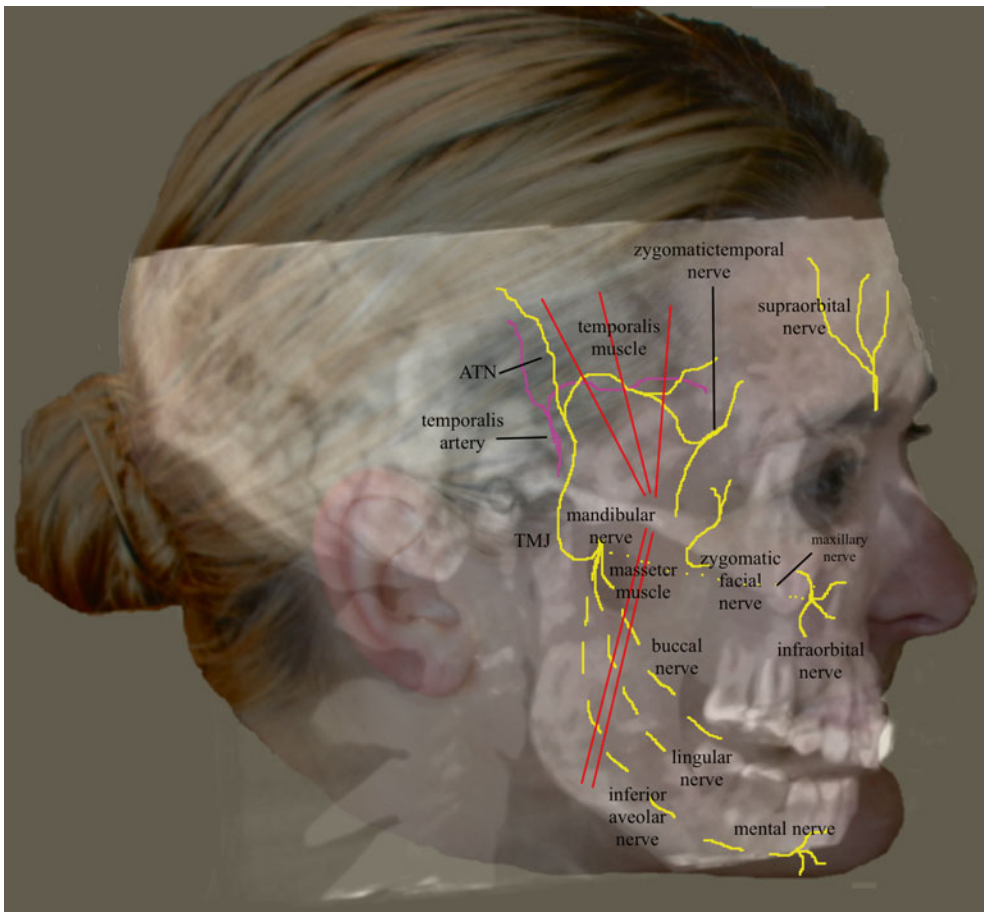


Fig. 23.4 Craniofacial nerves (Image courtesy of Andrea Trescot, MD)

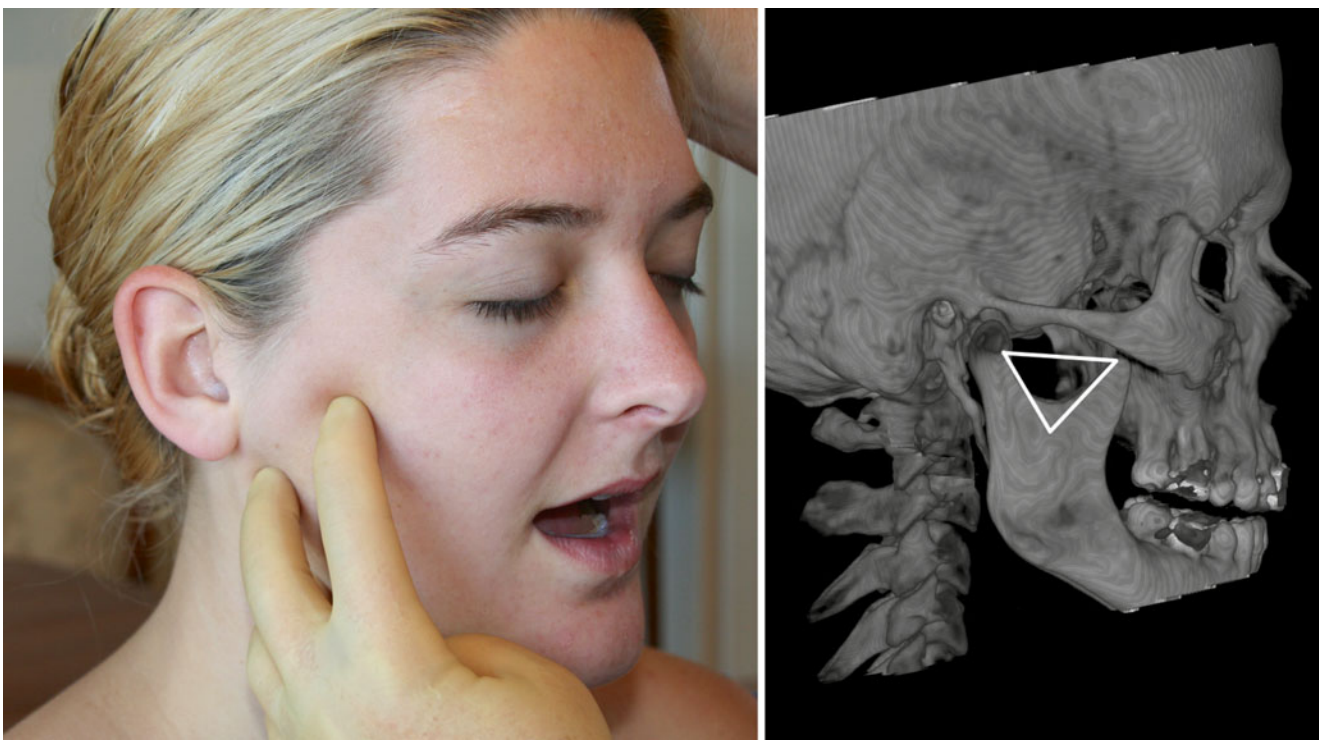


Fig. 23.5 Examination of the coronoid notch (Image courtesy of Andrea Trescot, MD)

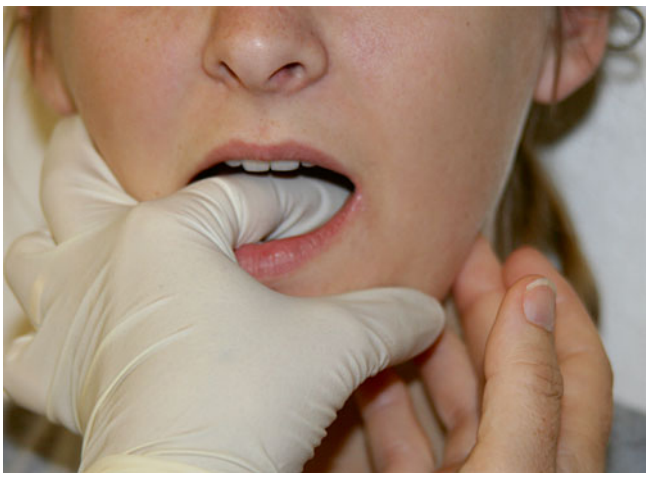


Fig. 23.6 Intraoral examination of the maxillary nerve (Image courtesy of Andrea Trescot, MD)

Table 23.3 Differential diagnosis of maxillary face pain

	Potential distinguishing features
Trigeminal neuralgia	Spontaneous and evoked paroxysm of pain in trigger zone. May be difficult to distinguish from maxillary nerve entrapment, as nerve entrapment may partially contribute to symptoms of trigeminal neuralgia
Atypical neuralgia	"Catch-all phrase" – maxillary nerve neuralgia may be a cause
Myofascial pain	Trigger point tenderness and spasm of masseter, temporalis, zygomaticus major and minor, levator labii superioris muscles
Temporomandibular facial pain	Pain over the TMJ, especially with opening and closing the jaw
Cluster headaches	Paroxysms of pain without facial tenderness; pain often located in retro-orbital, periorbital, and temporal regions. Associated with lacrimation, conjunctival injection, rhinorrhea
Multiple sclerosis	Accompanied by visual changes and neurologic deficits. MRI of brain shows demyelinating lesions
Sinus infection	Fever, X-rays, lab work, sinus drainage
Dental pathology	X-rays, tenderness to dental palpation

not associated with a change in sensation in the V2 distribution, as can be seen with maxillary nerve entrapment. *Temporomandibular joint* (*TMJ*) *pain* can be diagnosed by palpating and examining the joint during jaw movement. TMJ pain is not associated with neurologic symptoms such as paresthesia. *Cluster headaches* often present in young men and are located in retro-orbital, periorbital, and temporal regions, unilaterally. These headaches occur in clusters, as the name describes, and can be associated with ipsilateral lacrimation, conjunctival injection, and rhinorrhea. *Sinus infections* can cause facial pain worsened by applying pressure to the affected sinus(es) and can result in fever and increased white blood cell count. Diagnosis of sinus infection can be confirmed by imaging studies such as X-rays and, more rarely, CT scans. Despite the long list of differential diagnoses, maxillary nerve entrapment can be diagnosed with a careful history and physical exam (Table 23.4).

Table 23.4 Diagnostic tests for maxillary nerve entrapment

	Potential distinguishing features
Physical exam	Involvement of the maxillary nerve produces pain along the upper lip, teeth and gum, the side of the nose, the part of the cheek under the eye, and the lower eyelid. Also may be associated with paresthesia in the same region
Diagnostic injection	Maxillary nerve injection via intraoral or extraoral approaches or infraorbital injection may reduce intensity of pain (described below)
Ultrasound	Nader et al. [4] placed the ultrasound probe below the zygomatic arch, just anterior to the mandibular condyle, and identified rotundum foramen. An experienced physician may be able to identify a narrow foramen, especially in comparison to the contralateral foramen
MRI	MRI may be helpful in identifying vascular or bony malformations, as well as the size of rotundum foramen and infraorbital foramen
Arteriography	Arteriography may be helpful in identifying vascular compression or malformation in proximity to the maxillary nerve
X-ray	X-ray may be useful in identifying fractures, leading to entrapment of maxillary nerve
Electrodiagnostic studies	Electrodiagnostic studies may be helpful in identifying the location of maxillary nerve entrapment

Identification and Treatment of Contributing Factors

Vascular entrapment of the maxillary nerve near its origin can cause painful attacks in the distribution of the nerve [1]. Anatomical and radiological studies have shown that the rotundum foramen on the right side of the human cranium is significantly narrower than on the left side, and patients suffer from pain in the trigeminal distribution on the right side twice as much as the left [2]. The maxillary nerve may also be entrapped in cases of malar fractures and dental trauma. It is possible that the entrapment may occur more distally, in the infraorbital foramen. Entrapment of the maxillary nerve

can be caused by a tumor or fibrosis of the adjacent structures [1]. Trauma that results in anatomical distortion of facial bones and/or teeth can cause entrapment of the maxillary nerve. Also, tumors or vascular malformations in proximity to the nerve can cause entrapment. Lastly, patients with narrow rotundum foramen may be predisposed to maxillary nerve entrapment and consequential pain.

Injection Technique

There are several different approaches to the maxillary nerve. The original approach described in the early 1900s involved inserting a needle through the orbital cavity, exiting the infraorbital fissure [4, 5]. The more common approaches are described below.

Extraoral Landmark-Guided Approach

With the patient positioned sitting or supine, with the head in a neutral position, the coronoid notch is identified by having the patient open and close the mouth (Fig. 23.5). After a skin prep and subcutaneous local anesthetic infiltration, a 25-gauge 2 inch or 22-gauge 3.5 inch needle is placed perpendicular to the skin at the posterior and inferior aspects of the notch (Fig. 23.7a), which should be close to the middle of the zygoma. The needle is advanced until it encounters the lateral pterygoid plate at a depth of 4–5 cm. The needle is then redirected anteriorly and superiorly toward the upper root of the nose (Fig. 23.7b). The needle is again advanced within the pterygopalatine fossa until a paresthesia is obtained. A peripheral nerve stimulator will allow localization of the nerve without resorting to eliciting paresthesias. One to 2 cc of local anesthetic (with or without deposteroids) is injected in divided doses. Contraindications include local infection and coagulation.

Intraoral Landmark-Guided Approach

Dentists and oral surgeons traditionally use intraoral approaches to the maxillary nerve. Three different techniques can be used when performing an intraoral maxillary block (Fig. 23.8) [5].

Oblique Intraoral Approach

The cheek at the angle of the mouth is retracted until the first upper molar is seen. The needle is introduced through the mucosa over the tooth and advanced backward, passing tangential to the maxillary tuberosity. When contact with the bone is lost at a depth of 3–4 cm, the needle is then advanced 0.5 cm more and 2 ml of 1 % lidocaine is injected.

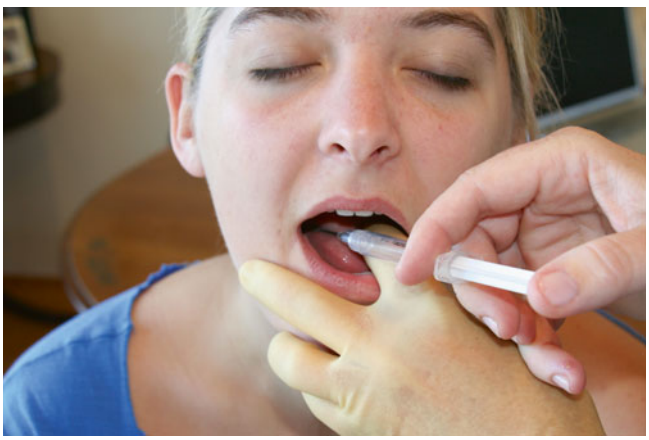


Fig. 23.8 Intraoral injection of the maxillary nerve (Image courtesy of Andrea Trescot, MD)

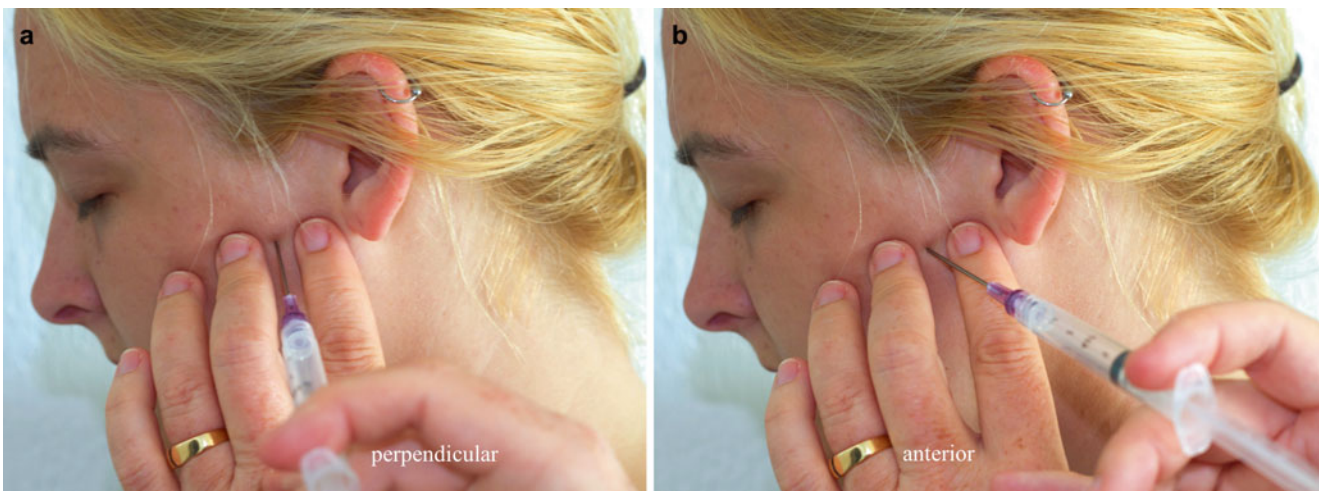


Fig. 23.7 (**a**) A 22-gauge needle is placed perpendicular to the skin at the posterior and inferior aspects of the coronoid notch. (**b**) The needle is advanced to the lateral pterygoid plate, then redirected anteriorly and superiorly. Extraoral injection of the maxillary nerve (Image courtesy of Andrea Trescot, MD)

Pterygomaxillary Intraoral Approach

When using the pterygomaxillary approach, the needle is introduced from the back of the upper molar tooth, directed upward and inward, almost perpendicularly to the tooth. The needle passes laterally to the angle formed by the tuberosity of the maxillary and the pterygoid process at a depth of 3–4 cm and reaches the sphenomaxillary fossa. Two ml of 1 % lidocaine is injected after the aspiration test.

Posterior Palatinal Intraoral Approach

For the posterior palatinal approach, the same technique as the pterygomaxillary route is used, passing the needle through the posterior palatinal foramen into the canal until the needle tip reaches the sphenomaxillary fossa, and 2 ml of 1 % lidocaine is administered.

Fluoroscopy-Guided Injection

The patient is positioned supine, and the midpoint of the zygomatic arch, the mandibular condyle, the coronoid process, and the mandibular notch areas are identified by fluoroscopy (Fig. 23.9). Like the landmark-guided approach, the needle is advanced to the pterygoid plate and then withdrawn and redirected anteriorly and superiorly at about 45° toward the upper root of the nose [5].

An alternative approach was described by Taika et al. [6]; the patient is positioned supine and the head rotated contralaterally approximately 60°, and the X-ray machine tilted caudally approximately 20° until the pterygopalatine fossa is visualized as a "vase" or "inverted vase" (Fig. 23.9) (position may need to be modified for a clear view of the fossa). The needle is advanced percutaneously below the zygomatic arch 3 cm anterior to the tragus and advanced to the top of the angle formed by the orbital floor and the posterior wall of the maxillary sinus into the fossa. The depth is confirmed on AP X-ray view.

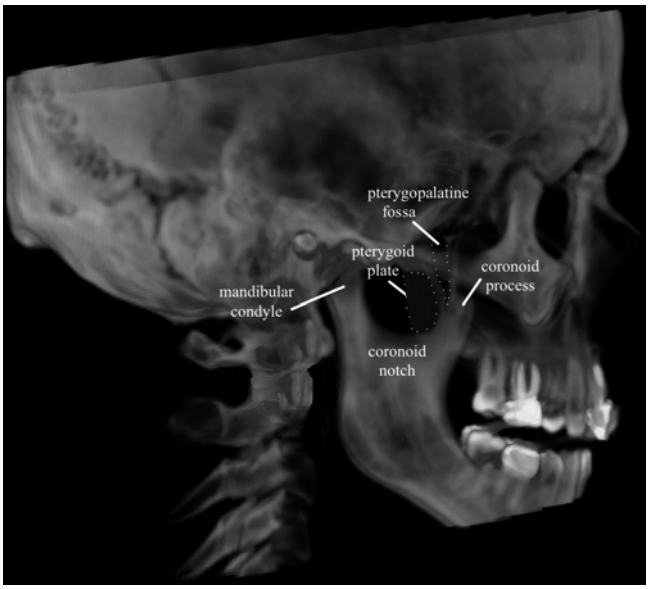


Fig. 23.9 Lateral skull X-ray showing fluoroscopic landmarks (Image courtesy of Andrea Trescot, MD)

Ultrasound-Guided Injection

Nader et al. [7] described a US approach to the maxillary nerve to treat trigeminal neuralgia. They placed the ultrasound probe below the zygomatic arch, just anterior to the mandibular condyle, and identified the mandibular condyle, the coronoid process, the infratemporal fossa, the lateral pterygoid muscle, and the lateral pterygoid plate (Fig. 23.10). They then advanced the needle from posterior to anterior and from lateral to medial through the *pterygomaxillary fissure* into the *pterygopalatine fossa*, passing through the *lateral pterygoid muscle* to the V2 division of the trigeminal nerve (Fig. 23.11). They were able to identify the pulsating maxillary artery, and X-rays confirmed flow of contrast proximally through the *foramen rotundum* onto the *gasserian ganglion*.

Infraorbital Injection

The infraorbital nerve is the terminal branch of the maxillary nerve (see Chap. 22). In cases with infraorbital nerve entrapment, this injection may be useful. The infraorbital foramen is situated 0.5–1 cm below the lower margin of the orbit. The needle is introduced through a point on the cheek 0.5–1 cm lateral to the midportion of the ala of the nose (Fig. 23.12). As soon as there is contact with the maxilla below the foramen, the needle is directed upward and backward, and the entrance to the foramen is felt. The needle should not be introduced into the foramen, and a small volume of local anesthetics (0.2–0.3 ml) should be used to reduce the risk of compression neuropathy.

Sphenopalatine Approach

Dr. Gabor Racz described an injection treatment for the maxillary nerve using a sphenopalatine ganglion (SPG) approach (personal communication). The SPG hangs down from the maxillary nerve in the sphenopalatine fossa. Dr. Racz has noted entrapment of the maxillary nerve/SPG after trauma, parotid surgery, or upper wisdom teeth extraction, causing scarring of the SPG and the palatine branch of the maxillary nerve. After access to the sphenopalatine fossa using a curved, blunt-tipped needle (Fig. 23.13a), he injects 1–2 cc of nonionic contrast (Fig. 23.13b) followed by 2–5 cc of local anesthetic with contrast to perform a hydrodissection (see Chap. 7) with "dramatic improvement."

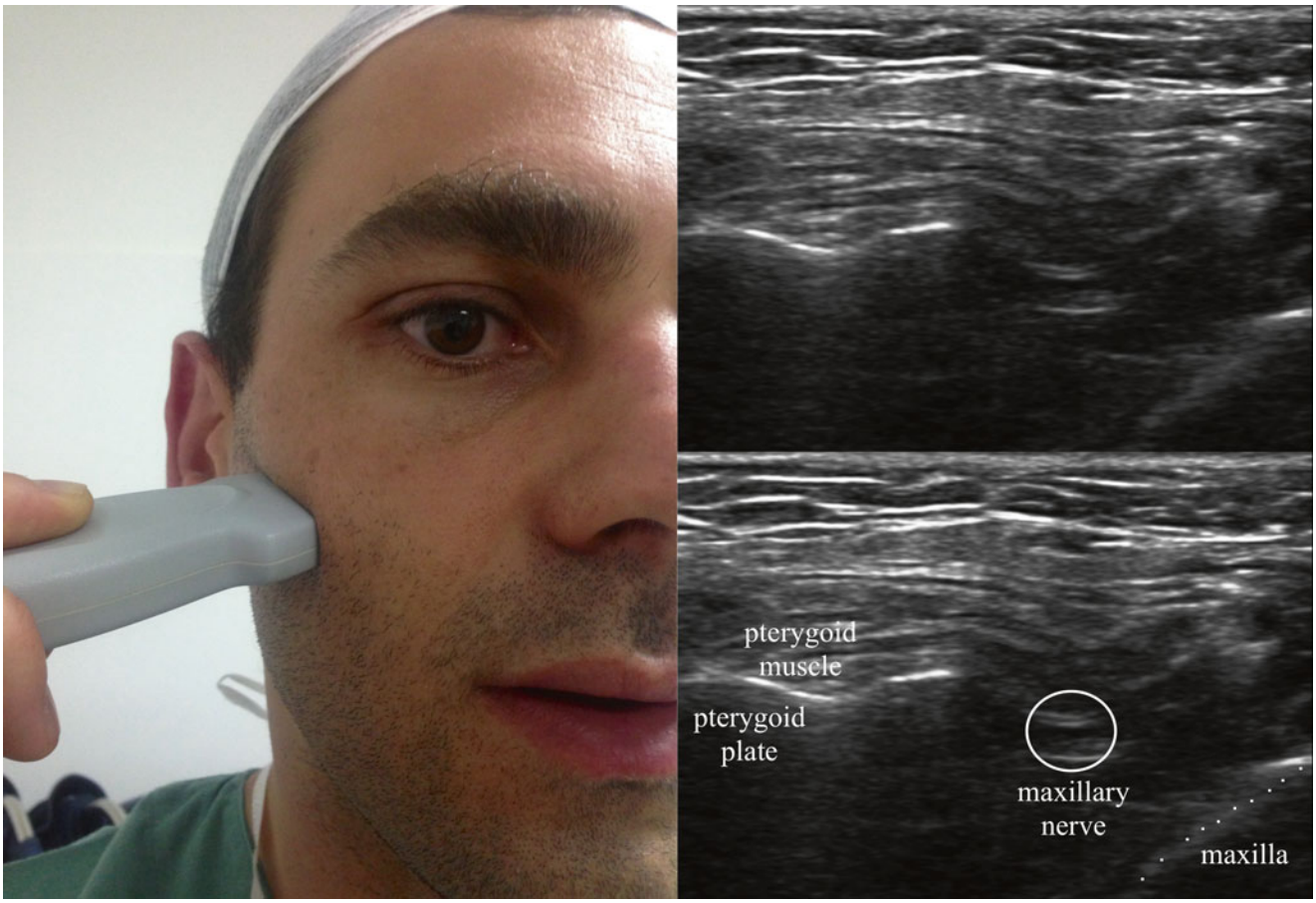


Fig. 23.10 Ultrasound image of the maxillary nerve (Image courtesy of Thiago Nouer, MD)

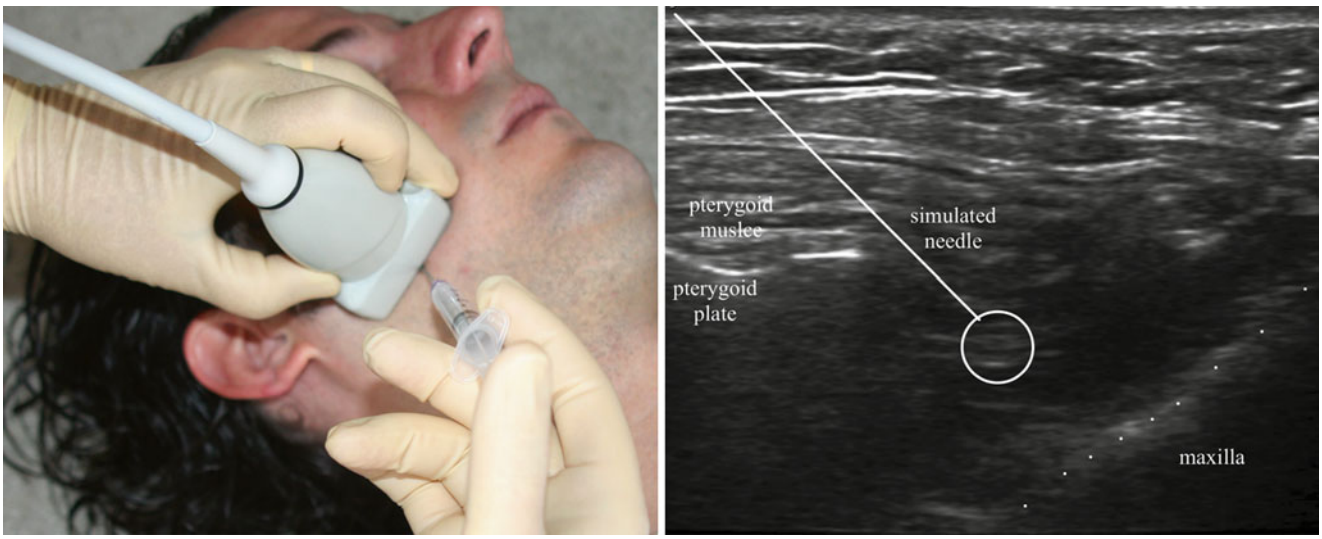


Fig. 23.11 Ultrasound-directed injection of the maxillary nerve (Image courtesy of Andrea Trescot, MD)

Trigeminal Approach

Maxillary nerve pathology can be addressed at the trigeminal ganglion. Using fluoroscopy, the C-arm is positioned to accomplish a submental view; with a contralateral oblique of the C-arm, the foramen ovale can be identified (Fig. 23.14).

Fig. 23.12 Infraorbital nerve injection (Image courtesy of Andrea Trescot, MD)

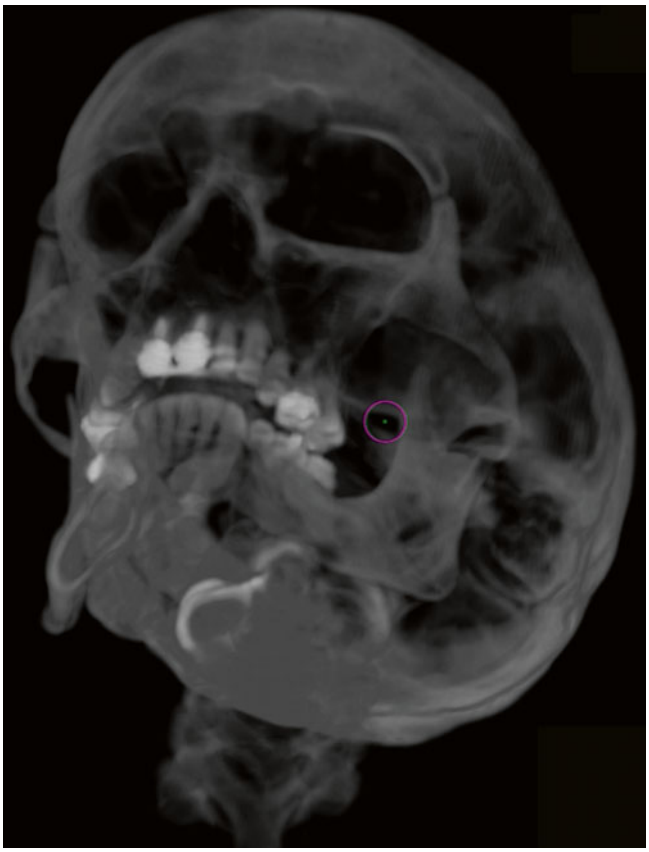


Fig. 23.14 Injection of the trigeminal ganglion (Image courtesy of Andrea Trescot, MD)

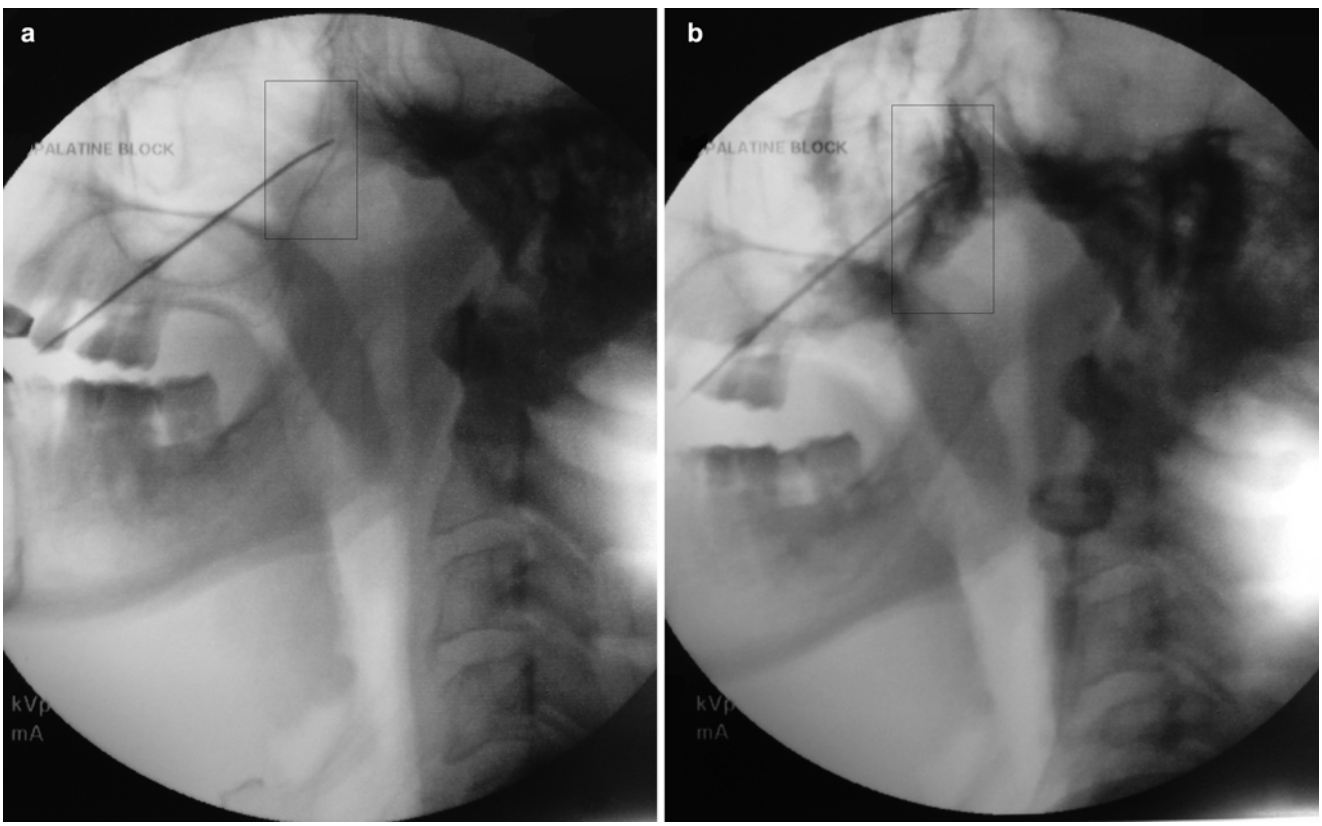


Fig. 23.13 (**a**) A curved, blunt tipped needle is placed in sphenopalatine fossa. Sphenopalatine ganglion injection. Note the contrast extending to the palate in image (**b**) (Image courtesy of Gabor Racz, MD)

Neurolytic/Surgical Treatments

Cryoneuroablation

There are no specific cryoneuroablation techniques described for cryoneuroablation of the maxillary nerve, although Dr. Andrea Trescot (personal communications) has used the percutaneous approach (see above) for cryoneuroablation on a few rare occasions.

Radiofrequency Lesioning

Erdine et al. [5] described pulsed radiofrequency lesioning for the maxillary nerve, using sensory (50 Hz, 0.3–0.6 V) and motor (2 Hz, 0.6–12 V) stimulation for confirmation and lesioning at 120–180 s at 42 °C for two cycles. Brusselmans et al. [8] also described PRF of the maxillary and mandibular nerve to treat atypical face pain. Conventional RF and PRF [7] are widely used for the treatment of trigeminal neuralgia at the trigeminal ganglion.

Chemical Neurolytic Procedures

Using the injection techniques above, chemical neurolytic procedures can be performed. Injection of 6 % phenol or absolute alcohol has been described to treat intractable pain of the maxillary nerve [5, 7]. Injections of glycerol, conventional RF and PRF [9], and balloon compression [10] have been used to treat the trigeminal ganglion at the foramen ovale (Fig. 23.14).

Surgery

Surgical treatments aimed to relieve pain specifically due to maxillary nerve entrapment are not widely performed. Zhu et al. [11] described resection of the maxillary nerve in 26 patients through a maxillary sinus route. After 24 months, 19 (73.08 %) of the patients had an excellent response, 5 (19.23 %) had a good response, 2 (7.69 %) had a fair response, and none (0 %) had a poor response. One patient had a recurrence with palatal pain 3 months after the operation.

Surgical techniques for trigeminal ganglion entrapment/compression are described below.

Microvascular Decompression

Microvascular decompression is the surgical procedure for pain induced by entrapment of the trigeminal nerve by vasculature. It is normally performed under general anesthesia. The skin is incised behind the ear and a 3 cm craniotomy performed. The dura is retracted to expose the trigeminal nerve, and the vascular elements compressing the nerve as it enters the pons are identified. Teflon felt is then used to pad the nerve away from the offending artery or vein. Large series have been published, and the initial efficacy is greater than 80 %. The recurrence rates, compared with those after other invasive treatments, are among the lowest (18 % pain recurrence in 25 years) [3].

Complications include chemical meningitis, ipsilateral hearing loss, and facial sensory loss or palsy. Mortality rates in experienced centers are less than 0.5 %. Serious morbidity includes dizziness, temporary facial palsy, cerebrospinal fluid leaks, meningitis, cerebellar stroke, and hearing loss, which may occur in 1–5 % of patients [3].

Open Trigeminal Rhizotomy

An alternative procedure for the treatment of trigeminal neuralgia is an open trigeminal rhizotomy or partial root section. In this procedure, the trigeminal nerve is partially cut just beyond the exit from the pons, causing some degree of permanent facial numbness. It is most effective for third division trigeminal neuralgia.

Percutaneous Procedures

Two percutaneous procedures for pain induced by trigeminal nerve have been described: percutaneous radiofrequency trigeminal gangliolysis (PRTG) and percutaneous balloon microcompression (PBM).

Percutaneous Radiofrequency Trigeminal Gangliolysis (PRTG)

PRTG is an outpatient procedure performed by placing a needle into the gasserian ganglion, through which an electrical current passes, heating the probe, and producing a thermal lesion in the ganglion. Of the percutaneous procedures, it has the lowest reported rate of pain recurrence, with the average patient experiencing 3 years of excellent pain reduction [4]. Pulsed radiofrequency on the trigeminal ganglion appears promising for the treatment of trigeminal neuralgia [12].

Percutaneous Balloon Microcompression (PBM)

With PBM, the operator inserts a balloon catheter through the foramen ovale into the region of the ganglion and inflates

it for 1–10 min. The patient is awake during the procedure, recovers quickly, and goes home the day of the procedure or the next day.

Summary of Neurolytic Techniques

Zakrzewska and Thomas [13] looked at 475 patients with trigeminal neuralgia; 145 underwent cryoneuroablation, 265 underwent radiofrequency lesioning, and 65 underwent microvascular decompression. These patients were followed for a mean of 45 months and then assessed by the clinician and by questionnaire. The cryoneuroablation patients had a reoccurrence of pain after a mean of 6 months, and the RF patients after 24 months, while only 38 % of the microvascular decompression patients had reoccurrence at 5 years. There was one operative death in the RF group. The patients were all satisfied with their care, but the post-procedure questionnaires showed a significant under-reporting of pain by the clinicians.

Pollack et al. [14] reviewed the costs involved in treating 126 patients who underwent 153 surgeries for trigeminal neuralgia; 33 underwent microvascular decompression, 51 underwent glycerol injections, and 69 underwent stereotactic radiosurgery. The cost per quality-adjusted pain-free year (QAPFY) was $6,342, $8,174, and $8,269 for glycerol injections, microvascular decompression, and stereotactic radiosurgery, respectively. However, because of the longer pain-free interval and the fewer repeat surgeries, microvascular decompression is predicted to be the most cost-effective surgery.

Complications

Because of the vascular nature of the compartment in which the maxillary nerve lies, intravascular injection is possible, and meticulous aspirations are essential. Local anesthetic toxicity may follow an intravascular injection. A hematoma may develop, and inadvertent puncture of dura is possible.

The close proximity of the orbit to this nerve means that the eye is likely to be involved in complications. Orbital swelling, anesthesia of the orbital tissues, ophthalmoplegia, loss of visual acuity, diplopia, or even temporary blindness can occur if the local anesthetic enters the infraorbital fissure. Damage to the vascular structures can cause hemorrhage into the orbit, and permanent blindness can occur.

Summary

Maxillary nerve entrapment, which is often underrecognized, may cause a variety of pain conditions. As a branch of the trigeminal nerve, the maxillary nerve travels from the base of the skull to the cheek. Recognition of maxillary pathology will potentially lead to treatment modalities for midface pain.

References

1. Schmidt RF, Yick F, Boghani Z, Eloy JA, Liu JK. Malignant peripheral nerve sheath tumors of the trigeminal nerve: a systematic review of 36 cases. Neurosurg Focus. 2013;34(3):E5.
2. Neto HS, Camilli JA, Marques MJ. Trigeminal neuralgia is caused by maxillary and mandibular nerve entrapment: greater incidence of right-sided facial symptoms is due to the foramen rotundum and foramen ovale being narrower on the right side of the cranium. Med Hypotheses. 2005;65(6):1179–82.
3. Olson S, Atkinson L, Weidmann M. Microvascular decompression for trigeminal neuralgia: recurrences and complications. J Clin Neurosci Off J Neurosurg Soc Australas. 2005;12(7):787–9.
4. Burchiel KJ, Steege TD, Howe JF, Loeser JD. Comparison of percutaneous radiofrequency gangliolysis and microvascular decompression for the surgical management of tic douloureux. Neurosurgery. 1981;9(2):111–9.
5. Erdine S, Racz GB, Noe CE. Somatic blocks of the head and neck. In: Raj PP, Lou L, Erdine S, Staats PS, Waldman SD, Racz GB, et al., editors. Interventional pain management: image-guided procedures. 2nd ed. Philadelphia: Saunders-Elsevier; 2008. p. 77–108.
6. Taira Y, Higa M, Iso A, Sasara T, Okuda Y. A method of radiographic guiding for maxillary nerve block (pterygopalatine fossa oblique view). J Anesth. 1998;12(4):212–4.
7. Nader A, Schittek H, Kendall MC. Lateral pterygoid muscle and maxillary artery are key anatomical landmarks for ultrasound-guided trigeminal nerve block. Anesthesiology. 2013;118(4):957.
8. Brusselmans G, Crombez E, Devulder J. Percutaneous pulsed radio frequency treatment of atypical facial pain: a study of 3 cases. Euro J Pain Suppl. 2011;5(S1):100.
9. Teixeira MJ, Siqueira SR, Almeida GM. Percutaneous radiofrequency rhizotomy and neurovascular decompression of the trigeminal nerve for the treatment of facial pain. Arq Neuropsiquiatr. 2006;64(4):983–9.
10. Broggi G. Percutaneous retrogasserian balloon compression for trigeminal neuralgia. World Neurosurg. 2013;79(2):269–70.
11. Zhu S, Rong Q, Chen S, Li X. Pterygopalatine fossa segment neurectomy of maxillary nerve through maxillary sinus route in treating trigeminal neuralgia. J Craniomaxillofac Surg. 2013;41(7):652–6.
12. Van Zundert J, Brabant S, Van de Kelft E, Vercruyssen A, Van Buyten JP. Pulsed radiofrequency treatment of the Gasserian ganglion in patients with idiopathic trigeminal neuralgia. Pain. 2003;104(3):449–52.
13. Zakrzewska JM, Thomas DG. Patient's assessment of outcome after three surgical procedures for the management of trigeminal neuralgia. Acta Neurochir (Wien). 1993;122(3–4):225–30.
14. Pollock BE, Ecker RD. A prospective cost-effectiveness study of trigeminal neuralgia surgery. Clin J Pain. 2005;21(4):317–22.

Mandibular Nerve Entrapment

24

Ava Yoon and Vinay Puttanniah

Introduction

The *mandibular nerve* (MN), the V3 branch of the *trigeminal ganglion*, innervates the motor branch of mastication and the sensory branches of the lower face. The nerve can be entrapped in the area of foramen ovale, as well as by vascular anomaly, fibrous dysplasia, scar tissue, and schwannoma, causing unilateral facial pain. Proper diagnosis and injection and/or surgical treatment can lead to improvement of symptoms.

Clinical Presentation (Table 24.1)

Involvement of the mandibular nerve produces pain in the chin, inferior oral cavity, lower teeth, and buccal tissues as well as the tongue (Fig. 24.1). Because the *auriculotemporal nerve* is a branch of the mandibular nerve (see Chap. 15), mandibular entrapment may also present as temple pain (Fig. 24.2). Pain may be neuropathic in character, with burning and shocking pains, intermittent or constant. There may also be decreased sensation to touch. Pain may be increased with chewing or yawning.

Anatomy (Table 24.2)

The temporal region can be divided into the *supratemporal* and *infratemporal fossa*, separated by the *zygomatic arch* (Fig. 24.3). Bony landmarks, containing the lower portions of the *temporalis* and *masseter muscles* as well as the *lateral* and *medial pterygoid muscles*, define the infratemporal fossa anteriorly, posteriorly, and superiorly. In addition, vasculature including the *middle meningeal artery*, *inferior alveolar artery*, *deep temporal artery*, and *buccal artery* can be found in the *infratemporal fossa*. Ligaments such as *pterygospinous* and *pterygoalar ligaments* are located in the fossa as well.

The MN, the largest branch of the *trigeminal ganglion*, is the only mixed motor/sensory division of the *trigeminal*

Table 24.1 Occupation/exercise/trauma history relevant to mandibular nerve entrapment

Trauma	Mandibular fracture
	Dental trauma
Entrapment	Lateral pterygoid muscle
	Foramen ovale
	Fibrous dysplasia
	Scar tissue
Tumor	Mandibular schwannoma

Fig. 24.1 Pattern of mandibular nerve pain (Image courtesy of Andrea Trescot, MD)

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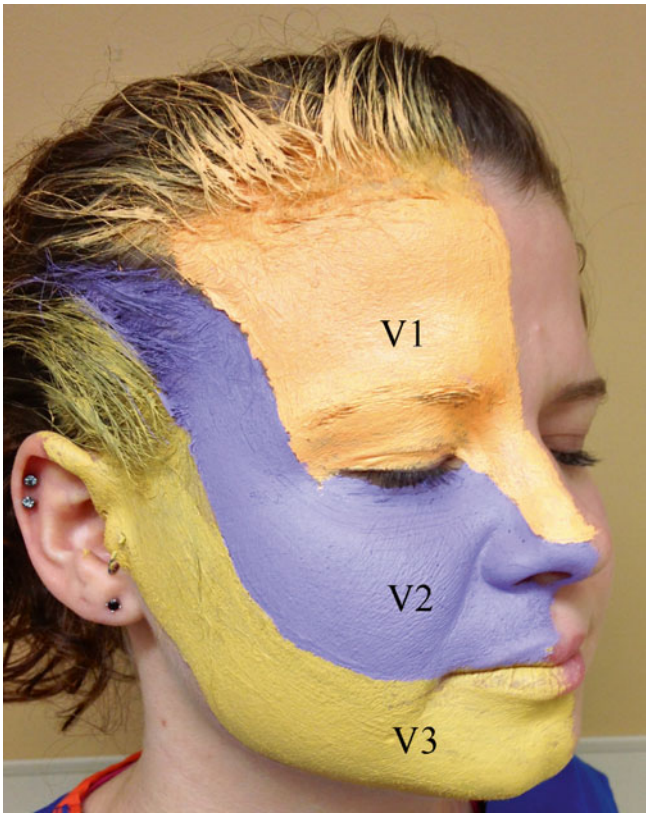

Fig. 24.2 Distribution of trigeminal nerve sensation. *V1* ophthalmic division/supraorbital nerve, *V2* maxillary division/maxillary nerve, *V3* mandibular division/mandibular nerve (Image courtesy of Terri Dallas-Prunskis, MD)

Table 24.2 Mandibular nerve anatomy

Origin	Trigeminal ganglion (TG)
General route	Sensory and motor roots exit through the foramen ovale then join into one trunk
Sensory distribution	Sensory root from the anterolateral portion of TG
Motor innervation	Motor root
Anatomic variability	Foramen ovale significantly more narrow on the right than the left [1]
Other relevant structures	Pterygoid plate, lateral pterygoid muscle

ganglion (TG) (Fig. 24.4). The MN is made of a large sensory root originating from the anterolateral portion of the trigeminal ganglion and a small motor root passing below the ganglion in a separate fascial compartment, uniting with the sensory root. The two roots exit the middle cranial fossa through the *foramen ovale* and combine into a single trunk immediately outside of the foramen ovale. The MN then travels through the *infratemporal fossa*, which contains the *lateral pterygoid muscle* (LPM). The nerve traverses anteriorly and inferiorly deep in the infratemporal fossa, just anterior to the middle meningeal artery.

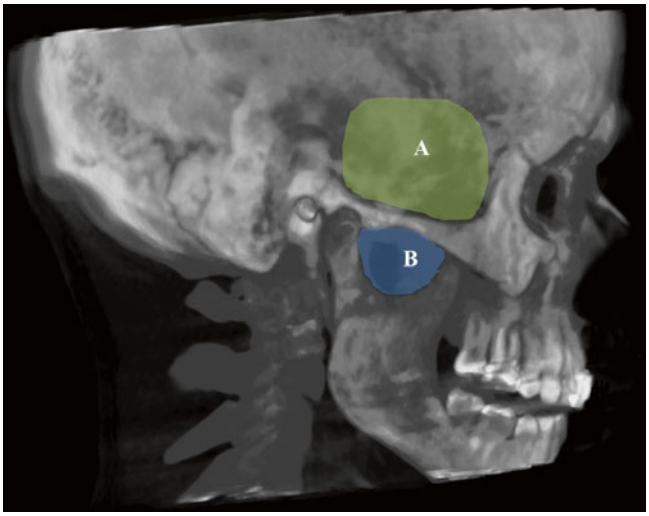

Fig. 24.3 Lateral skull showing the supratemporal fossa (*A*) and infratemporal fossa (*B*) (Image courtesy of Andrea Trescot, MD)

The mandibular nerve gives off two small branches, the *nervus spinosus,* which supplies the dura and the nerve to the pterygoid muscle. It then divides into a small anterior and a large posterior trunk. The *anterior trunk* travels between the roof of the infratemporal fossa and the LPM and is composed of motor fibers (the *masseteric nerve*, the *buccal nerve*, and the *deep temporal nerve*) that innervate the muscles of mastication (the *mylohyoid*, the anterior belly of the *digastric muscle*, the *tensor veli palatini*, and *tensor tympani muscle*), the *anterior deep temporal branch*, the *posterior deep temporal branch*, and a small sensory branch that supplies the mucous membrane and skin over the muscles. The large *posterior trunk* is composed mostly of sensory fibers to the temple and lower jaw, and it travels medial to the LPM. It is divided into the *auriculotemporal* (see Chap. 15) (Fig. 24.3), *lingual*, *mylohyoid*, and *inferior alveolar* nerves (Fig. 24.5) [2]. The inferior alveolar nerve travels laterally to enter the mandible on the medial surface of the mandible through the *mandibular foramen*. The terminal portion of the inferior alveolar nerve is the *mental nerve* (see Chap. 25) (Fig. 24.5), which innervates the chin.

To locate the anatomy of the mandibular nerve from an external point of view, imagine an inverted triangle with one segment formed by the neck of the mandible, another formed by the coronoid process, and the base formed by the line connecting the two (Fig. 24.6). Deep to this triangle is the pterygoid plate, and the mandibular nerve is found posterior to this region (Fig. 24.7).

Entrapment

The mandibular nerve has several possible areas of entrapment. One possible area is the *foramen ovale*. If the diameter of the foramen ovale is too small and/or the size of the nerve

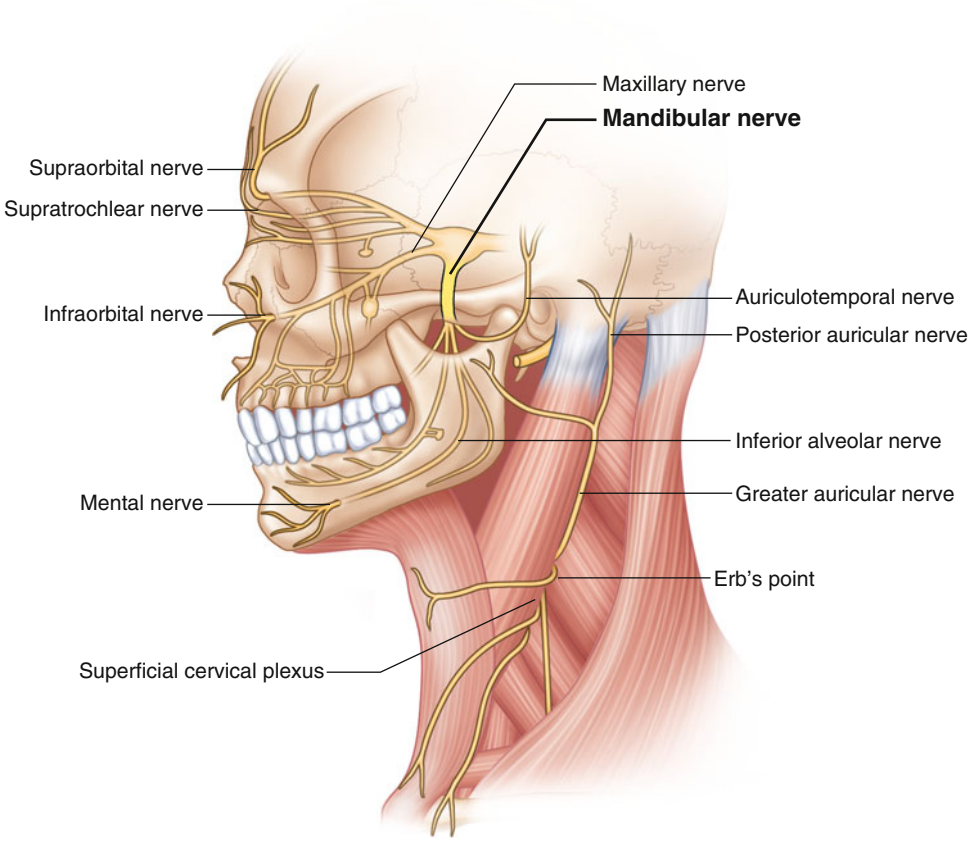

Fig. 24.4 Nerve anatomy of the face (Image courtesy of Springer)

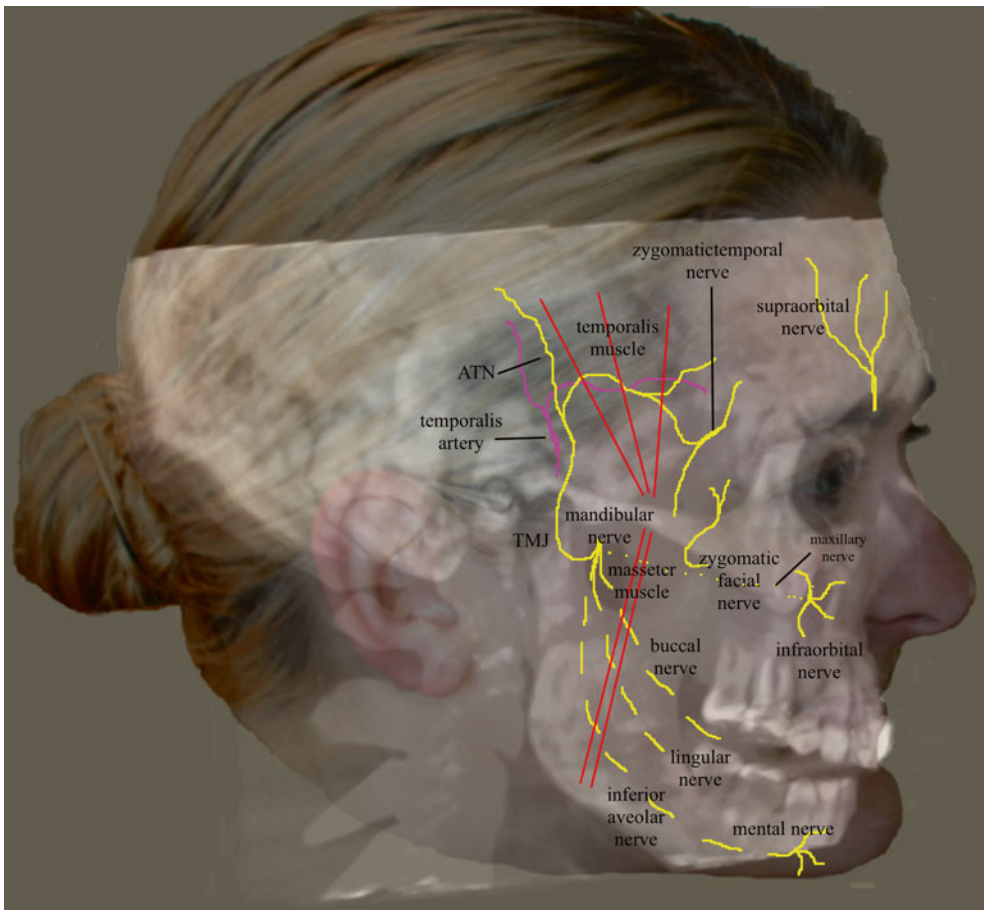

Fig. 24.5 Craniofacial nerves (Image courtesy of Andrea Trescot, MD)

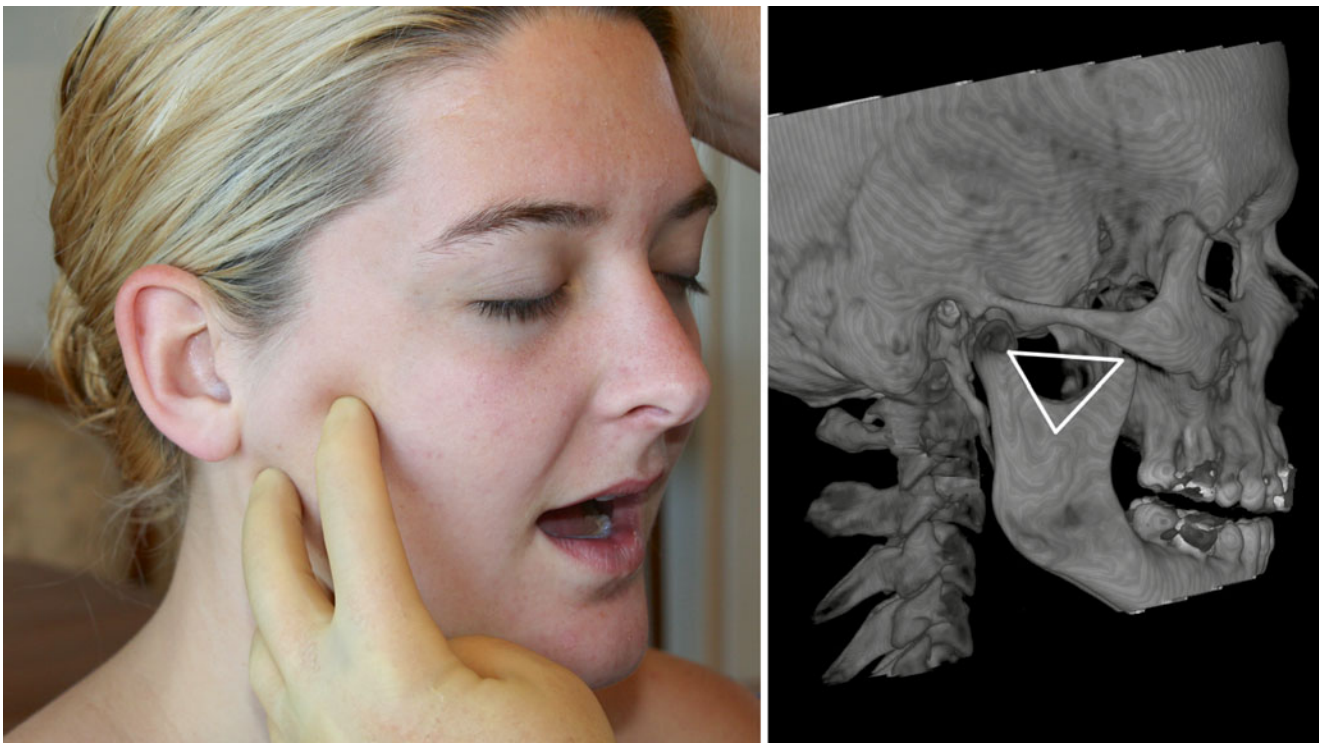

Fig. 24.6 Examination of the coronoid notch (Image courtesy of Andrea Trescot, MD)

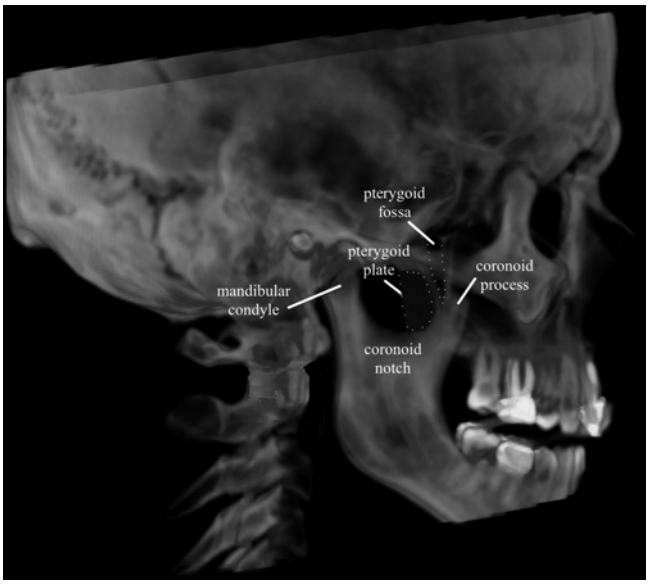

Fig. 24.7 Location of pterygoid plate (Image courtesy of Andrea Trescot, MD)

traversing the foramen is too large, the nerve can be entrapped in this region. According to Neto and colleagues [1], the foramen ovale is significantly narrower on the right side compared to the left, potentially explaining the predominance of right-sided trigeminal neuralgia.

According to Piagkou et al. [3], the *infratemporal fossa* is a common site of mandibular entrapment. Abnormalities in these structures can cause entrapment of the mandibular nerve in the infratemporal fossa. There can be partial or complete calcification of the *pterygospinous* or *pterygoalar ligaments*, causing compression of the posterior trunk of the mandibular nerve. The nerve can be displaced or compressed by an enlarged *lateral pterygoid plate*, or it can be trapped between the *medial and lateral pterygoid muscles*. Hypertrophy of the *lateral pterygoid muscle* (LPM) has been identified as a cause of chronic face and jaw pain [4], especially with chewing. It is also possible that entrapment can be caused by vasculature, especially in the setting of vascular anomaly, as well as tumors such as mandibular schwannoma and fibrous dysplasia. In addition, the mandibular nerve can be entrapped by scar tissue after trauma or surgery.

The clinical result of this entrapment depends on the site of entrapment. Compression of the MN motor branches can lead to paresis or weakness in the innervated muscles, whereas compression of the sensory branches can provoke neuralgia or paresthesia. Compression of the *lingual nerve* (LN) is associated with numbness, hypoesthesia, or even anesthesia of the tongue, loss of taste in the anterior two thirds of the tongue, anesthesia of the lingual gums, pain, and speech articulation disorders.

Entrapment of the posterior division of the mandibular nerve is not uncommon [2], with one study showing entrapment in 6 % of 52 cadavers, primarily at the level of the LPM [4]. The posterior trunk of the mandibular division of the tri-

geminal nerve normally descends deep to the LPM. In 3 of 52 dissections, the 3 main branches of the posterior trunk (*lingual*, *inferior alveolar,* and *auriculotemporal nerves*) were observed to pass through the medial fibers of the lower belly of the LPM. The *mylohyoid* and *anterior deep temporal nerves* also were observed to pass through the LPM in other specimens. These nerve entrapments in the infratemporal fossa provide new information concerning the anatomic and clinical relationships between the mandibular nerve and the lateral pterygoid muscle. These findings support the hypothesis that a spastic condition of the LPM may be causally related to compression of an entrapped nerve that leads to numbness, pain, or both in the respective areas of nerve distribution. Anil et al. dissected 20 mandibular nerves and noted that the nerve was "fixed" between the foramen ovale and the mandibular foramen and was sometimes compressed between the medial and lateral pterygoid muscles or by "neurovascular anatomic variations" [5].

In 1934, Costen suggested a relationship between the MN and the temporomandibular joint (TMJ), and the Costen's syndrome includes symptoms of impaired hearing, ear "stuffiness," ear pain, dizziness, sinus-like pain, headaches, and trismus [6]. Cascone et al. [7] dissected 8 cadavers (16 TMJs) and found that in all specimens, the MN was located at the anteromedial aspect of the mandibular condyle and, during its course, was close to the TMJ capsule

Physical Exam

A thorough examination of the teeth, jaw, and sinuses is performed to exclude other causes of pain such as infection. The neurological exam includes an assessment of which nerve is involved based on the location of pain. Involvement of the mandibular nerve produces pain in the chin, inferior oral cavity and teeth, and buccal tissues. If the individual is examined during an episode of pain, involuntary twitching of the facial muscles along the affected nerve branch may be seen. As the mandibular branch supplies motor function to the temporalis, masseter, and pterygoid muscles, a prolonged or severe entrapment of the nerve may result in weakness or atrophy of these muscle groups. Having the patient open their mouth tests the strength of the pterygoid muscles. If the pterygoid muscle is weak, the chin will deviate toward the side of weakness. The masseter muscle is tested using palpation of the muscle anterior to the ear and superior to the mandible as the patient clenches teeth.

To locate the mandibular nerve, the coronoid notch is identified by palpating just below the zygoma, while having the patient open and close the mouth slightly (Fig. 24.6). Applying pressure to this location does not usually induce pain, since the nerve is deep to the coronoid notch, though the region may be sore from concomitant inflammation.

The mandibular nerve can be more effectively examined intraorally, with the non-examining hand on the cheek and the examining finger intraoral. This finger should carefully palpate the LPM and the inferior alveolar foramen (Fig. 24.8).

Differential Diagnosis (Table 24.3)

There are many causes of mandibular nerve entrapment (Table 24.1) and a fairly large list of differential diagnoses of lower jaw pain (Table 24.3), but a careful history and physical exam help distinguish these from mandibular nerve entrapment. Trigeminal neuralgia can be caused by mandibular nerve entrapment, making these two diagnoses often difficult to distinguish. Myofascial pain involving the *masseter*, *zygomaticus*, and *lateral pterygoid* muscles can be identified by direct palpation of these muscle groups and is not associated with change in sensation in the V3 distribution, as can be seen with mandibular nerve entrapment. Temporomandibular joint (TMJ) pain can be diagnosed by palpating and examining the joint during jaw movement, and it is not associated with neurologic symptoms such as paresthesias (Table 24.4).

Identification and Treatment of Contributing Factors

The MN entrapment can be caused by narrowing at the foramen ovale, as well as by tissue fibrosis, compression by vasculature (especially in the setting of vascular anomaly), and tumors such as mandibular schwannoma and fibrous dysplasia. Also, as discussed earlier, the nerve can be extrinsically compressed from LPM, and sometimes the *medial pterygoid muscle* (MPM) can contribute to entrapment as well.

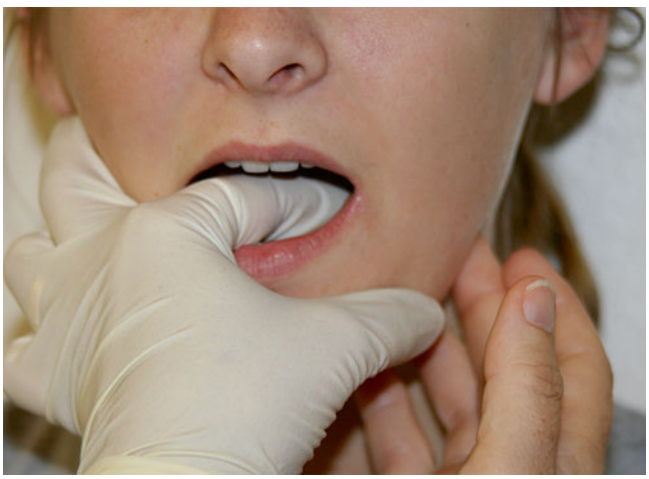

Fig. 24.8 Intraoral examination of the mandibular nerve (Image courtesy of Andrea Trescot, MD)

Table 24.3 Differential diagnosis of lower jaw pain

	Potential distinguishing features
Trigeminal neuralgia	Spontaneous and evoked paroxysm of pain in trigger zone. May be difficult to distinguish from mandibular nerve entrapment as nerve entrapment may partially contribute to symptoms of trigeminal neuralgia
Atypical neuralgia	"Catch-all phrase" – mandibular nerve neuralgia may be a cause
Myofascial pain	Trigger point tenderness and spasm of masseter, zygomaticus, and lateral pterygoid muscles
Temporomandibular facial pain	Pain over the TMJ, especially with opening and closing the jaw
Multiple sclerosis	Accompanied by visual changes and neurologic deficits. MRI of brain shows demyelinating lesions
Maxillary nerve entrapment	Maxillary distribution of pain
Dental pathology	X-rays, tenderness to dental palpation

Table 24.4 Diagnostic tests for mandibular nerve entrapment

	Potential distinguishing features
Physical exam	Tenderness on intraoral exam with the practitioner's finger palpating the LPM and inferior alveolar foramen
Diagnostic injection	Extraoral or intraoral
Ultrasound	Ultrasound is useful for diagnosing anatomical abnormalities. Fractures, enlarged vasculature, and muscles can be visualized
MRI	MRI head can be used to study the mandibular nerve and its surrounding structures
Arteriography	Arteriography can be a sensitive study in diagnosing abnormal vasculature
X-ray	X-ray is useful for displaying bony abnormalities such as fractures that can contribute to mandibular nerve entrapment
Electrodiagnostic studies	Electrodiagnostic studies are a useful tool to locate the entrapped portion of the mandibular nerve

Fig. 24.9 (**a**, **b**) Extraoral injection of the mandibular nerve (Image courtesy of Andrea Trescot, MD)

Valeriy felt that "conservative" or "expectant" care was not advised, noting that all 16 patients he studied had failed conservative care, and the earlier that offending implants were removed, the faster the resolution of the pain [8]. Fukuda et al. [9] felt that preoperative CT imaging is essential for patients undergoing implant placement surgery, to identify the anatomy as accurately as possible in order to decrease the risk of nerve injury. If nerve injury is identified intraoperatively, Misch and Resnik [10] describe the intraoperative topical application of the IV formulation of dexamethasone 4 mg/cc, 1–2 cc applied for 1–2 min. They also recommend removal of any implants associated with the nerve injury. Gatot and Tovi reported on the use of prednisone for postdental trauma to the inferior alveolar nerve [11].

Injection Technique

Landmark-Guided Extraoral Approach

With the patient positioned sitting or supine and the head in a neutral position, the coronoid notch is identified by having the patient open and close the mouth (Fig. 24.6). The initial approach for blocking this nerve is identical to that for blocking the maxillary nerve (see Chap. 23). After a skin prep and subcutaneous local anesthetic infiltration, a 25-gauge 2 inch or 22-gauge 3.5 inch needle is introduced perpendicular to the skin at the posterior and inferior aspects of the notch (Fig. 24.9a), which should be close to the middle of the zygoma. The needle is advanced until it encounters the lat-

eral pterygoid plate at a depth of about 4–5 cm. Then, the needle is walked backward off the lateral pterygoid plate, maintaining the same depth as the plate until paresthesia of the lower lip, lower jaw, or ipsilateral tongue or ear is obtained (Fig. 24.9b). A peripheral nerve stimulator will allow localization of the nerve without resorting to eliciting paresthesias. One to 2 cc of local anesthetic (with or without deposteroids) is injected in divided doses. It should never be necessary to advance the needle more than 5.5 cm beyond the skin in the extraoral technique. If paresthesia is not obtained at this depth, the needle should be withdrawn and the landmarks reconsidered. Contraindications include local infection and coagulation abnormalities.

Intraoral Approach

To perform the intraoral technique, retract the cheek with the index finger or a retractor until the second upper molar tooth is seen. A 3.5 inch needle is inserted into the mucous reflection above the mucosa of the tooth, directed posteriorly, upward, and inward toward the infratemporal plate (Fig. 24.10). The direction of the needle from the lateral view should be toward the midpoint of the zygomatic arch; from the frontal view, it should be toward the outer canthus. At a depth of 4–5 cm, the needle will contact the infratemporal plate, and at that area paresthesia should be sought. When the patient feels paresthesia, 2 cc of local anesthetic with or without deposteroid is injected.

Fluoroscopic-Guided Technique

There are two different fluoroscopic approaches to the mandibular nerve – peripheral and trigeminal. The peripheral approach is a modification of the landmark-guided approach.

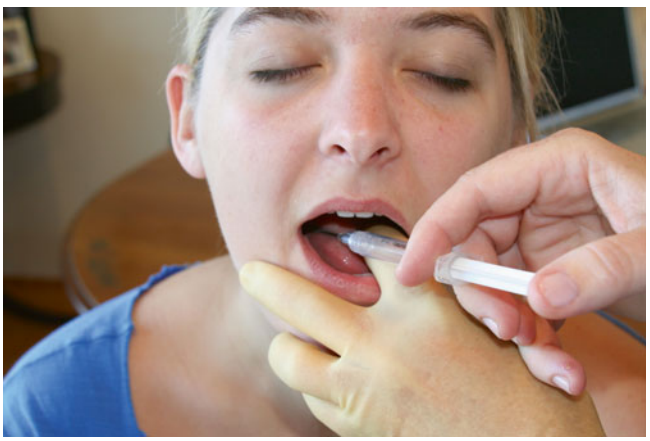

Fig. 24.10 Intraoral injection of the mandibular nerve (Image courtesy of Andrea Trescot, MD)

The pterygoid plate is identified by fluoroscopy (Fig. 24.11); after an appropriate skin prep and subcutaneous infiltration, a 22-gauge 3.5 inch Quincke needle is advanced under fluoroscopic control to the pterygoid plate and then withdrawn slightly and redirected posteriorly. A paresthesia (or muscle twitch from a peripheral nerve stimulator) will confirm the location.

The mandibular nerve can also be injected at the foramen ovale. Under fluoroscopic guidance, using a submental view, the foramen ovale is identified and the needle advanced into the foramen (Fig. 24.12). The reader is referred to specific interventional texts for details of this injection, which are beyond the scope of this chapter [12].

Chang Chien et al. [13] described the fluoroscopic injection of the mandibular nerve for TMJ disorder in three people, using a foramen ovale approach with a submental view (Fig. 24.13).

Ultrasound-Guided Technique (US)

Ultrasound has been used successfully to find and inject the MN [14]. The probe is placed horizontally on the side of the face, just below the zygoma and above the mandibular notch, anterior to the mandibular condyle, with the patient's mouth slightly open (Fig. 24.14a). The *zygomatic bone*, LPM, *lateral pterygoid plate*, mandible, and *maxillary artery* are then identified (Fig. 24.14b). The injection can be performed from an in-plane approach from a lateral to medial and posterior to anterior direction, deep to the LPM, or out-of-plane, in an inferior to superior direction.

Neurolytic/Surgical Treatments

Cryoneuroablation

There are no specific cryoneuroablation techniques described for cryoneuroablation of the mandibular nerve, although Trescot (personal communications) has used the percutaneous approach (see above) for cryoneuroablation on a few rare occasions. More commonly used are cryoneuroablation treatments of the peripheral branches of the mandibular nerve, including the ATN (see Chap. 15), inferior alveolar, and mental (see Chap. 25) nerves.

Radiofrequency Lesioning (RF)

Erdine et al. [15] described pulsed radiofrequency lesioning (PRF) for the maxillary and mandibular nerves, using sensory (50 Hz, 0.3–0.6 V) and motor (2 Hz, 0.6–12 V)

Fig. 24.11 Fluoroscopic anatomy of the mandibular notch (Image courtesy of Andrea Trescot, MD)

stimulation for confirmation and lesioning at 120–180 s at 42 °C for 2 cycles. Similarly, Brusselmans et al. [16] described three patients with persistent idiopathic facial pain who were treated with PRF of the maxillary (see Chap. 23), or mandibular nerve, or both, using two 45 V cycles for 2 min. Conventional RF and pulsed RF [17] are widely used for the treatment of trigeminal neuralgia at the trigeminal ganglion.

Chemical Neurolytic Procedures

Using the injection techniques above, chemical neurolytic procedures can be performed. Six percent phenol or absolute alcohol has been described [15, 18]. Wilkinson [19] described 60 injections of 6 % phenol in glycerol onto the peripheral trigeminal branches (including the mandibular nerve) to treat 18 patients with tic douloureux: 80 % noted total or marked relief for a median of 9 months (though 30 % had relief for 2 years). Trigeminal ganglion injections (Fig. 24.10) have also been used for injections of glycerol, conventional, and pulsed radiofrequency lesioning [20], and balloon compression [21].

Martos-Diaz et al. [22] described the injection of 100 units of botulinum toxin in 4 cc of saline guided by EMG and TMJ arthroscopy for LPM dystonia.

Percutaneous Procedures

Two percutaneous procedures for pain induced by the trigeminal nerve have been described: percutaneous radiofrequency trigeminal gangliolysis (PRTG) and percutaneous balloon microcompression (PBM).

Percutaneous Radiofrequency Trigeminal Gangliolysis (PRTG)

PRTG is an outpatient procedure performed by placing a needle into the gasserian ganglion, through which an electrical current passes, which heats the probe, producing a thermal lesion in the ganglion. Of the percutaneous procedures, it has the lowest reported rate of pain recurrence, with the average patient experiencing 3 years of excellent pain relief [23]. Pulsed radiofrequency on the trigeminal

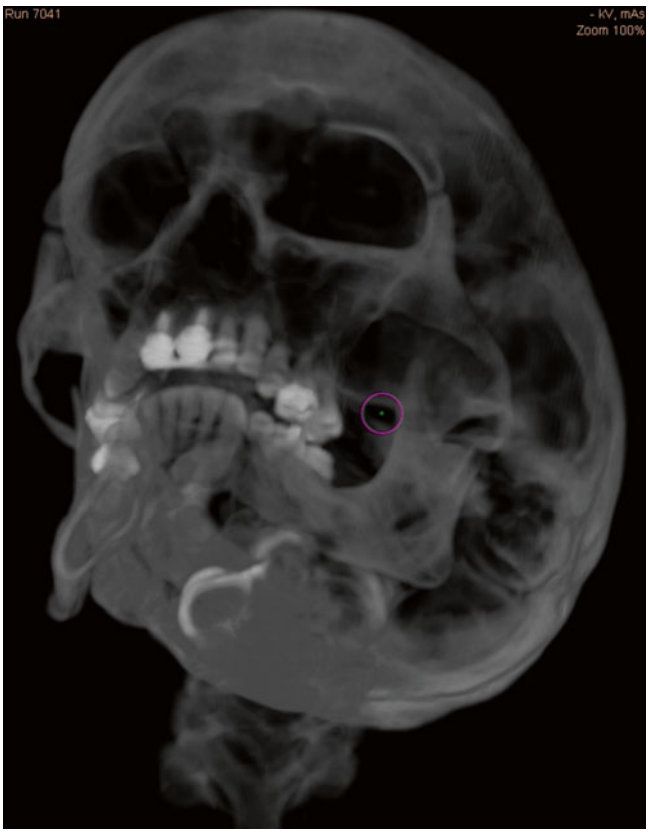

Fig. 24.12 Injection site for a submental approach to the trigeminal ganglion (Image courtesy of Andrea Trescot, MD)

ganglion appears promising for the treatment of trigeminal neuralgia [17].

Percutaneous Balloon Microcompression (PBM)

Mullan et al. first described balloon compression of the trigeminal ganglion in 1980 [24]. With PBM, the operator percutaneously inserts a balloon catheter through the foramen ovale into Meckel's cave and inflates it for 1–10 min. The patient is awake during the procedure, recovers quickly, and goes home the day of the procedure or the next day [21]. The major advantage of this technique is the possibility of treating first-branch (V1) trigeminal neuralgia while preserving corneal-eyelid reflex [25].

Surgery

Surgical treatments aimed to relieve pain specifically due to maxillary mandibular nerve entrapments are not widely performed. Surgical techniques for trigeminal nerve entrapment/compression are described below.

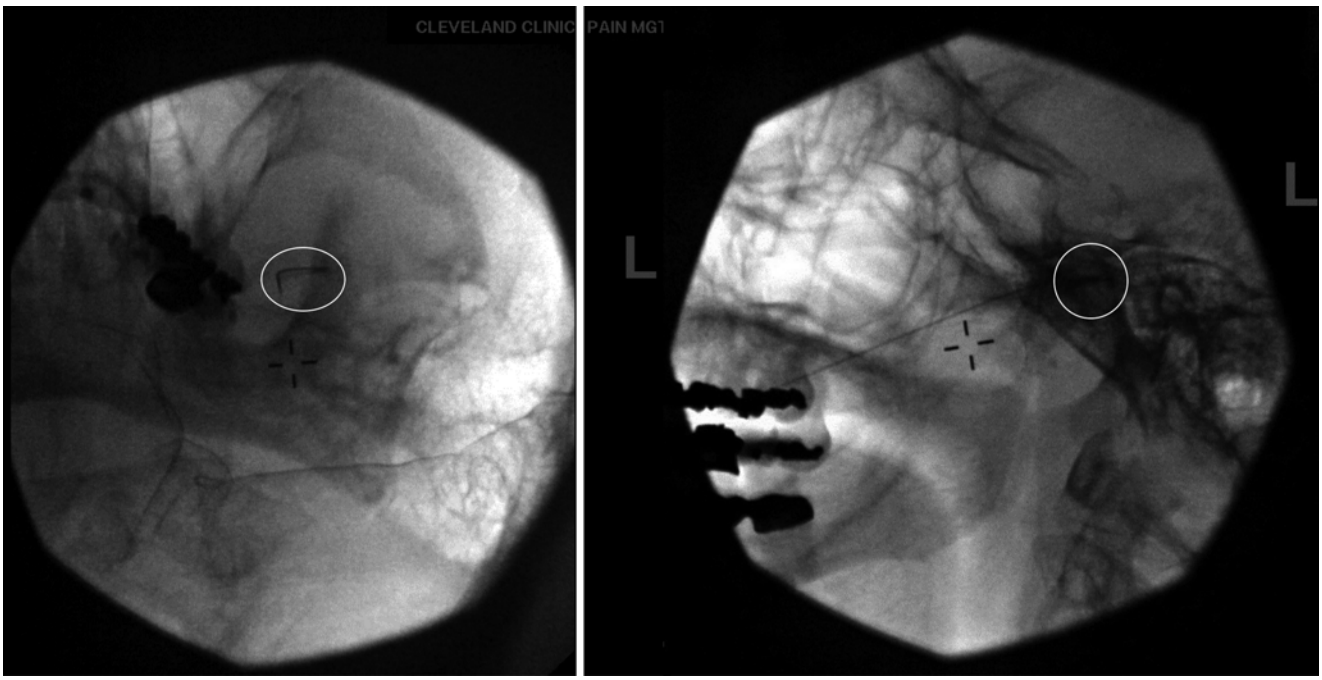

Fig. 24.13 Fluoroscopic mandibular nerve injection from a foramen ovale approach (Image courtesy of George Chang Chien, from Chang Chien et al. [13] with permission)

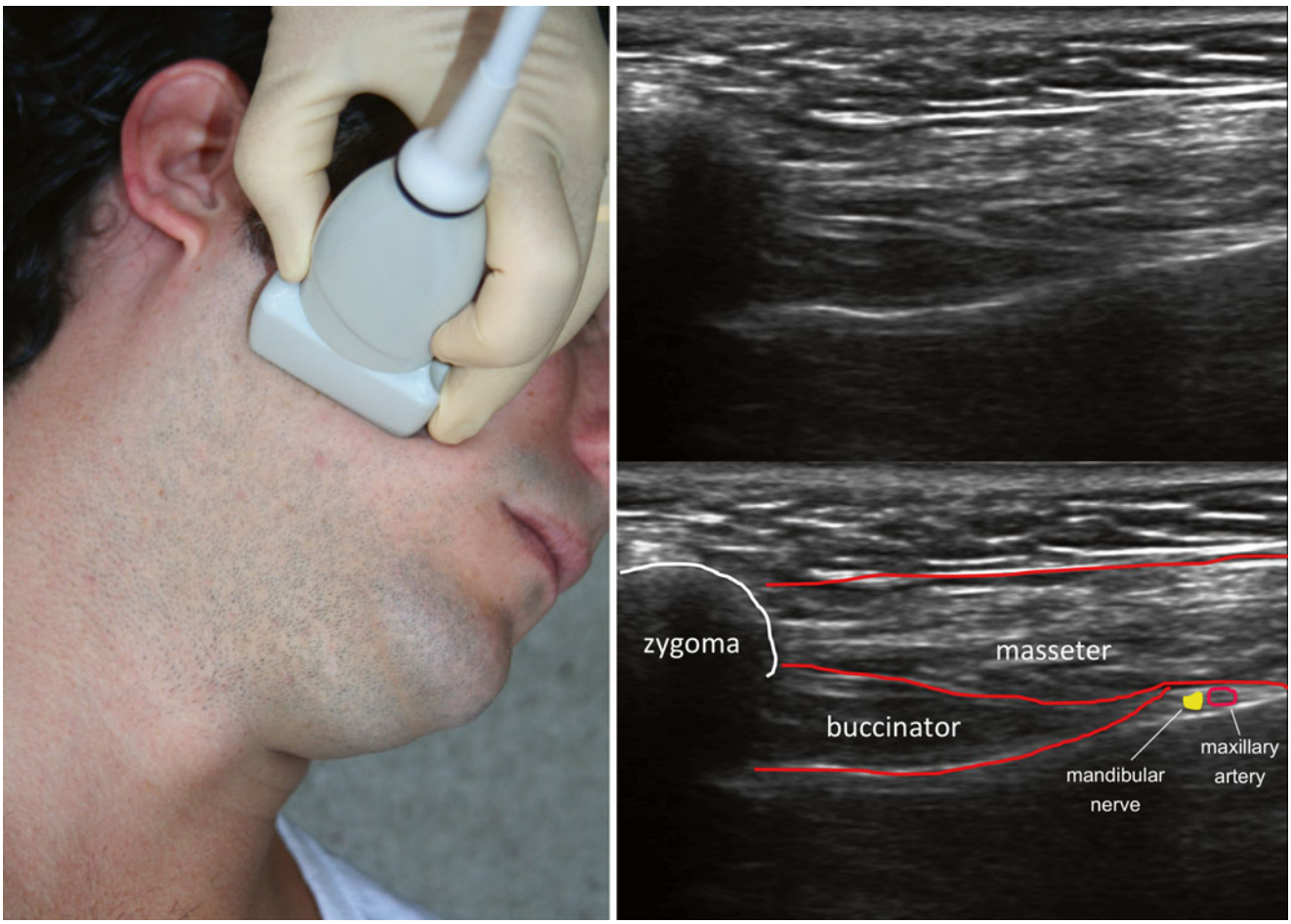

Fig. 24.14 Ultrasound evaluation of the mandibular nerve (Image courtesy of Andrea Trescot, MD)

Microvascular Decompression

Microvascular decompression is the surgical procedure for pain induced by entrapment of the trigeminal nerve by vasculature. It is normally performed under general anesthesia. The skin is incised behind the ear, and a 3 cm craniotomy performed. The dura is retracted to expose the trigeminal nerve, and the vascular elements compressing the nerve as it enters the pons are identified. Teflon felt is then used to pad the nerve away from the offending artery or vein. Large series have been published, and the initial efficacy is greater than 80 %. The recurrence rates, compared with those after other invasive treatments, are among the lowest (18 % pain recurrence in 25 years) [18].

Complications include chemical meningitis, ipsilateral hearing loss, and facial sensory loss or palsy. Mortality rates in experienced centers are less than 0.5 %. Serious morbidity (which includes dizziness, temporary facial palsy, cerebrospinal fluid leaks, meningitis, cerebellar stroke, and hearing loss) may occur in 1–5 % of patients [18].

Open Trigeminal Rhizotomy

An alternative procedure for the treatment of trigeminal neuralgia is open trigeminal rhizotomy or partial root section. In this procedure, the trigeminal nerve is partially cut just beyond the exit from the pons, causing some degree of permanent facial numbness. It is most effective for third-division trigeminal neuralgia.

Summary of Neurolytic and Surgical Techniques

Zakrzewska and Thomas [26] looked at 475 patients with trigeminal neuralgia; 145 underwent cryoneuroablation, 265 underwent radiofrequency lesioning, and 65 underwent microvascular decompression. These patients were followed for a mean of 45 months and then assessed by the clinician and by questionnaire. The cryoneuroablation patients had a reoccurrence of pain after a mean of 6 months, and the RF patients after 24 months, while only 38 % of the microvascular

decompression patients had reoccurrence at 5 years. There was one operative death in the RF group. The patients were all satisfied with their care, but the questionnaires showed a significant underreporting of pain by the clinicians.

Pollack et al. [27] reviewed the costs involved in treating 126 patients who underwent 153 surgeries for trigeminal neuralgia: 33 underwent microvascular decompression, 51 underwent glycerol injections, and 69 underwent stereotactic radiosurgery. The cost per quality adjusted pain-free year (QALY) was $6342, $8174, and $8269 for glycerol injections, microvascular decompression, and stereotactic radiosurgery, respectively. However, because of the longer pain-free interval and the fewer repeat surgeries, microvascular decompression is predicted to be the most cost-effective surgery.

Complications

As with all injections, there is a risk of bleeding and infection, as well as the risk of local anesthetic toxicity. If the needle is advanced too deep while walking posteriorly off the lateral pterygoid plate, the tip of the needle can puncture the pharynx. There is a very close relation of the mandibular nerve with the middle meningeal artery, thus making meticulous aspirations necessary. Hemorrhage in the cheek often occurs during and following the block via the extraoral route. Hematoma of the face and subscleral hematoma of the eye may occur.

There can be trauma to nerves in the region, particularly the branches of the mandibular nerve (inferior alveolar and lingual). Lingual nerve damage is more common than inferior alveolar nerve damage; when an inferior alveolar nerve block causes permanent nerve impairment, the lingual nerve is affected about 70 % of the time, while the inferior alveolar nerve is affected only 30 % of the time [28]. According to dissections performed by Pogrel et al. [28], the lingual nerve was showed to be unifascicular at the level of the inferior alveolar block site (which was multifascicular). Their conclusion was that there "is no known way to avoid the remote possibility of nerve damage resulting from an inferior alveolar nerve block."

There have been reports of immediate and delayed facial nerve palsy after inferior alveolar nerve injections, presumably because of the proximity to the facial nerve [29]. Ezirganli and Kazancioglu [30] described "anemia" after inferior alveolar injections, though the description is more consistent with blanching of the cutaneous vessels, rather than a general intravascular depletion of red blood cells.

Summary

The mandibular nerve is the V3 branch of the trigeminal ganglion, which innervates the lower face. The involvement of this nerve produces pain in the chin, inferior oral cavity, lower teeth, buccal tissues, and tongue as well as temple regions.

Usually, a careful history and physical will lead to proper diagnosis, and the injection and/or surgical treatment may result in improvement of symptoms.

References

1. Neto HS, Camilli JA, Marques MJ. Trigeminal neuralgia is caused by maxillary and mandibular nerve entrapment: greater incidence of right-sided facial symptoms is due to the foramen rotundum and foramen ovale being narrower on the right side of the cranium. Med Hypotheses. 2005;65(6):1179–82.
2. Speciali JG, Gonçalves DA. Auriculotemporal neuralgia. Curr Pain Head Rep. 2005;9:277–80.
3. Piagkou M, Demesticha T, Skandalakis P, Johnson EO. Functional anatomy of the mandibular nerve: consequences of nerve injury and entrapment. Clin Anat. 2011;24(2):143–50.
4. Schmidt BL, Pogrel MA, Necoechea M, Kearns G. The distribution of the auriculotemporal nerve around the temporomandibular joint. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1998;86(2):165–8.
5. Anil A, Peker T, Turgut HB, Gulekon IN, Liman F. Variations in the anatomy of the inferior alveolar nerve. Br J Oral Maxillofac Surg. 2003;41(4):236–9.
6. Costen JB. A syndrome of ear and sinus symptoms dependent upon disturbed function of the temporomandibular joint. Ann Otol Rhinol Laryngol. 1934;43:1–15.
7. Cascone P, Fatone FM, Paparo F, Arangio P, Iannetti G. Trigeminal impingement syndrome: the relationship between atypical trigeminal symptoms and anteromedial disk displacement. Cranio. 2010;28(3):177–80.
8. Valeriy L. Doctor's approach in case of mandibular injury with dental implant. http://www.alpha-dent.net/en/doctors-approach-case-mandibular-nerve-injury-dental-implant. Accessed 7/1/15.
9. Fukuda K, Ichinohe T, Kaneko Y. Pain management for nerve injury following dental implant surgery at Tokyo Dental College Hospital. Int J Dent. 2012;2012:209.
10. Misch CE, Resnik R. Mandibular nerve neurosensory impairment after dental implant surgery: management and protocol. Implant Dent. 2010;19(5):378–86.
11. Gatot A, Tovi F. Prednisone treatment for injury and compression of inferior alveolar nerve: report of a case of anesthesia following endodontic overfilling. Oral Surg Oral Med Oral Pathol. 1986;62(6):704–6.
12. Day M. Trigeminal ganglion block. In: Manchikanti L, Singh V, editors. Interventional techniques in chronic nonspinal pain. Paducah: ASIPP Publishing; 2007. p. 213–20.
13. Chang Chien GC, Chin SL, Cheng J. Mandibular nerve block for painful temporomandibular disorder: a case series. AAPM Annual Meeting; National Harbor, MD2015.
14. Waldman SD. Ultrasound-guided mandibular nerve block. In: Waldman SD, editor. Comprehensive atlas of ultrasound-guided pain management injection techniques. Philadelphia: Lippincott, Williams, and Wilkins; 2014. p. 52–60.
15. Erdine S, Racz GB, Noe CE. Somatic blocks of the head and neck. In: Raj PP, Lou L, Erdine S, Staats PS, Waldman SD, Racz GB, et al., editors. Interventional pain management: image-guided procedures. 2nd ed. Philadelphia: Saunders-Elsevier; 2008. p. 77–108.
16. Brusselmans G, Crombez E, Devulder J. Percutaneous pulsed radiofrequency treatment of atypical facial pain: a study of 3 cases. Eur J Pain. 2011;5(S1):100.
17. Van Zundert J, Brabant S, Van de Kelft E, Vercruyssen A, Van Buyten JP. Pulsed radiofrequency treatment of the Gasserian

ganglion in patients with idiopathic trigeminal neuralgia. Pain. 2003;104(3):449–52.
18. Olson S, Atkinson L, Weidmann M. Microvascular decompression for trigeminal neuralgia: recurrences and complications. J Clin Neurosci: Off J Neurosurg Soc Aust. 2005;12(7):787–9.
19. Wilkinson HA. Trigeminal nerve peripheral branch phenol/glycerol injections for tic douloureux. J Neurosurg. 1999;90(5): 828–32.
20. Teixeira MJ, Siqueira SR, Almeida GM. Percutaneous radiofrequency rhizotomy and neurovascular decompression of the trigeminal nerve for the treatment of facial pain. Arq Neuropsiquiatr. 2006;64(4):983–9.
21. Broggi G. Percutaneous retrogasserian balloon compression for trigeminal neuralgia. World Neurosurg. 2013;79(2):269–70.
22. Martos-Diaz P, Rodriguez-Campo FJ, Bances-del Castillo R, Altura-Guillen O, Cho-Lee GY, Mancha-de la-Plata M, et al. Lateral pterygoid muscle dystonia. A new technique for treatment with botulinum toxin guided by electromyography and arthroscopy. Med Oral Patol Oral Cir Bucal. 2011;16(1):e96–9.
23. Burchiel KJ, Steege TD, Howe JF, Loeser JD. Comparison of percutaneous radiofrequency gangliolysis and microvascular decompression for the surgical management of tic douloureux. Neurosurgery. 1981;9(2):111–9.
24. Mullan S, Duda EE, Patronas NJ. Some examples of balloon technology in neurosurgery. J Neurosurg. 1980;52(3):321–9.
25. Lichtor T, Mullan JF. A 10-year follow-up review of percutaneous microcompression of the trigeminal ganglion. J Neurosurg. 1990;72(1):49–54.
26. Zakrzewska JM, Thomas DG. Patient's assessment of outcome after three surgical procedures for the management of trigeminal neuralgia. Acta Neurochir (Wien). 1993;122(3–4):225–30.
27. Pollock BE, Ecker RD. A prospective cost-effectiveness study of trigeminal neuralgia surgery. Clin J Pain. 2005;21(4):317–22.
28. Pogrel MA, Schmidt BL, Sambajon V, Jordan RC. Lingual nerve damage due to inferior alveolar nerve blocks: a possible explanation. J Am Dent Assoc. 2003;134(2):195–9.
29. Tzermpos FH, Cocos A, Kleftogiannis M, Zarakas M, Iatrou I. Transient delayed facial nerve palsy after inferior alveolar nerve block anesthesia. Anesth Prog. 2012;59(1):22–7.
30. Ezirganli S, Kazancioglu HO. An unforeseen complication arising from inferior alveolar nerve block: is anemia possible? J Craniofac Surg. 2013;24(6):2178–9.

Mental Nerve Entrapment

25

Sydney E. Rose and Amitabh Gulati

Introduction

The mental nerve (MN) arises from the sensory portion of the *inferior alveolar nerve* (IAN) and is a terminal branch of the *mandibular division* (V3) of the trigeminal nerve (CN V). It is one of the several trigeminal nerve entrapments that can cause facial pain. The MN is a somatic sensory nerve that is susceptible to injury and compression as it exits through the mental foramen. *Mental nerve neuropathy* (MNN), also known as *numb chin syndrome* (NCS) is a sensory neuropathy characterized by numbness and/or paresthesias in the region of the lower lip and chin. A seemingly benign presentation, numb chin syndrome is easy to overlook. Yet, NCS is an important clinical presentation to recognize, particularly because non-dental or orofacial associated MNNs have unfortunately been associated with an array of malignancies, and more often than not MNN is a harbinger for diagnosis of advanced metastatic disease. The presentation of NCS should never be taken lightly. Any patient presenting with numbness or dysesthesias in the region of the lower mouth, unrelated to any obvious dental or orofacial causes, should urgently undergo a thorough malignancy work-up.

Clinical Presentation (Table 25.1)

Charles Bell was the first person to describe MNN. In 1830, in his monograph: *The Nervous System of the Human Body*, Bell described his patient, an elderly woman with advanced breast cancer, who complained of left-sided numbness in her lower lip. She was found to have a bony metastasis in her left mandible that was compressing her left IAN [22]. Over a century later, Roger and Pallais described a patient with sim-

Table 25.1 Occupation/exercise/trauma history relevant to mental nerve entrapment

Trauma	Dental trauma [1]
	Dental implants [2]
	Facial trauma
	Blow to the face
	Chin augmentation [3]
Infection	Periapical infection [4]
Mental foraminal stenosis	Paget's disease [5]
Neuropathy	HIV [6]
	Diabetes mellitus
	Sickle cell anemia [7, 8]
	Amyloidosis
	Multiple sclerosis
	Anemia
	Syphilis
	Vasculitis [9]
	Sarcoidosis
	Trigeminal neuralgia [10]
	Bisphosphonates [11]
	Postherpetic neuralgia [12]
Neoplasm	Metastatic cancer
	Lymphoma [6, 13, 14]
	Large cell lung cancer [15]
	Leukemia [16]
	Prostate [17]
	Squamous cell [18]
	Multiple myeloma [19–21]
	Primary oral cancer

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ilar symptoms to Bell's, except that her mental neuropathy was the initial sign of an undiagnosed cancer [23]. Calverley and Mohnac coined the phrase "numb chin syndrome" in 1963 when they published on five patients with metastatic cancer who had numbness and tingling of the lower lip and chin [24].

MNN has multiple etiologies (Table 25.1) and clinically presents with numbness or other abnormal sensation (tingling, burning, pins, and needles) localized to a discrete area of the lower maxillary/mandibular region of the face (Fig. 25.1). Patients often describe having the feeling of a swollen lip – similar to the sensation in one's mouth after undergoing dental anesthesia. Loss of sensitivity often results in biting of the lower lip, skin breakdown, and even scars. Pain is not typically a feature of MNN [15, 25, 26]. Patients may also report feeling a swelling or lump in the lower mouth. The majority of patients with NCS report symptoms to be unilateral; however, bilateral symptoms have also been reported [26]. The initial presentation of patients with MNN is often to a nearby urgent care center or emergency room [15]. In addition to orofacial conditions, MNN has also been associated with a plethora of systemic diseases including HIV, diabetes mellitus, sickle cell anemia, amyloidosis, and multiple sclerosis (see Table 25.1). Since MNN is so easily overlooked, it is imperative that internists and emergency clinicians are trained to recognize MNN as a sentinel symptom of a potentially life-threatening condition and urgently refer the patient for the appropriate management.

Fig. 25.1 Pattern of pain from mental neuralgia (Image courtesy of Andrea Trescot, MD)

Anatomy (Table 25.2)

The MN is a terminal sensory branch of the *trigeminal nerve*. It innervates the gingiva of the incisor region, the mucosa and skin of the lower lip and jaw, and the skin of the chin (Fig. 25.2) [27]. Numerous anatomical variants, including multiples, deviance of location, and course, have all been reported. Anatomical variations can put patients at increased risk for damage during any sort of anesthetic block, dental procedure, or surgical work in the region (Fig. 25.3) [28–30]. Normal anatomy is discussed below. Note that this includes having two mental nerves, one supplying each side of the face. The relay of sensory information begins with sensory stimuli to the skin of the chin or lower lip or in the mucous membranes of the lower lip. Sensations are broadcasted as electrical signals along an ascending neuronal pathway to the cortex of the brain, where they are ultimately processed and interpreted cognitively [27]. For clarity, as well as consistency with other anatomic descriptions in this book, relevant anatomy will be discussed beginning in the base of the skull and followed peripherally.

The *trigeminal ganglion*, also known as the *semilunar ganglion*, is a collection of first-order sensory cell bodies of CN V. The ganglion is located on the anterior side of the base of the skull, in a depression of the petrous portion of the temporal bone known as *Meckel's cave*. Three divisions: *ophthalmic* (V1), *maxillary* (V2, a sensory portion only) (see Chap. 23), and *mandibular* (V3) (see Chap. 24) emerge from this ganglion, each exiting the cranium via a unique foramen [31, 32]. The mental nerve is a terminal branch of the man-

Table 25.2 Mental nerve anatomy

Origin	Trigeminal ganglion
General route	Mandibular nerve, foramen ovale, posterior trunk, inferior alveolar nerve, mandibular foramen, internal surface of mandible, mandibular canal, mental foramen, mental nerve
Sensory distribution	Gingiva of incisor, mucosa of skin of lower lip and jaw, skin of chin
Motor innervation	None
Anatomic variability	Multiple branches, location of mental foramen
Other relevant structures	Mandible, mental foramen, depressor anguli oris muscle

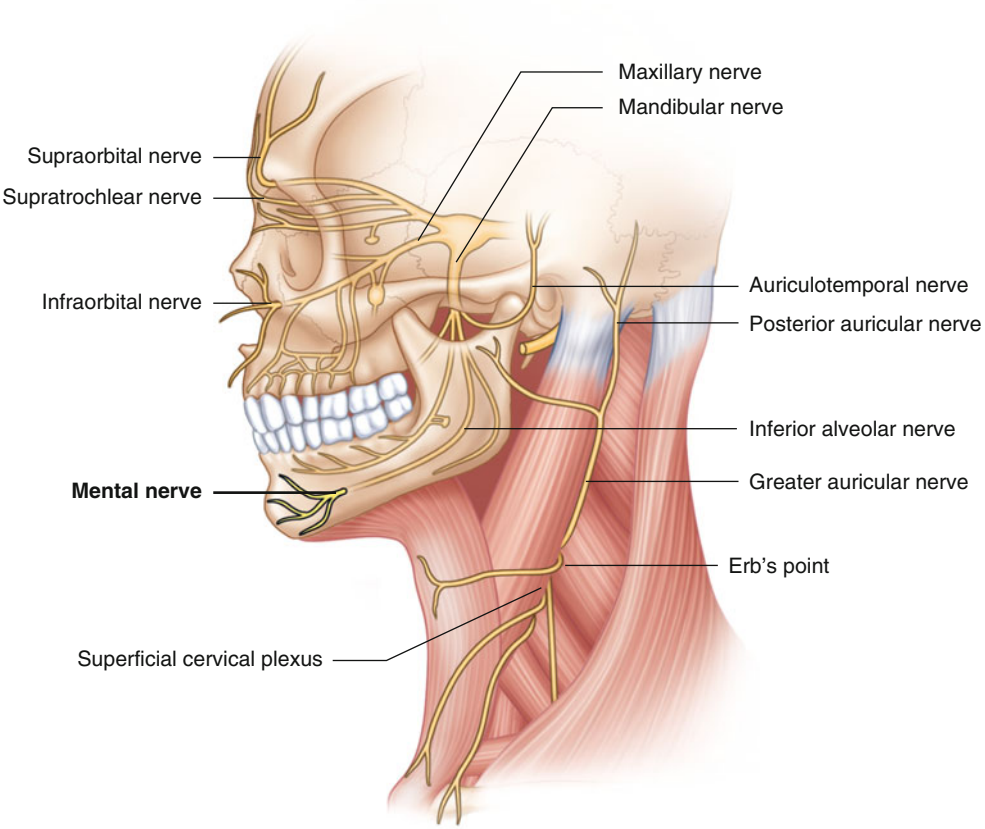

Fig. 25.2 Mental nerve anatomy (Image courtesy of Springer)

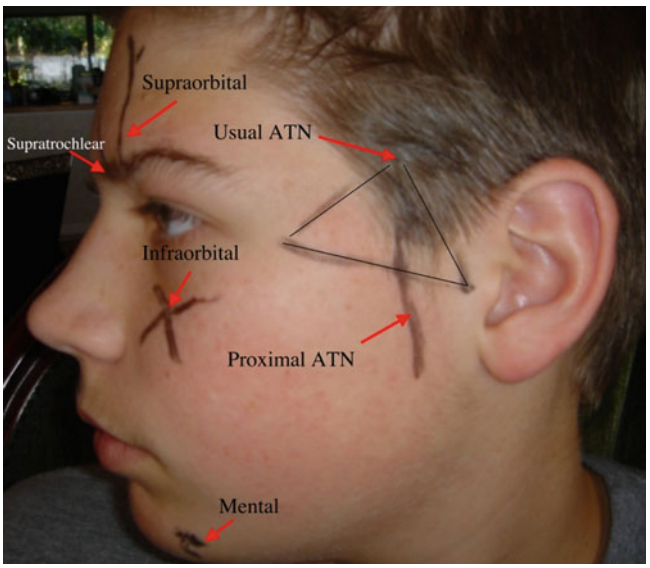

Fig. 25.3 Surface landmarks of the nerves of the face (Image courtesy of Andrea Trescot, MD)

dibular nerve, and thus our focus will be narrowed to V3, as detailed below (Figs. 25.4 and 25.5).

The mandibular nerve is the largest division of CN V. It exits the bottom of the skull through the foramen ovale into the infratemporal fossa. Here, it immediately sends off two branches on its medial side: *nervous spinous* and the nerve to the medial pterygoid muscle, before dividing further into anterior and posterior trunks. The small anterior trunk of V3 primarily carries V3 motor fibers, while the larger posterior trunk consists mostly of V3 sensory fibers [33–35].

At the superior border of the *lateral pterygoid muscle*, the posterior trunk of V3 gives rise to three branches: the *auriculotemporal nerve* (see Chap. 15), the *inferior alveolar nerve* (IAN), and the *lingual nerve*. The auriculotemporal nerve departs from the dorsal aspect of the trunk and loops rostrally. Starting between the lateral pterygoid muscle and the *tensor veli palatini*, the lingual and IAN nerves descend caudally and anteriorly in the *interpterygoid fascia* between the *medial pterygoid muscle* and the ramus of the mandible, in a space known as the *pterygomandibular space* [33, 36]. The IAN runs posterior and lateral to the lingual nerve. As it courses between the *sphenomandibular* ligament and ramus of the mandible, it sends off the mylohyoid nerve (supplying motor innervation to the *mylohyoid muscle* and the anterior belly of the *digastric muscle*) prior to entering the *mandibular foramen*. Once it crosses through the foramen, the IAN travels anteriorly in the mandibular canal along the interior surface of the mandible and below the apices of the lower teeth. At the *mental foramen*, located at approximately the second premolar tooth, the IAN forks into two terminal

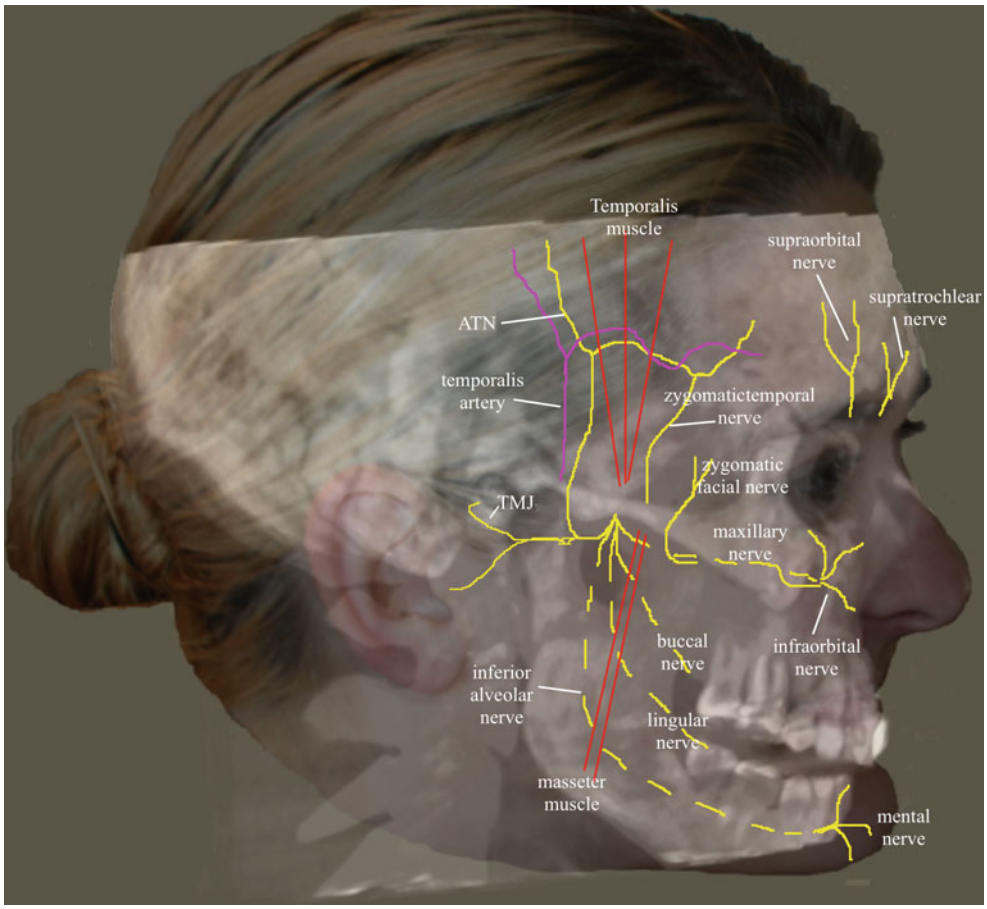

Fig. 25.4 Nerves of the face (Image courtesy of Andrea Trescot, MD)

Fig. 25.5 Sensory areas of the trigeminal and cervical nerve branches: *A* supraorbital nerve, *B* infraorbital nerve, *C* mental nerve, *D* buccal nerve, *E* lacrimal nerve, *F* auriculotemporal nerve, *G* superficial cervical plexus, *H* posterior auricular nerve/great auricular nerve, *I* occipital nerve (Image courtesy of Terri Dallas-Prunskis, MD)

branches [34]. The mental branch pierces through the mental foramen, while the lateral branch continues on in the interior of the mandible as the *incisive nerve* [35].

The MN emerges from the mental foramen on the outer surface of the mandible deep to the *depressor labii inferioris muscle*. It courses superficially toward the skin. Behind the *depressor anguli oris muscle*, the MN ramifies into three sensory input collecting branches, which continue peripherally to the mucosa and skin. One branch gathers information from sensory receptors in the skin of the chin, while the remaining two collect and relay sensory information from receptors located in the mucous membrane and the skin of the lower lip [2, 37, 38].

The oral-maxillofacial region has the highest density of sensory receptors per body surface area and thus presents immeasurable opportunities for nerve injury [27, 32]. A detailed knowledge of local anatomy is essential to analyzing and manipulating any facial tissue. The mental foramen is a strategic marker for dental and OMF surgeons to avoid mental nerve injury. Smith and colleagues analyzed the location of the mental foramen by dissecting cadavers and measuring the palpable anatomical landmarks of the face. They identified that the mental foramen was 64.2 ± 6.4 mm medial to the angle of the mandible and 12.9 ± 1.6 mm superior to the inferior border of the mandible [39]. Agthong and his team looked at mental foramina in reference to soft tissue cadaver landmarks such as the *cheilion* (corner of the mouth) and showed that, on average, the mental foramen was 21.83 ± 3.26 mm inferior and 5.56 ± 3.37 mm medial to the cheilion. Additionally, they noted direct distance between the cheilion and mental foramen was 22.74 ± 2.96 mm [40]. Just like the nerve, several anatomic variations of the mental foramen have been discovered. Variances have been found in location, size, redundancy, and even path of mental nerve emergence [41–47]. Differences have also been documented between age, gender, and ethnicity groups [7, 48–50]. The mental foramen is an important landmark when it comes to analyzing mental nerve entrapment, regardless of whether it is due to iatrogenic or pathologic causes. Data on its position can help clinicians avoid, diagnose, and treat mental nerve injuries [2, 51].

The accessory mental nerve and foramen have more recently been recognized and identified as a potential cause of unexpected mental nerve trauma [29, 52]. A literature review suggested that the incidence of accessory mental foramen (AMF) was 2–13 % of cadavers evaluated [52]. The authors also suggested that surgeons who were planning a procedure that would involve "exfoliation" of the periosteum of the anterior mandible should first obtain a CT of the jaw; if there seemed to be a difference in the size of the left vs right mental foramen, this could indicate the presence of an AMF.

Pathophysiology

Paresthesias are symptoms of burning, numbness, or prickling in an area on the body due to nerve injury. Etiology may be local or systemic, and the most common mechanisms of nerve injury are mechanical, thermal, or chemical modalities [4]. Mechanical injury may involve over-stretching, compressing, partially or totally resecting the nerve or even lacerating it. Focal mechanical causes of injury include trauma and compression. This can be spontaneous or iatrogenic and can include mandibular fractures, oral surgery, dental work (extractions, endodontic overfilling, and implants), foreign bodies, tumor expansion, impacted teeth, and infections (osteomyelitis, periapical, and peri-implant infections) [1].

Chemical damage to the mental nerve can occur locally from toxic endodontic filling materials such as paraformaldehyde, corticosteroids, and eugenol. Dental irrigation chemicals like sodium hypochlorite are also culprits [53]. Lastly, chemical injury can be caused internally by the body's own local inflammatory markers and cytokines released by cells fighting infection [54].

Thermal damage to the MN is rare. The main thermal source of MNN comes from oral surgeons using specific mechanical handpieces for too long in the same spot, causing the bone to overheat [55].

The pathophysiology of MNN caused by distant malignancies is not well understood. Several mechanisms for the symptoms have been proposed. Some even believe that the etiology is not determinable in the majority of cases [56].

Lesions potentially causing NCS have been found both peripherally and centrally. Peripherally, metastatic lesions to the mandible causing direct compression of either the mental or inferior alveolar nerve are thought to be the primary culprit causing MNN [22]. Perineural invasion of the mental nerve is another common finding in malignant MNN [10, 55, 57]. Cancers typically responsible for these lesions are breast, prostate, lung, kidney, stomach, and thyroid cancer. Centrally, lymphatic or hematologic spread to the CNS, paraneoplastic syndrome, leptomeningeal seeding, and bony metastases to the interior base of the skull have also been implicated as culprits causing MNN [13, 55]. It is important to note that the histology and origin of the primary tumor most likely influence the pathogenesis of MNN [55].

Physical Exam

The single most important step in the diagnosis of mental nerve neuropathy is recognizing the potential clinical significance of chin or lip numbness [58]. A comprehensive history and physical exam should be conducted on any patient complaining of abnormal sensations in the lower lip, gums, or chin, especially if the sensations are unilateral. A

clinical exam should be done intraorally and extraorally, and examination of the nostrils, gums, tongue, and inner cheeks should not be overlooked. Tenderness over the mental foramen can be examined extraorally (Fig. 25.6) (Video 25.1) or intraorally (Fig. 25.7). Distribution of sensory dysfunction should be tested with light touch, pain, and temperature stimuli. Motor function should be assessed and can be evaluated by asking the patient to make chewing movements and at the same time noting any asymmetry of the lower jaw [4]. Additional mechanical, electrical, thermal, or chemical tests can be carried out as needed to elicit specific subjective responses.

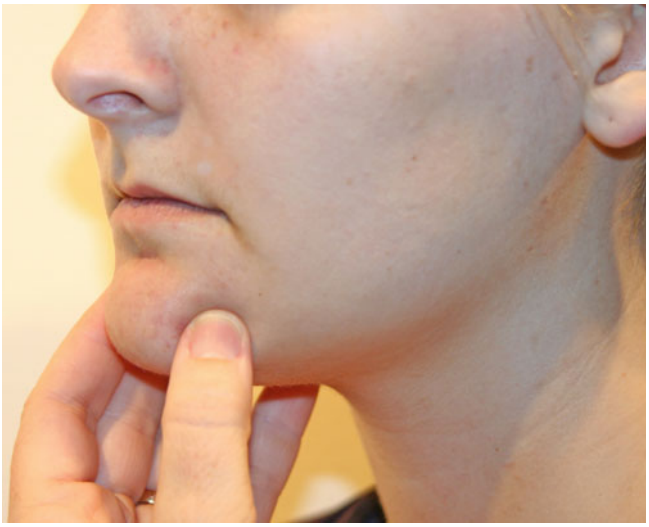

Fig. 25.6 Extraoral mental nerve exam (Image courtesy of Andrea Trescot, MD

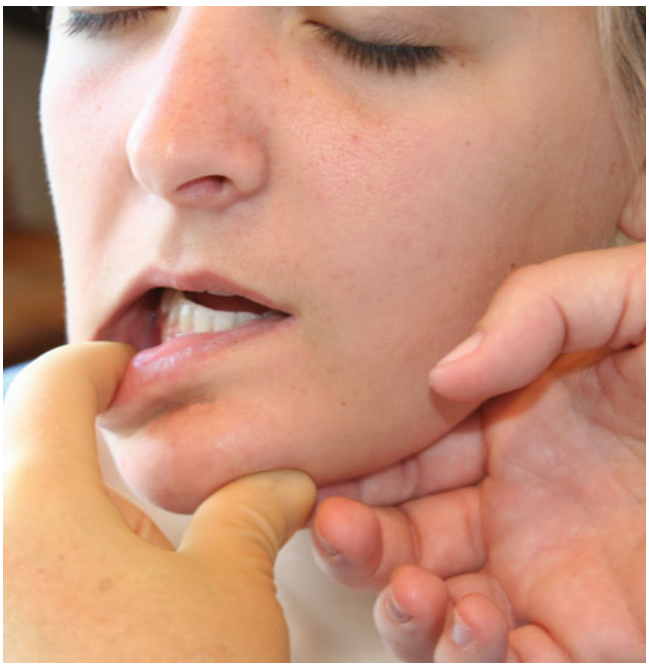

Fig. 25.7 Intraoral mental nerve exam (Image courtesy of Andrea Trescot, MD)

Cancer must be considered in any patient in whom maxillofacial trauma (spontaneous or iatrogenic), other obvious dental process, or osseous disease as the cause of nerve damage can be excluded [59]. These patients need thorough oncologic work-ups and should be immediately referred to an appropriate specialist.

Since sensory abnormalities around the chin and lower jaw can be caused by lesions located peripherally or centrally along the trigeminal nerve path, investigation should not only be focused on local symptoms but rather cover the entire course [60]. Typically, focal sensory disturbances of only the lower mouth and chin suggest a peripheral lesion, such as a bony mandibular metastasis compressing the mental or inferior alveolar nerve. Sensory loss encompassing a broader area, however, or even signs of motor involvement (jaw deviation, loss of muscle tone in the lower mouth, or ipsilateral temporalis muscle wasting), may signal a more proximal V3 lesion [61].

Differential Diagnosis (Table 25.3)

The differential diagnosis of mental nerve neuropathy is vast, and various etiologies have been reported [6, 8, 9, 26]. Evaluation of MNN begins with a detailed history of the sensory alteration and its evolution. The majority of cases are due to local pathologic and iatrogenic causes [59]. Patients with a history of dental work requiring intraoral blocks at the mental foramen, trauma to the jaw, third molar extractions, lower jaw root canal therapy, or surgery manipulating the lower jaw are at increased risk for MNN [62]. However, neoplastic disease is more frequently described in association with NCS than any other systemic disease, and it should be ruled out first.

Table 25.3 Differential diagnosis of "numb chin syndrome" and chin pain

	Potential distinguishing features
Dental pathology	X-rays, tomogram; physical exam shows tenderness over the lower incisors
Jaw tumor or cyst	X-rays, CT
Mandibular fracture	High-resolution CT – nonanatomic linear lucencies
Vasculitis [9]	ESR and CRP elevated
Osteonecrosis of the jaw [11]	X-rays, CT scan
Sickle cell disease [8]	Sickle cells on blood smear
Metastatic cancer	X-ray, CT scan, MRI depending on lesion location

Clinical exams are usually insufficient for identifying an etiology of NCS, and thus complete radiographic imaging of the trigeminal nerve is warranted [63]. Panoramic jaw radiography, computerized tomographic (CT) scanning, magnetic resonance imaging (MRI), and nuclear bone scintigraphy can all be useful in obtaining diagnosis. Since most malignant MNNs are caused by metastases to the mandible, panoramic radiography of the jaw is a good start. Osteoblastic lesions or osteolytic defects have been found on radiography at pretty much every location along the mandible and many times in the region of the mental foramen. Any abnormal results should be followed up with a bone scan (nuclear bone scintigraphy) and a biopsy of the lesion, as necessary [26, 64].

CT imaging of the brain and skull base may reveal mass lesions, parenchymal brain metastases, or leptomeningeal invasion of V3 near Meckel's cave [14, 64]. Additionally, multiplane CT imaging of the mandible may also be useful, since it is more sensitive than panoramic radiography for osteoblastic and osteolytic lesions, osteomyelitis, and tumor invasion of the inferior alveolar nerve [65].

MRI with gadolinium is another imaging modality that may be helpful in evaluating a patient for a central process, especially if radiographs and CT show no abnormalities or are inconclusive [64, 65]. Some recommend scanning the mandible and inferior alveolar nerve as well [55, 64].

If the above imaging studies fail to reveal a lesion, lumbar puncture and cytological analysis of CSF may be indicated to look for signs of infectious or carcinomatous meningitis due to leptomeningeal metastases, both of which have been documented as NCS etiologies [26]. Electrophysiologic studies are not routinely done for trigeminal nerve evaluation. Additionally, sensory nerve conduction studies have been described but are not sensitive and have limited use in clinical practice. See Table 25.4.

Table 25.4 Diagnostic tests for mental nerve

	Potential distinguishing features
Physical exam	Unilateral anesthesia of the chin, lower lip, and gingiva
Diagnostic injection	Mental nerve block with resolution of dysesthesia
Ultrasound	Diminished echogenicity, increased diameter (edematous), and CSA
MRI with contrast	Surrounding edema, soft tissue lesions, and nerve invasion
Arteriography	Not routine
CT	Bone destruction/reabsorption of mandible
Nuclear bone scintigraphy	Bone metastases and increased radioactive dye uptake
Electrodiagnostic studies	Not reliable

Identification and Treatment of Contributing Factors

Remodeling of the mandible after dental extractions (Fig. 25.8) or boney overgrowth in conditions such as Paget's disease can entrap the mental nerve. Mental nerve neuropathy has been described in a multitude of malignancies. Frequently the sign of cancer recurrence and a marker of metastatic disease, non-orofacial MNN is also commonly the initial presentation of cancer. In fact, Massey and colleagues found that in 47 % of their patients with MNN secondary to systemic cancer, numb chin syndrome preceded the diagnosis of the primary tumor [15, 56]. Unfortunately, when MNN is the initial presentation, it typically is a harbinger for advanced disease [64].

The most commonly associated primary malignancy with MNN is breast cancer. There is no sex predilection of NCS, per se; the occurrence of such is related to the sex distribution of the underlining malignancies. Thus, incidence of MNN is higher in women, as more women than men have breast cancer [66]. Lymphoproliferative malignancies are also frequently diagnosed in NCS; in fact, acute lymphoblastic leukemia accounts for a significant number of cases in children [6, 16]. Some of the multiple malignancies that have been associated with NCS include prostate, lung, kidney, stomach, thyroid, ovarian, and renal cell cancers, melanomas, sarcomas, and multiple myeloma [15, 17–21, 57, 61].

The mean age of MNN secondary to a systemic cancer diagnosis is approximately 48 years. Symptoms are more likely to be unilateral (85 %) than bilateral (15 %). Numbness

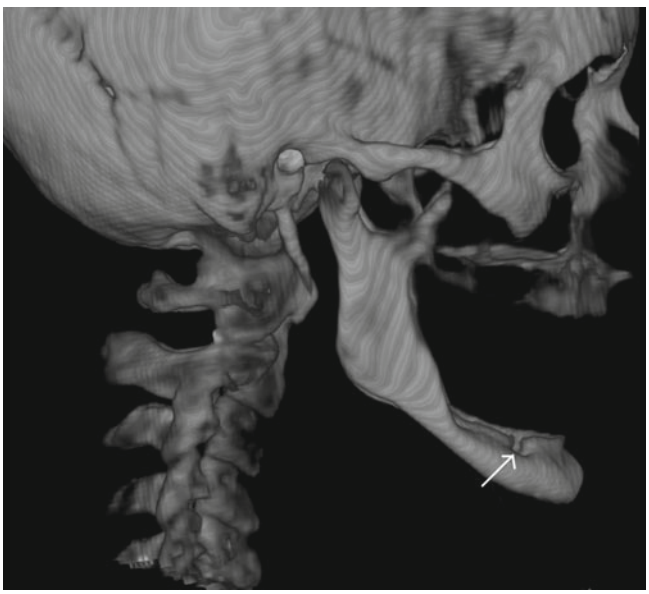

Fig. 25.8 Remodeling of mandible and mental foramen in an edentulous patient. *Arrow* shows mental foramen partially unroofed, exposing mental nerve to direct trauma (Image courtesy of Andrea Trescot, MD)

of the lower lip and chin are the most frequent complaints, while "pain" is reported much less frequently [66, 67].

Several nonmalignant systemic diseases have also been implicated as a cause of MNN. They include demyelinating conditions (e.g., multiple sclerosis), diabetes mellitus, amyloidosis, HIV, sickle cell anemia, syphilis, vasculitis, and sarcoidosis [6, 8, 15, 26, 62, 64].

It cannot be emphasized how important it is to recognize the seemingly benign complaint of lower lip or chin numbness as a potential sentinel signal of malignancy. A detailed history and physical exam may aid in distinguishing an etiology clinically; however, imaging studies play an important role in identifying the etiology of NCS.

The differential diagnosis of MNN is so broad that the first step in treatment is identifying an underlying cause. Management is then tailored to the underlying disease and pathophysiologic mechanisms causing NCS.

For example, in cases of orofacial or dental trauma, treatment may entail removing an implant, removal of surgical scar tissue, draining an abscess, or oral antibiotics. Sickle cell disease patients will sometimes experience transient MNN; treatment is similar to that of managing of an acute sickle cell crisis, such as hydration and systemic opioids for pain [7].

When MNN is due to cancer, management is directed mainly at the primary malignancy. Unfortunately, the finding of NCS in cancer patients is usually associated with advanced metastatic disease, and treatment is palliative. Some patients with skull base metastases have shown regression of their symptoms with local radiation. Others with leptomeningeal lesions have showed improvement with either whole brain irradiation or intrathecal chemotherapy [55]. Prognosis of patients with NCS in the setting of malignancy is extremely grim. Mandibular bone metastases seem to be associated with more aggressive disease, as median survival for these patients following NCS diagnosis is under 5 months. Most patients with leptomeningeal metastases survive for about 12 months following diagnosis [62, 66].

Injection Technique

Landmark-Guided Technique

The patient is placed in a supine or sitting position, with the head supported. The mental nerve can be injected percutaneously or intraorally. For the percutaneous injection, the mental foramen is palpated with the index and middle finger of the examining hand. The skin is cleaned and prepped in sterile fashion with alcohol. With the target identified, a 25 or 27 gauge 1/2 inch needle is inserted at a 90° angle to the skin until bony contact is made (Fig. 25.9) (Video 25.2). Care is taken during needle insertion to not insert the needle tip into the mental foramen, as this can precipitate paresthesias along the distribution of the mental nerve. Injections can also be done intraorally, which avoids cosmetic issues (Fig. 25.10). Following negative aspiration, a volume of 0.5–1 mL of local anesthetic with or without a deposteroid solution is injected. Some authors recommend using larger volumes of injectate, such as 3–4 mL; however, Trescot [68] recommends smaller volumes to avoid the potential increase in entrapment from the injectate. This procedure can be repeated; with recurrence of pain, other longer-lasting procedures such as radiofrequency thermocoagulation, pulsed radiofrequency, cryoneurolysis, or peripheral field stimulation can be performed (see below).

Fluoroscopic-Guided Technique

The use of fluoroscopy can be implemented for better visualization of the mental foramen. The C-arm should be moved in a cephalad rotation until the infraorbital foramen is viewed

Fig. 25.9 Percutaneous (extraoral) mental nerve injection (Image courtesy of Andrea Trescot, MD)

Fig. 25.10 Intraoral mental nerve injection (Image courtesy of Andrea Trescot, MD)

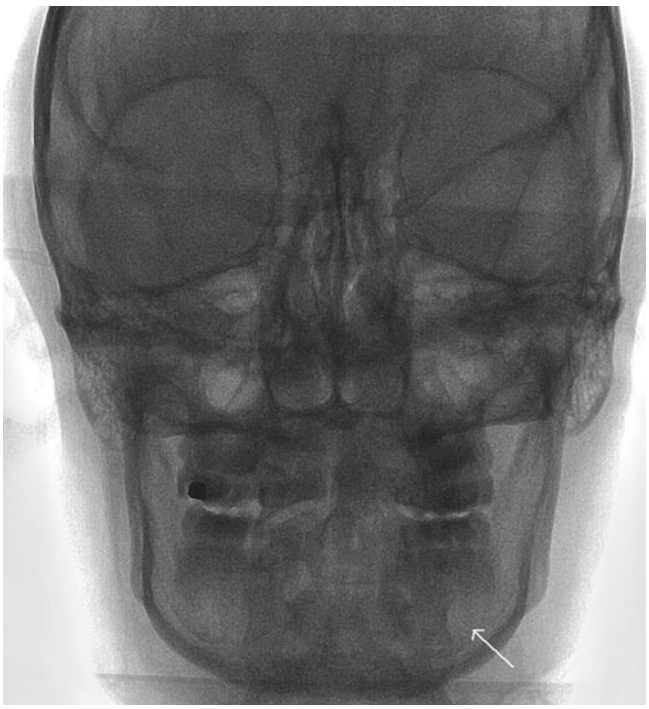

Fig. 25.11 Fluoroscopic image of the mental foramen at the *arrow* (Image courtesy of Andrea Trescot, MD)

(Fig. 25.11). Once the foramen is identified, the procedure is performed in the same fashion as the blind technique, with needle placement just inferior to the infraorbital foramen.

The mental nerve, as a branch of the trigeminal nerve, can be also injected at the mandibular nerve (see Chap. 24) and the foramen ovale (Fig. 25.12).

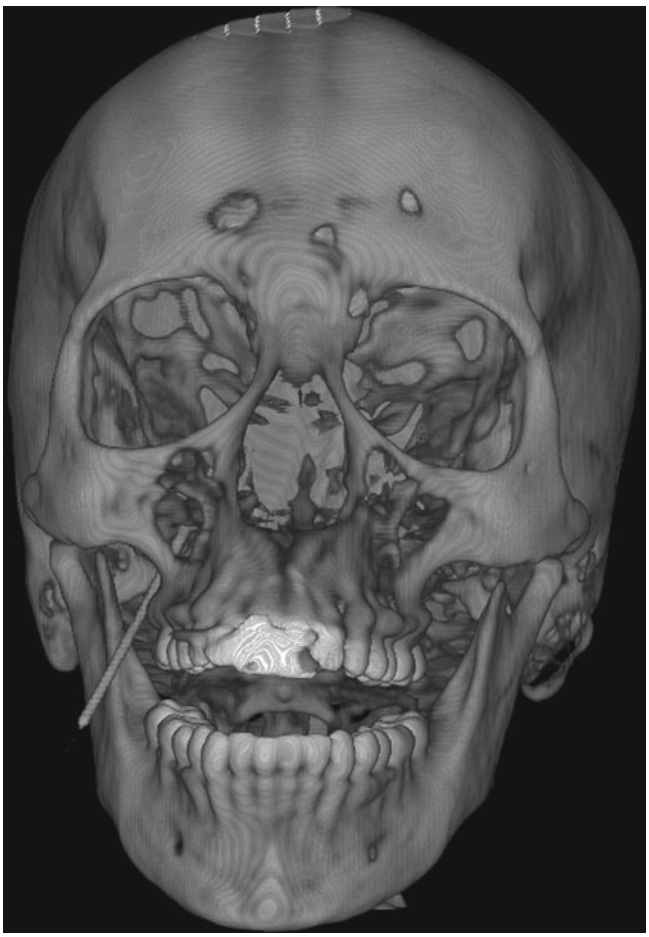

Fig. 25.12 3-D image of injection of the trigeminal nerve at the foramen ovale (Image courtesy of Andrea Trescot, MD)

Ultrasound-Guided Technique

Ultrasound (US) guidance to block the mental nerve can be beneficial over the blind or fluoroscopic approaches. With direct visualization of the mental foramen, proper needle placement can be achieved while also avoiding inadvertent needle placement into the foramen (Fig. 25.13). The US technique would be performed in the same fashion as the conventional landmark-guided technique but with improved visualization of the target [69, 70].

Neurolytic/Surgical Techniques

Cryoneuroablation

Cryoneuroablation has been an effective therapeutic technique for MNN. Zakrzewska and Nally [71] used cryoneuroablation to treat 145 patients with paroxysmal trigeminal neuralgia, including the MN, and followed these patients for 1 month to 6 years. The mean length of relief for cryoneuroablation of the MN was 17 months; all patients regained sensation within 2–3 months, long before the return of pain (if any). Trescot described the use of cryoneuroablation for the treatment of craniofacial pain syndromes including MNN [72].

Radiofrequency Lesioning

Radiofrequency ablation (RFA) is an interventional technique in which high-frequency currents are targeted at nervous tissue. Although there are potential concerns regarding neuritis and neuromas when performing conventional radiofrequency lesioning [69, 73], pulsed radiofrequency (PRF) ablation has repeatedly shown to be a safe, nondestructive technique [74]. PRF is different from classic RF in that high-frequency electric currents are delivered in short bursts. Park et al. [73] described the successful use of ultrasound for PRF in three patients with post-herpetic neuralgia.

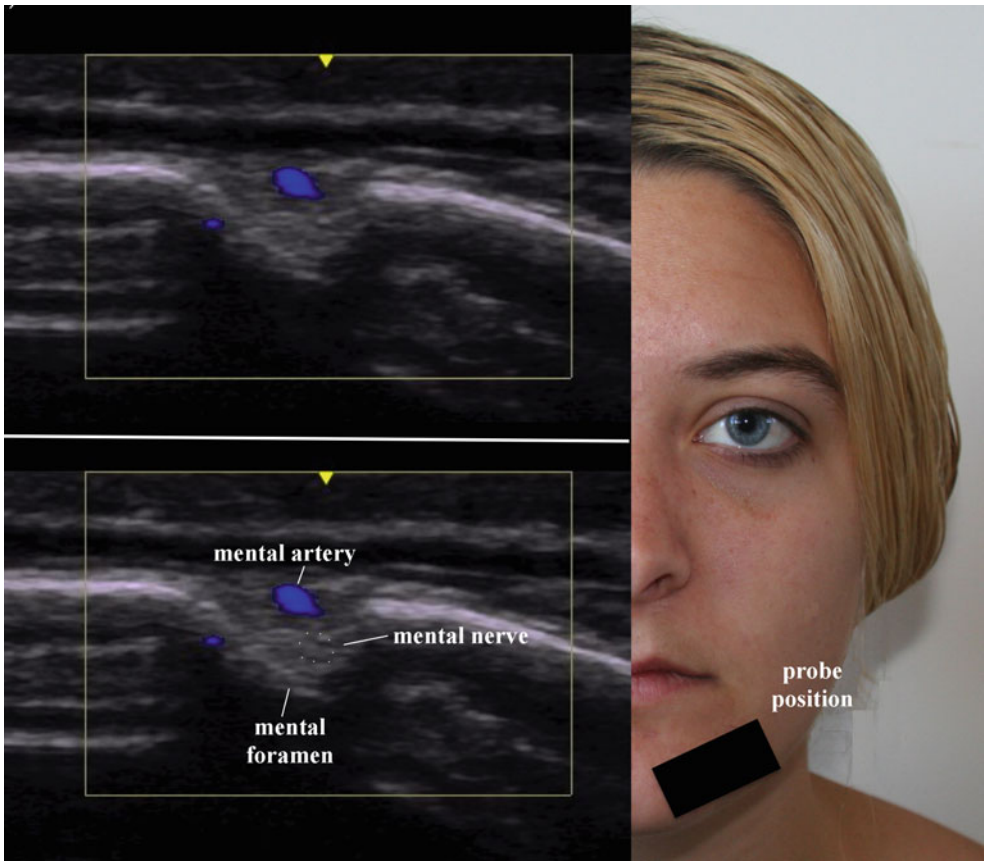

Fig. 25.13 Ultrasound visualization of the mental nerve (Image courtesy of David Spinner, MD)

Chemical Neurolysis

Chemical neurolytic techniques involve the use of ethyl alcohol, phenol, or botulinum toxin to treat neuropathic pain. These agents destruct nerves and are believed to promote long-term analgesia by locally destroying the structures responsible for relaying pain signals [75]. In his *Atlas of Interventional Pain Management*, Waldman describes the technique of performing a 6.5 % aqueous phenol neurolytic block of the inferior alveolar nerve when pain secondary to malignancy is intractable [76]. While chemical neurolysis was extensively used to treat neuropathic pain in the past, it has fallen out of favor with the advent of newer analgesics and safer interventional techniques including cryoneuroablation and PRF [69, 77, 78].

Peripheral Nerve Stimulation

Peripheral nerve stimulation is a technique performed by pain physicians in which a stimulating lead is placed near the known anatomic location of the affected peripheral nerve via an open surgical or percutaneous approach. The lead transmits electrical currents to stimulate the distal subcutaneous distribution of pain (as subjectively described by the patient). The underlying concept is that by stimulating the painful tissue, the peripheral nerve related to the pain is also indirectly stimulated. Although the precise mechanism is unknown, it is believed that PNS modulates the nervous system with electrical signals, resulting in local inflammatory mediator changes as well as changes in gene expression.

PNS of the trigeminal nerve (and its branches) for postherpetic, post-traumatic, and postsurgical neuropathic pain has resulted in significant success. Both Johnson et al. and Feletti and his colleagues have shown that stimulation of the trigeminal nerve, or branches thereof, is effective in treating facial neuralgia refractory to medical treatment [12, 79]. Improvement has been documented in pain relief, quality of life, and functional capacity.

Complications

Complications from mental nerve injections are rare but can include bleeding at the site, which is easily remedied by compression over the injection site. Other complications are worsening pain after injection, neuritis with neurolytic techniques, and bruising related to any subcutaneous bleeding from associated procedures.

Summary

Unilateral numbness of the lower gums, lower lip, or chin, with no obvious orofacial or dental etiology, should be considered of neoplastic etiology until proven otherwise. In addition to a thorough history and physical exam, radiologic evaluation should begin with panoramic radiography of the mandible. A CT scan of the head is necessary to evaluate for brain or skull base lesions. An MRI can be useful for leptomeningeal metastases if the CT is unremarkable. Additionally, nuclear bone scintigraphy may also be helpful. Lastly, if imaging studies are unrevealing, lumbar puncture and cytological examination of the CSF should be performed. Management of MNN is focused on the driving etiology; however, if associated with malignancy, prognosis is extremely poor, with an average survival of 6.9 months [18, 66].

Although a seemingly benign symptom, numbness in the lower mouth or chin regions is often an ominous sign, and patients presenting with such complaints, regardless of age, sex, or race, should be urgently investigated for an occult malignancy [10, 61].

References

1. Wolford LM, Stevao EL. Considerations in nerve repair. Proc (Bayl Univ Med Cent). 2003;16(2):152–6.
2. Greenstein G, Tarnow D. The mental foramen and nerve: clinical and anatomical factors related to dental implant placement: a literature review. J Periodontol. 2006;77(12):1933–43.
3. Wever I, Hwang S, Choroomi S, Mooney W. Delayed mental nerve neuralgia following chin augmentation. Case Rep Otolary. 2013;ID 860634, 3 pages.
4. Di Lenarda R, Cadenaro M, Stacchi C. Paresthesia of the mental nerve induced by periapical infection: a case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2000;90(6):746–9.
5. Friedman P, Sklaver N, Jr. Klawans HL. Neurologic manifestations of Paget's disease of the skull. Dis Nerv Syst. 1971;32(12):809–17.
6. Chand V, Sweeney C, Agger WA. Mental neuropathy in patients with AIDS-associated malignant lymphoma. Clin Infect Dis. 1997;24(3):521–2.
7. Mestoudjian P, Steichen O, Stankovic K, Lecomte I, Lionnet F. Sickle cell disease, a benign cause of numb chin syndrome. Am J Med. 2008;121(10):E1.
8. da Fonseca M, Oueis HS, Casamassimo PS. Sickle cell anemia: a review for the pediatric dentist. Pediatr Dent. 2007;29(2):159–69.
9. Abilleira S, Bowler JV. The numb chin syndrome as an early manifestation of giant-cell (temporal) arteritis: a case report. Headache. 2005;45(10):1411–3.
10. Penarrocha-Diago M, Mora-Escribano E, Bagan JV. Neoplastic trigeminal neuropathy: presentation of 7 cases. Med Oral Patol Oral Cir Bucal. 2006;11(2):E106–11.
11. Zadik Y, Benoliel R, Fleissig Y, Casap N. Painful trigeminal neuropathy induced by oral bisphosphonate-related osteonecrosis of the jaw: a new etiology for the numb-chin syndrome. Quintessence Int. 2012;43(2):97–104.
12. Johnson MD, Burchiel KJ. Peripheral stimulation for treatment of trigeminal postherpetic neuralgia and trigeminal posttraumatic neuropathic pain: a pilot study. Neurosurgery. 2004;55(1):135–4.
13. Fan Y, Luka R, Noronha A. Non-Hodgkin lymphoma presenting with numb chin syndrome. BMJ Case Rep. 2011: on line.
14. Sweeney CJ, Agger WA. AIDS-related lymphomas with neurologic manifestations. West J Med. 1997;167(1):40–4.
15. Marinella MA. Metastatic large cell lung cancer presenting with numb chin syndrome. Respir Med. 1997;91(4):235–6.
16. Sasaki M, Yamazaki H, Aoki T, Ota Y, Sekiya R, Kaneko A. Bilateral numb chin syndrome leading to a diagnosis of Burkitt's cell acute lymphocytic leukemia: a case report and literature review. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2011;111(3):e11–6.
17. Requena A, Riera-Mestre A, Formiga F, Vidaller A, Pujol R. Metastatic prostate carcinoma presenting as numb chin syndrome in elderly people. J Am Geriatr Soc. 2008;56(3):581–2.
18. Narendra H, Ray S. Numb chin syndrome as a manifestation of metastatic squamous cell carcinoma of esophagus. J Cancer Res Ther. 2009;5(1):49–51.
19. Divya KS, Moran NA, Atkin PA. Numb chin syndrome: a case series and discussion. Br Dent J. 2010;208(4):157–60.
20. Dispenzieri A, Kyle RA. Neurological aspects of multiple myeloma and related disorders. Best Pract Res Clin Haematol. 2005;18(4):673–88.
21. Miera C, Benito-Leon J, dela Fuente M, de la Serna J. Numb chin syndrome heralding myeloma relapse. Muscle Nerve. 1997;20(12):1603–6.
22. Furukawa T. Charles bell's description of numb chin syndrome. Neurology. 1988;38(2):331.
23. Rogers R, Paillas J. Le signe du mentonnier (paresthesie et anesthesie unilaterale) revelateur d'un processus metastatique. Rev Neurol. 1937;2:751–2.
24. Calverley JR, Mohnac AM. Syndrome of the numb chin. Arch Intern Med. 1963;112:819–21.
25. Rozman C, Erill S. Dysesthesia of the chin, a valuable diagnostic fact in diffuse bone neoplasms. Med Clin (Barc). 1965;44(4):248–52.
26. Laurencet FM, Anchisi S, Tullen E, Dietrich PY. Mental neuropathy: report of five cases and review of the literature. Crit Rev Oncol Hematol. 2000;34(1):71–9.
27. Kim MK. Head & neck anatomy. 5th ed. Seoul: Dental & Medical Publishing; 2001.
28. Kqiku L, Weiglein A, Kamberi B, Hoxha V, Meqa K, Stadtler P. Position of the mental foramen in Kosovarian population. Coll Antropol. 2013;37(2):545–9.
29. Gupta S, Soni JS. Study of anatomical variations and incidence of mental foramen and accessory mental foramen in dry human mandibles. Natl J Med Res. 2012;2(1):28–30.
30. Leite GM, Lana JP, de Carvalho Machado V, Manzi FR, Souza PE, Horta MC. Anatomic variations and lesions of the mandibular canal detected by cone beam computed tomography. Surg Radiol Anat. 2014;36(8):795–804.
31. Scheid RC, Weiss G, Woelfel JB. Woelfel's dental anatomy. 8th ed. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins Health; 2012.
32. Hiatt JL, Gartner LP. Textbook of head and neck anatomy. 4th ed. Philadelphia: Wolters Kluwer Health/Lippincott William & Wilkins; 2010.
33. Agur AMR, Grant JCB. Grant's atlas of anatomy. 13th ed. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2011.
34. Alsaad K, Lee TC, McCartan B. An anatomical study of the cutaneous branches of the mental nerve. Int J Oral Maxillofac Surg. 2003;32(3):325–33.
35. Netter FH, Hansen JT, Lambert DR. Netter's clinical anatomy. 1st ed. Carlstadt: Icon Learning Systems; 2005.

36. Khoury JN, Mihailidis S, Ghabriel M, Townsend G. Applied anatomy of the pterygomandibular space: improving the success of inferior alveolar nerve blocks. Aust Dent J. 2011;56(2):112–21.
37. Sinnatamby CS, Last RJ. Last's anatomy: regional and applied. 12th ed. Edinburgh: Churchill Livingstone/Elsevier; 2011.
38. Mraiwa N, Jacobs R, Moerman P, Lambrichts I, van Steenberghe D, Quirynen M. Presence and course of the incisive canal in the human mandibular interforaminal region: two-dimensional imaging versus anatomical observations. Surg Radiol Anat. 2003;25(5–6):416–23.
39. Smith JD, Surek CC, Cortez EA. Localization of the supraorbital, infraorbital, and mental foramina using palpable, bony landmarks. Clin Anat. 2010:on line.
40. Agthong ST, Huanmanop, Chentanez V. Anatomical variations of the supraorbital, infraorbital, and mental foramina related to gender and side. J Oral Maxillofac Surg. 2005;63(6):800–4.
41. Arzouman MJ, Otis L, Kipnis V, Levine D. Observations of the anterior loop of the inferior alveolar canal. Int J Oral Maxillofac Implants. 1993;8(3):295–300.
42. Jacobs R, Mraiwa N, Van Steenberghe D, Sanderink G, Quirynen M. Appearance of the mandibular incisive canal on panoramic radiographs. Surg Radiol Anat. 2004;26(4):329–33.
43. Ritter L, Neugebauer J, Mischkowski RA, Dreiseidler T, Rothamel D, Richter U, et al. Evaluation of the course of the inferior alveolar nerve in the mental foramen by cone beam computed tomography. Int J Oral Maxillofac Implants. 2012;27(5):1014–21.
44. Bavitz JB, Harn SD, Hansen CA, Lang M. An anatomical study of mental neurovascular bundle-implant relationships. Int J Oral Maxillofac Implants. 1993;8(5):563–7.
45. Kieser J, Kuzmanovic D, Payne A, Dennison J, Herbison P. Patterns of emergence of the human mental nerve. Arch Oral Biol. 2002;47(10):743–7.
46. Kuzmanovic DV, Payne AG, Kieser JA, Dias GJ. Anterior loop of the mental nerve: a morphological and radiographic study. Clin Oral Implants Res. 2003;14(4):464–71.
47. Driscoll CF. Bifid mandibular canal. Oral Surg Oral Med Oral Pathol. 1990;70(6):807.
48. Wang TM, Shih C, Liu JC, Kuo KJ. A clinical and anatomical study of the location of the mental foramen in adult Chinese mandibles. Acta Anat. 1986;126(1):29–33.
49. Ngeow WC, Yuzawati Y. The location of the mental foramen in a selected Malay population. J Oral Sci. 2003;45(3):171–5.
50. Ngeow WC, Dionysius DD, Ishak H, Nambiar P. Effect of ageing towards location and visibility of mental foramen on panoramic radiographs. Singapore Dent J. 2010;31(1):15–9.
51. Al-Ani O, Nambiar P, Ha KO, Ngeow WC. Safe zone for bone harvesting from the interforaminal region of the mandible. Clin Oral Implants Res. 2013;24(Suppl A100):115–21.
52. Iwanaga J, Saga T, Tabira Y, Nakamura M, Kitashima S, Watanabe K, et al. The clinical anatomy of accessory mental nerves and foramina. Clin Anat. 2015;28(7):848–56.
53. Morse DR. Endodontic-related inferior alveolar nerve and mental foramen paresthesia. Compend Contin Educ Dent. 1997;18(10): 963–8.
54. Morse DR. Infection-related mental and inferior alveolar nerve paresthesia: literature review and presentation of two cases. J Endod. 1997;23(7):457–60.
55. Lossos A, Siegal T. Numb chin syndrome in cancer patients: etiology, response to treatment, and prognostic significance. Neurology. 1992;42(6):1181–4.
56. Massey EW, Moore J, Schold Jr SC. Mental neuropathy from systemic cancer. Neurology. 1981;31(10):1277–81.
57. Burt RK, Sharfman WH, Karp BI, Wilson WH. Mental neuropathy (numb chin syndrome). A harbinger of tumor progression or relapse. Cancer. 1992;70(4):877–81.
58. DeGowin EL. Diagnostic reasoning. In: DeGowin EL, editor. Bedside diagnostic examination. New York: Macmillan Publishing; 1987. p. 1–13.
59. Lata J, Kumar P. Numb chin syndrome: a case report and review of the literature. Indian J Dent Res. 2010;21(1):135–7.
60. Carroll CG, Campbell WW. Multiple cranial neuropathies. In: Donofrio P, editor. Textbook of peripheral neuropathy. New York: Demos Medical Publishing; 2012. p. 407–20.
61. Baskaran R, Krishnamoorthy K, Smith M. Numb chin syndrome – a reflection of systemic malignancy. World J Surg Oncol. 2006;4:2.
62. Evans RW, Kirby S, Purdy RA. Numb chin syndrome. Headache. 2008;48(10):1520–4.
63. Penarrocha Diago M, Bagan Sebastian JV, Alfaro Giner A, Escrig Orenga V. Mental nerve neuropathy in systemic cancer. Report of three cases. Oral Surg Oral Med Oral Pathol. 1990;69(1):48–51.
64. Bar-Ziv J, Slasky BS. CT imaging of mental nerve neuropathy: the numb chin syndrome. AJR Am J Roentgenol. 1997;168(2):371–6.
65. Marinella MA. Ophthalmoplegia: an unusual manifestation of hypocalcemia. Am J Emerg Med. 1999;17(1):105–6.
66. Galan Gil S, Penarrocha Diago M, Penarrocha Diago M. Malignant mental nerve neuropathy: systematic review. Med Oral Patol Oral Cir Bucal. 2008;13(10):E616–21.
67. Friedrich RE. Mental neuropathy (numb chin syndrome) leading to diagnosis of metastatic mediastinal cancer. Anticancer Res. 2010;30(5):1819–21.
68. Trescot AM. Headache: is it a migraine? Think again. Tech Reg Anesth Pain Manag. 2005;9:68–72.
69. Lim SM, Park HL, Moon HY, Kang KH, Kang H, Baek CH, et al. Ultrasound-guided infraorbital nerve pulsed radiofrequency treatment for intractable postherpetic neuralgia – a case report. Kor J Pain. 2013;26(1):84–8.
70. Tsui BC. Ultrasound imaging to localize foramina for superficial trigeminal nerve block. Can J Anaesth. 2009;56(9):704–6.
71. Zakrzewska JM, Nally FF. The role of cryotherapy (cryoanalgesia) in the management of paroxysmal trigeminal neuralgia: a six year experience. Br J Oral Maxillofac Surg. 1988;26(1):18–25.
72. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
73. Park HG, Park PG, Kim WJ, Park YH, Kang H, Baek CW, et al. Ultrasound-assisted mental nerve block and pulsed radiofrequency treatment for intractable postherpetic neuralgia: three case studies. Kor J Pain. 2014;27(1):81–5.
74. Cohen SP, Van Zundert J. Pulsed radiofrequency: rebel without cause. Reg Anesth Pain Med. 2010;35(1):8–10.
75. Kus M, Dobrogowski J. Chemical neurolysis in advanced neoplasms. Pol Tyg Lek. 1985;40(6):175–7.
76. Waldman SD. Atlas of interventional pain management. 3rd ed. Philadelphia: Saunders/Elsevier; 2009.
77. Eisenberg E, Carr DB, Chalmers TC. Neurolytic celiac plexus block for treatment of cancer pain: a meta-analysis. Anesth Analg. 1995;80(2):290–5.
78. Rowe J, Caprio AJ. Chronic pain: challenges and opportunities for relieving suffering. N C Med J. 2013;74(3):201–4.
79. Feletti A, Santi GZ, Sammartino F, Bevilacqua M, Cisotto P, Longatti P. Peripheral trigeminal nerve field stimulation: report of 6 cases. Neurosurg Focus. 2013;35(3):E10.

Glossopharyngeal Nerve Entrapment

26

Agnes R. Stogicza and Andrea M. Trescot

Introduction

Glossopharyngeal neuralgia (GPN) is a relatively rare but severe and often misdiagnosed condition, characterized by shooting and stabbing pain at the ear canal, at the pharynx, at the angle of the jaw, and at the base of the tongue. It has also been referred to as *stylalgia* or *Eagle's syndrome*; these latter names describe the enlarged or elongated styloid process that can be one of the causes of GPN.

Clinical Presentation (Table 26.1)

In 1910, Weisenburg described a patient with lancinating pain of the throat and ear that he diagnosed as "an unusual *tic douloureux*" (Fig. 26.1) [1]. GPN is a pain condition of the *glossopharyngeal nerve* (GN), analogous to *trigeminal neuralgia* or *tic douloureux*. According to the International Headache Society [2], GPN is defined as unilateral neuralgia with severe transient, stabbing pain experienced in the ear, base of the tongue, back of the nose, tonsillar fossa, and larynx or beneath the angle of the jaw (Fig. 26.2) [3]. The pain is therefore felt in the distribution of the auricular and pharyngeal branches of the vagus nerve, as well as that of the glossopharyngeal nerve. It is commonly provoked by swallowing, talking, laughing, chewing, and coughing and may remit and relapse, similar to what is seen in tic douloureux. There may be a fear of eating because of the pain, and there have been reported cases of concomitant syncope [3], presumably because of vagal stimulation and subsequent bradycardia. Idiopathic and symptomatic GPNs are similar in clinical symptoms but can be differentiated based on etiology (see Table 26.1). Diagnostic criteria are summarized in Table 26.2. Trigeminal neuralgia can be seen in combination with GN.

The pain distribution may be mainly in the tympanic or in the oropharyngeal region. The patient will describe their GPN triggers in several ways: the *oropharyngeal type* is triggered mainly by swallowing, talking, and yawning, whereas the *tympanic type* is provoked by loud sounds or tactile sensations at the outer auditory canal. Pressure over the styloid process may provoke a pain attack, but it is not a necessary criterion of GPN.

GPN attacks evolve somewhat slower than trigeminal neuralgia, with a mean duration of 30 s. It is an excruciating, sharp, stabbing pain. The pain attacks can reoccur several times a day or over weeks, months, or years. Swallowing, chewing, talking, sneezing, and coughing are the most common trigger factors, but some of the patients also notice the paroxysm from hot, cold or acidic drinks, head movements or touching the ear [5].

Other less common features include tinnitus, vomiting, vertigo, swelling sensation and involuntary movements [6]. Many patients have been described in the literature with asystole, convulsions, and syncope associated with GPN. This condition is called *vagoglossopharyngeal neuralgia* (VGN) [6, 7]. These reactions occur due to the complex

Table 26.1 Occupation/exercise/trauma history relevant to glossopharyngeal nerve entrapment

Compression	Elongated styloid process (Eagle's syndrome)
	Vascular compression
Trauma	Tonsillectomy
	Craniotomy
	Radical neck dissection
	Motor vehicle accident, upper cervical spine injury
Neuropathy	Demyelinating disease

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Fig. 26.1 Patient described pain patterns of GPN (Image courtesy of Andrea Trescot, MD)

Fig. 26.2 Pattern of pain associated with GPN (Image courtesy of Andrea Trescot, MD)

anatomical relationship between the *intermedius nerve*, *vagus nerve*, and GPN [8] (see "Anatomy" section).

Secondary GPN is less common than idiopathic GPN, and it can be caused by tumor mass, vascular malformations along the course of the nerve, or ossification of the *stylohyoid ligament* (*Eagle's syndrome*) (Fig. 26.3) [4].

Table 26.2 Diagnostic criteria of GPN according to the international classification of headache disorders, 2004

Idiopathic GPN	Secondary GPN
Paroxysmal attacks of facial pain lasting from a fraction of a second to 2 min	
Pain has all of the following characteristics:	
Unilateral location	
Distribution within the posterior part of the tongue, tonsillar fossa, and pharynx or beneath the angle of the lower jaw and/or in the ear	
Sharp, stabbing, and severe; precipitated by swallowing, chewing, talking, coughing, and/or yawning	
Attacks are stereotypical for that individual patient	
There is no clinically evident neurological deficit	Neurologic deficit may be present at the area supplied by glossopharyngeal nerve
Other causes have been ruled out by history, physical examination, and/or special investigations	A causative lesion has been demonstrated by special investigations and/or surgery

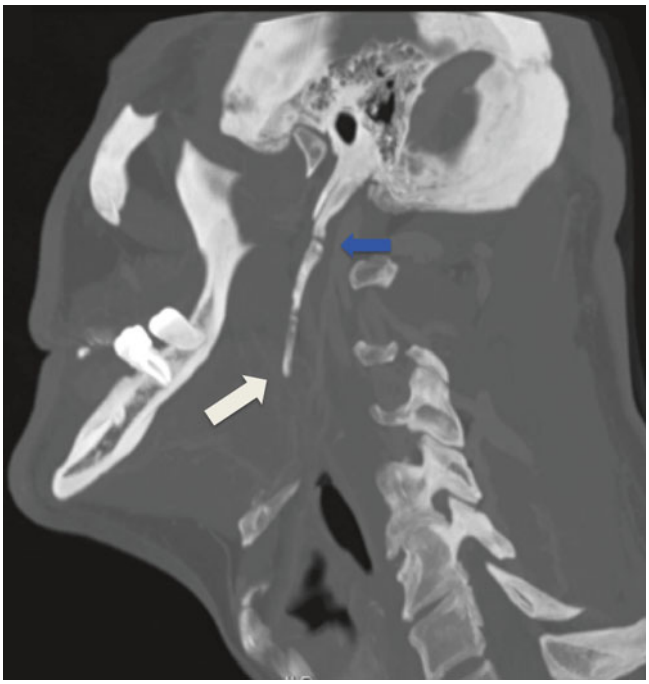

Fig. 26.3 CT scan showing elongated styloid process (*white arrow*). Note fracture line (*blue arrow*) (Image courtesy of Christ DeClerck, MD)

An unusual clinical use of GN injections is in the treatment of intractable hiccups. Since the GN is part of the reflex arc of the hiccup, anesthetizing the GN (with potential denervation) has been reported as useful [9, 10]. Injections and subsequent lysis may also be useful to treat throat and neck pain from cancer infiltration [11].

Although considered "uncommon," Singh et al. [3] felt that GPN was "not as uncommon as has been reported in the

literature" but rather was underdiagnosed due to the variety of presentations, as well as lack of awareness of the disease. The incidence is 0.7/100,000/year according to Manzoni [12]. Katusic et al. compared the epidemiologic incidence of trigeminal neuralgia versus GPN; they found an incidence of 4.7/100,000/year for trigeminal neuralgia as opposed to 0.8/100,000/year for GPN [13]. Bruyn [5] reviewed 55 years of records at Mayo Clinic and could only identify 217 cases, with another 304 that he found in the literature.

Anatomy (Table 26.3)

The *styloid process* is the calcified proximal attachment of the *stylohyoid ligament*, extending in a caudal and ventral direction from the temporal bone just below the auditory meatus to the hyoid bone. The GN arises from the medulla and exits the skull with the *vagus* and *spinal accessory nerves* (see Chap. 27) through the *jugular foramen*, descends through the narrow space between the transverse process of the atlas (C1) and the styloid process (Figs. 26.4 and 26.5), passes anterior to the carotid artery and deep to the styloid, and then turns to the tongue where it passes through the tonsillar fossa to enter the pharynx. The internal carotid artery, internal jugular vein, and vagus nerve are separated from the external carotid artery by the styloglossus muscle, the stylopharyngeus muscle, and the GN (Fig. 26.6).

The GN is a mixed cranial nerve with sensory, motor, and autonomic components. Sensory innervation includes the *eustachian tube*, middle ear, and mastoid; the oropharynx and soft palate; the posterior third of the tongue (including taste); the *vallecula*; the anterior surface of the epiglottis (by the lingual branch); the walls of the pharynx (by the pharyngeal branch); and the tonsils (by the tonsillar branch). Therefore, the GN is part of the gag reflex.

Table 26.3 Anatomy of the glossopharyngeal nerve

Origin	Jugular foramen
General route	Descends anterior to carotid artery and deep to styloid; through tonsillar fossa to tongue
Sensory distribution	Eustachian tube, middle ear, and mastoid; posterior third of the tongue; vallecula; anterior surface of the epiglottis; pharynx; tonsils
Autonomic innervation	Parasympathetic fibers to the parotid gland, the carotid body, and the carotid sinus
Motor innervation	Stylopharyngeus muscle

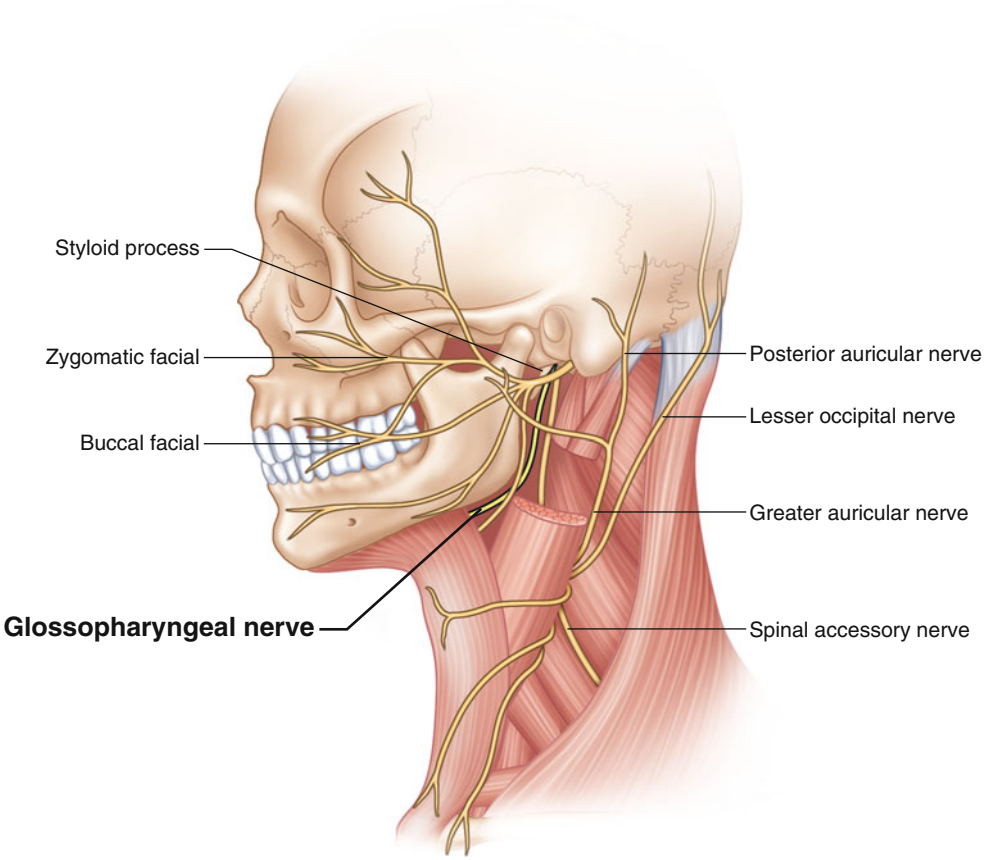

Fig. 26.4 The anatomy of the glossopharyngeal nerve (Image by Springer)

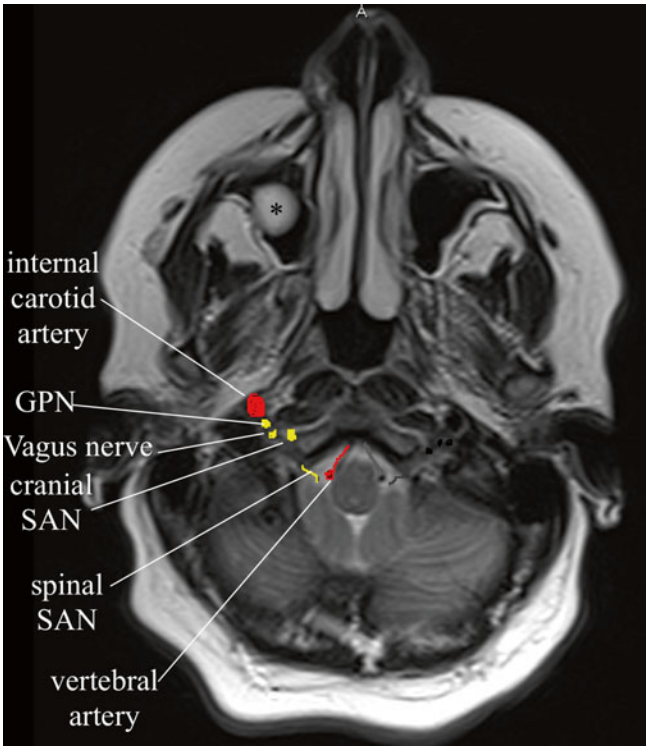

Fig. 26.5 MRI axial image at the level of the foramen magnum. *GPN* glossopharyngeal nerve, *SAN* spinal accessory nerve (cranial and spinal divisions); * = maxillary sinus cyst (Image courtesy of Andrea Trescot, MD)

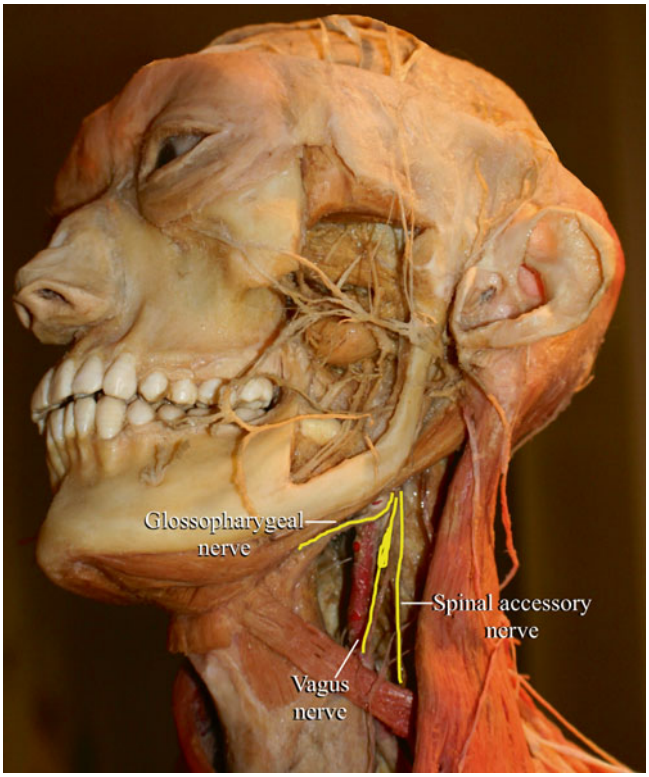

Fig. 26.6 Dissection of the head and neck, showing the relationship between the spinal accessory nerve, the glossopharyngeal nerve, and the vagus nerve in the neck next to the carotid artery (Image modified from an image from *Bodies, The Exhibition,* with permission. Image courtesy of Andrea Trescot, MD)

The GN provides motor innervation to the striated *stylopharyngeus muscle*. The autonomic innervation includes the secretomotor parasympathetic fibers to the parotid gland, the carotid body, and the carotid sinus (via the *nerve of Hering*).

Entrapment

Idiopathic GPN is just that – idiopathic, and not attributed to any specific, well understood entrapment. However, Eagle's syndrome of GPN with an elongated styloid process is felt to be due to an entrapment of the GN at the styloid (Fig. 26.3). Eagle himself described entrapment of the GN at the tip of the styloid from scarring after a tonsillectomy [14].

Physical Exam

Physical exam yields minimal information; history and clinical symptoms aid more in suggesting the diagnosis. The styloid process can be found by creating an imaginary line from the mastoid to the angle of the mandible; the styloid should be found halfway between these two structures (Fig. 26.7) (Video 26.1). Pressure over the styloid process may provoke a pain attack or replicate the pain, but it is not a necessary criterion of GPN (Fig. 26.8).

Differential Diagnosis (Table 26.4)

Trigeminal Neuralgia/Tongue Pain

GPN, like trigeminal neuralgia, can be caused by neurovascular compression. According to Singh et al. [3], three MRA radiologic findings aid this diagnosis:

1. High-origin *posterior inferior cerebellar artery* (PICA)
2. The PICA making an upward loop
3. The PICA coursing and compressing the supra-olivary fossette [15]

However, if the offending vessel is the anterior inferior cerebellar artery (AICA), GPN is difficult to diagnose before surgery because it is difficult to visualize on MRA [15]. They also noted that bilateral pain, constant pain (instead of intermittent), and multiple daily bouts of pain are all "bad prognostic signs."

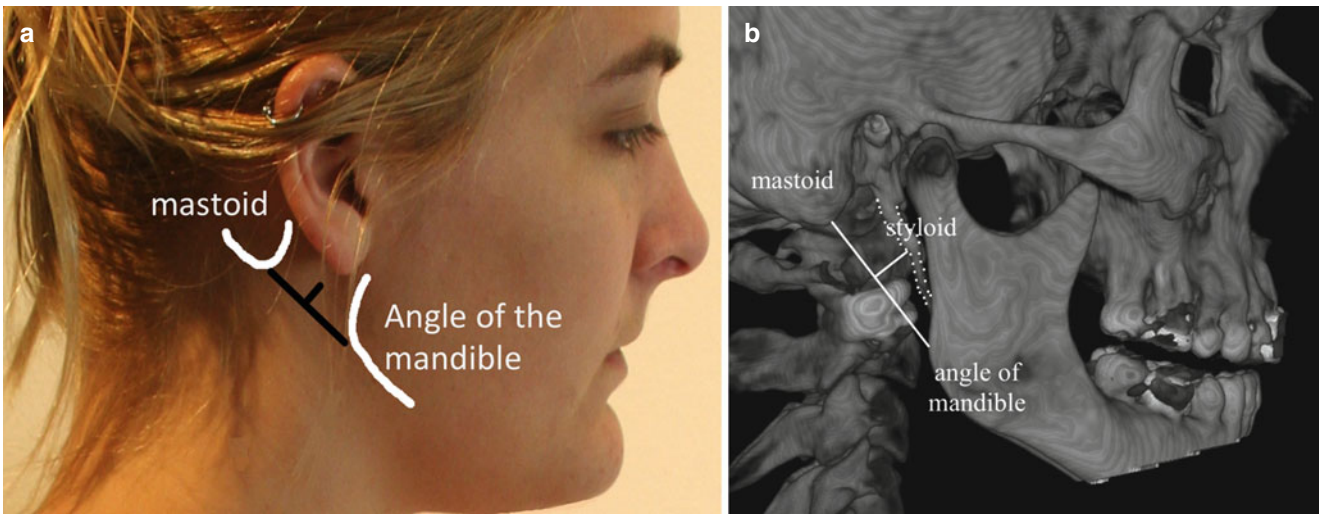

Fig. 26.7 Surface anatomy (**a**) and 3D X-ray image (**b**) of the styloid process (*outlined*). Imaginary line is drawn from the mastoid to the angle of the mandible; styloid process will be found at the halfway point of this line (Image courtesy of Andrea Trescot, MD)

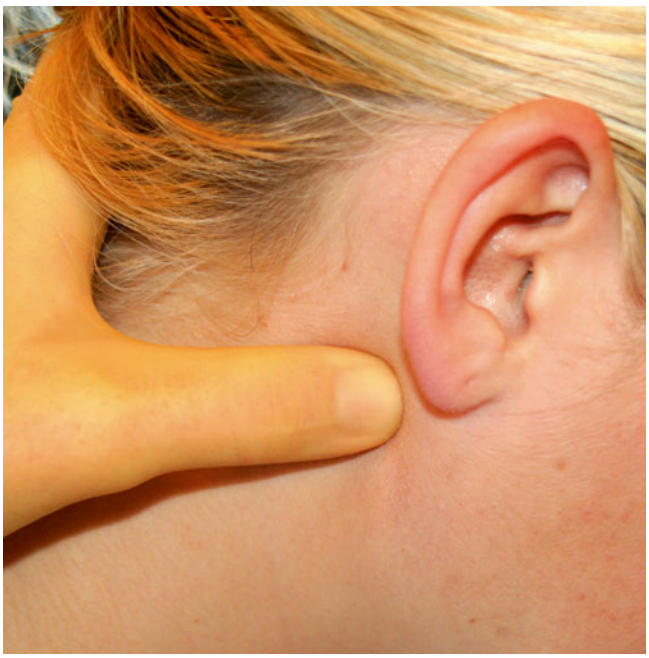

Fig. 26.8 Pressure over the styloid process may reproduce the pain of glossopharyngeal nerve entrapment (Image courtesy of Andrea Trescot, MD)

GPN can also be caused by cerebellopontine angle masses, oropharyngeal tumors, arachnoiditis, and multiple sclerosis [3]. There are multiple pathologies that can have similar presentations, leading to the difficulty in diagnosis. Diverse conditions such as temporal arteritis, retropharyngeal abscess, and mandibular neuralgia can mimic GPN (see Table 26.4). Imaging of the neck should also be done to rule out tumors of the hypopharynx, larynx, or angle of the jaw [3]. Table 26.5 lists the diagnostic tests for GN entrapment.

Table 26.4 Differential diagnosis of neck and throat pathology

	Potential distinguishing features
Trauma of the skull base or upper cervical vertebrae	History of trauma, X-ray/CT
Neoplasms of the pharynx, head, or neck, or post irradiation	MRI evidence of tumor
Infection of head, neck, pharynx (abscess, tuberculosis)	Fever, elevated WBC, elevated ESR/CRP
Eagle's syndrome	X-rays showing elongated styloid process
Temporal arteritis	Elevated ESR, tenderness over temporal artery
Mandibular neuralgia	See Chap. 24

ESR erythrocyte sedimentation rate, *CRP* C-reactive protein

Identification and Treatment of Contributing Factors

Trauma, infection, tumor, and postsurgical or postirradiation state of the head, neck, pharynx and skull base are the obvious contributing factors to secondary GPN. The treatment of GPN includes the treatment of the secondary causes of GPN, such as tumor resection, AV malformation embolization, styloid resection and microvascular decompression (MVD) (see *Surgery* section below).

Injection Technique

Injections can give rapid relief and confirm the diagnosis. The injections can be done with or without steroids [3]. These injections are indicated to diagnose and potentially treat GPN, idiopathic face pain, pain from pharyngeal cancer,

Table 26.5 Diagnostic tests for glossopharyngeal nerve entrapment

	Potential distinguishing features
Physical exam	Tenderness over the styloid process
Diagnostic injections	Confirms diagnosis if no more than 1 mL local anesthetic used at the styloid process or at the tonsil
Ultrasound	Useful to exclude other upper cervical soft tissue pathology
MRI/MRA	Useful to exclude other upper cervical soft tissue pathology and concurrent pathology as well as vascular compromise
Arteriography (3D-CTA)	MRA is preferred if feasible [3]
X-ray	Useful to identify bony anomalies/trauma at upper cervical spine, including elongated styloid
EKG (during an attack)	To rule out associated cardiac arrhythmias (VGN)
Electrodiagnostic studies	Not useful

refractory hiccups and to provide anesthesia of pharyngeal procedures (such as awake intubations). Testing the gag reflex confirms the success of the injection.

Landmark-Guided Technique

If the styloid process is palpable, it may be reasonable to attempt a landmark-guided injection of the GN, with a clear understanding of the anatomy. Since the carotid artery is directly behind the styloid process, there is a significant risk of vascular injection and bleeding. With the patient positioned supine and the head turned slightly away, the non-injecting fingers straddle the styloid, which is about 2 cm posterior from the angle of the mandible and slightly anterior and 1–2 cm caudad to the mastoid (Fig. 26.7). The needle is advanced onto the styloid process (usually about 3 cm in depth) and then redirected slightly posteriorly (Fig. 26.9) (Video 26.2). After careful aspiration, 1 cc of local anesthetic and steroid is injected. As this is a highly vascular area, consider using a non-particulate steroid.

Injection of the GN at the lower lateral portion of the tonsillar fossa was originally developed as an anesthetic technique for conscious intubations. Using a longer needle (consider placing a distal bend in the needle), a site just below the palatopharyngeal fold is targeted with the local anesthetic, as shown on Fig. 26.10. The injection is submucosal, at a depth of about 0.5 cm, and care must be taken to avoid the tonsillar artery. Local anesthetic spray is recommended to decrease the gag reflex. Note that this injection does not anesthetize the tympanic branch of the GN and therefore may not address ear pain.

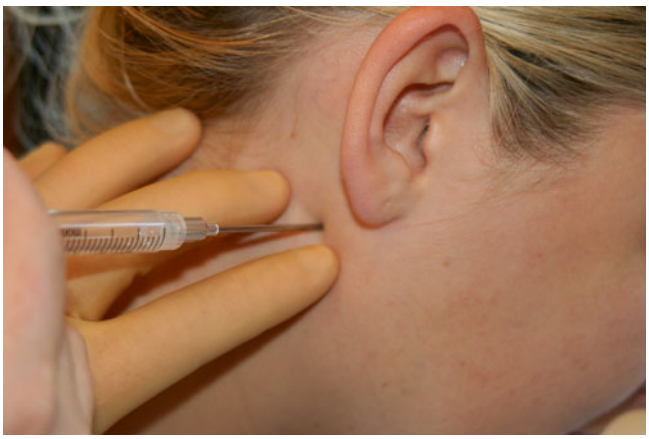

Fig. 26.9 Landmark-guided injection of the glossopharyngeal nerve at the styloid process (Image courtesy of Andrea Trescot, MD)

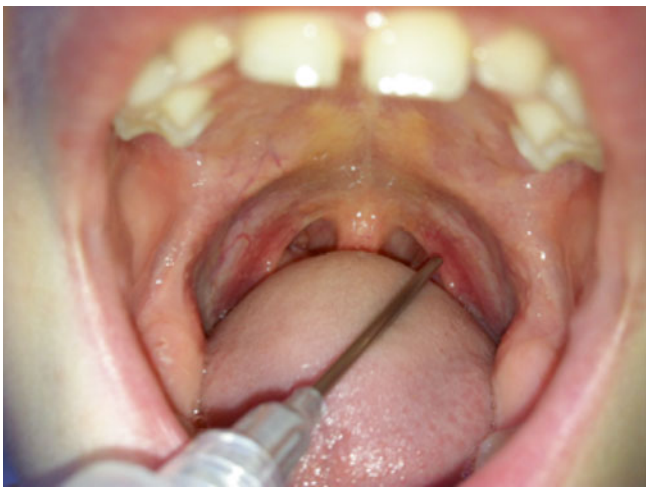

Fig. 26.10 Landmark guided injection of the tonsillar branch of the glossopharyngeal nerve (Image courtesy of Agnes Stogicza, MD)

Fluoroscopy-Guided Technique

The patient is positioned supine; if the styloid is visible on a true lateral X-ray (Fig. 26.11), the needle can be advanced directly onto the styloid and then redirected slightly posteriorly. A peripheral nerve stimulator (PNS) can help separate the GN from the spinal accessory nerve (see Chap. 27) or the vagus nerve. Because of the close proximity of the carotid artery and internal jugular vein, use of a curved, blunt-tipped needle would be prudent in this region.

Ultrasound-Guided Technique (US)

Bedder and Lindsay [16] described an US approach to the GN in 1989 to treat pain from cancer of the throat and tongue. The linear US probe is placed obliquely between the mastoid and the angle of the jaw on the lateral neck (Fig. 26.12), which allows observation of the blood vessels to facilitate

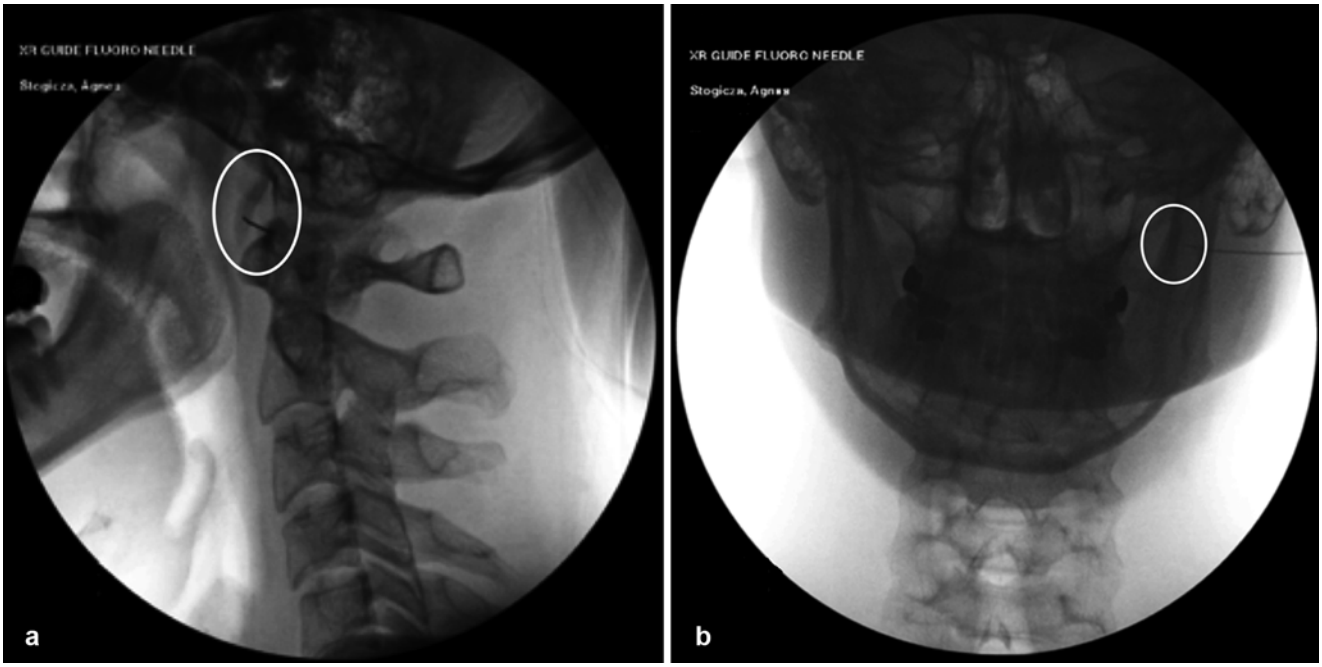

Fig. 26.11 Fluoroscopy guided injection of glossopharyngeal nerve. Lateral view shows the needle placed onto the styloid process. (**a**) Lateral view; (**b**) anterior/posterior view (Image courtesy of Agnes Stogicza, MD)

Fig. 26.12 Location of ultrasound probe for the glossopharyngeal nerve (Image by Springer)

avoidance during in-plane injection. The styloid process is seen as a bone shadow slightly anterior to the mastoid. The target GN is located just posterior to the carotid artery which is visualized with slight rotation of the US probe (Fig. 26.13). Of note the two most superficial blood vessels that are seen are the retromandibular vein and the external jugular vein. Immediately under the SCM one can see the occipital artery at this level, and just deeper to these structures will be the internal jugular and the internal carotid seen. A combined US- and fluoroscopic-directed injection might combine the best of both techniques, allowing for easier identification of the styloid (fluoroscopy) while identifying the nearby vascular structures with US (Fig. 26.14).

CT-Guided Technique

Although not published in the literature, an injection under CT guidance should be possible, since the styloid is visible on CT, and a CT with contrast would show the vasculature (Fig. 26.15).

Neurolytic Technique

Neurolysis of the GN may be indicated for patients with refractory pain in the posterior third of the tongue, oropharynx, and angle of the jaw.

Cryoneuroablation

Cryoneuroablation at the styloid process is not recommended because of the proximity of the carotid artery and the lack of visualization of the target itself. In addition, the vagus and spinal accessory nerve also pass through this area, and it would be difficult to avoid lysis of these nerves.

As a result, the usual site for cryoneuroablation is at the tonsillar fossa. The patient is placed supine and the tongue retracted medially. The nerve is located at the inferior portion of the tonsillar pillar. The mucosa is anesthetized with a topical spray or pledget, and 1 mL of saline with epinephrine is infiltrated for hemostasis. The 12-gauge introducer is then advanced subcutaneously, and the 2-mm probe is advanced through the catheter. Sensory stimulation should refer to the

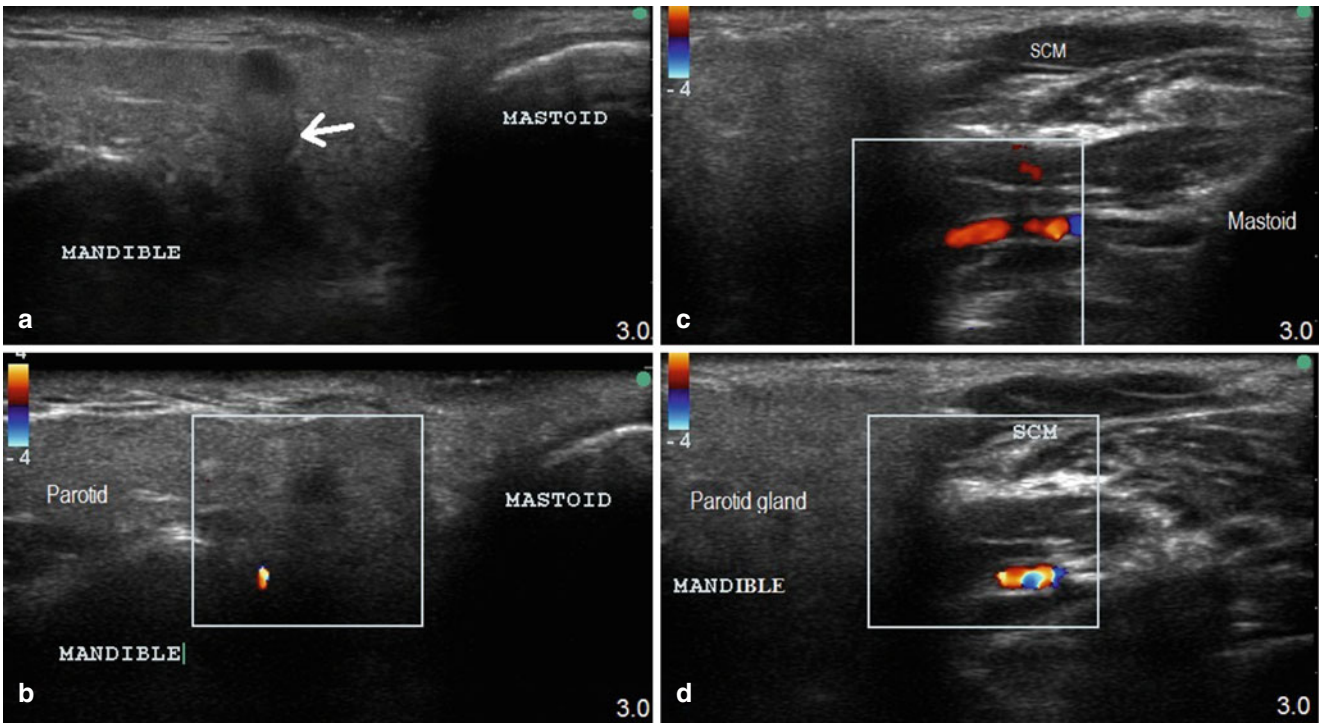

Fig. 26.13 (**a**) linear probe is placed between the angle of the mandible and the mastoid process. (**b**) Styloid shadow (*arrow*), parotid gland and retromandibular vein identified. (**c**) Slight rotation of the US probe allows visualizing the mastoid, the SCM and the carotid artery underneath. Mandible not in view. (**d**) Slight rotation of the US probe in the opposite direction than in "image C" allows visualizing the mandibular angle and the carotid under the SCM. Mastoid not in view (Image courtesy of Agnes Stogicza, MD)

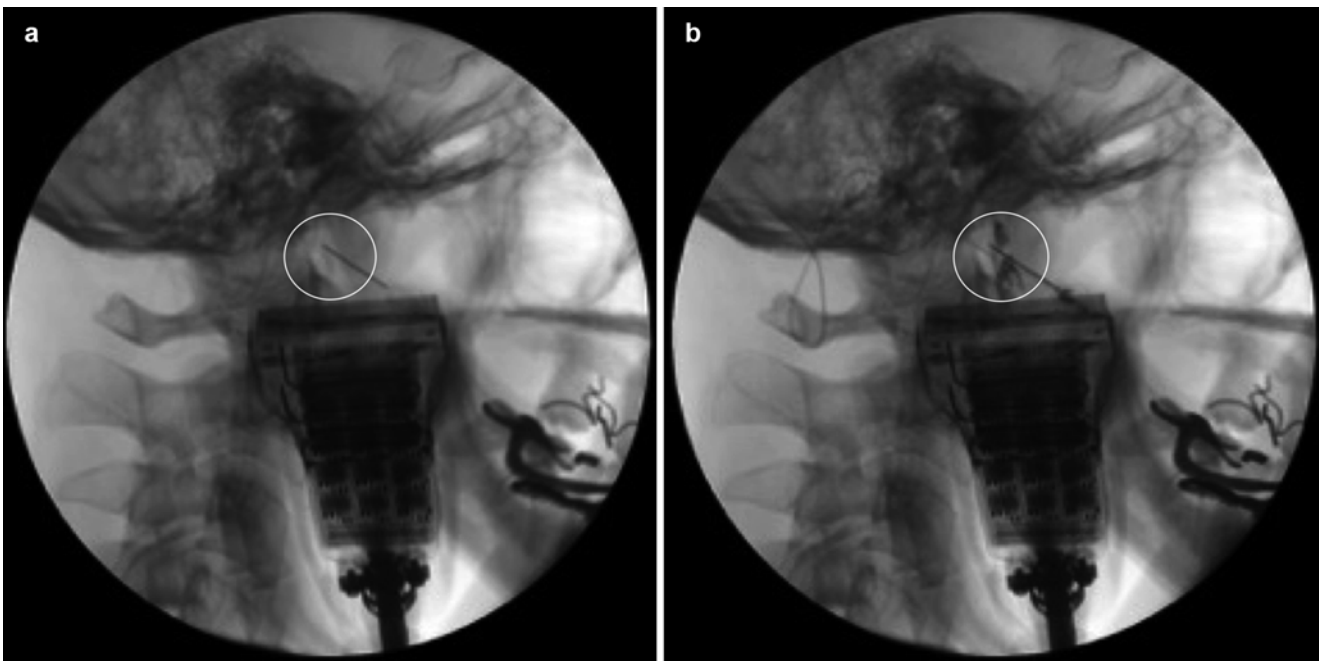

Fig. 26.14 Combined fluoroscopy/ultrasound images showing the glossopharyngeal nerve injection, with needle seen within the *white circle*. (**a**) Needle placement; (**b**) injection of contrast (Image courtesy of Christ DeClerck, MD)

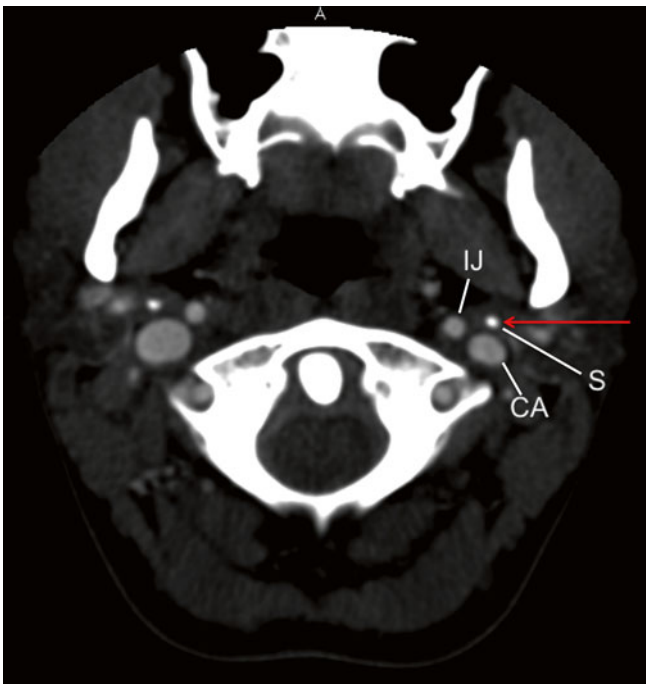

Fig. 26.15 Axial CT image with contrast at the level of the styloid. *IJ* internal jugular vein, *CA* carotid artery, *S* styloid. The *red arrow* represents the proposed injection path (Image courtesy of Andrea Trescot, MD)

ear and throat, and there may be a throat motor stimulation. Care must be used to avoid the palatine artery. This may be done most easily at surgical tonsillectomy, when the nerve is exposed and can be lesioned under direct vision. The newer, smaller diameter (20 Gauge), blunt-tipped cryo probes may allow safer procedure performance, maybe allowing ablation even at the styloid level.

Pulsed Radiofrequency Lesioning

Pulsed radiofrequency lesioning (PRF) can be performed either at the styloid level or at the tonsillar fossa. The needle approach is the same as described above, but, before lesioning, the target nerve must be identified by nerve stimulation and must refer sensation to the ear or throat. PRF ablation is then performed at 42 °C for 90–120 s.

Mollinedo et al. [17] described a patient with bilateral elongated styloid processes (6.3 cm on the left and 6.4 cm on the right) who underwent PRF for left-sided GPN with 3 months of relief.

Alcohol, Phenol, and Glycerol

Koyyalagunta and Burton [18] described the injection of 1 cc of 6 % phenol or absolute alcohol at the styloid process for cancer of the tongue, tonsils, and hypopharynx. Yue and Zhang described the injection of glycerol in eight "sick" children for intractable GPN [19].

Surgical Techniques

Shin and colleagues [4] described surgical resection in seven patients with an elongated styloid, using a lateral transcutaneous approach; all had "complete" relief for at least 12 months. Resnic et al. [20] noted that microvascular decompression provided "complete relief" in 76 % of the cases and a "substantial improvement" in an additional 16 %; Sampson et al. [21] noted pain relief 10 years after MVD.

Complications

Because of the vessels nearby, there is a risk of injection into the carotid artery or the internal jugular vein (from the extraoral approach) or tonsillar artery (from an oral approach), which could cause serious bleeding or seizure (from local anesthetic in the carotid artery). Reflex tachycardia and hypertension could occur from paralysis of the vagus nerve. The injection can cause difficulty with swallowing or hoarseness (from anesthetizing the *recurrent laryngeal nerve*); because of the airway obstruction that could occur with bilateral vocal cord paralysis, patients should not undergo bilateral GN injections at the same time. There can also be local anesthetic spillover onto the spinal accessory nerve (see Chap. 27), causing weakness of the trapezius muscle.

Summary

GPN is an uncommon, but probably underdiagnosed cause of ear, throat, and jaw pain. Like trigeminal neuralgia, there can be entrapment by intracranial vascular structures, but there can also be irritation from an elongated styloid process. As is seen in all of these nerve entrapments, proper use of diagnostic injections is key for proper diagnosis and treatment.

References

1. Weisenburg TH. Cerebellopontine tumour diagnosed for six years as tic douloureux: the symptoms of irritation of the ninth and twelfth cranial nerves. JAMA J Am Med Assoc. 1910;54:1600–4.
2. Headache Classification Subcommittee of the International Headache Society. The international classification of headache disorders. 2nd ed. Cephalalgia. 2004;24 Suppl 1:9–160
3. Singh PM, Kaur M, Trikha A. An uncommonly common: glossopharyngeal neuralgia. Ann Indian Acad Neurol. 2013;16(1):1–8.

4. Shin JH, Herrera SR, Eboli P, Aydin S, Eskandar EH, Slavin KV. Entrapment of the glossopharyngeal nerve in patients with Eagle syndrome: surgical technique and outcomes in a series of 5 patients. J Neurosurg. 2009;111(6):1226–30.
5. Bruyn GW. Glossopharyngeal neuralgia. Cephalalgia. 1983;3(3): 143–57.
6. Bohm E, Strang RR. Glossopharyngeal neuralgia. Brain J Neurol. 1962;85:371–88.
7. Rossitti SL, Balbo RJ, Sperlescu A. Vagoglossopharyngeal neuralgia. Arq Neuropsiquiatr. 1991;49(1):73–9.
8. Ozenci M, Karaoguz R, Conkbayir C, Altin T, Kanpolat Y. Glossopharyngeal neuralgia with cardiac syncope treated by glossopharyngeal rhizotomy and microvascular decompression. Europace Eur Pacing Arrhythmias Card Electrophysiol J Work Group Card Pacing Arrhythmias Card Cell Electrophysiol Eur Soc Cardiol. 2003;5(2):149–52.
9. Babacan A, Ozturk E, Kaya K. Relief of chronic refractory hiccups with glossopharyngeal nerve block. Anesth Analg. 1998; 87(4):980.
10. Gallacher BP, Martin L. Treatment of refractory hiccups with glossopharyngeal nerve block. Anesth Analg. 1997;84(1):229.
11. Wallace MS, Leung AV, McBeth MD. Malignant pain. In: Raj PP, editor. Textbook of regional anesthesia. New York: Churchill Livingstone; 2002. p. 572.
12. Manzoni GC, Torelli P. Epidemiology of typical and atypical craniofacial neuralgias. Neurol Sci. 2005;26(2):S65–7.
13. Katusic S, Williams DB, Beard CM, Bergstralh EJ, Kurland LT. Epidemiology and clinical features of idiopathic trigeminal neuralgia and glossopharyngeal neuralgia: similarities and differences, Rochester, Minnesota, 1945–1984. Neuroepidemiology. 1991;10(5–6):276–81.
14. Eagle WW. Symptomatic elongated styloid process: report of two cases of styloid process – carotid artery syndrome with operation. Arch Otolaryngol. 1949;49:490–503.
15. Hiwatashi A, Matsushima T, Yoshiura T, Tanaka A, Noguchi T, Togao O, et al. MRI of glossopharyngeal neuralgia caused by neurovascular compression. AJR Am J Roentgenol. 2008;191(2):578–81.
16. Bedder MD, Lindsay D. Glossopharyngeal nerve block using ultrasound guidance: a case report of a new technique. Reg Anesth. 1989;14(6):304–7.
17. Mollinedo FT, Esteban SL, Vega CG, Orcasitas AC, Maguregi AA. Pulsed radiofrequency treatment in a case of Eagle's syndrome. Pain Pract. 2013;13(5):399–404.
18. Koyyalagunta D, Burton AW. The role of chemical neurolysis in cancer pain. Curr Pain Headache Rep. 2010;14(4):261–7.
19. Yue WL, Zhang Y. Peripheral glycerol injection: an alternative treatment of children with glossopharyngeal neuralgia. Int J Pediatr Otorhinolaryngol. 2014;78(3):558–60.
20. Resnick DK, Jannetta PJ, Bissonnette D, Jho HD, Lanzino G. Microvascular decompression for glossopharyngeal neuralgia. Neurosurgery. 1995;36(1):64–8; discussion 8–9.
21. Sampson JH, Grossi PM, Asaoka K, Fukushima T. Microvascular decompression for glossopharyngeal neuralgia: long-term effectiveness and complication avoidance. Neurosurgery. 2004;54(4):884–9; discussion 9–90.

Part IV

Chest Wall Peripheral Nerve Entrapment Syndromes

Amitabh Gulati

Introduction

Thoracic pain, which can affect almost 3 % of primary care encounters, can be a diagnostic dilemma [1]. Chest wall pain (CWP) made up 44.6 % of the 672 consecutive thoracic pain patients that presented to a group of primary care offices [1]. Peripheral nerve injury can occur anywhere along the intercostal nerve as each nerve leaves from a thoracic nerve root, causing thoracic and abdominal wall pain. Two intercostal nerves (left and right side) exit from each thoracic level (T1-T12) to innervate the thoracic chest and abdominal wall as well as the parietal pleura and peritoneum. As posterior, lateral, and anterior intercostal branches arise, compression of the branches can lead to segmental pain syndromes. Of these, compression of the anterior cutaneous nerve (ACNES) may lead to abdominal wall pain symptoms (Chap. 42). The purpose of this section is to describe entrapments along the intercostal nerve and its branches, as well as other causes of chest wall pain, including suprascapular, long thoracic, axillary, spinal accessory, or dorsal scapular nerves. The relevant anatomy, clinical presentations, and treatment options will be presented.

Entrapment of the intercostal nerve and its branches should be distinguished from thoracic radiculopathy, which may present with similar clinical symptoms. While not common, degenerative disk disease, facet arthropathy, and malignancy may present in the thoracic spine. Compression of the exiting nerve root may present with similar pain patterns as entrapment of the intercostal nerve (especially proximal compression). Thus, radiologic imaging (MRI or CT of the chest wall and/or thoracic spine) should be considered to evaluate entrapment syndromes of this area.

Reference

1. Verdon F, Burnand B, Herzig L, Junod M, Pecoud A, Favrat B. Chest wall syndrome among primary care patients: a cohort study. BMC Fam Pract. 2007;8:51.

Spinal Accessory Nerve Entrapment

27

Agnes R. Stogicza

Introduction

The *spinal accessory nerve* (SAN), the 11th cranial nerve (CNXI), has a long superficial course in the neck, which makes it vulnerable to injury that is rarely recognized. SAN was traditionally believed to be a pure motor nerve, but later anatomical studies have confirmed that it has both sensory and motor components [1]. Entrapment of the SAN and/or chronic *compartment syndrome of the trapezius muscle* may cause chronic debilitating pain after *flexion-extension trauma*, without radiologic or electrodiagnostic evidence of injury. SAN palsy presents with isolated *sternocleidomastoid muscle* (SCM) and *trapezius muscle* weakness and atrophy ("*sloping shoulder syndrome*"), as well as shoulder and neck pain. The SAN is also sometimes simply called the "*accessory nerve*" [2, 3].

Clinical Presentation (Table 27.1)

Patients with SAN palsy present with ipsilateral neck, shoulder, neck, and occipital pain and headache (Figs. 27.1 and 27.2) or persistent contralateral spasm of the SCM and trapezius muscles, with asymmetric neckline, *winged scapula*, and sometimes *torticollis.* Pain is a common complaint (86 % of patients with SAN injury) [17], which may occur immediately, before the weakness and atrophy have developed. There may also be sensory changes over the angle of the jaw, the ear, the shoulder and the chest, which has been attributed to concomitant damage of the great auricular nerve (Chap. 16) [17].

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A sudden acceleration-deceleration incident (whiplash), especially with head turned (as in a motor vehicle accident in which the patient was looking in the rear view or side view mirror at impact), put the SAN at particular risk because it is stretched in this position and tethered at the level of the styloid [4, 5]. The SAN can also be injured iatrogenically during procedures such as radical neck dissections, lymph node biopsies or other *posterior triangle* interventions, often without radiologic or electrodiagnostic evidence of injury [18]. Walvekar and Li state that SAN injury occurs in 3–8 % of patients after cervical lymph node biopsy and up to 80 % of patients who have had a radical neck dissection [6].

Initially after denervation, the trapezius and SCM may be swollen; later they begin to atrophy. SAN injury causes paresis of both muscles on the ipsilateral side and unopposed contraction of the muscles on the contralateral side, leading to torticollis (Fig. 27.3), a symptom that can divert attention from the actual pathology. Often compensatory hypertrophy of the levator scapulae muscle is present on the ipsilateral side. As the patient develops weakness of the SCM and trapezius muscles, traction on the brachial plexus by the unsupported shoulder can result in a painful "*shoulder syndrome*" [8],

Table 27.1 Occupation/exercise/trauma history relevant to spinal accessory nerve entrapment

Trauma	Flexion-extension injuries [4, 5]
	Falls [5]
	Sports injuries [5]
	Superficial "love bite" on the neck [6]
Surgery	Cervical lymph node biopsy [7]
	Radical neck dissection despite nerve-sparing
	Techniques (30–40 %) [8–11]
	Carotid endarterectomy [12]
	Face lift [13]
	Rotator cuff repair [14]
	Internal jugular vein cannulation (3–8 %) [15]
Neuritis	Varicella infection [16]

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Fig. 27.1 Patient complaint of pain from spinal accessory nerve entrapment (Image courtesy of Andrea Trescot, MD)

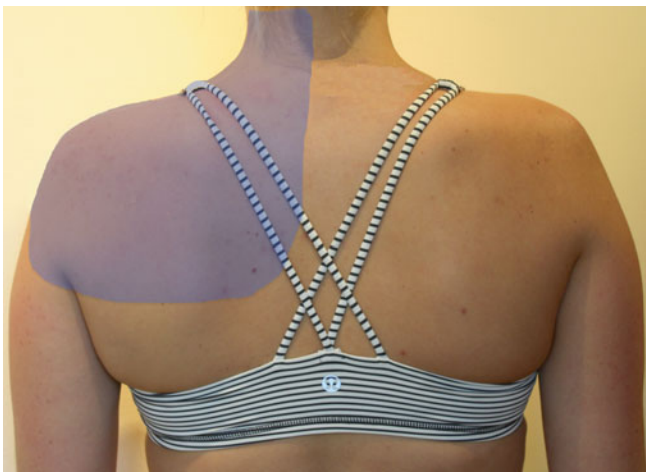

Fig. 27.2 Pain pattern associated with spinal accessory nerve entrapment (Image courtesy of Andrea Trescot, MD)

with an eventual frozen shoulder (*adhesive capsulitis)*. There may be a winged scapula (Fig. 27.4), a droop and internal rotation in the ipsilateral shoulder (Figs. 27.5 and 27.6) and

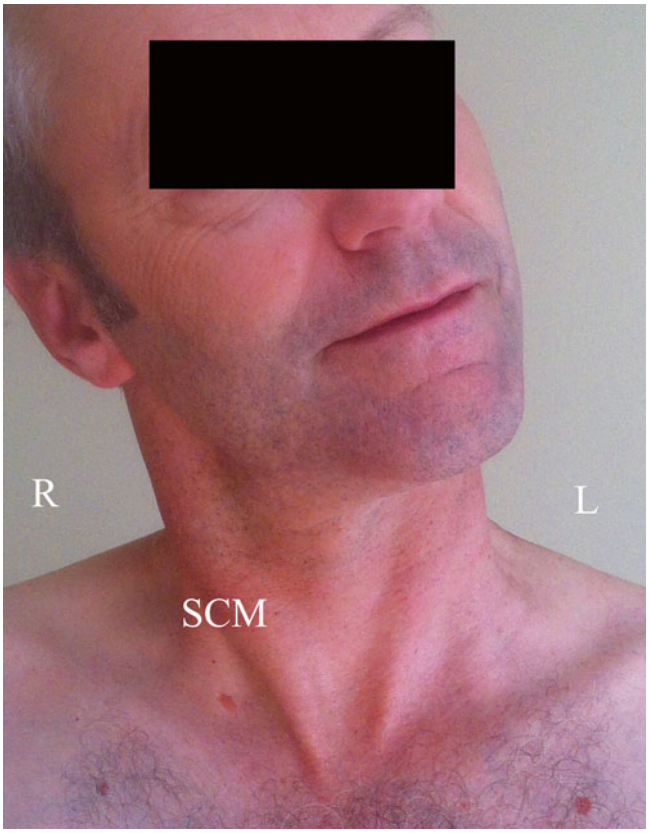

Fig. 27.3 Example of torticollis, causing shortening of the right sternocleidomastoid muscle, tilting the head to the right and turning it to the left (Image courtesy of Agnes Stogicza, MD)

atrophy of the trapezius and SCM (Figs. 27.5 and 27.6). The winging is usually most obvious when the patient actively externally rotates the shoulder against resistance. Subsequently the abnormal stresses on the clavicle can result in sternoclavicular joint hypertrophy or subluxation [19]. Pain in the shoulder will increase when its weight is not supported, and patients have decreased strength for overhead activities, such as putting away dishes. The most common sign is limited sustained abduction of the shoulder [6]. Lastly, there may be pain and spasm of the muscles on the contralateral side due to their unopposed function that diverts attention from the actual pathology.

Nystrom et al. [5] described the characteristics of 30 patients with whiplash injuries to the SAN, which included four clinical signs:

1. Asymmetric posture, typically with the shoulder elevated on the side of the greatest pain (lower on the pathologic side)
2. Decreased and painful motion in the neck and shoulder
3. Tenderness to palpation along the horizontal portion of the upper trapezius
4. Greater than 50 % reduction in pain and increased mobility following infiltration of local anesthetic into the upper trapezius

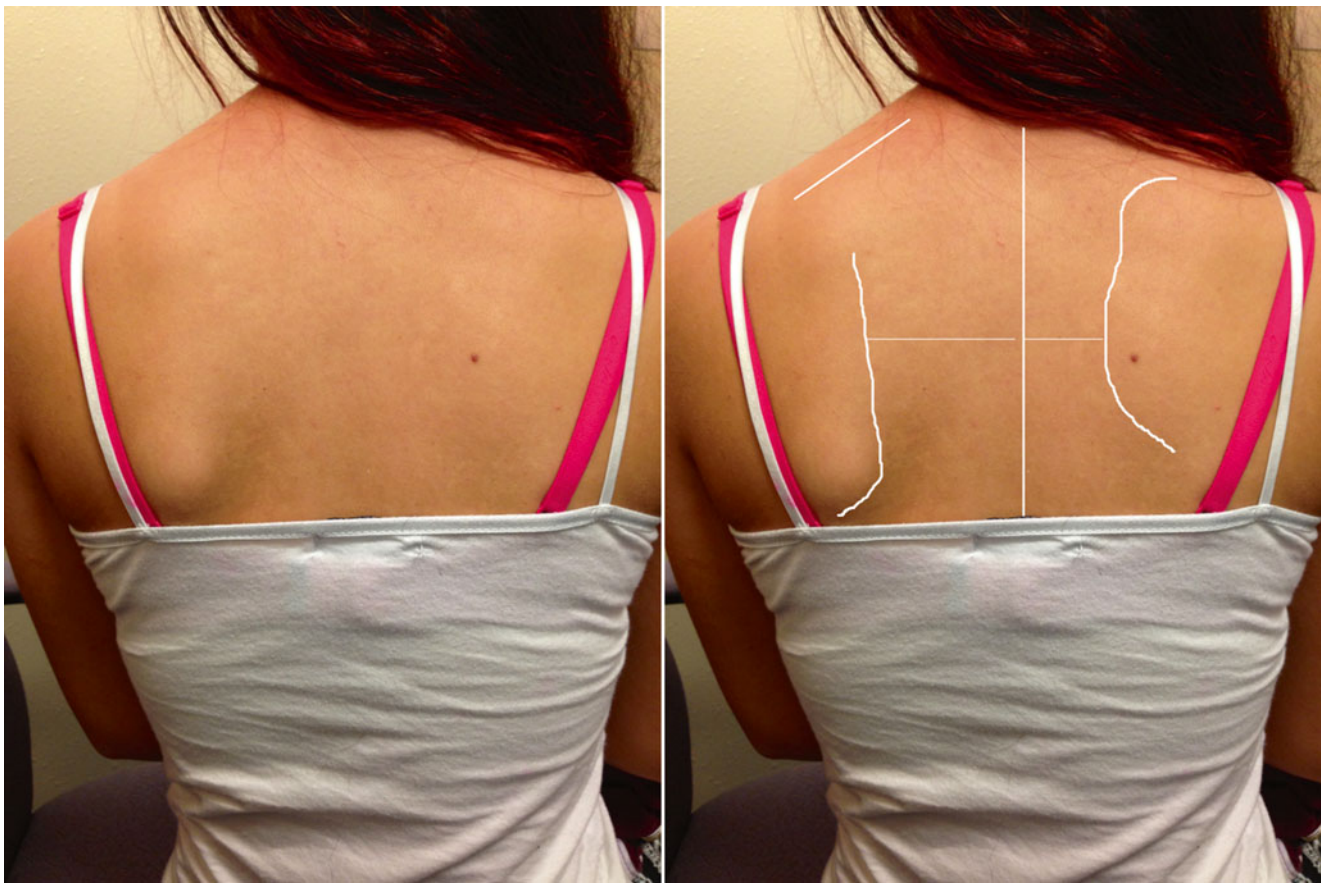

Fig. 27.4 Lateral winging of the left scapula after an MVA; winging is usually most obvious when the patient actively externally rotates the shoulder against resistance (Image courtesy of Andrea Trescot, MD)

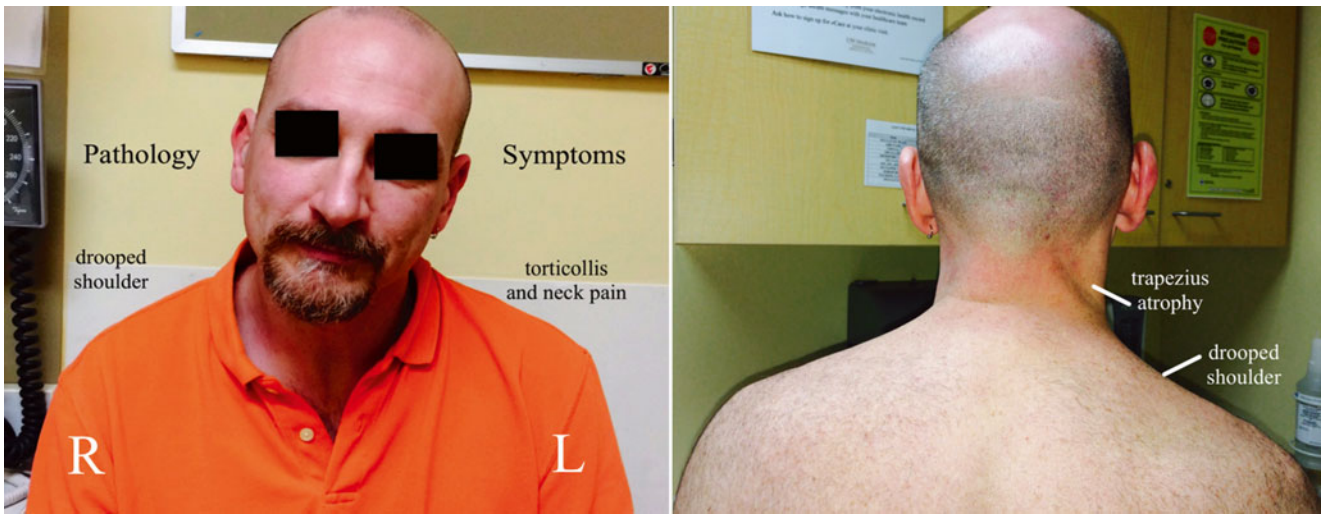

Fig. 27.5 Example of drooped shoulder from spinal accessory neuralgia. Note that the pathology (the spinal accessory neuralgia, trapezius atrophy) is on the right, but the symptoms (torticollis, shoulder pain) are on the left (Image courtesy of Agnes Stogicza, MD)

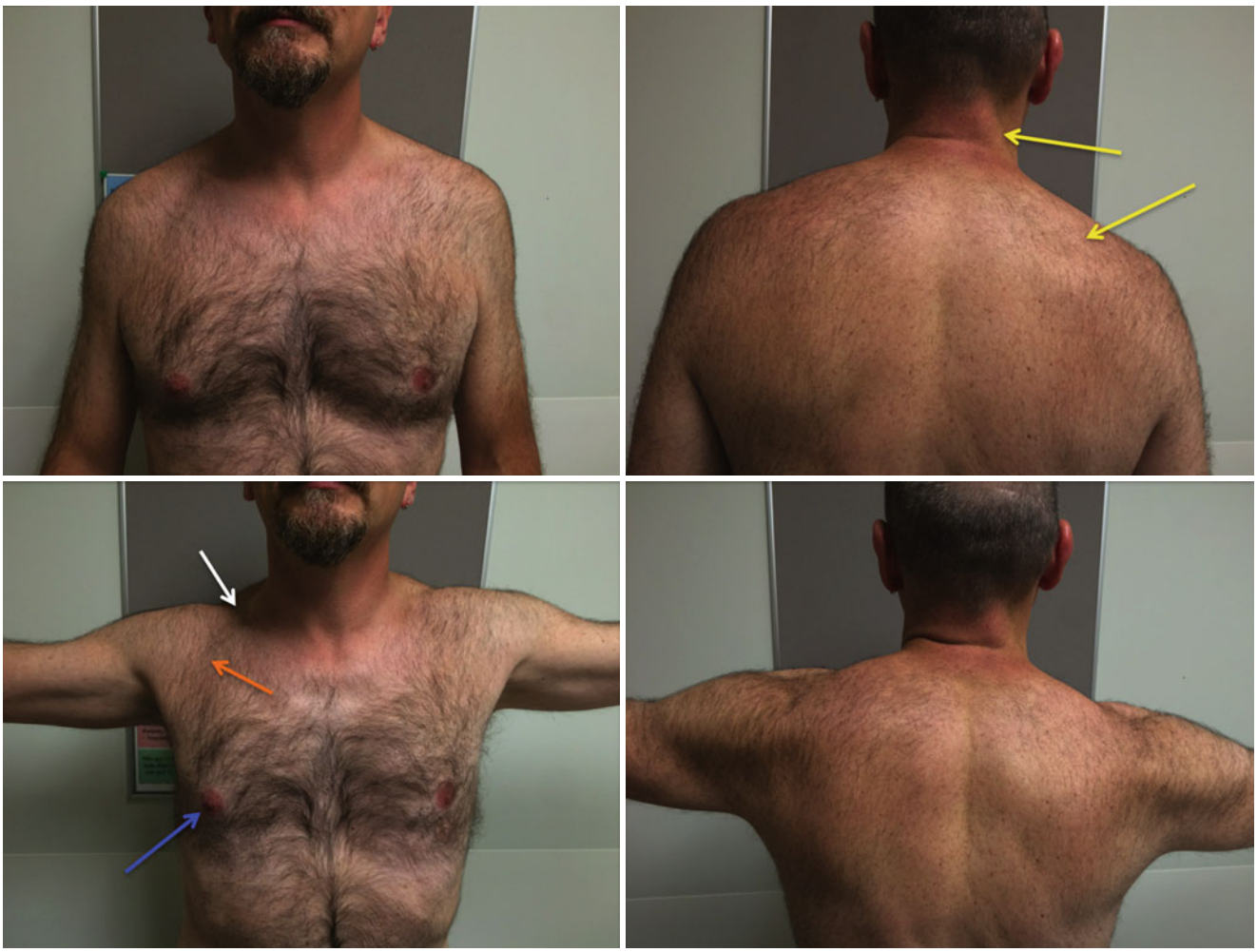

Fig. 27.6 Example of spinal accessory nerve lesion resulting in trapezius wasting, shoulder drop and asymmetry of the neck and shoulder. *Yellow arrows* point to the atrophied trapezius muscle, causing subclavicular and pectoral asymmetry. *White arrow* shows supraclavicular wasting, *orange arrow* shows subclavicular pitting and *blue arrow* shows pectoral drooping (Image courtesy of Agnes Stogicza MD)

27 of the 30 patients had head pain and headaches. After surgery to release the SAN (see *Surgery* section below), 10 of the 27 patients reported "complete relief," while 22 had 50 % relief. The authors concluded that some of the most common symptoms found in chronic flexion-extension injuries (e.g. headaches, stiffness of the neck and pain in the shoulder/neck region) may be due to either primary injury or secondary dysfunction of the spinal accessory nerve and/or the trapezius muscle.

Anatomy (Table 27.2)

The SAN is the 11th cranial nerve and had been traditionally thought to consist of two parts: spinal and cranial. The *cranial root* begins in the *nucleus ambiguous* in the medulla. The *spinal root* originates from a cluster of motor neuron cell bodies in the *accessory nucleus*, located in the lateral part of the anterior horn of the first five segments of the spinal cord, then travels cephalad through the foramen magnum where it was thought to join the cranial section. Once the two parts "join," the SAN exits the cranium through the *jugular foramen* with the *vagus nerve* and *glossopharyngeal* nerve (Figs. 27.7 and 27.8) [2], traveling behind the *styloid process* (Fig. 27.9). Even while traveling together, the spinal and cranial components make few if any distinct connections [3]; it is increasingly thought that what we commonly refer to as the SAN carries motor fibers from the cervical spinal cord and sensory fibers of unclear origin [1].

The spinal branch travels under the posterior belly of the *digastric muscle*; crosses the internal jugular vein, anterior or posterior to the occipital artery; and then passes beneath the SCM. It emerges posterior of the SCM, joins with fibers from C3 to C4, and then travels obliquely down across the floor of the posterior cervical triangle (on top of the levator scapula) to enter the trapezius muscle (Fig. 27.10); that distal section

Table 27.2 Spinal accessory nerve anatomy (CN XI)

Origin	*Cranial*: nucleus ambiguus (controversial) [3]
	Spinal: spinal accessory nucleus
General route	*Cranial*: joins spinal section inside cranium (controversial) [3]
	Spinal: travels cephalad to join cranial section
	Combined: exits cranium through jugular foramen. Cranial section joins vagus. Spinal section travels under digastric, posterior to SCM, enters trapezius
Sensory distribution	None, just communicating branches with vagus nerve, greater and posterior auricular nerve (GAN/PAN) (see Chap. 16), lesser occipital nerve (LON) (see Chap. 18)
Motor innervation	Trapezius (partial): also receives innervation from cervical plexus
	SCM (partial): also receives innervation from cervical plexus
Anatomic variability	Multiple connections to other nerves, including GAN/PAN, LON, trigeminal and hypoglossal nerves, cervical plexus, stellate ganglion, mandibular branch of the facial nerve, accessory phrenic nerve, brachial plexus
Other relevant structures	*Styloid process*
	Posterior cervical triangle (also known as the *lateral cervical region*): a region of the lateral neck bounded by the SCM anteriorly, the trapezius posteriorly and the clavicle inferiorly. It contains many blood vessels, most notably the external jugular vein, as well as lymph nodes and the trunks of the brachial plexus
	Cervical plexus: the ventral rami of C1–C4 unite and provide sensory and motor innervation to the anterior and lateral neck, including direct contributions to the SCM and trapezius muscles

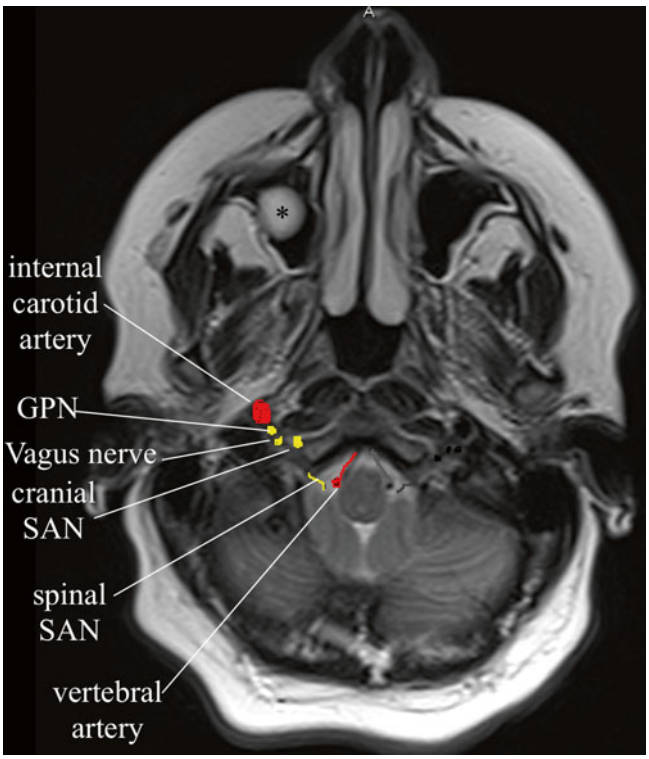

Fig. 27.7 MRI axial image at the level of the foramen magnum. *GPN* glossopharyngeal nerve, *SAN* spinal accessory nerve (cranial and spinal divisions), * = incidental maxillary sinus cyst (Image courtesy of Andrea Trescot, MD)

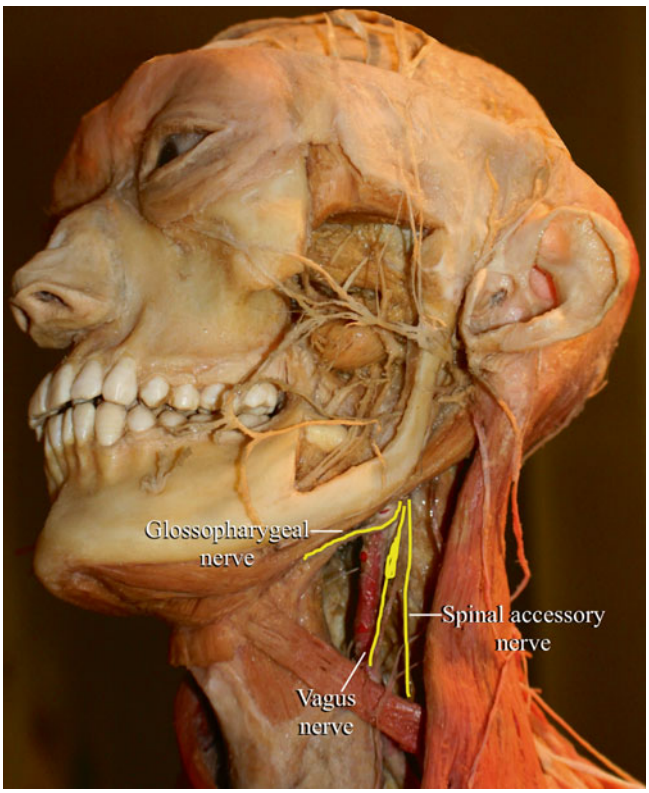

Fig. 27.8 Dissection of the head and neck, showing the relationship between the spinal accessory nerve, the glossopharyngeal nerve, and the vagus nerve in the neck next to the carotid artery (Image modified from an image from *Bodies, The Exhibition,* with permission) (Image courtesy of Andrea Trescot, MD)

is coiled and of variable length, depending on position (4–5 cm when lax with the chin pointing forward, but 9–10 cm when the chin is pointing to the opposite shoulder) [9].

The SAN innervates the trapezius and SCM muscles but has multiple connections to other nerves, likely contributing to the variable signs and symptoms associated with its injury. For instance, the *great or posterior auricular nerve* (Chap. 16) (which arises from C2 to C3) may

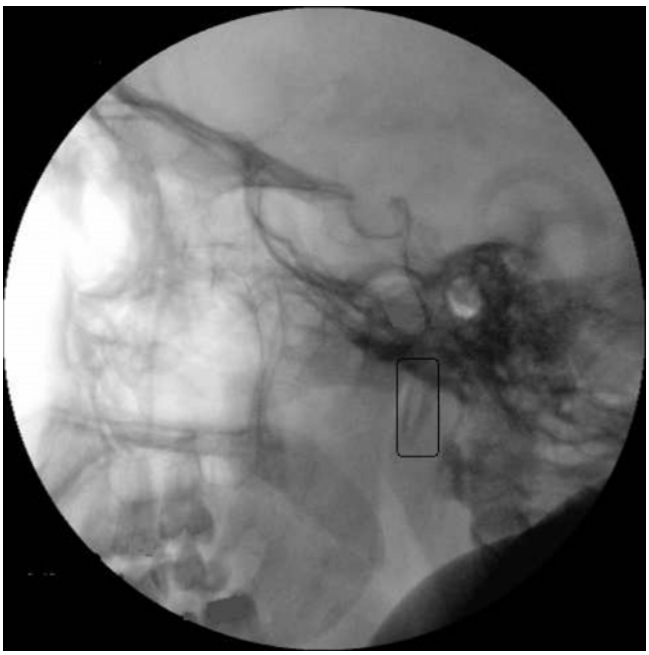

Fig. 27.9 X-ray image of the styloid process (*outlined*) (Image courtesy of Andrea Trescot, MD)

serve as a conduit between the SAN, the *lesser occipital nerve* (Chap. 18), and the lower divisions of the *trigeminal nerve* [9]. In the same way, the *cervical plexus*, which arises from C2, C3, to C4, connects the SAN to the *hypoglossal nerve* (via the ansa hypoglossi), the *stellate ganglion* and the *mandibular branch of the facial nerve* [10]. The SAN is also connected to the *accessory phrenic nerve* and the *brachial plexus* [16].

Entrapment

Because the SAN has been considered a pure motor nerve, the pain from SAN injury has been attributed to SAN entrapment caused by either trapezius compartment syndrome described below or SAN injury and entrapment at higher levels due to trauma. Patients often have ipsilateral of contralateral neck, shoulder, and occipital pain as well as headache and persistent muscle spasm. Nystrom et al. [5] described 16 patients with chronic whiplash symptoms and SAN entrapment after falls or sports injuries. At surgery,

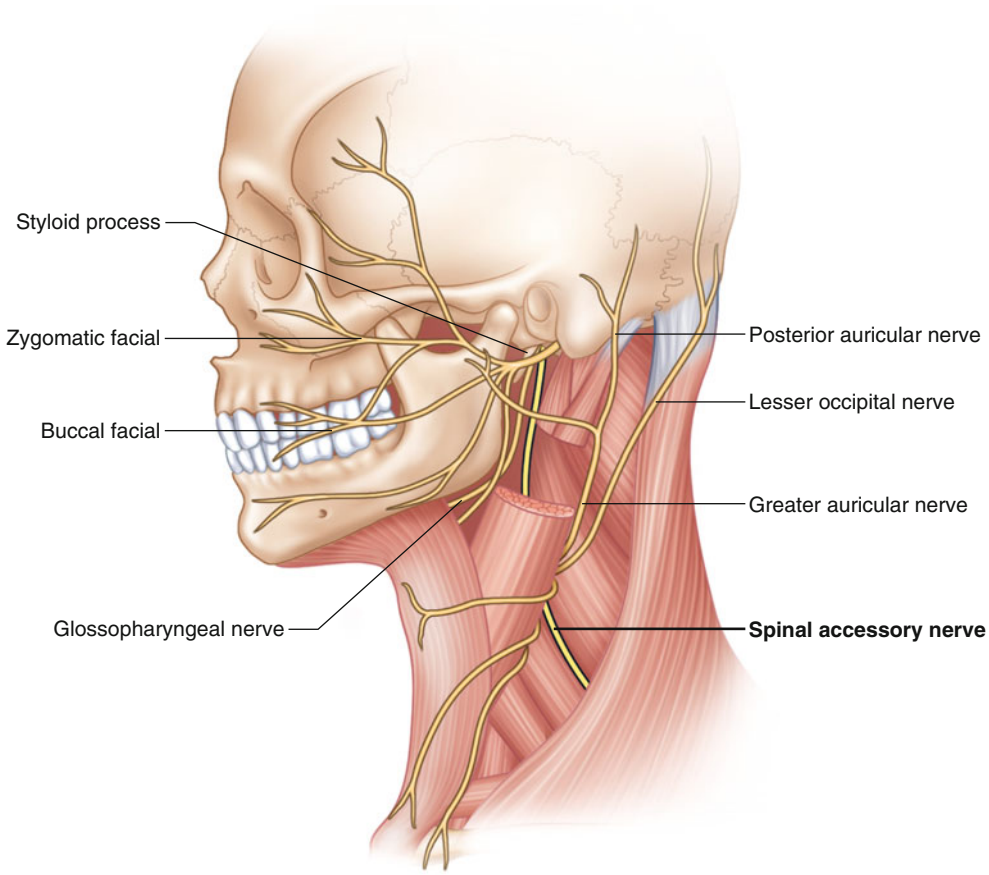

Fig. 27.10 Anatomy of the proximal spinal accessory nerve (Image courtesy of Springer)

they discovered adhesions between the SAN and underlying fascia. If the site is proximal, both the SCM and trapezius muscles will be involved; if it is at the more usual site in the posterior triangle, the trapezius alone is affected.

The *trapezius compartment syndrome* is thought to be due to mechanical drooping of the shoulder causing traction neuritis of the brachial plexus [20–23]. However, histologic evaluation of SAN samples has shown a high proportion of small unmyelinated C fibers, so that the entrapped SAN theoretically could directly carry pain signals [1].

Ewing and Martin first described SAN injury during radical neck dissections in 1952 [24]. This nerve is susceptible to injury because it is small (<2 mm diameter and therefore hard to see surgically) and has a long course across the posterior triangle of the neck, where it is intimately associated with many sets of lymph nodes [1], and postoperative scarring can contribute to entrapment symptoms. Motor neuron disorders, tumors or other neurologic pathology can be responsible for SAN entrapment. Spontaneous SAN dysfunction has also been reported [25].

Physical Exam

Physical examination focuses on assessment of the trapezius and SCM muscles. The patient is positioned standing with both scapulae visible, and areas of shoulder or neckline asymmetry, atrophy, swelling, taut bands and pain are noted (Fig. 27.6). The most consistent physical finding is weakness of arm abduction. The mechanism of injury should be considered when deciding if the muscles on one side have increased tone or the other side shows decreased activity. Tapping or applying pressure along the path of the SAN may elicit tenderness, especially where the SAN is tethered at the styloid process. The non-examining hand stabilizes the head on the contralateral side, while the examining thumb palpates the styloid process to elicit paresthesias (Fig. 27.11).

The other prominent feature of SAN palsy is lateral winging of the scapula. Have the patient stand with the arms by their side, flex the elbow to 90° and externally rotate the shoulder against the examiner's hand to elicit this sign (Fig. 27.4) [26]. If the SAN injury is proximal, SCM weakness on one side may cause unopposed contraction on the other, leading to torticollis (Fig. 27.5).

Restrepo et al. [17] noted a "*subclavicular pit*" (a concavity in the deltopectoral groove that results in a more clear outline of the clavicle) in six patients with EMG-confirmed SAN palsy; the patients were also noted to have a "*pectoral drooping,*" with a prominence and lateral deflection of the breast (Fig. 27.6).

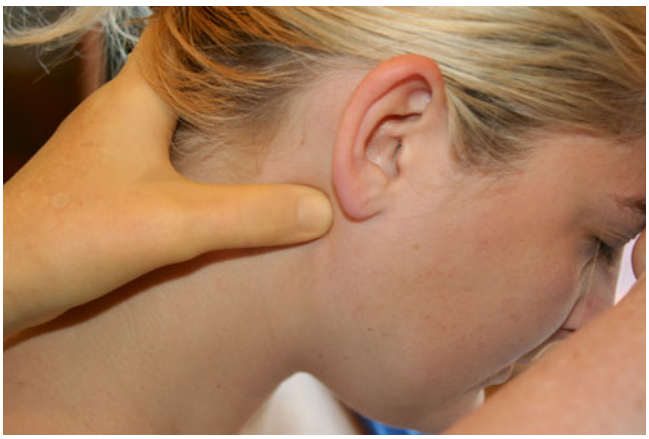

Fig. 27.11 Physical exam of the proximal spinal accessory nerve: the non-examining hand stabilizes the head on the contralateral side, while the examining thumb palpates the styloid process to elicit paresthesias (Image courtesy of Andrea Trescot, MD)

Table 27.3 Differential diagnosis of shoulder and neck pain

	Potential distinguishing features
Long thoracic nerve	Winged scapula on flexion or abduction, not external rotation
Myofascial spasms	No trapezius weakness or atrophy
Cervical facet pathology	Paravertebral tenderness, spondylosis on X-ray
Cervical radiculopathy	Dermatomal pain pattern, sensory changes, muscle weakness in a myotomal distribution, decreased reflexes

Differential Diagnosis (Table 27.3)

Diagnosis is mainly based on history (surgery, trauma or neurologic disorders) and physical exam. SAN palsy can be part of many pathologic lesions, mainly benign neoplasms that affect the 9th–11th cranial nerves. Complex 9th, 10th, and XIth cranial neuropathies (*Vernet syndrome*) usually imply a disease or tumor in the medulla, along the basal cistern, in the jugular foramen, or in nasopharyngeal carotid space; these neuropathies are also associated with aneurysms or basal skull fractures [16] (Fig. 27.7).

Lesions of the long thoracic nerve (see Chap. 30) may give similar winging of the scapula, but the lateral winging seen in long thoracic nerve pathology is elicited by forward flexion of the affected shoulder, while the medial winging of SAN palsy is accentuated by arm abduction or external shoulder rotation [2]. Myofascial trigger points in the cervical and shoulder muscles can give similar pain patterns, but they are not associated with trapezius weakness and atrophy or winged scapula [11]. The comparison of types of winging is found on Table 27.4, and the pattern of shoulder muscle atrophy due to nerve entrapments is found on Table 27.5 (Fig. 27.12).

Table 27.4 Comparison of winging from long thoracic, spinal accessory, and dorsal scapular nerve pathology

Nerve	Muscles involved	Type of winging	Provocative maneuvers
Spinal accessory	Trapezius	Lateral winging with drooping shoulder	Resisted arm abduction or external rotation
Long thoracic	Serratus anterior	Medial winging	Forward elevation and pushing with outstretched arms (wall push-up)
Dorsal scapular (a rare cause of subtle winging)	Rhomboid and levator scapulae	Scapula shifted laterally and dorsally	Slowly lowering arm from forward elevation

Table 27.5 Relationship between nerve entrapment and muscle atrophy

Nerve entrapment	Muscle atrophy
Spinal accessory nerve	Trapezius muscle and sternocleidomastoid muscle
Suprascapular nerve (Chap. 28)	Supraspinatus and infraspinatus muscle
Axillary nerve (Chap. 31)	Teres minor
Long thoracic nerve (Chap. 30)	Serratus anterior muscle
Dorsal scapular nerve (Chap. 32)	Rhomboid and/or levator scapula muscle

Diagnostic Tests (Table 27.6)

CT or MRI can help identify primary disease of SAN, e.g., neoplasm; it also helps to visualize muscle atrophy or hypertrophy of the SCM and trapezius caused SAN palsy and compensatory hypertrophy of other muscles (Fig. 27.12). Since the trapezius muscle may have dual nerve supply from the cervical plexus, it can retain some of its function in the face of SAN palsy, which might make the clinical picture less obvious. Other symptoms that result from the trapezius and SCM dysfunction, such as myofascial pain syndromes,

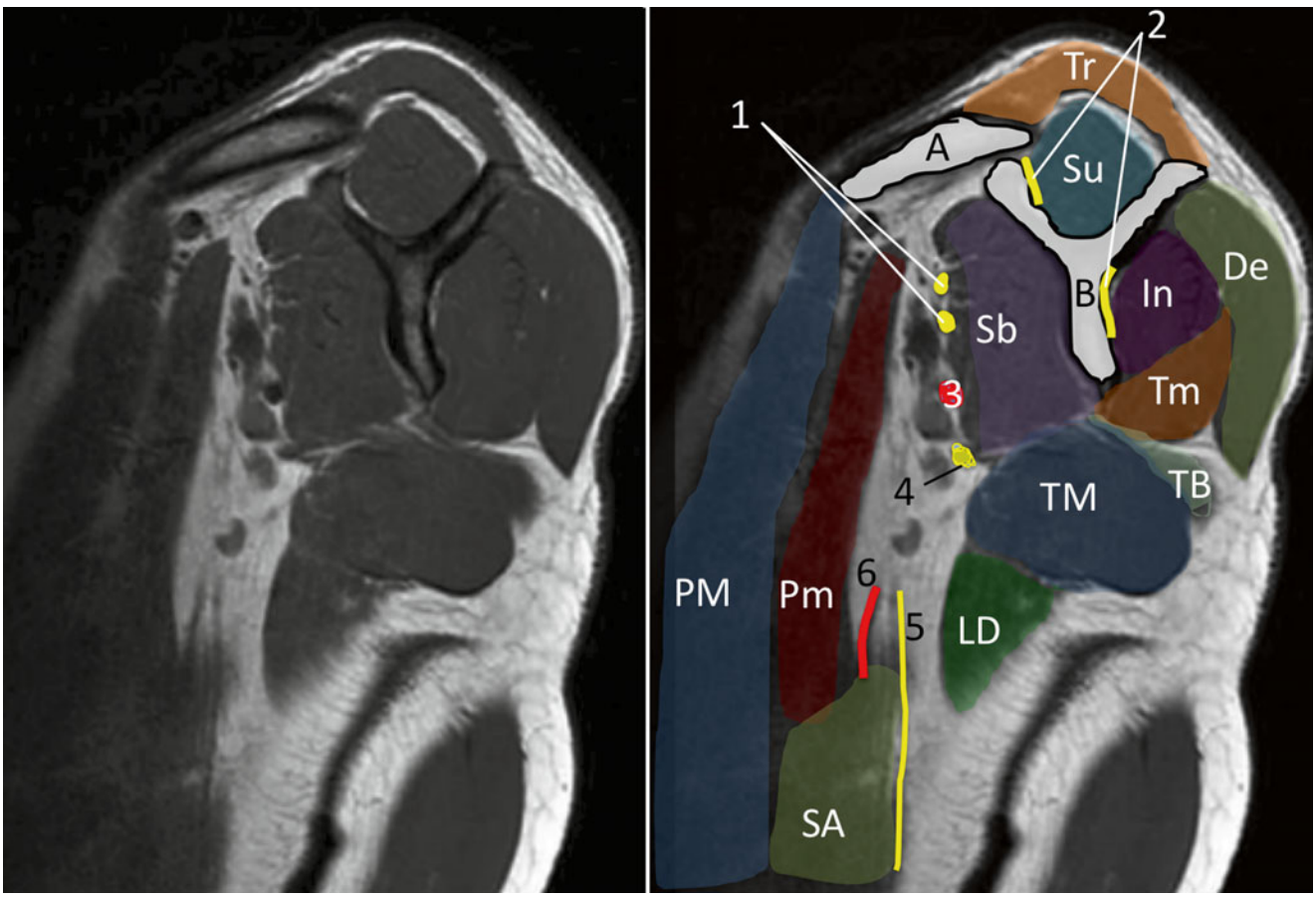

Fig. 27.12 Sagittal MRI image of the scapular structures. *A* clavicle, *B* scapula, *De* deltoid muscle, *In* infraspinatus muscle, *LD* latissimus dorsi muscle, *PM* pectoralis major muscle, *Pm* pectoralis minor muscle, *Sb* subscapularis muscle, *Su* suprascapular muscle, *TM* teres major muscle, *Tm* teres minor muscle, *Tr* trapezius muscle, *TB* triceps brachii muscle, *1* brachial plexus, *2* suprascapular nerve, *3* axillary artery, *4* suprascapular nerve, *5* long thoracic nerve, *6* long thoracic artery (Image courtesy of Andrea Trescot, MD)

Table 27.6 Diagnostic tests for spinal accessory nerve entrapment

	Potential distinguishing features
Physical exam	Scapular winging, tenderness at styloid, SCM/trapezius spasm or atrophy
Diagnostic injection	Pain relief with early injection may be diagnostic. In the face of atrophy or torticollis, injections may be useful on the contralateral side
Ultrasound	Visualizes nerve, possibly scarring or disruption in posterior triangle
MRI	May show atrophy of trapezius and SCM, scarring or neoplasm
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies	Useful in prevention, somewhat useful in confirming diagnosis

contralateral paresthesias and radiculitis, can further complicate the diagnosis.

Variations in presentation can also be attributed to the specific anatomic level of SAN injury, the amount of collateral tissue damage and subjective pain thresholds. EMG can aid in diagnosis of SAN palsy, but neither the CT and MRI nor the EMG changes are necessary [7].

Clinical symptoms, including the level of shoulder dysfunction, do not necessarily correlate with the electrophysiologic integrity of the SAN [27]. However, range of motion (ROM) testing has shown a positive correlation with EMG. EMG can also be used to track trapezius recovery, plan physical therapy and to monitor SAN function intraoperatively.

Identification and Treatment of Contributing Factors

Forward head posture and posterior cervical ligamentous laxity, because of the already compromised ergonomics, may contribute to accelerated disability, brachial plexus entrapment, thoracic outlet syndrome (see Chap. 33) and misdiagnosis. Early physical therapy for postoperative shoulder dysfunction is "mandatory" [8].

Several authors have described procedures to limit intraoperative damage to SAN, including intraoperative electrophysiologic monitoring [8, 28] and attention to positioning to avoid hyperextension and traction on the SAN.

Friedenberg et al. [29] looked at 56 spinal accessory neuropathy patients (confirmed by EMG) at Mayo Clinic over a 22-year period. Good functional recovery was generally observed, regardless of the results of the electrodiagnostic studies, and no electrodiagnostic findings correlated with poor outcome. However, involvement of the dominant limb, scapular winging and impaired arm abduction were all associated with poor outcome.

Injection Technique and Results

Landmark-Guided Technique

The landmark-guided technique should only be attempted in patients with a neck thin enough to palpate the styloid process. With the head supported, palpate and straddle the styloid process with index and middle fingers of the non-examining hand (Fig. 27.13). A 27-gauge 1.5-inch needle is usually long enough to reach the styloid process; the needle should touch bone and then be redirected posteriorly. A peripheral nerve stimulator (PNS) may help to identify the nerve more accurately. 1 to 2 ccs (1 cc if using PNS) of local anesthetic with or without steroids is injected in divided doses after negative aspiration. Since there are highly vascular structures nearby (specifically, the carotid artery), serious consideration should be given to using non-particulate steroids or no steroid at all, to mitigate the risk of a steroid particle vascular occlusion. Although this complication has not been reported for this particular injection, reports of disasters after injection of particulate steroids into other blood vessels should cause the clinician great concern.

Waldman [30] described a more distal approach to the SAN. With the patient supine and head turned to the contralateral side, the patient is asked to raise their head against the resistance of the examiner's hand in order to identify the posterior border of the upper third of the SCM (Fig. 27.14). The needle is advanced through the skin to a depth of about three-fourths of an inch and 10 cc of local anesthetic with deposteroid infiltrated in a fan configuration.

Fluoroscopy-Guided Technique

With the patient positioned supine, the styloid process is identified (Fig. 27.15). Under fluoroscopic guidance, the needle is advanced to the styloid process and then directed posteriorly. The use of a 25 g 2 inch needle with

Fig. 27.13 Landmark-guided injection of the glossopharyngeal nerve at the styloid process. The non-injecting fingers straddle the styloid process (Image courtesy of Andrea Trescot, MD)

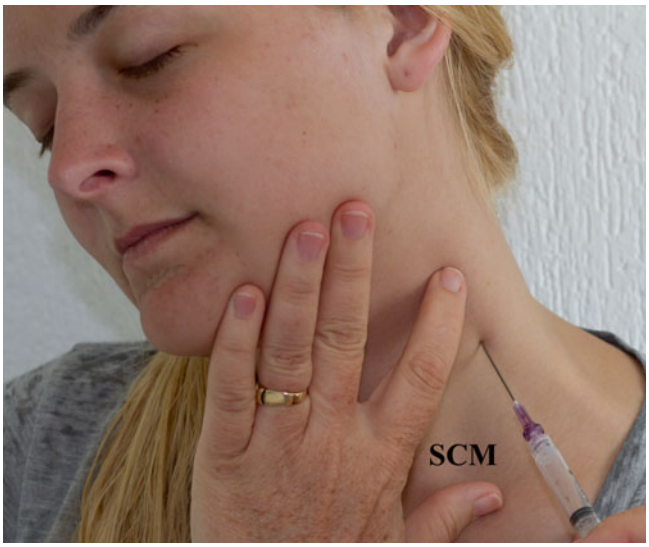

Fig. 27.14 Distal cervical injection of the spinal accessory nerve (Image courtesy of Andrea Trescot, MD)

peripheral nerve stimulator (PNS) will facilitate identification of the SAN by eliciting a scapular twitch. 1 to 2 ccs (1 cc if using PNS) of local anesthetic (with or without steroids) is injected in divided doses after negative aspiration.

Ultrasound-Guided Technique

Bodner et al. [31] originally described the US evaluation of the SAN. Ultrasound is less efficient visualizing the SAN at the level of the styloid process or above, but it easily shows the SAN in the posterior triangle. The probe is placed over the posterior triangle in a horizontal fashion, so the posterior border of SCM, levator scapulae muscle and possibly the anterior border of trapezius are visualized (Fig. 27.16). The nerve is then identified as a round hypoechoic structure in the connective tissue between SCM and levator scapulae muscle. As the nerve is tracked caudally, it moves superficially and posteriorly toward trapezius.

Canella et al. [32] evaluated the SAN by US in 7 cadavers and 15 volunteers and noted that bone landmarks were not useful for the accurate localization of the SAN. More recently, Mirjalili and colleagues [33] studied 50 healthy volunteers using US; the SAN could be identified in all the subjects running superficially across the posterior triangle with either a straight (56 %) or tortuous (44 %) course at a depth of about 3 mm. They noted that 58 % of the nerves divided into two to four branches before penetrating trapezius, which could lead to confusion at surgery.

Although not yet described in the literature, the injection can be performed by either an out-of-plane approach or an in-plane approach without major risks in well-trained hands, considering the very superficial location of SAN at this level.

Neurolytic/Surgical Techniques

Cryoneuroablation/Radiofrequency Lesioning

Since the SAN is primarily a motor nerve, neurolytic procedures are less common but may be appropriate for pain or to complete a partial denervation. There is most likely a potential for ultrasound-guided cryoablation or pulsed radiofrequency of SAN at the level of the posterior triangle, but no report has been published so far.

Pulsed radiofrequency lesioning of the SAN at the styloid has been performed using a combined fluoroscopy/ultrasound technique (personal communication Dr. Christ Declerck), which allowed precise identification of the styloid (fluoroscopy), while ultrasound confirmed the lack of vascular contact (Fig. 27.17).

Chemical Neurolysis

Because of the multitude of critical nerves and blood vessels in this region, alcohol or phenol would not be recommended.

Surgical Techniques

Nystrom et al. [5] performed surgical fasciectomies on 30 consecutive patients with chronic SAN pain after flexion-extension injuries, an average of 41 months after injury.

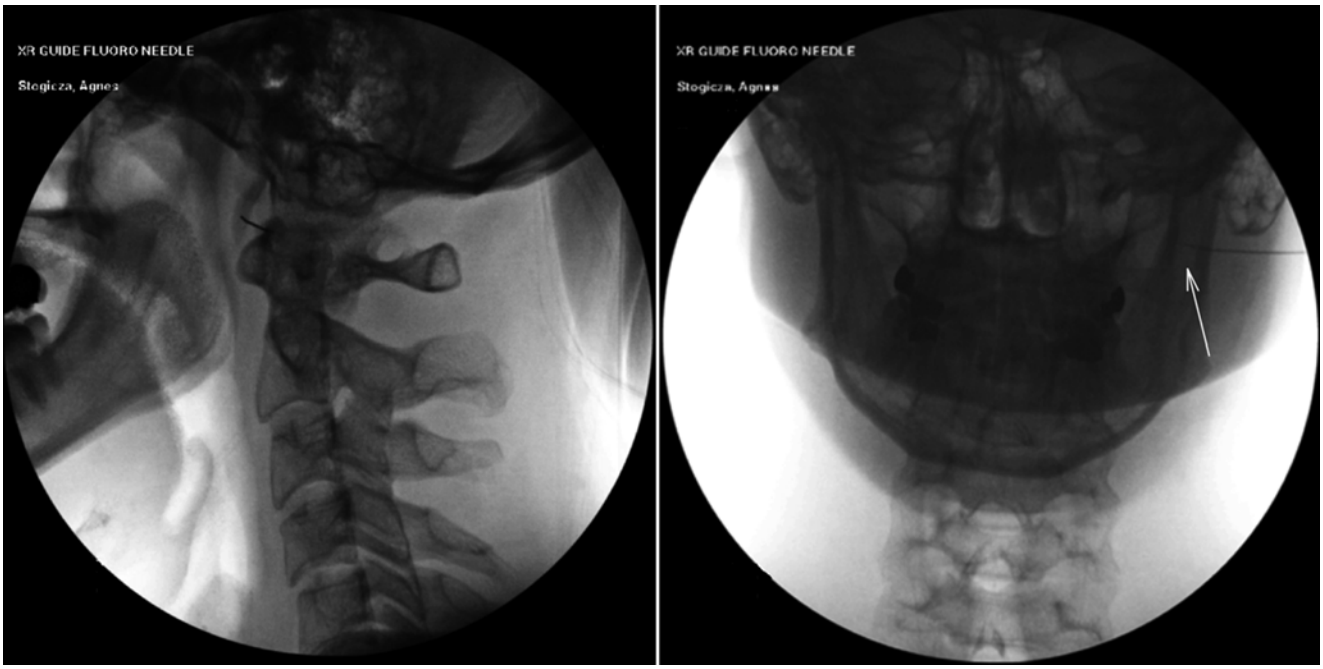

Fig. 27.15 Fluoroscopy-guided injection of the spinal accessory nerve. The lateral view shows the left and right styloid processes overlapping and a 22-gauge 4 cm needle placed onto the styloid process. After bony contact, the needle is redirected posteriorly. The anteroposterior view shows the needle placed onto the styloid process, marked by the white arrow (Image courtesy of Agnes Stogicza, MD)

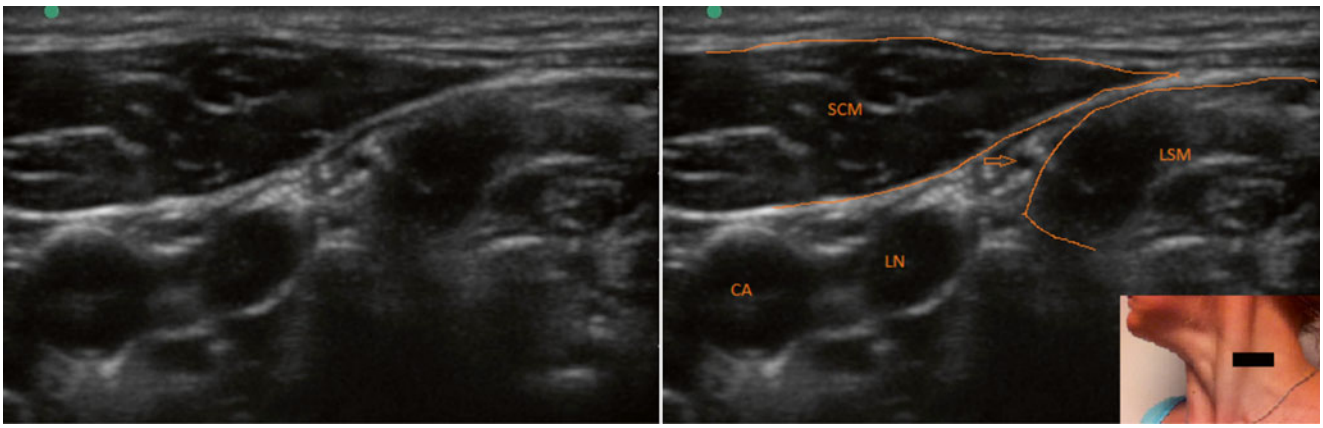

Fig. 27.16 Ultrasound image of the spinal accessory nerve (marked by the *arrow*) at the posterior triangle. *SCM* sternocleidomastoid muscle, *LSM* levator scapulae muscle, *CA* carotid artery, *LN* lymph node. Internal jugular vein is compressed (Image courtesy of Agnes Stogicza, MD)

Hagert and Christenson [34] reported that they treated chronic compartment syndrome of the trapezius and entrapment of the SAN in patients with a history of "*arm overuse syndrome,*" with a pain pattern similar to the pattern of pain seen in patients with chronic flexion-extension injuries. They described spinal accessory nerve decompression at the level of trapezius, recommending the removal of the thickened fascia, including the septa between bundles of the muscle.

Surgical repair of the spinal accessory nerve or muscle transfer is performed in patients with direct trauma of the SAN and also with spontaneous trapezius palsy [28]. Chandawarkar and colleagues [7] reported on the treatment of six patients with SAN dysfunction after cervical lymph node biopsies. Pain was the most common symptom and loss of sustained arm abduction was the most common finding. Three patients had a primary nerve repair and the other three patients had nerve grafting. All six were pain-free postoperatively, with varying degrees of motor function recovery. The authors stressed the need for prevention as well as early intervention rather than "watchful waiting."

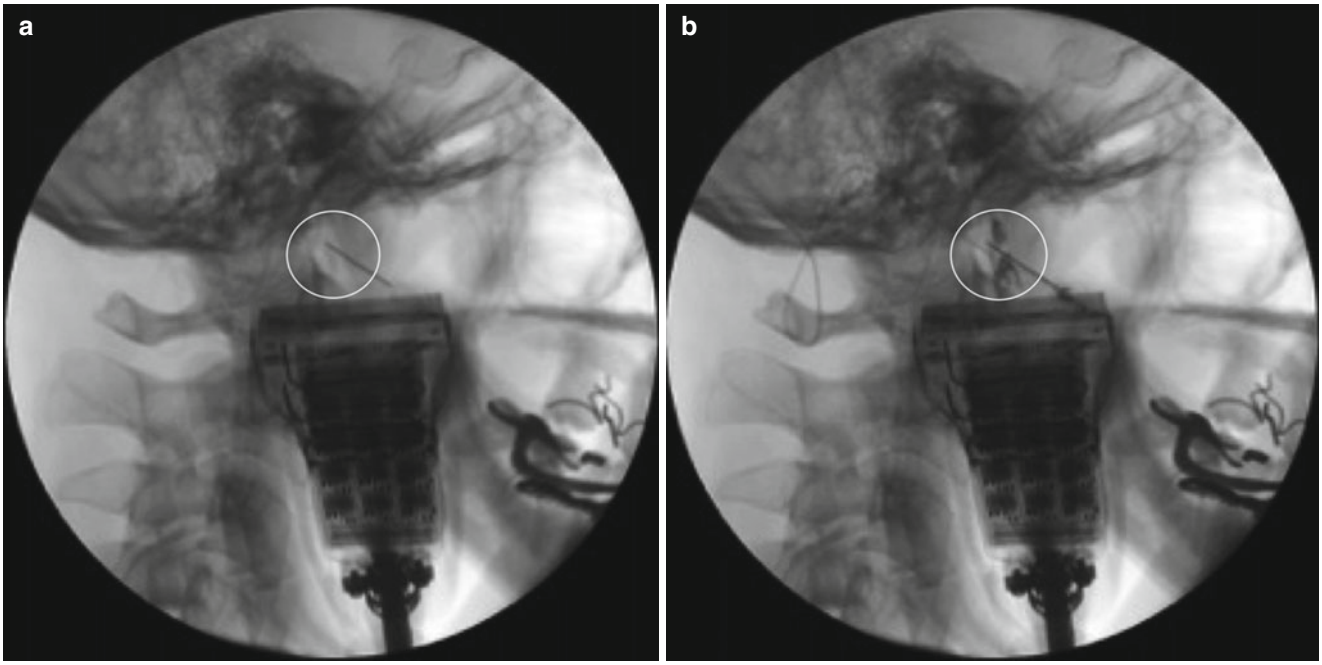

Fig. 27.17 Combined fluoroscopy/ultrasound-directed pulse radiofrequency lesioning of the spinal accessory nerve (Image courtesy of Christ DeClerck, MD General Hospital Sint Jan, Bruges AV, Belgium)

If SAN injury is diagnosed within 1 year, microsurgical reconstruction should be considered [35]. If diagnosed later, surgically repositioning the functioning shoulder muscles, known as the *Eden-Lange procedure,* can be performed; the insertions of the *levator scapulae*, *rhomboideus minor*, and *rhomboideus major* muscles are transferred, which relieves pain, corrects deformity, and improves function in patients with irreparable injury to the spinal accessory nerve [36, 37]. Treatment is less likely to succeed when the patient is older than 50 or the SAN palsy was due to a radical neck dissection, penetrating injury, or spontaneous palsy [28].

Complications

The SAN sits directly over the carotid artery and jugular vein, so intravascular injections are potentially encountered when injecting SAN at the styloid level. At the styloid level, the SAN is also in close relation with vagus, glossopharyngeal and hypoglossal nerves, and there may be unexpected anesthesia or neurolytic effect.

More distal injections (e.g., posterior triangle or in the trapezius muscle) carry significantly less risk when appropriate attention is paid to the depth, the external and internal jugular veins, the carotid artery and the apex of the lung. Ultrasound use, with constant visualization of the needle, should mitigate the risk of injuring these structures.

Summary

Injury to the SAN can be difficult to diagnose without a high index of suspicion. A careful history, identifying a flexion-extension injury or surgical trauma, as well as a careful physical exam, looking for trapezius and SCM atrophy and scapular winging, can lead to the correct diagnosis and therefore appropriate treatment.

References

1. Bremner-Smith AT, Unwin AJ, Williams WW. Sensory pathways in the spinal accessory nerve. J Bone Joint Surg Br. 1999;81(2): 226–8.
2. Tummala RP, Coscarella E, Morcos JJ. Surgical anatomy of the jugular foramen. Oper Tech Neurosurg. 2005;8:2–5.
3. Ryan S, Blyth P, Duggan N, Wild M, Al-Ali S. Is the cranial accessory nerve really a portion of the accessory nerve? Anatomy of the cranial nerves in the jugular foramen. Anat Sci Int. 2007; 82(1):1–7.
4. Bodack MP, Tunkel RS, Marini SG, Nagler W. Spinal accessory nerve palsy as a cause of pain after whiplash injury: case report. J Pain Symptom Manage. 1998;15(5):321–8.
5. Nystrom NA, Champagne LP, Freeman M, Blix E. Surgical fasciectomy of the trapezius muscle combined with neurolysis of the spinal accessory nerve; results and long-term follow-up in 30 consecutive cases of refractory chronic whiplash syndrome. J Brachial Plex Peripher Nerv Inj. 2010;5:7.
6. Walvekar RR, Li RJ. Accessory nerve injury: medscape. 2012. Available from: http://emedicine.medscape.com/article/1298684-overview.

7. Chandawarkar RY, Cervino AL, Pennington GA. Management of iatrogenic injury to the spinal accessory nerve. Plast Reconstr Surg. 2003;111(2):611–7; discussion 8–9.
8. Cappiello J, Piazza C, Nicolai P. The spinal accessory nerve in head and neck surgery. Curr Opin Otolaryngol Head Neck Surg. 2007;15(2):107–11.
9. Brown H, Burns S, Kaiser CW. The spinal accessory nerve plexus, the trapezius muscle, and shoulder stabilization after radical neck cancer surgery. Ann Surg. 1988;208(5):654–61.
10. Cappiello J, Piazza C, Giudice M, De Maria G, Nicolai P. Shoulder disability after different selective neck dissections (levels II–IV versus levels II–V): a comparative study. Laryngoscope. 2005; 115(2):259–63.
11. Kizilay A, Kalcioglu MT, Saydam L, Ersoy Y. A new shoulder orthosis for paralysis of the trapezius muscle after radical neck dissection: a preliminary report. Eur Arch Otorhinolaryngol. 2006; 263(5):477–80.
12. Woodward G, Venkatesh R. Spinal accessory neuropathy and internal jugular thrombosis after carotid endarterectomy. J Neurol Neurosurg Psychiatry. 2000;68(1):111–2.
13. Millett PJ, Romero A, Braun S. Spinal accessory nerve injury after rhytidectomy (face lift): a case report. J Shoulder Elbow Surg. 2009;18(5):e15–7.
14. Williams Jr GR. Painful shoulder after surgery for rotator cuff disease. J Am Acad Orthop Surg. 1997;5(2):97–108.
15. Leipzig B, Suen JY, English JL, Barnes J, Hooper M. Functional evaluation of the spinal accessory nerve after neck dissection. Am J Surg. 1983;146(4):526–30.
16. Jo YR, Chung CW, Lee JS, Park HJ. Vernet syndrome by varicella-zoster virus. Ann Rehabil Med. 2013;37(3):449–52.
17. Restrepo CE, Tubbs RS, Spinner RJ. Expanding what is known of the anatomy of the spinal accessory nerve. Clin Anat. 2015; 28(4):467–71.
18. Aker PD, Gross AR, Goldsmith CH, Peloso P. Conservative management of mechanical neck pain: systematic overview and meta-analysis. BMJ. 1996;313(7068):1291–6.
19. Piazza C, Cappiello J, Nicolai P. Sternoclavicular joint hypertrophy after neck dissection and upper trapezius myocutaneous flap transposition. Otolaryngol Head Neck Surg. 2002;126(2):193–4.
20. Nahum AM, Mullally W, Marmor L. A syndrome resulting from radical neck dissection. Arch Otolaryngol. 1961;74:424–8.
21. Sunderland S. Nerves and nerve injuries. 2nd ed. London: Churchill Livingstone; 1978.
22. Gabel G, Nunley JA. Spinal accessory nerve. In: Gelberman RH, editor. Operative nerve repair and reconstruction. Philadelphia: Lippincott; 1991. p. 445–52.
23. Kline DG, Hudson AR. Nerve injuries: operative results for major nerve injuries, entrapments, and tumors. Philadelphia: W.B. Saunders; 1995.
24. Ewing MR, Martin H. Disability following radical neck dissection; an assessment based on the postoperative evaluation of 100 patients. Cancer. 1952;5(5):873–83.
25. Ozdemir O, Kurne A, Temucin C, Varli K. Spontaneous unilateral accessory nerve palsy: a case report and review of the literature. Clin Rheumatol. 2007;26(9):1581–3.
26. Chan PK, Hems TE. Clinical signs of accessory nerve palsy. J Trauma. 2006;60(5):1142–4.
27. Erisen L, Basel B, Irdesel J, Zarifoglu M, Coskun H, Basut O, et al. Shoulder function after accessory nerve-sparing neck dissections. Head Neck. 2004;26(11):967–71.
28. Teboul F, Bizot P, Kakkar R, Sedel L. Surgical management of trapezius palsy. J Bone Joint Surg Am. 2005;87(Suppl 1(Pt 2)): 285–91.
29. Friedenberg SM, Zimprich T, Harper CM. The natural history of long thoracic and spinal accessory neuropathies. Muscle Nerve. 2002;25(4):535–9.
30. Waldman SD. Spinal accessory nerve block. In: Waldman SD, editor. Atlas of interventional pain management. 3rd ed. Philadelphia: Saunders Elsevier; 2013. p. 108–10.
31. Bodner G, Harpf C, Gardetto A, Kovacs P, Gruber H, Peer S, et al. Ultrasonography of the accessory nerve: normal and pathologic findings in cadavers and patients with iatrogenic accessory nerve palsy. J Ultrasound Med. 2002;21(10):1159–63.
32. Canella C, Demondion X, Abreu E, Marchiori E, Cotten H, Cotten A. Anatomical study of spinal accessory nerve using ultrasonography. Eur J Radiol. 2011;82(1):56–61.
33. Mirjalili SA, Muirhead JC, Stringer MD. Ultrasound visualization of the spinal accessory nerve in vivo. J Surg Res. 2012;175(1):e11–6.
34. Hagert CG, Christenson JT. Hyperpressure in the trapezius muscle associated with fibrosis. Acta Orthop Scand. 1990;61(3):263–5.
35. Donner TR, Kline DG. Extracranial spinal accessory nerve injury. Neurosurgery. 1993;32(6):907–10; discussion 11.
36. Wiater JM, Bigliani LU. Spinal accessory nerve injury. Clin Orthop Relat Res. 1999;368:5–16.
37. Bertelli JA, Ghizoni MF. Refinements in the technique for repair of the accessory nerve. J Hand Surg Am. 2006;31(8):1401–6.

Suprascapular Nerve Entrapment: Shoulder

28

Christopher J. Burnett and Helen W. Karl

Introduction

The *suprascapular nerve* (SN) is a mixed sensory and motor nerve that originates from the upper trunk of the *brachial plexus* and can be a cause of shoulder pain and weakness. There are two primary sites for entrapment of the SN the clinical presentation varies depending on the site of entrapment. Patients with proximal SN entrapment (at the suprascapular notch) primarily complain of poorly localized posterolateral shoulder pain and weakness. Diagnosis is made by injection at the suprascapular notch, using a peripheral nerve stimulator (PNS), fluoroscopy, ultrasound (US), or CT scan. Treatment includes cryoneuroablation, pulsed radiofrequency (PRF), surgery, or peripheral stimulation. Entrapment of the distal SN at the spinoglenoid notch causes much less pain and is discussed in Chapter 34.

Clinical Presentation (Table 28.1)

Signs and symptoms of SN entrapment depend on the location of nerve compression. Entrapment at the *suprascapular notch* results in significant, sudden onset of shoulder pain due to compression of the deep sensory fibers innervating the *glenohumeral* and *acromioclavicular joints* [2, 10]. The pain is described as a dull ache in the posterolateral aspect of the shoulder and scapular regions (Fig. 28.1) that may radiate into the ipsilateral shoulder, arm, or neck (Fig. 28.2). There can be limitations in abduction and external rotation. This condition is seen primarily in athletes or people performing repetitive overhead motions (e.g., weight lifting, baseball, tennis, swimming, carpentry). It can be triggered by a traumatic or other acute event, such as a lifting injury with the arm internally rotated (like carrying a heavy suitcase), but the onset is typically insidious, involving the dominant arm in patients (usually male) from 20 to 50 years of age. The patient may complain of shoulder weakness or fatigue, particularly in abduction and external rotation of the arm. Any forward

Table 28.1 Occupation/exercise/trauma history relevant to suprascapular nerve entrapment

Repetitive shoulder movements, especially external rotation and abduction, which usually cause entrapment at the spinoglenoid fossa [1]	Sports: baseball [2]; weight lifting [3]; swimming [2]; dancing [2]; tennis [4]; volleyball [2, 3]
	Carpentry [5]
Surgical positioning	Knee-chest position with the scapula protracted [2]
Carrying heavy objects [3]	Meat packers [3]
	Newsreel cameramen [3]
	Roofers [6]
Stretch and direct trauma	Fracture of scapula, humerus, clavicle [3]
	Anterior shoulder dislocation [3]
	Shoulder surgery [7]
	Skeet shooting [8]
Space occupying lesions are often associated with a trauma history	Ganglion cyst [3, 5]; lipoma [2]; hematoma [3]; tumor [3]
Other	Insertion of the spinoglenoid ligament onto the scapulohumeral joint, causing tension on the ligament (trapping the nerve) with arm movements [9]

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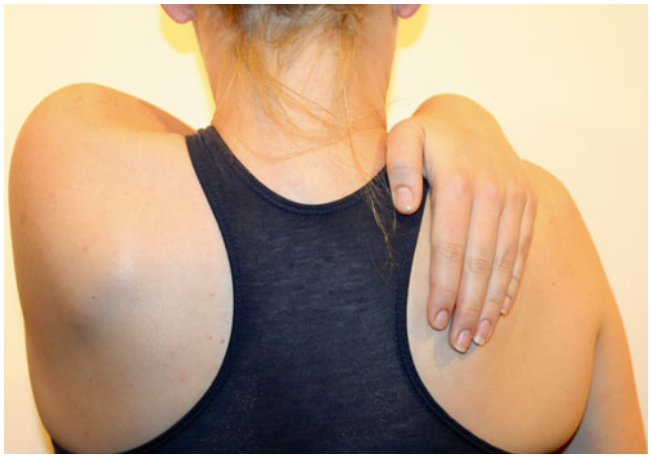

Fig. 28.1 Patient description of proximal suprascapular nerve pain (Image courtesy of Andrea Trescot, MD)

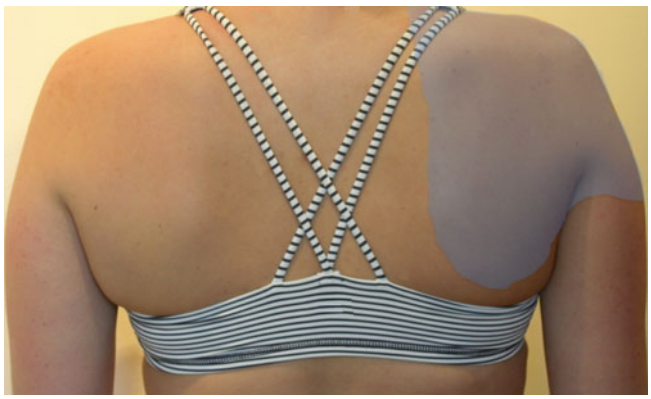

Fig. 28.2 Pain pattern associated with suprascapular nerve entrapment (Image courtesy of Andrea Trescot, MD)

movement of the scapula can elicit pain, including movements as simple as reaching across the chest. Patients can also develop a "*frozen shoulder*," (adhesive capsulitis), one cause of which is unwillingness to move the shoulder joint due to pain [1, 2, 10].

If the entrapment occurs more distally, at the *spinoglenoid notch*, the patient will have isolated atrophy and weakness of the infraspinatus muscle. In this situation, pain is largely absent because the deep sensory fibers to the shoulder joint exit proximal to this entrapment site [2, 3]. Liveson et al. [8] reviewed 13 reported cases of entrapment at the spinoglenoid notch most patients did not complain of pain or weakness, but rather came to medical attention because of insidious infraspinatus atrophy. They felt that the lack of weakness complaints was due to compensation by other muscles.

Brachial neuritis can also cause suprascapular neuropathy, though, in this case, weakness is not confined to the supraspinatus and infraspinatus muscles [2].

Anatomy (Table 28.2)

The suprascapular nerve originates from the fibers of the fifth and sixth cervical nerve roots, with frequent contributions (15–22 % of cases) [4] from the fourth cervical nerve root. It branches from the upper trunk of the brachial plexus and runs across the posterior cervical triangle adjacent to the omohyoid muscle. It passes under the *coracoclavicular ligament* and the *trapezius muscle* [4], then posteriorly to pass through the *suprascapular notch* (also known as the *supraspinatus notch* or *incisura scapulae*) (Fig. 28.3). Anatomic [11] and radiologic [4] studies show that the suprascapular notch can have a wide variety of shapes, ranging from almost flat, to u-shaped, to a deep groove, and even to a closed circle. The *transverse scapular ligament* encloses the superior portion of the suprascapular notch. This ligament is of variable thickness and can become partially or completely ossified, which can contribute to entrapment neuropathy. The position of the nerve in the notch is variable, making injection using anatomic landmarks alone difficult. Both the *suprascapular artery* and the *suprascapular vein* usually cross the upper edge of the scapula above the suprascapular ligament (Fig. 28.3) [1, 3, 10, 12], though a recurrent branch of the suprascapular artery may accompany the nerve through the notch [4]. Interestingly, the suprascapular notch has been found to be absent in 8% of 423 cadavers studied [13].

After passing through the suprascapular notch, the nerve travels underneath the *supraspinatus muscle* and provides its motor innervation. The SN, in combination with the *lateral pectoral nerve* [7], also supplies sensory branches to the shoulder capsule, *glenohumeral joint*, the *acromioclavicular* (*AC*) *joint*, the *coracoclavicular ligament*, and the

Table 28.2 Suprascapular nerve anatomy

Origin	A direct branch of the upper trunk of the brachial plexus (C4-C6)
General route	Crosses the posterior neck, under the trapezius, toward the suprascapular foramen, through the suprascapular notch/foramen, and then through the spinoglenoid notch
Sensory distribution [7]	Glenohumeral joint (shoulder joint)
	Acromioclavicular joint (shoulder joint)
	Subacromial bursa
	A patch of skin on the lateral upper shoulder
Motor innervation: 2 of the 4 rotator cuff muscles	Supraspinatus: Abducts humerus, especially the first 20–30°
	Infraspinatus: externally rotates humerus. The posterior deltoid and teres minor muscles also perform this function, so isolated infraspinatus muscle dysfunction may be asymptomatic [4]

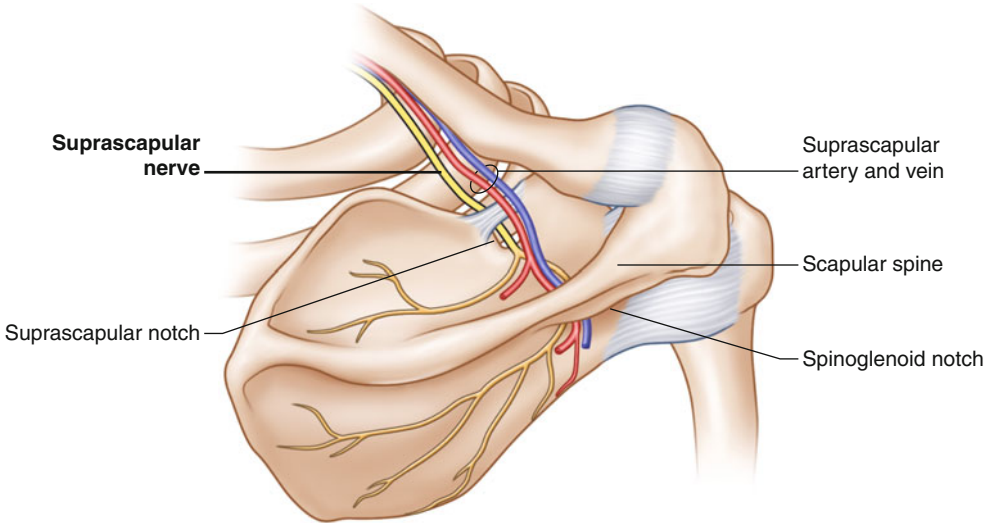

Fig. 28.3 Anatomy of the suprascapular nerve; note the suprascapular nerve (in *yellow*) as it passes underneath the transverse scapular ligament in the suprascapular notch. Note also that the suprascapular artery and vein lie above the transverse scapular ligament. Sensory branches from the shoulder joint not shown (Image courtesy of Springer)

subacromial bursa [4]. It has been estimated that the suprascapular nerve provides up to 70 % of the sensory innervation of the shoulder joint [10]. After innervating the supraspinatus muscle, the suprascapular nerve travels inferolaterally toward the rim of the *glenoid fossa*. It then makes a tight turn around the lateral edge of the scapular spine, beneath the *spinoglenoid ligament* (also known as the *inferior transverse scapular ligament*) [3], which creates a fibro-osseous tunnel called the *spinoglenoid notch* (or *infraspinatus notch*) through which the suprascapular nerve courses toward the *infraspinatus fossa* (Fig. 28.4). Here, the nerve provides motor innervation to the *infraspinatus muscle* [2, 3, 10, 14]. There is anatomic and clinical evidence for a small cutaneous branch, which in some people provides sensory innervation to a patch of skin on the lateral aspect of the upper shoulder [6, 15].

Entrapment

The anatomical course of the SN presents three distinct potential sites of entrapment: cervical origin, suprascapular notch, and spinoglenoid notch. The most common site of its entrapment is at the suprascapular notch. Kopell and Thompson [16] first described entrapment of the suprascapular nerve at the suprascapular notch in 1959. The nerve has little freedom of movement there, while the shoulder and scapula are extremely mobile. Rotator cuff tears can contribute to SN entrapment as the supraspinatus and/or infraspinatus muscle retracts [10]. The SN can be impinged by the sharp inferior border of the suprascapular ligament at the site of angulation at the notch, or by bony overgrowth, ligament thickening, or collocation of the artery and the nerve in the canal.

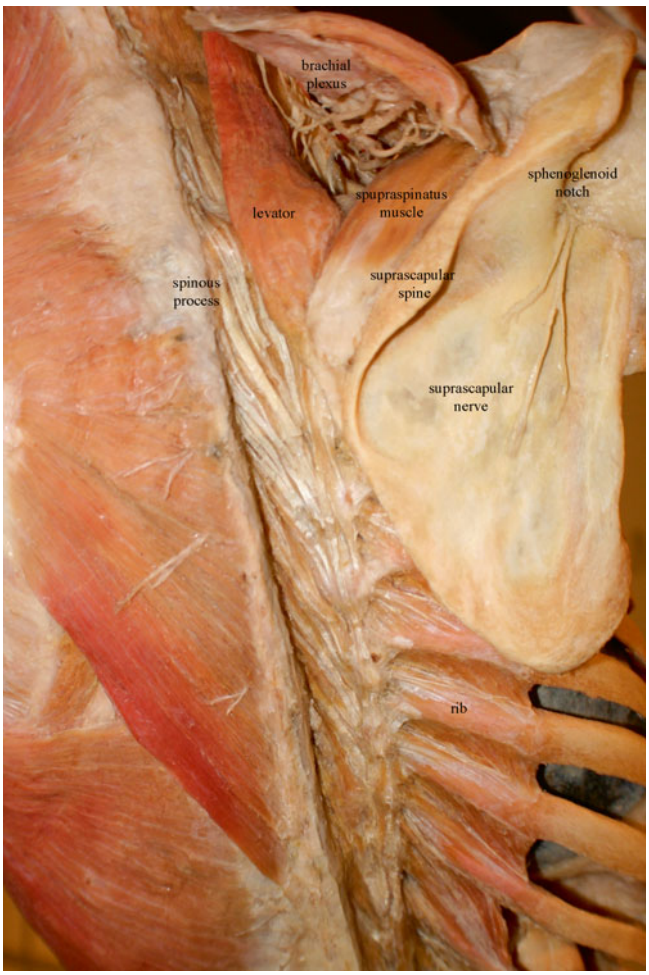

Fig. 28.4 Suprascapular anatomy, modified from an image from *Bodies*, *The Exhibition*, with permission (Image courtesy of Andrea Trescot, MD)

Similarly, spinoglenoid notch entrapment, first described in 1981 [17], occurs because the notch is a relatively fixed tunnel through which the nerve passes, limiting its mobility and predisposing to entrapment (see Chap. 34) [1, 2, 8, 10, 13]. Liveson and colleagues [8] suggest that the spinoglenoid entrapment may be more common than the entrapment at the suprascapular notch and needs to be recognized to avoid treatment of the wrong condition.

Physical Examination

Begin the physical examination of a patient with suspected SN entrapment by inspecting for atrophy of the supraspinatus or infraspinatus muscles. Atrophy is most easily visualized in the infraspinatus muscle, because the trapezius muscle can obscure the supraspinatus (Fig. 28.5). The clinician should evaluate the active range of motion of the shoulder, comparing it to the contralateral side, and looking specifically for limitation of abduction in the plane of the scapula or limitation of external rotation with the arm held in 90° of abduction. Specific provocative tests may reveal supraspinatus and/or infraspinatus weakness (see below) [18]. The clinician should then palpate the suprascapular notch to elicit pain (Video 28.1). The notch can be found by placing a hand on the patient's affected shoulder with the fingertips on the clavicle. The clinician then presses their thumb along the distal third of the scapular spine ("the Vulcan death grip") (Fig. 28.6). The examiner should then move their thumb inferiorly and laterally to palpate the spinoglenoid notch and evaluate for potential entrapment at that site [1, 2, 10, 14, 19]. Weakness in the initial 20–30° of arm abduction and forearm external rotation in comparison to the non-affected side may be noted. The *cross-body adduction test* described by Kopell and Thompson [16] (Fig. 28.7), which is performed by having the patient elevate the arm to 90° and forcibly adduct the arm across their chest, can tighten the spinoglenoid ligament and compress the nerve to reproduce the patient's symptoms.

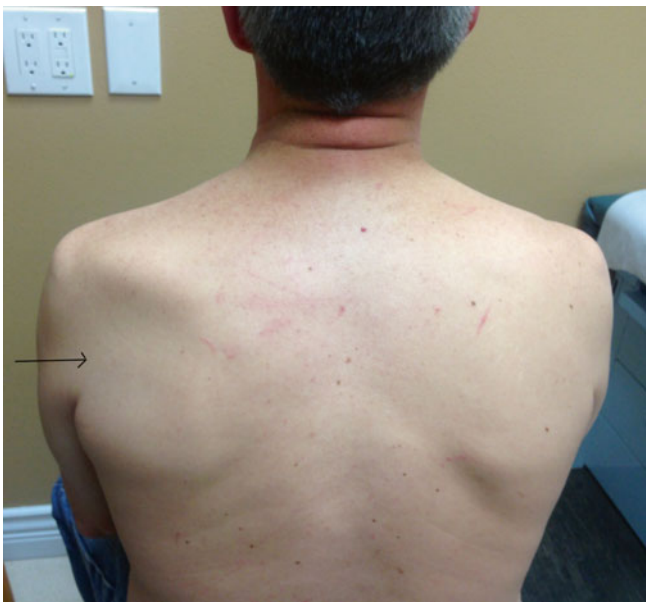

Fig. 28.5 Physical exam of supraspinatus and infraspinatus atrophy from suprascapular nerve entrapment. *Arrow* shows left infraspinatus atrophy (Image courtesy of Andrea Trescot, MD)

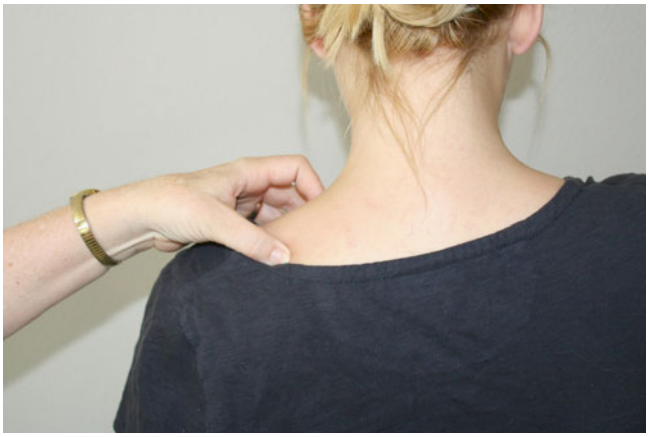

Fig. 28.6 Palpation of the suprascapular notch to elicit pain (Image courtesy of Andrea Trescot, MD)

Fig. 28.7 Cross-body adduction test (Image courtesy of Andrea Trescot, MD)

Differential Diagnosis (Table 28.3)

The differential diagnosis of shoulder pain includes *rotator cuff pathology* [10] or other joint related abnormalities, *shoulder impingement syndrome* [23], *labral tear*, *adhesive capsulitis* [10], *Parsonage-Turner syndrome* [2, 10], *thoracic outlet syndrome* (Chap. 33), and pain referred from cervical structures [23]. Parsonage-Turner syndrome almost always

Table 28.3 Differential diagnosis of chronic shoulder pain

	Potential distinguishing features
C5-6 radiculopathy	Usually have neck pain, decreased arm reflexes, as well as shoulder weakness [2]
Upper thoracic radiculopathy	MRI of the thorax will show HNP
Brachial plexopathy	Usually have weakness beyond the SN distribution; reflex and sensory changes [2]
Rotator cuff injury	Pain with passive movement; pain on palpation other than at suprascapular notch [2, 10]. Seen well on MRI
Glenohumeral joint pathology	Both active and passive range of motion are limited [12, 20]
Paralabral/ganglion cyst	Usually associated with labral tears; seen well on MRI [5, 19]
Varicosities	Compression in spinoglenoid notch [21]
Adhesive capsulitis	"Frozen shoulder" [12]. More likely in older patients and those with diabetes or thyroid disease [22]
Brachial neuritis (Parsonage-Turner)	Involvement of other nerves of the brachial plexus; frequently a sudden onset; may be bilateral. Patients are generally older, perhaps with a history of recent vaccination or infection [4, 10]

Table 28.4 Relationship between muscle pathology and nerve entrapment

Denervated muscle	Suspected nerve entrapment
Supraspinatus and infraspinatus muscle	Suprascapular nerve at suprascapular notch
Trapezius muscle	Spinal accessory nerve (Chap. 27)
Isolated infraspinatus muscle	Suprascapular nerve at spinoglenoid (Chap. 28)
Serratus anterior muscle	Long thoracic nerve (Chap. 30)
Teres minor muscle	Axillary nerve at quadrilateral space (Chap. 31)
Rhomboid and/or levator muscle	Dorsal scapular nerve at scalene muscle (Chap. 32)

Table 28.5 Diagnostic tests for suprascapular nerve entrapment

	Potential distinguishing features
Physical exam	Atrophy of supraspinatus, infraspinatus; weakness with abduction/external rotation; cross-body adductor test
Ultrasound	May show edema of the muscle
Electrodiagnostic studies	Needle EMG should show axonal loss limited to SN distribution. Motor NCSs may show reduced amplitude
MRI	Edema, atrophy, or fatty infiltration of supraspinatus and/or infraspinatus muscles

affects the SN but may involve the axillary nerve (Chap. 31) in 50 % of the cases [10]. The possibility of axillary nerve entrapment should also be considered.

Diagnostic Studies (Tables 28.4 and 28.5)

Diagnosis may be aided by electrodiagnostic studies, magnetic resonance imaging, or high-resolution ultrasonography. X-rays may show an unusual suprascapular notch. EMG/NCV can show denervation changes, such as an increase in distal motor latency and signs of denervation

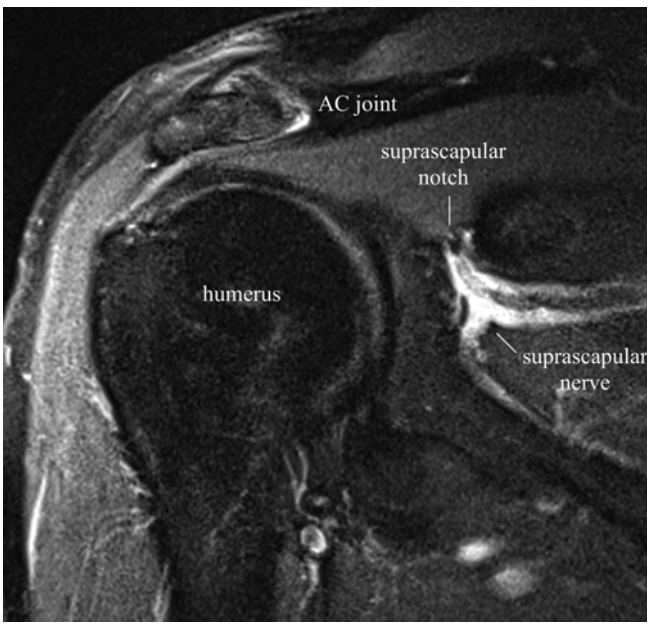

Fig. 28.8 MRI showing the suprascapular nerve (Image courtesy of Andrea Trescot, MD)

(increased spontaneous activity, positive sharp waves, fibrillation, polyphasic activity, decreased amplitude evoked potentials, and single unit recruitment of normal motor unit potentials) [4]. MRI (Fig. 28.8) may show atrophy, tumors, or cysts and may identify other causes of shoulder pain, such as rotator cuff tears or cervical radiculopathy. Edema of the denervated muscle (supraspinatus and/or infraspinatus) (Figs. 28.9 and 28.10) (Table 28.4) may be the surest and earliest characteristic sign of SN entrapment [4] and can help to differentiate SN entrapment from pure rotator cuff tears. US may also be useful for diagnosis; Polguj et al. [24] looked at 60 shoulders, comparing the evaluation of the suprascapular notch by US and CT; US was able to correctly evaluate the shape and size of the suprascapular notch in 42 of those patients.

Hill et al. [25] retrospectively reviewed 65 consecutive patients diagnosed with SN neuropathy and contacted them

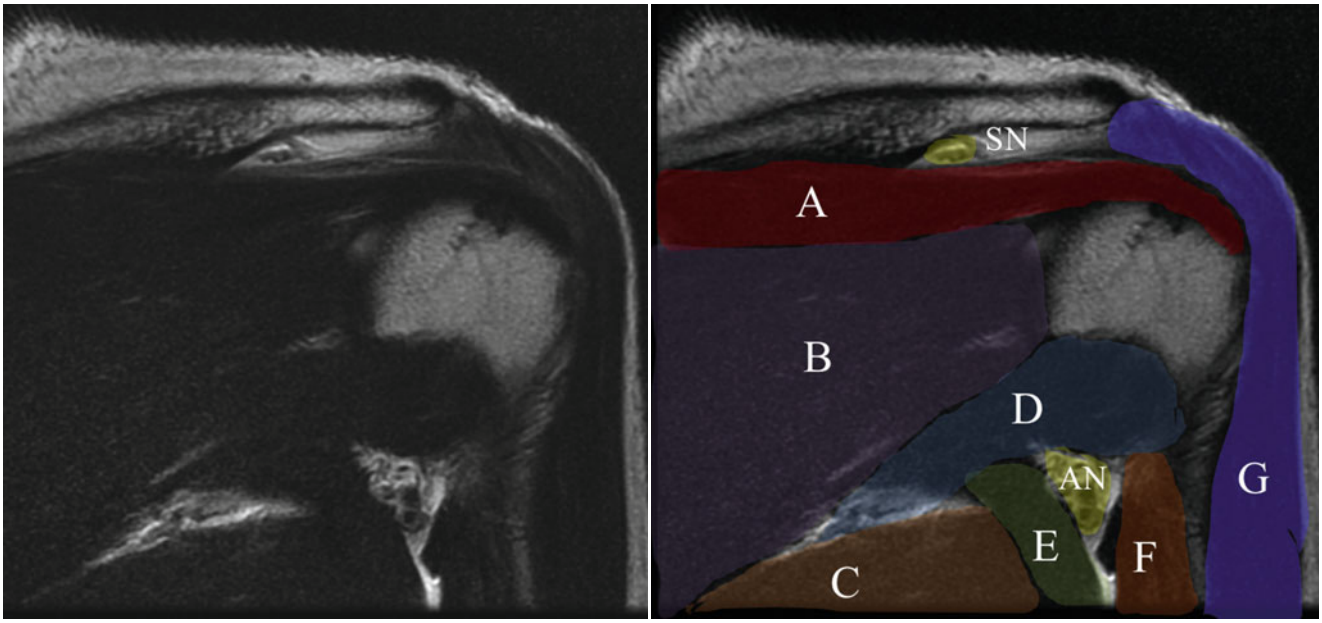

Fig. 28.9 Labeled MRI of the shoulder. *SN* suprascapular nerve, *AN* axillary nerve and axillary vessels, *A* supraspinatus muscle, *B* infraspinatus muscle, *C* teres major, *D* teres minor, *E* long head of the triceps muscle, *F* short head of the triceps, *G* deltoid muscle (Image courtesy of Andrea Trescot, MD)

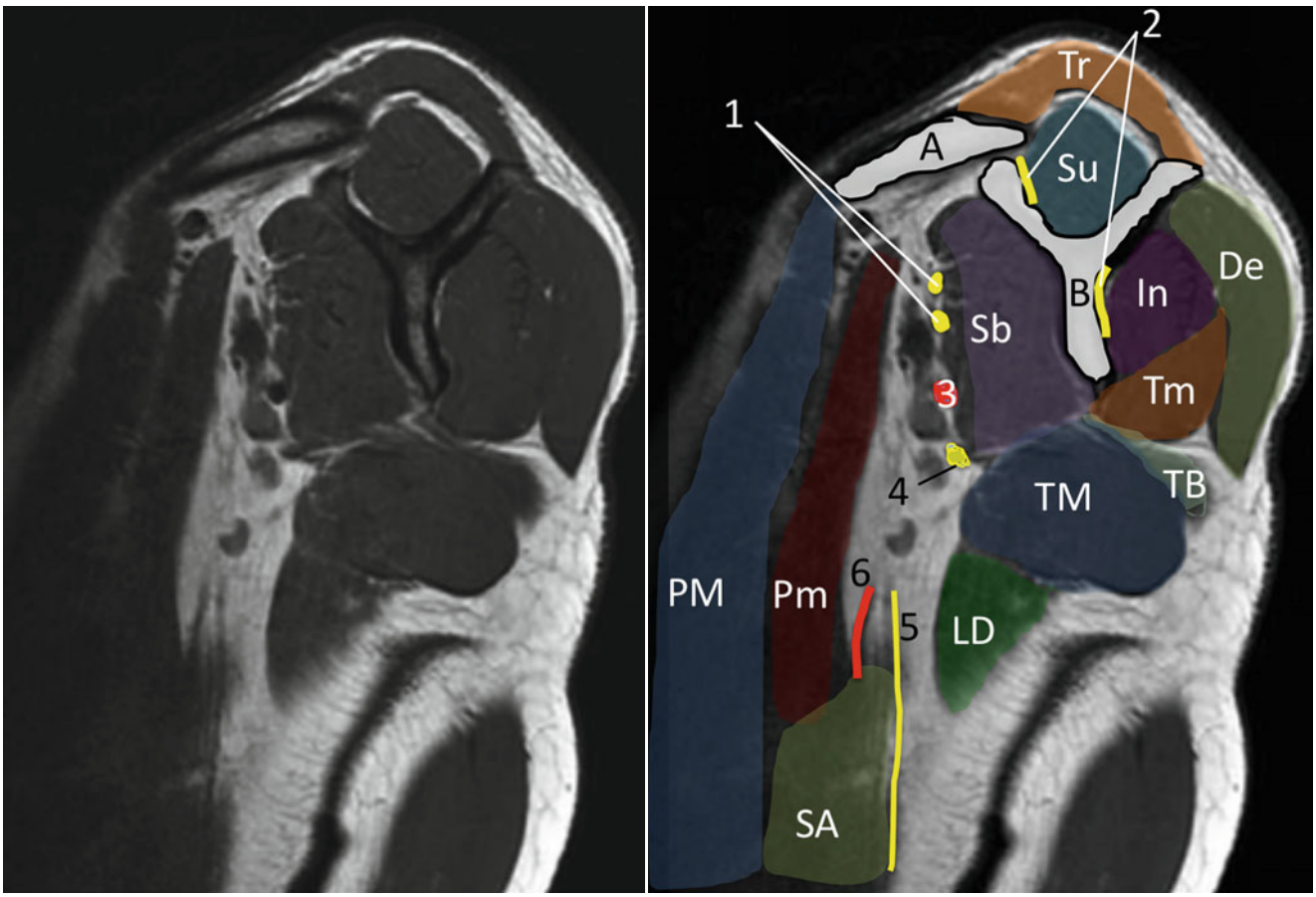

Fig. 28.10 Sagittal MRI image of the scapular structures. *A* clavicle, *B* scapula, *De* deltoid muscle, *In* infraspinatus muscle, *LD* latissimus dorsi muscle, *PM* pectoralis major muscle, *Pm* pectoralis minor muscle, *Sb* subscapularis muscle, *Su* suprascapular muscle, *TM* teres major muscle, *Tm* teres minor muscle, *Tr* trapezius muscle, *TB* triceps brachii muscle, (*1*) brachial plexus, (*2*) suprascapular nerve, (*3*) axillary artery, (*4*) suprascapular nerve, (*5*) long thoracic nerve, (*6*) long thoracic artery (Image courtesy of Andrea Trescot, MD)

5 years later. Fibrillation potentials were observed in 81 % of the patients. Seventy-five percent underwent at least one MRI, while 11 % had CT scan and five patients underwent US evaluation (four of the five also had an MRI; there was complete concordance between those two tests regarding cyst identification). Labral and rotator cuff tears were seen in more than 40 % of the patients.

The diagnosis of an axillary nerve (AN) entrapment depends on a high level of suspicion from the clinician. AN (Chap. 31) or SN entrapment should be considered when there appear to be several shoulder muscles involved [10]. Physical exam and diagnostic injections provide significant diagnostic clues, which can be confirmed by MRI, US, and electrodiagnostic studies.

Identification and Treatment of Contributing Factors

One common contributing factor of SN entrapment arises from overuse of the muscles of the rotator cuff. Because this nerve supplies the muscles that allow for the abduction and external rotation of the arm, overuse of these muscles, especially the supraspinatus, can lead to muscle spasms. These spasms can potentially trap the suprascapular nerve as it enters the scapular notch.

The suprascapular notch itself is highly variable in shape and size, which may contribute to impingement. Similarly, the transverse scapular ligament can vary significantly in its thickness and shape and may also be partially or completely calcified [10, 11].

Mass lesions can also contribute to SN entrapment, specifically a lipoma or ganglion cyst near the nerve. The latter is often associated with labral tears and has a tendency to form near the spinoglenoid notch [19].

Injection Techniques

Landmark-Guided Injection Technique

There are multiple approaches for landmark-guided injections of the SN. We will describe two different approaches, one with and one without the use of a peripheral nerve stimulator. To perform the first landmark-guided technique, the patient is placed in a seated or prone position with their arms to their sides. The clinician begins by inserting a 25-gauge, 2-inch needle at the distal third of the scapula and directing it vertically down to the scapular spine (Video 28.1). The needle is then advanced anteriorly and superiorly, maintaining contact with the scapula until it is felt to advance into the scapular notch (Fig. 28.11). After negative aspiration for blood or air, the planned injectate can be administered. Pneumothorax is a significant clinical concern with this technique, as the cupola of the lung lies in close proximity to the nerve at this location [26].

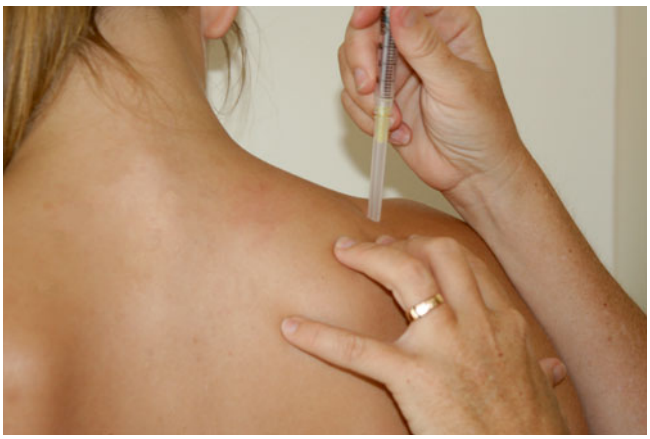

Fig. 28.11 Vertical approach for landmark-guided injection of the suprascapular nerve (Image courtesy of Andrea Trescot, MD)

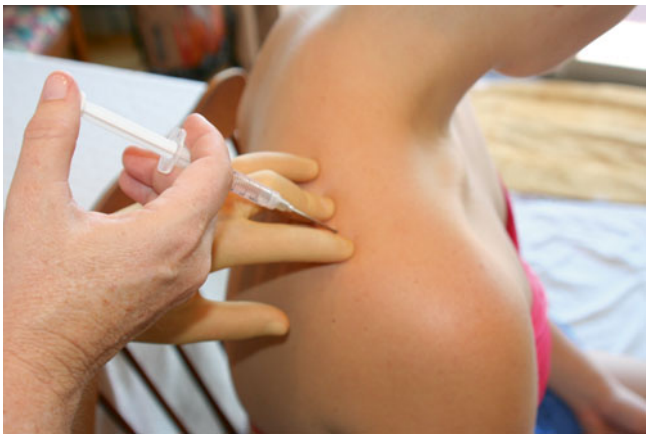

Fig. 28.12 Horizontal approach for landmark-guided injection of the suprascapular nerve (Image courtesy of Andrea Trescot, MD)

The second landmark-guided technique uses a horizontal instead of vertical needle position. In this approach, the patient is positioned in the seated or prone position with their arms relaxed at the sides. The distal third of the scapular spine is palpated, and a 25-gauge, 2-inch needle is advanced just above the scapular spine, perpendicular to the skin (Fig. 28.12). This technique is easily modified to add a peripheral nerve stimulator (Fig. 28.13). Initial target needle position is the posterior wall of the lateral border of the scapular spine, parallel to the floor and lateral to the scapular notch. The needle is advanced medially in the plane of the scapular spine with the use of continuous motor stimulation until the needle tip is near the nerve, as evidenced by external rotation of the shoulder, indicating contraction of the infraspinatus and supraspinatus muscles. After negative aspiration for blood or air, the planned injectate can be administered. By utilizing the bone as a backdrop, the risk of pneumothorax is

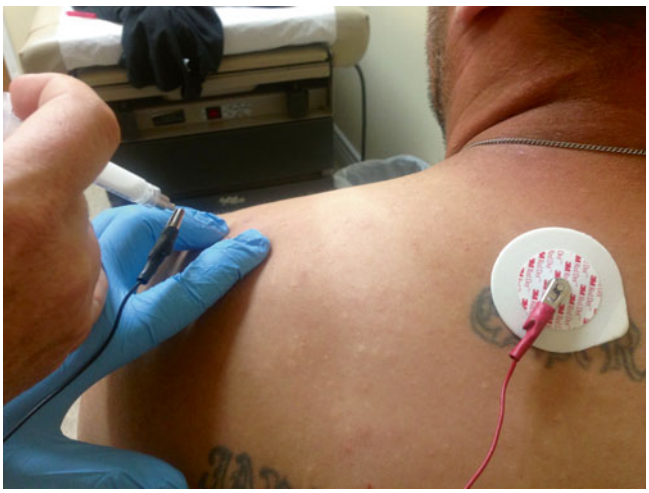

Fig. 28.13 Horizontal suprascapular landmark-guided injection with peripheral nerve stimulator (Image courtesy of Andrea Trescot, MD)

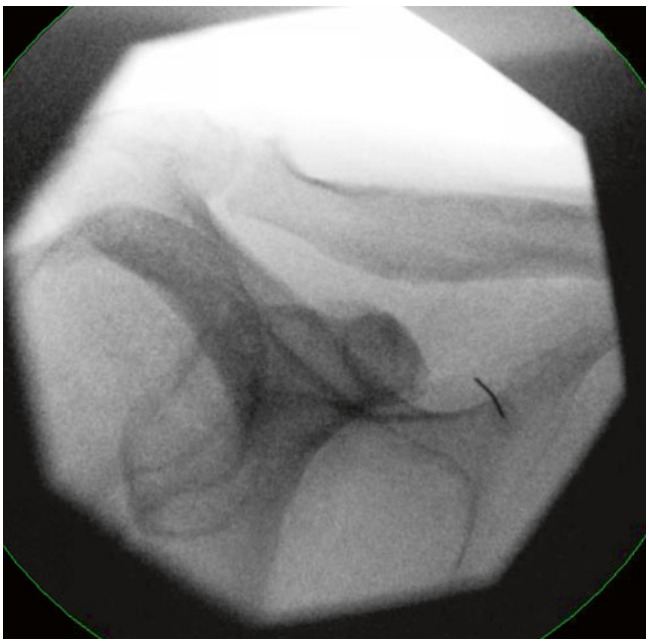

Fig. 28.14 Fluoroscopic image of suprascapular notch located just above the needle (Image courtesy of Christopher Burnett, MD)

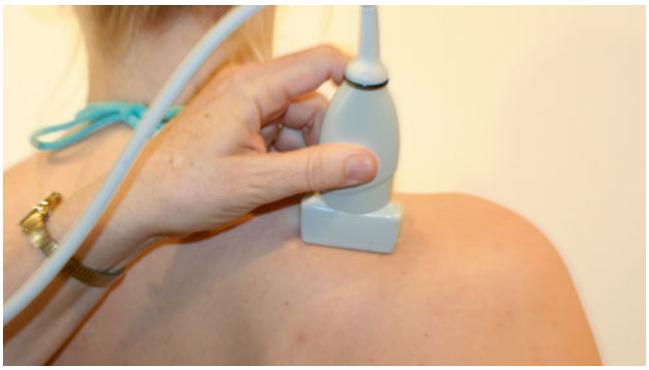

Fig. 28.15 Ultrasound transducer placed over the suprascapular notch (Image courtesy of Andrea Trescot, MD)

negligible. Having the patient place their hand on the opposite shoulder further decreases the risk of pneumothorax, by moving the scapula away from the chest wall and underlying pleura [19].

Fluoroscopic-Guided Injection Technique

Using the fluoroscopic-guided technique, the patient is placed prone on the fluoroscopy table, with the arms at the sides and the head turned to the contralateral side. Initially, straight AP fluoroscopic imaging is used to identify the T2 and T3 levels. Cephalocaudal tilt of the fluoroscope will help optimize the view of the suprascapular notch. The skin and subcutaneous tissue at the inferior aspect of the suprascapular notch is anesthetized, and a 22- or 25-gauge, 2-inch needle is advanced toward this target in a coaxial fluoroscopic view until contact is made with the scapula (Fig. 28.14). The needle is then withdrawn slightly and advanced in the cephalad direction no more than 1 cm toward the suprascapular notch. A paresthesia may be encountered, either from direct needle contact with the nerve or as a result of muscle spasm compressing the nerve. Many advocate the use of a nerve stimulator for improved safety and efficacy. Careful aspiration should be performed for blood or air. Contrast can be injected to further verify needle placement, and the planned injectate can be administered [12].

Ultrasound-Guided Injection Technique (US)

US injections of the SN were first described by Harmon and Hearty in 2007 [27]. The patient is placed in a seated position with their ipsilateral hand resting on their contralateral shoulder. The initial US scan is performed in the sagittal plane at the superior border of the scapula, with the transducer placed parallel to the scapular spine, to identify the pleura at a depth of approximately 4 cm (Fig. 28.15). The US probe is moved laterally and then cephalad to visualize the suprascapular fossa. The transducer is moved in small increments laterally and/or superiorly until the suprascapular notch is visualized (Fig. 28.16). The SN will appear as a round, hyperechoic structure beneath the transverse scapular ligament. A 22-gauge, 2-inch (or longer) needle is inserted in the longitudinal axis of the ultrasound beam (in-plane) and advanced under full US visualization until the needle tip is in proximity to the SN. The use of a stimulating needle (such as in Fig. 28.17) or a peripheral nerve stimulator can aid

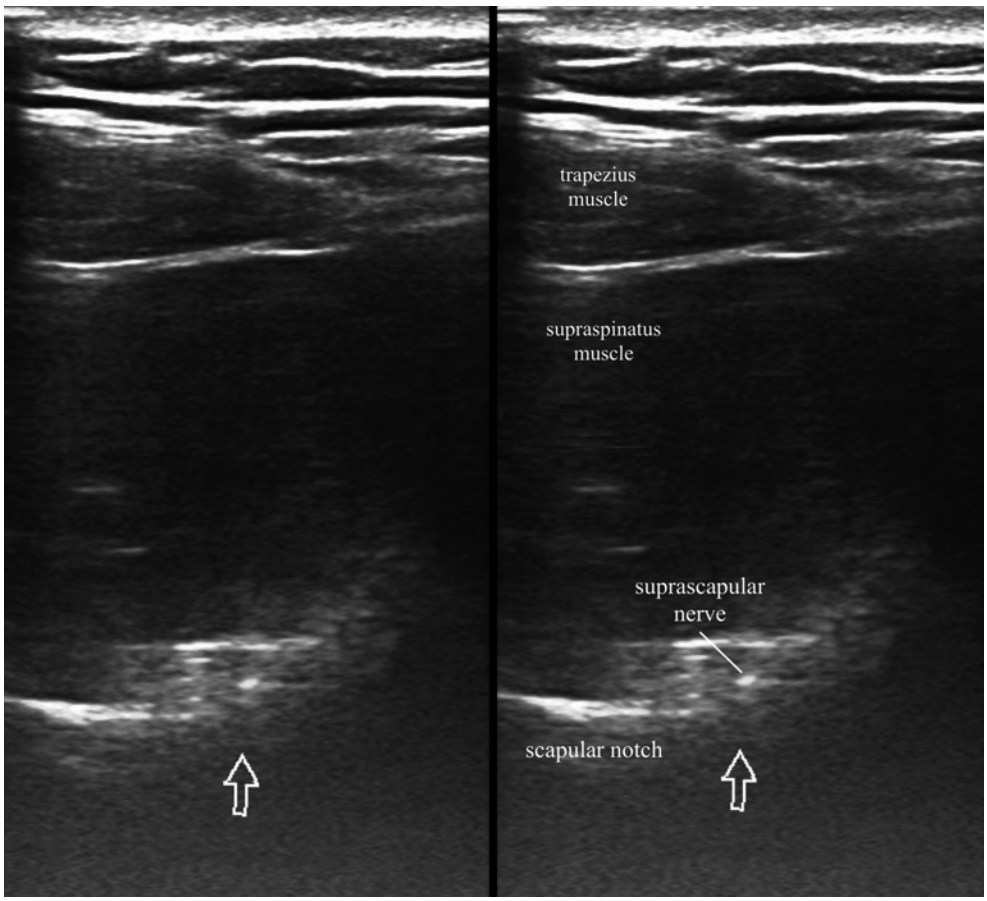

Fig. 28.16 Composite ultrasound image of suprascapular notch and suprascapular nerve (Image courtesy of Andrea Trescot, MD)

in localization of the nerve. It is important not to go too deep and to visualize the needle at all times. The needle is introduced in-plane either from medial to lateral or lateral to medial. If a stimulator is used, you will see a rhythmic external rotation and abduction of the shoulder. After threshold current is established and after a negative aspiration for blood or air, the planned injectate can be administered. Real-time imaging can be used to watch local anesthetic spread around the nerve [27].

More recently, an anterior approach to the SN has been described, which visualizes the suprascapular nerve branching off the superior trunk of the brachial plexus, deep to the omohyoid muscle (Fig. 28.18) [28]. The authors initially scanned 60 volunteers at both the suprascapular notch and the supraclavicular region and then placed needles into cadavers using the same technique to confirm the location. They were able to visualize the SN in the supraclavicular region in 81 % of the volunteers, but only 36 % of the time could the SN be seen at the suprascapular notch; they were able to place the needle correctly in cadavers 95 % of the time. Unfortunately, this is in close proximity to the brachial plexus (which potentially increases the risk of unintended spread of local anesthetic), as well as the pleura (potentially increasing the risk of pneumothorax).

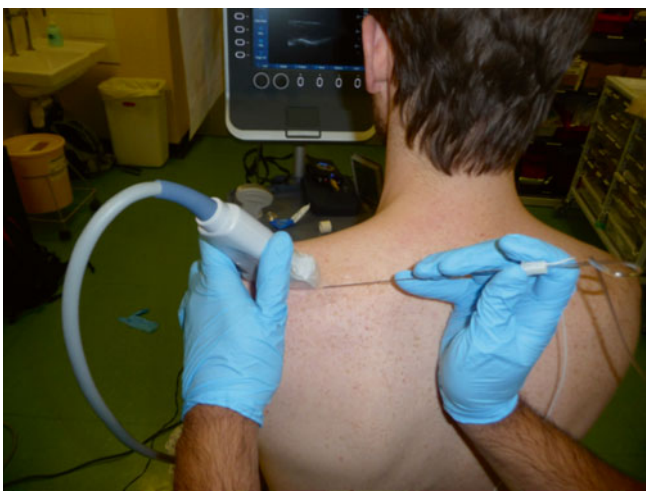

Fig. 28.17 Ultrasound-directed injection of the suprascapular nerve (Image courtesy of Christopher Burnett, MD)

CT Guided Injection

Schneider-Kolsky et al. [29] performed SN injections under CT guidance on 40 consecutive patients, noting sustained (>3 weeks) relief in 10 of 35 patients. Shanahan and col-

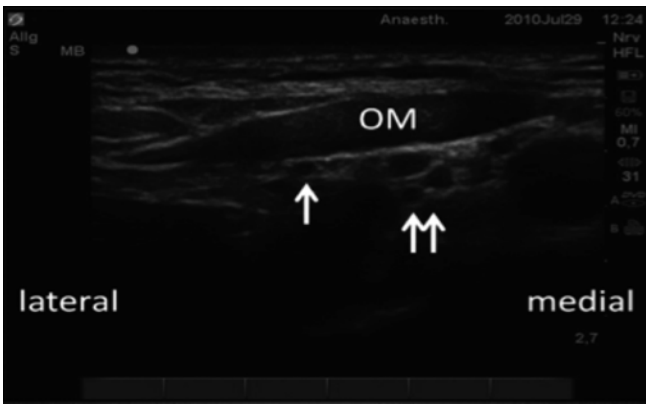

Fig. 28.18 Ultrasound image of supraclavicular anterior approach to the suprascapular nerve. *Single arrow* = suprascapular nerve, *double arrow* = brachial plexus; *OM* omohyoid muscle (Image courtesy of Dr. Róbert Rapčan, Bardejov, Slovakia)

leagues [22] randomized 77 shoulders to either landmark-guided (blind) suprascapular procedures (using 11 cc of local anesthetic and steroid) or CT-guided suprascapular injections (using 3 cc of local anesthetic and steroid) and found similar efficacy; neither group had any complications, and both techniques offered similar efficacy.

Neurolytic/Surgical Technique

Cryoneuroablation

Trescot [30] described a cryoneuroablation technique for the SN. The patient is positioned seated (for blind or US-directed procedures) (Fig. 28.19) or prone (for fluoroscopic-directed procedures). A 12-gauge introducer is advanced into the suprascapular notch, parallel to the direction of the nerve and perpendicular to the scapula. Consider using fluoroscopy or US if the superior border of the scapula is not easily palpated. The 2.0-mm probe is then advanced through the catheter, and the nerve is identified using sensory or motor stimulation, this being one of the few nerves with significant motor function that is amenable to cryoneuroablation.

Radiofrequency Lesioning

The use of either pulsed radiofrequency ablation (at 42 °C for 120 s) [31] or thermal radiofrequency ablation (at 80 °C for 60 s) [12] of the SN has been described and found to be a safe and effective way to obtain longer-term pain resolution.

Neuromodulation

Peripheral field stimulation has been growing as a treatment option. George Arcos, DO, described five patients with

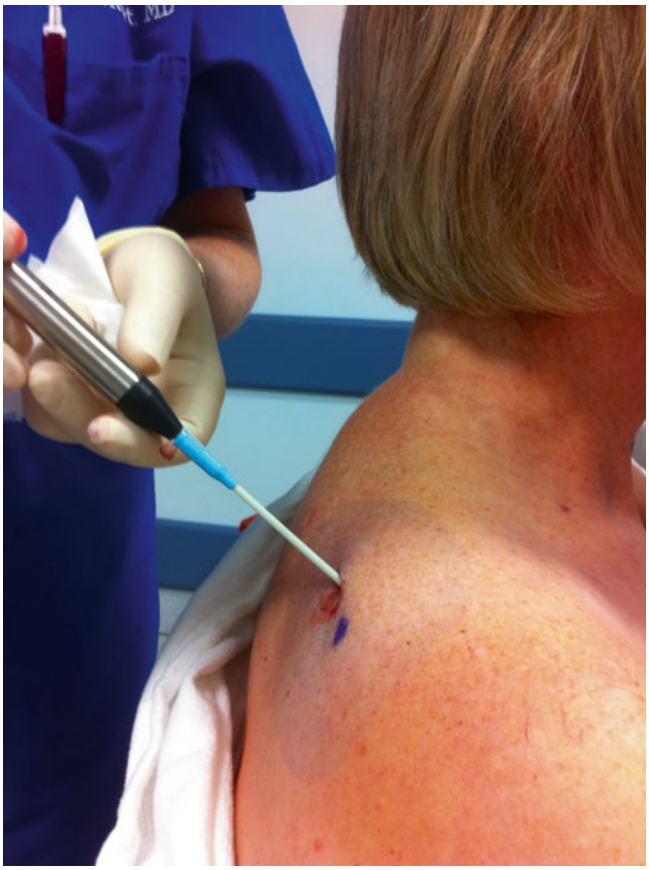

Fig. 28.19 Cryoneuroablation of the suprascapular nerve (Image courtesy of Andrea Trescot, MD)

chronic, persistent shoulder pain and dysfunction, with the earliest cases placed in approximately August 2011. He places two leads, and the vertical lead is placed 1 cm lateral to the spinous process, with cranial tip at the superior scapular border. The second lead is then placed parallel to the spine of the scapula, with a cranial to caudal trajectory (Fig. 28.20). The leads converge at the superior medial scapular border. By "stacking" anodal current, he states that he is able to cover pain in the entire intrascapular region, or laterally to the shoulder, axilla, and even proximal arm. He states that the patients are all at full function, with no opioid use (personal communication). Elahi and Reddy described a patient with intractable shoulder pain after seven shoulder surgeries (including hemiathroplasty) who was treated with a suprascapular peripheral nerve stimulator placed under US guidance (Fig. 28.21) [32].

Surgery

Surgical decompression of the SN may be considered in patients who have significant muscle wasting, weakness, or intractable pain and who have failed non-operative therapy. Traditionally, an open surgical approach was used to decompress the suprascapular or spinoglenoid notch. More recently,

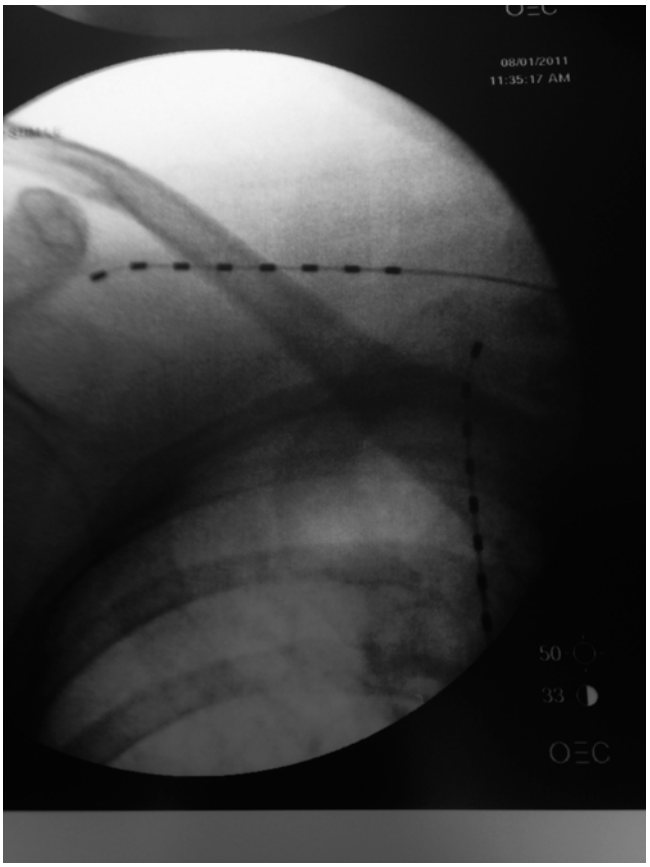

Fig. 28.20 Peripheral stimulation of the suprascapular nerve (Image courtesy of George Arcos, DO)

arthroscopic surgical approaches have been described for SN decompression [2, 20, 33]. Shah et al. [34] described the results of 27 patients who underwent arthroscopic decompression of the SN confirmed preoperatively with MRI and EMG findings, and followed them for an average of 22.5 months (with three lost to follow-up). Seventy-one percent (17/24) noted sustained pain relief and return of function.

Complications

Complications for SN block include pneumothorax, direct needle trauma to the neurovascular bundle, intravascular injection, or infection [26].

Summary

The SN is a cause of sometimes vague shoulder and scapular pain. Recognition of this pathology can save patients unnecessary and often fruitless evaluations. Use of the SN injection for diagnosis as well as for treatment of conditions such as adhesive capsulitis (frozen shoulder) may offer significant relief.

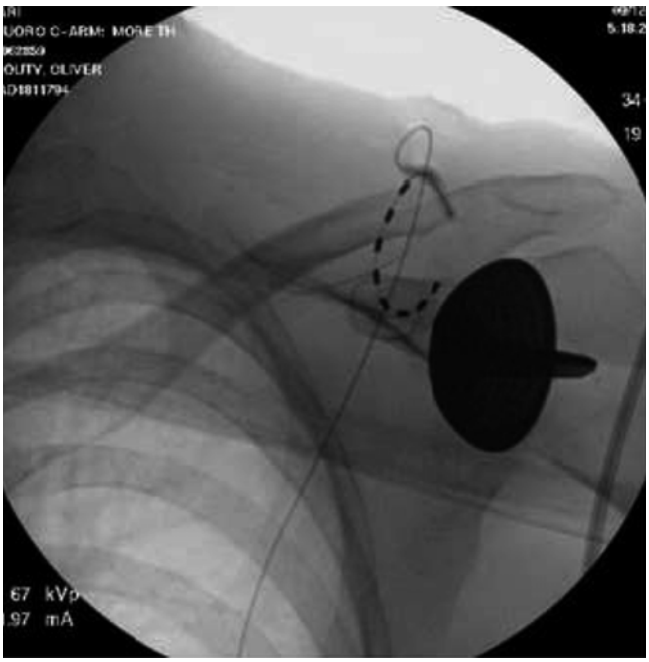

Fig. 28.21 Fluoroscopic imaging of a peripheral suprascapular stimulation lead placed under ultrasound guidance (Image from Elahi and Reddy [32], with permission from the American Society of Interventional Pain Physicians)

References

1. Linda DD, Harish S, Stewart BG, Finlay K, Parasu N, Rebello RP. Multimodality imaging of peripheral neuropathies of the upper limb and brachial plexus. Radiographics. 2010;30(5):1373–400.
2. Shapiro BE, Preston DC. Entrapment and compressive neuropathies. Med Clin N Am. 2009;93(2):285–315, vii.
3. Kim DH, Murovic JA, Tiel RL, Kline DG. Management and outcomes of 42 surgical suprascapular nerve injuries and entrapments. Neurosurgery. 2005;57(1):120–7; discussion –7.
4. Blum A, Lecocq S, Louis M, Wassel J, Moisei A, Teixeira P. The nerves around the shoulder. Eur J Radiol. 2013;82(1):2–16.
5. Iannotti JP, Ramsey ML. Arthroscopic decompression of a ganglion cyst causing suprascapular nerve compression. Arthroscopy. 1996;12(6):739–45.
6. Harbaugh KS, Swenson R, Saunders RL. Shoulder numbness in a patient with suprascapular nerve entrapment syndrome: cutaneous branch of the suprascapular nerve: case report. Neurosurgery. 2000;47(6):1452–5; discussion 5–6.
7. Ebraheim NA, Whitehead JL, Alla SR, Moral MZ, Castillo S, McCollough AL, et al. The suprascapular nerve and its articular branch to the acromioclavicular joint: an anatomic study. J Should Elb Surg. 2011;20(2):e13–7.
8. Liveson JA, Bronson MJ, Pollack MA. Suprascapular nerve lesions at the spinoglenoid notch: report of three cases and review of the literature. J Neurol Neurosurg Psychiatry. 1991;54(3):241–3.
9. Aiello I, Serra G, Traina GC, Tugnoli V. Entrapment of the suprascapular nerve at the spinoglenoid notch. Ann Neurol. 1982;12(3):314–6.
10. Budzik JF, Wavreille G, Pansini V, Moraux A, Demondion X, Cotten A. Entrapment neuropathies of the shoulder. Magn Reson Imaging Clin N Am. 2012;20(2):373–91, xii.
11. Rengachary SS, Burr D, Lucas S, Brackett CE. Suprascapular entrapment neuropathy: a clinical, anatomical, and comparative study. Part 3: comparative study. Neurosurgery. 1979;5(4):452–5.
12. Simopoulos TT, Nagda J, Aner MM. Percutaneous radiofrequency lesioning of the suprascapular nerve for the management of chronic shoulder pain: a case series. J Pain Res. 2012;5:91–7.

13. Natsis K, Totlis T, Tsikaras P, Appell HJ, Skandalakis P, Koebke J. Proposal for classification of the suprascapular notch: a study on 423 dried scapulas. Clin Anat. 2007;20(2):135–9.
14. Dong Q, Jacobson JA, Jamadar DA, Gandikota G, Brandon C, Morag Y, et al. Entrapment neuropathies in the upper and lower limbs: anatomy and MRI features. Radiol Res Pract. 2012; 2012:230679.
15. Safran MR. Nerve injury about the shoulder in athletes, part 1: suprascapular nerve and axillary nerve. Am J Sports Med. 2004;32(3):803–19.
16. Kopell HP, Thompson WA. Pain and the frozen shoulder. Surg Gynecol Obstet. 1959;109(1):92–6.
17. Ganzhorn RW, Hocker JT, Horowitz M, Switzer HE. Suprascapular-nerve entrapment. J Bone Joint Surg Am. 1981;63(3):492–4.
18. Burbank KM, Stevenson JH, Czarnecki GR, Dorfman J. Chronic shoulder pain: part I. Evaluation and diagnosis. Am Fam Physician. 2008;77(4):453–60.
19. Neal S, Fields KB. Peripheral nerve entrapment and injury in the upper extremity. Am Fam Physician. 2010;81(2):147–55.
20. Toussaint CP, Zager EL. What's new in common upper extremity entrapment neuropathies. Neurosurg Clin N Am. 2008;19(4):573–81, vi.
21. Carroll KW, Helms CA, Otte MT, Moellken SM, Fritz R. Enlarged spinoglenoid notch veins causing suprascapular nerve compression. Skelet Radiol. 2003;32(2):72–7.
22. Burbank KM, Stevenson JH, Czarnecki GR, Dorfman J. Chronic shoulder pain: part II. Treatment. Am Fam Physician. 2008;77(4):493–7.
23. Hoskins WT, Pollard HP, McDonald AJ. Quadrilateral space syndrome: a case study and review of the literature. Br J Sports Med. 2005;39(2):e9.
24. Polguj M, Synder M, Kwapisz A, Stefanczyk K, Grzelak P, Podgorshi M, et al. Clinical evaluation of the shape of the suprascapular notch—an ultrasonographic and computed tomography comparative study: application to shoulder pain syndromes. Clin Anat. 2015;28:774–9.
25. Hill LJ, Jelsing EJ, Terry MJ, Strommen JA. Evaluation, treatment, and outcomes of suprascapular neuropathy: a 5-year review. PMR. 2014;6(9):774–80.
26. Shanahan EM, Smith MD, Wetherall M, Lott CW, Slavotinek J, FitzGerald O, et al. Suprascapular nerve block in chronic shoulder pain: are the radiologists better? Ann Rheum Dis. 2004;63(9):1035–40.
27. Harmon D, Hearty C. Ultrasound-guided suprascapular nerve block technique. Pain Phys. 2007;10(6):743–6.
28. Siegenthaler A, Moriggl B, Mlekusch S, Schliessbach J, Haug M, Curatolo M, et al. Ultrasound-guided suprascapular nerve block, description of a novel supraclavicular approach. Reg Anesth Pain Med. 2012;37(3):325–8.
29. Schneider-Kolsky ME, Pike J, Connell DA. CT-guided suprascapular nerve blocks: a pilot study. Skelet Radiol. 2004;33(5):277–82.
30. Trescot AM. Cryoanalgesia in interventional pain management. Pain Phys. 2003;6(3):345–60.
31. Gofeld M, Restrepo-Garces CE, Theodore BR, Faclier G. Pulsed radiofrequency of suprascapular nerve for chronic shoulder pain: a randomized double-blind active placebo-controlled study. Pain Pract. 2013;13(2):96–103.
32. Elahi F, Reddy CG. Neuromodulation of the suprascapular nerve. Pain Phys. 2014;17(6):E769–73.
33. Lafosse L, Tomasi A, Corbett S, Baier G, Willems K, Gobezie R. Arthroscopic release of suprascapular nerve entrapment at the suprascapular notch: technique and preliminary results. Arthroscopy. 2007;23(1):34–42.
34. Shah AA, Butler RB, Sung SY, Wells JH, Higgins LD, Warner JJ. Clinical outcomes of suprascapular nerve decompression. J Should Elb Surg. 2011;20(6):975–82.

Intercostal Nerve Entrapment: Chest Wall

29

Sola Olamikan, Amitabh Gulati, and Andrea M. Trescot

Introduction

Intercostal neuralgia (IN), a relatively rare condition, consists of nerve pain secondary to intercostal nerve pathology. The term "intercostal" refers to the location between two ribs. In contrast to more common chest wall pain syndromes such as *costochondritis*, acute rib fracture, or *Tietze's syndrome*, which are musculoskeletal in origin, the pain of intercostal neuralgia is neuropathic in origin. The most common causes of intercostal neuralgia nerve damage-related pain are postherpetic neuralgia, thoracic surgery, and diabetic thoracic neuropathy [1]. Other causes of intercostal neuralgia include direct nerve injury, stretching, entrapment, and inflammation. Direct nerve injury may be due to physical trauma or as an aftereffect of surgery. Stretching injuries include traction on the chest wall from the expanding gravid uterus. Entrapment can be caused by neoplasm, sarcoidosis, and pleural mesothelioma. The pain roughly parallels the rib over the affected nerve. The condition has also been referred to as "*intercostal nerve syndrome*." At times, it can be extremely painful, to the point of being debilitating.

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Clinical Presentation (Table 29.1)

Intercostal neuralgia (IN) is easily misdiagnosed due to the ambiguity of its symptoms. Patients may complain of "rib pain" (Fig. 29.1), but they may also describe "chest pain" or upper abdominal pain. The history of onset may be clearly related to a specific event such as blunt trauma or rib fracture or nerve injury such as *shingles* or *thoracotomy* and *medial sternotomy*, but the onset may also be insidious with no identifiable preceding event. The pain is often perceived as stabbing, sharp, spasm-like, tearing, tender, aching, or gnawing. The pain may wrap around the chest in a dermatomal pattern or radiate from the back toward the front of the chest in a band-like pattern (Fig. 29.2). It may occur in sporadic episodes, or it may be dull and constant. The pain may intensify with exertion such as with heavy lifting, twisting, or turning the torso. Breathing, coughing, laughing, or sneezing may be painful. It may be associated with numbness and tingling. Pain may start in the back and wrap around the front, or it may be felt only in

Table 29.1 Occupation/exercise/trauma history relevant to intercostal entrapment

Surgery	Thoracotomy, median sternotomy
Neuropraxia, stretching injury	Pregnancy, with tension on nerves by gravid uterus; retractor trauma during thoracotomy; frequent, prolonged coughing
Trauma	Rib fracture or rib contusion, breast surgery, prolonged positioning
Neuropathy	Postherpetic neuralgia, diabetic peripheral neuropathy, pleuritis
Entrapment	Scar at chest tube insertion sites; entrapment at lateral rectus border (ACNE syndrome – see Chap. 42); pleural fibrosis [1]; osteoporotic compression, degenerative disk disease, scoliosis, rib articulation arthritis
Intercostobrachial nerve injury	Injured during mastectomies and axillary node dissection
Postoperative neuroma	Thoracotomy, breast implant [2]

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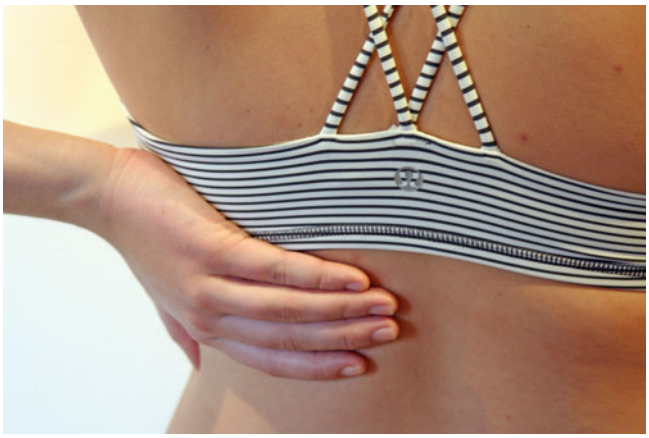


Fig. 29.1 Patient description of intercostal nerve pain (Image courtesy of Andrea Trescot, MD)

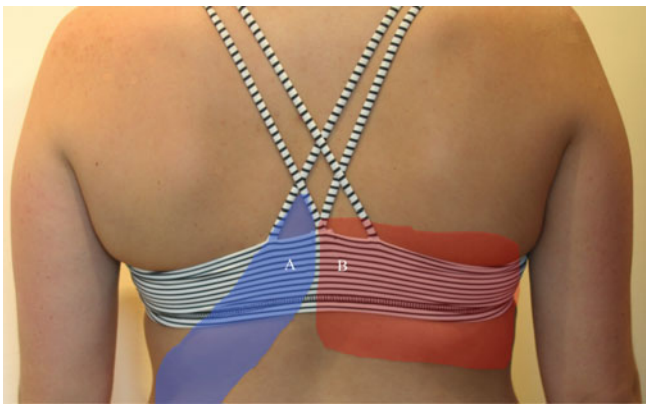
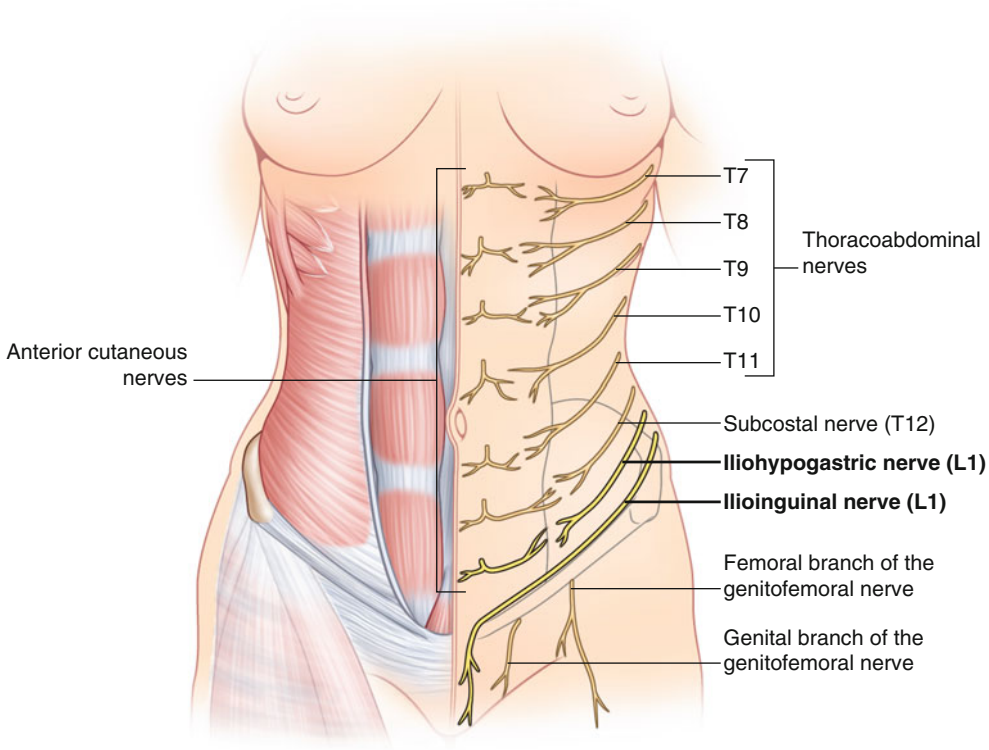


Fig. 29.2 Pain patterns of intercostal neuralgia; *A* dermatomal pattern, *B* "band-like" pattern (Image courtesy of Andrea Trescot, MD)

Fig. 29.3 Anatomy of the abdominal wall (Image by Springer)



the front or back. If the subcostal nerve is involved, patients may believe they are suffering from gallbladder disease.

The lower intercostal nerves actually travel around to the upper abdomen and can get trapped at the edge of the rectus abdominis (Fig. 29.3), the muscle that runs vertically from the bottom of the breastbone to the top of the pubic bones causing an *anterior cutaneous nerve entrapment syndrome* (ACNE syndrome) discussed in Chap. 42. Allodynia may be present.

Trejjo-Gabriel-Galan et al. [1] described intercostal neuralgia caused by compression of the nerves from a fibrotic mass from a prior tuberculosis infection with chronic pleuritis.

Anatomy (Table 29.2)

Intercostal nerves are the anterior rami of the first 11 thoracic spinal nerves. The anterior ramus of the 12th thoracic nerve travels to the abdomen as the subcostal nerve. The intercostal nerve travels from the foramen, under the inferior edge of the rib with the intercostal artery and vein, to the anterior chest or abdominal wall. Each intercostal nerve enters the corresponding intercostal space between the *posterior intercostal membrane* and the *parietal pleura*. The inner layer (the innermost intercostal muscle) does not exist at this point,

Table 29.2 Intercostal nerve anatomy

Origin	Anterior rami of the first 11 thoracic nerve roots; T12 anterior ramus is called the subcostal nerve (not technically an "intercostal" nerve because there is no 13th rib)
General route	Main intercostal runs in subcostal groove to the anterior chest or abdominal wall – four branches
	Gray ramus communicans connects to the sympathetic chain
	Posterior cutaneous innervates the muscles and skin of the paraspinal area
	Lateral cutaneous separates from the main nerve in the anterior axillary line and provides the skin sensation to the chest and abdominal wall
	T2 (sometimes also T3) lateral cutaneous nerve is called intercostobrachial, crosses axilla to medial arm
	Anterior cutaneous provides innervation of the abdominal wall, piercing the fascia of the abdominal wall at the lateral border of the rectus abdominis muscle
Sensory distribution	Skin of the chest wall and abdominal wall
Motor innervation	Intercostal muscles and abdominal wall
Anatomic variability	Collateral nerves connect with a variable number of nerves
Other relevant structures	Subcostal groove, lateral rectus border

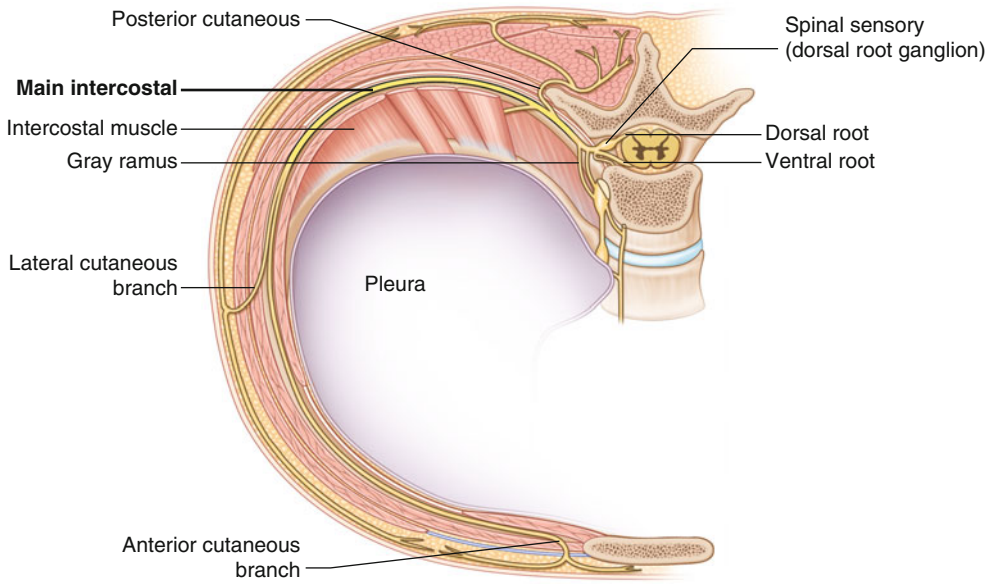


Fig. 29.4 Branches of the intercostal nerve (Image by Springer)

and only the parietal pleura is present on the inner side. The intercostal nerve then travels forward with the intercostal vessels in the subcostal groove of the corresponding rib between the innermost intercostal and internal intercostal muscles (Fig. 29.4). The first six intercostal nerves terminate within their respective intercostal spaces in the anterior chest. The seventh, eighth, and ninth leave their intercostal spaces anteriorly (after innervating the structures within) and pass to the anterior abdominal wall (Fig. 29.3).

The *main intercostal nerve* runs in the subcostal groove contained in a fibrous sheath with the intercostal vessels. A typical intercostal nerve has four major branches [3] (Fig. 29.4). The *gray ramus communicans* consists of unmyelinated postganglionic fibers that interface with the sympathetic chain. The *posterior cutaneous intercostal branch* innervates the muscles and skin of the paraspinal area. The *lateral cutaneous nerve* courses with the main intercostal nerve until it penetrates the *intercostal muscles* to arrive at the skin along the midaxillary line, and the *anterior cutaneous nerve*, providing innervation of the abdominal wall, pierces the fascia of the abdominal wall at the lateral border of the rectus abdominis muscle. At this point, the nerve, accompanied by an epigastric artery and vein, makes a sharp turn anteriorly to supply sensation to the anterior abdominal wall; the nerve passes through a firm fibrous ring as it transits the fascia, which results in a site of entrapment. Although traditionally considered unilateral structures, occasionally a given intercostal nerve will cross the midline to provide sensation to the contralateral side.

There is also a *collateral branch* with a less predictable path, though it usually travels near the superior border of the rib below for at least part of its route. Usually, neighboring intercostal nerves communicate with each other via variable additional nerve branches.

Table 29.3 Branches of intercostal nerves (see Fig. 29.4)

Nerve	Anatomic course and function
Rami communicans	Connects the intercostal nerves to the sympathetic trunk
Collateral branch	Runs parallel to main nerve on upper border of the rib below
Posterior cutaneous	Innervates the muscles and skin of the paraspinal area
Lateral cutaneous branch	Innervates the skin on the side of the thoracic wall by dividing into anterior and posterior branches
Anterior cutaneous branch	Terminal portion of the intercostal nerves, which innervate the skin near the midline of the chest
Muscular branches	Supply all the muscles of the intercostal spaces
Pleural branches	Sensory branches to the parietal pleura
Peritoneal sensory branches	Similar to the pleural sensory branches but arise from the lower intercostal nerves and supply the abdominal peritoneum

The 12th intercostal nerve, also called the *subcostal nerve*, is unique in that it joins with the first lumbar nerve root, thus becoming part of the lumbar plexus (Chap. 43) (Table 29.3).

Causes of Intercostal Neuralgia

A variety of mild to severe diseases, disorders, and conditions may lead to the development of intercostal neuralgia. A chest or rib injury, such as a fractured rib or bruised chest sustained in a motor vehicle accident or participation in sports that involve high speeds or contact with other athletes (e.g., skiing, snowboarding, football, wrestling, and rugby), can be precipitating events. Thoracic shingles, along the path of the intercostal nerve, is the most common site of herpes zoster eruptions (55 % of the cases) [4].

In pregnancy, the increasing size of a growing baby alters the structure of the torso and rib cage such that pressure on the nerves between the ribs can occur. Intercostal neuralgia in pregnancy has been associated with pain and numbness in the ribs, abdomen, and back.

Oesch et al. [5] documented 14 cases of intercostal neuralgia resulting from an impingement of the intercostal nerve at the level of the anterior rectus sheath, presenting as acute or chronic abdominal pain, also known as *anterior cutaneous nerve entrapment* (*ACNE*) *syndrome*, as described in Chap. 42. The authors were able to identify the correct diagnosis upon noting a positive *Carnett test* (pain intensification during palpation while contracting the abdominal muscles by raising the head while lying supine) and relief of pain after injecting local anesthesia at the point of maximal tenderness.

The first intercostal nerve joins the brachial plexus through a small branch and is very small compared to the other intercostal nerves. The *intercostobrachial nerve* (ICBN) is the lateral cutaneous branch of the second intercostal nerve. It crosses the axilla to the medial side of the arm and joins with a filament from the medial brachial cutaneous nerve. It then pierces the fascia and supplies the skin of the upper half of the medial and posterior part of the arm and axilla. A second intercostobrachial nerve is frequently given off from the lateral cutaneous branch of the third intercostal nerve, as well as sometimes from the fourth intercostal nerve. It supplies filaments to the axilla and medial side of the arm. Women who undergo a partial or radical mastectomy including an axillary lymph node dissection are at extreme risk for transection of this nerve. Subsequent pain and sensory deficit are localized to the axilla and/or arm in the distribution of innervation of this nerve [6]. In addition, the ICBN contributes to the innervation of the posterior forearm via connections with the posterior antebrachial cutaneous nerve and contributions to the upper anterior chest wall secondary to connections with the *long thoracic nerve* (see Chap. 30).

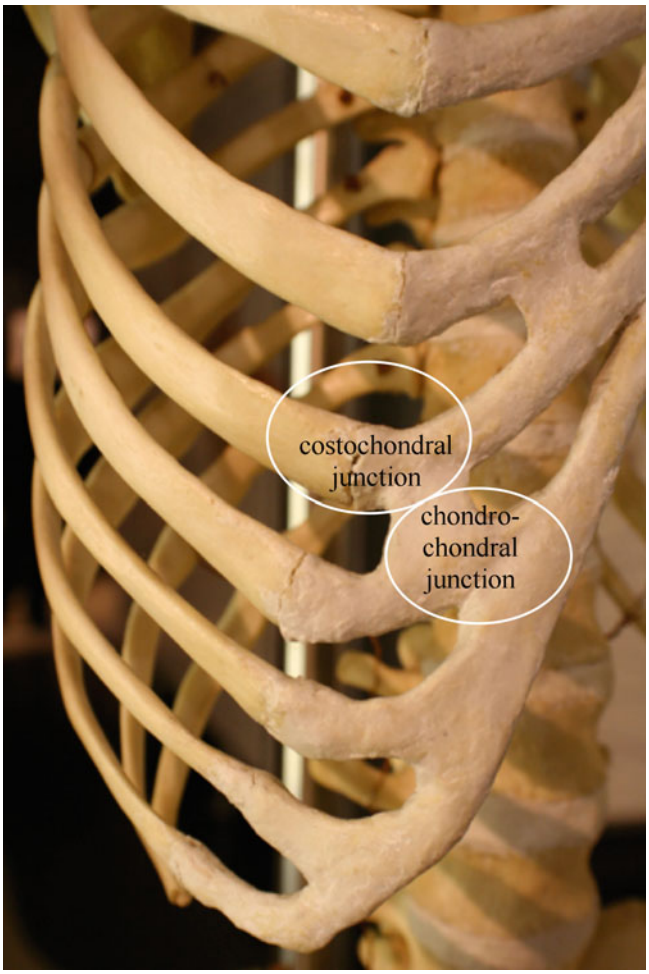


Fig. 29.5 Costochondral and chondrochondral junctions (Image courtesy of Andrea Trescot, MD)

The intercostal nerve also innervates the *costochondral junction* (where the rib attaches to the cartilage) and the *chondrochondral junction* (where the cartilage attaches to the cartilage above) (Fig. 29.5). These junctions are traumatized by

forces less than those necessary to fracture the rib itself (visualize how easy it is to pop the cartilage off the end of a barbequed spare rib), but will not show up on X-ray. The patient may describe a sharp, painful, slipping, or popping sensation in the anterior chest with movement; smokers, perhaps because they have poor blood flow to the tissues or because of chronic cough, seem to be most susceptible.

Intercostal nerve injury has long been suspected as a complication of *thoracotomy* procedures. Rogers et al. [7] recorded motor evoked potentials from intercostals muscles in 13 patients undergoing thoracotomy. Intercostal nerves functioned normally before and after entering the pleural space. After the rib retractor was removed, there was a total conduction block in the nerve immediately above the incision in every patient. In the nerves above this, six had a total block, one had a partial block, and three had normal conduction. There was a total conduction block in the nerve immediately below the incision in all but one patient. Of the nerves below this, four had a total block, two had a partial block, and three had normal conduction. In a single patient where rib retraction was not employed, there was no impairment of the intercostal nerves throughout the operation. This study demonstrates that intercostal nerve injury occurs routinely due to rib retraction during thoracotomy.

Wildgaard et al. [8] reviewed literature published from 2000 to 2008 on post-thoracotomy pain syndrome. Although the authors noted the limit of their ability to compare data from the various studies, they concluded that intercostal nerve injury does appear to be the most important pathogenic factor in chronic post-thoracotomy pain. Intercostal neuromas have also been identified as a source of pain after aesthetic and reconstructive breast implant surgery [2].

Shingles (also termed *herpes zoster* or *zoster*) is a disease caused by reactivation of a previous infection with the herpes zoster virus (the same virus that causes *chicken pox*, termed *varicella zoster virus*). After a chicken pox outbreak, the virus remains dormant for many years, usually in the roots of sensory nerves. Upon reactivation, it typically causes unilateral pain, burning, or tingling and a blistering rash in the sensory distribution of the nerve. Shingles can involve an intercostal nerve and results in a painful rash along the chest or abdominal wall. The risk of the disease increases with age, with about half of all cases occurring among men and women 60 years of age or older. Conditions that may trigger reactivation include stress, fatigue, weakened immune system, cancer, and HIV. As many as 25 % of people over 50 years old with shingles develop postherpetic neuralgia [9], where significant pain remains even after the rash has resolved. It can be very difficult to treat.

Atypical chest pain is a common complaint among patients with *complex regional pain syndrome* (*CRPS*), formerly *reflex sympathetic dystrophy* (RSD) or *causalgia*. It is a chronic systemic disease characterized by severe pain, swelling, and changes in the skin. CRPS is an uncommon form of chronic pain that usually affects an arm or leg. It normally develops after an injury, surgery, stroke, or heart attack, but the pain is out of proportion to the severity of the initial injury, if any. In CRPS affecting the arm, the *intercostobrachial nerve* (ICBN) has been implicated. It is connected to the *brachial plexus* and innervates the axilla, medial arm, and anterior chest wall. By connecting to the brachial plexus, the intercostobrachial nerve can become sensitized and produce *atypical chest pain* [6].

Rasmussen et al. [6] described evidence of ICBN sensitization in CRPS; they evaluated 40 patients and controls, finding that 94 % of CRPS patients reported a history of chest pain versus only 19 % of the controls.

Physical Exam

The physical examination will generally reveal minimal physical findings, unless there is a history of previous thoracic or subcostal surgery, or cutaneous findings of herpes zoster involving the thoracic dermatomes. In contrast to musculoskeletal chest wall pain syndromes, the patient does not attempt to splint or protect the affected area. Careful sensory examination of the affected dermatomes may reveal decreased sensation or allodynia. With significant motor involvement of the subcostal nerve, the patient may complain that his or her abdomen bulges out (Fig. 29.6). Pressure or stretching over the site of the nerve injury will typically reproduce symptoms (Video 29.1) (Fig. 29.7). Finally, the single most diagnostic test for intercostal neuralgia is an intercostal nerve block.

Differential Diagnosis (Table 29.4)

The diagnosis of intercostal neuralgia is typically one of exclusion. Cardiac disease and abdominal pathology should be ruled out. Although uncommon, *thoracic disk herniation* should be excluded. Intercostal nerve conduction study has proved to be an accurate technique in diagnosis of thoracic radiculopathy. Johnson et al. [11] performed intercostal nerve conduction studies in 161 patients, 80 of whom had subsequent posterior rhizotomy (81 % of those noted relief of pain with surgery). The only significant complication of the intercostal nerve conduction study is an 8.8 % incidence of pneumothorax. Pradhan and Taly [12] then developed a surface nerve conduction study for the intercostal nerves.

Chen et al. described two patients with pain after shingles, presumed because of postherpetic neuralgia; both patients complained of burning, shooting, aching pain, and allodynia (consistent with neuropathic pain), localized to the site of the previous lesions. However, they were able to identify *trigger points* in the intercostal muscles just below the hyperesthetic region; injections of local anesthetic in

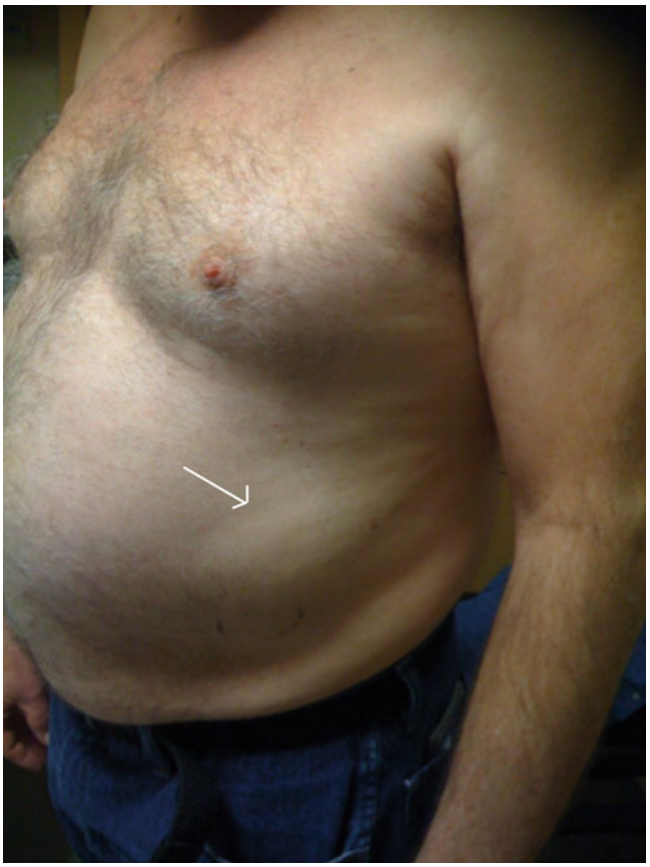


Fig. 29.6 Bulging abdominal wall caused by intercostal nerve injury – *arrow* shows the abdominal wall bulge (Image courtesy of Andrea Trescot, MD)

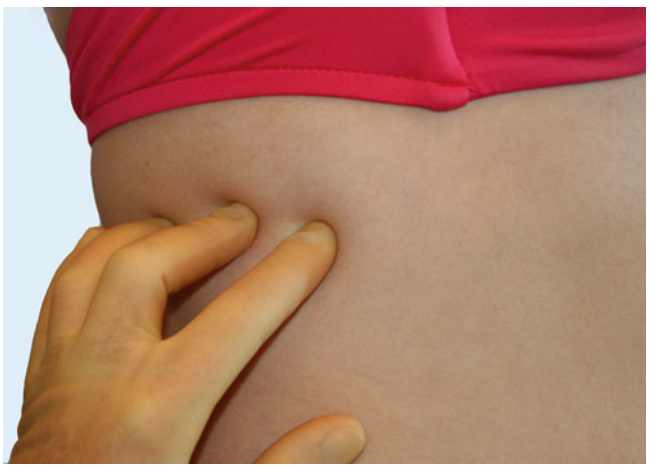


Fig. 29.7 Palpation of the intercostal nerve (Image courtesy of Andrea Trescot, MD)

this region replicated the pain and then abolished the pain. Although the patients required several sessions of injections, both were virtually pain-free 6 months later. The authors felt that the relief was unlikely to be due to an intercostal nerve block, since they injected only 1 cc of local anesthetic at the superior (not inferior) edge of the rib. However, it is conceivable that, by relaxing the intercostal muscle, they released an entrapment of the intercostal nerve (Table 29.5).

Table 29.4 Differential diagnosis of chest wall pain

	Potential distinguishing features
Cardiac pathology	Positive EKG
Myofascial pain	Palpable trigger points in the intercostal space (can be seen after shingles and mimic postherpetic neuralgia) [10]
Rib fracture	X-rays or bone scan will show fracture
Costochondritis	Tenderness to palpation at costochondral border
Pleurisy	Auscultation revels "rubbing" sound; chest X-ray shows pleural fluid
Hepatic disease	Elevated liver function tests; enlarged liver by palpation and US
Thoracic radiculopathy	Thoracic herniated disk by MRI

Table 29.5 Diagnostic tests for intercostal neuralgia

	Potential distinguishing features
Physical exam	Tenderness over the rib or rib cartilage junction
Diagnostic injection	Intercostal nerve block is diagnostic
Ultrasound	Useful for nerve localization
MRI/CT	May show rib fracture
Arteriography	Nondiagnostic
X-ray	May show rib fracture or calcification of cartilage
Electrodiagnostic studies	May show conduction slowing

Identification and Treatment of Contributing Factors

Chronic cough or increased abdominal girth (obesity, pregnancy, ascites) will put tension on the intercostal nerve. As noted above, smokers appear to be more likely to have rib nonunion or nonhealing cartilage issues.

For thoracic disk herniations, percutaneous discectomy can offer relief. For chronic cough leading to costochondral pathology, the use of cough suppressants may help. Topical medications can give relief from postherpetic neuralgia.

Intercostal Nerve Block Techniques

The technique may be performed with landmark guidance, with fluoroscopic guidance, or with the use of ultrasound guidance. Radiological guidance is advised for neurolytic blocks.

An intercostal nerve block with local anesthetic and corticosteroid serves as a diagnostic test for intercostal neuralgia and offers short-term (and potentially long-term) therapeutic relief. The immediate effect is usually from the local anesthetic injected. This wears off in a few hours. The depo-steroid starts working in about 3–5 days, and its effect can last for several days to a few months. The injections can be done

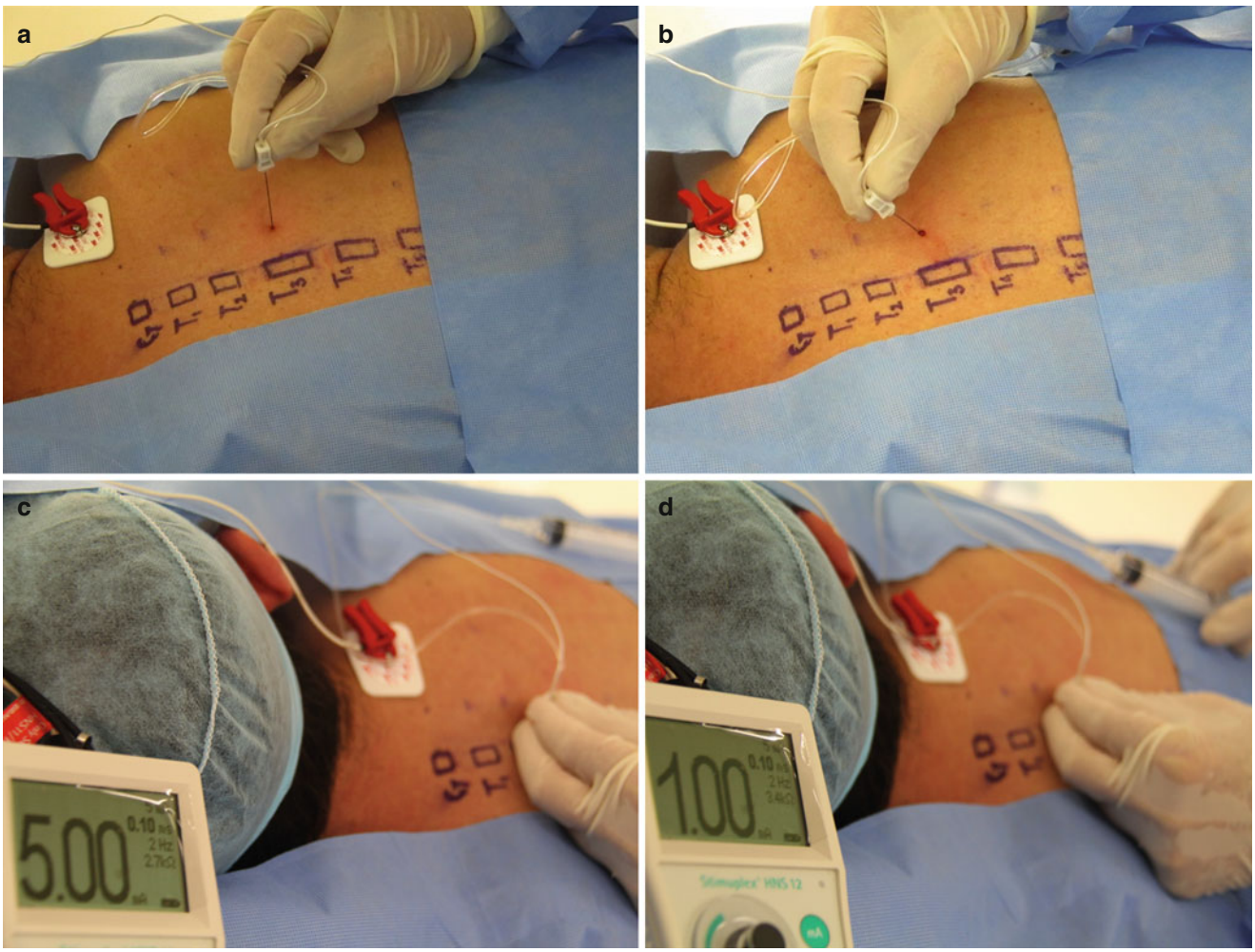


Fig. 29.8 Landmark-guided intercostal nerve block. (**a**) The needle is placed perpendicular to the skin; (**b**) the needle is walked inferiorly; (**c**) stimulator is used to identify the nerve location at a higher voltage; (**d**) stimulator is used at a lower voltage to identify the nerve (Image courtesy of Thialgo Nouer Frederico, MD)

about 1 week apart and only if needed. If the first injection does not relieve symptoms in about a week or 2, a second injection may be recommended.

Landmark-Guided Injection

The patient is placed in the prone position with the patient's arm hanging loosely off the side of the table. Alternatively, this block can be done with the patient in the sitting or lateral position. Because of the overlapping innervation of the chest and upper abdominal wall, the intercostal nerves above and below the nerve suspected of subserving the painful condition will likely need to be injected as well.

There are several sites for injecting an intercostal nerve, based on the location of pathology. Perhaps the most common site is at the angle of the rib about 7 cm lateral to the midline in adults. The landmark-guided ("blind") injection should be reserved for those patients in whom the ribs and rib interspace can be reliably palpated.

The skin is first drawn cephalad about 1 cm with the palpating hand, and a 1.5–2 inch (5 cm) 22- to 27-gauge (for single-shot injection) short-bevel needle is introduced through the chosen site of entry at a 20° cephalad angle, with the bevel facing cephalad. The needle is advanced until it contacts the rib at a depth of less than 1 cm (for most non-obese patients). At this moment, a small amount of local anesthetic may be injected to anesthetize the periosteum. With the palpating hand holding the needle firmly and resting securely on the patient's back, the injecting hand gently walks the needle caudally while the skin is allowed to move back over the rib. The needle is now advanced 3 mm, still maintaining the 20° tilt angle cephalad. A subtle "give" or "pop" of the fascia of the internal intercostal muscle may be felt, especially if a short-bevel needle is used. As the distance from the posterior aspect of the rib to the pleura averages 8 mm, advancement of the needle much beyond 3 mm increases the risk of pneumothorax. Paresthesia, while not actively sought, confirms needle placement. A peripheral nerve stimulator may aid in confirmation (Fig. 29.8). After

negative aspiration to ensure the needle is not in a blood vessel or intrapleural, local anesthetic and steroid are injected. It is desirable to block at least one intercostal nerve cephalad and one caudad due to overlapping innervation.

Because the perpendicular approach has a significant risk of *pneumothorax*, an alternative approach places the needle more parallel to the bottom of the rib, sliding the needle up underneath the inferior edge of the rib (Video 29.2) (Fig. 29.9).

Fluoroscopic-Guided Technique

Use of fluoroscopy allows precise localization of the rib, potentially decreasing the risk of pneumothorax. The patient is positioned prone, supine, or lateral, depending on the site of pain. Instead of placing the needle perpendicular to the rib

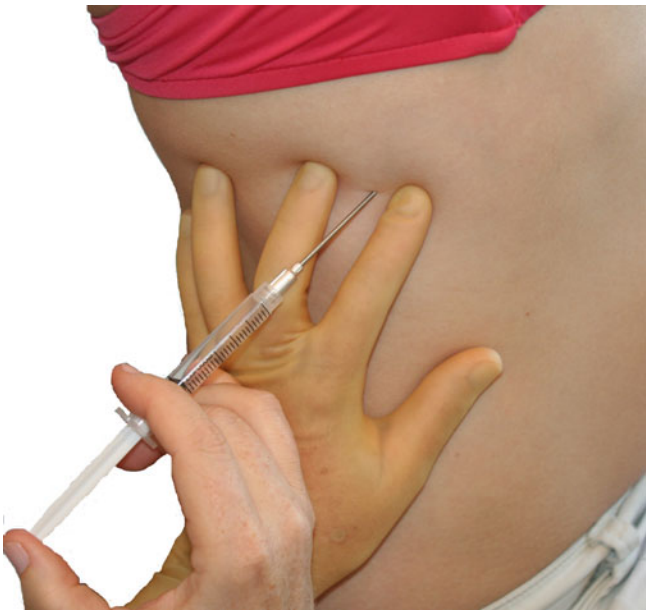


Fig. 29.9 Landmark-guided intercostal nerve block (alternative technique) – the needle is parallel to the inferior rib (Image courtesy of Andrea Trescot, MD)

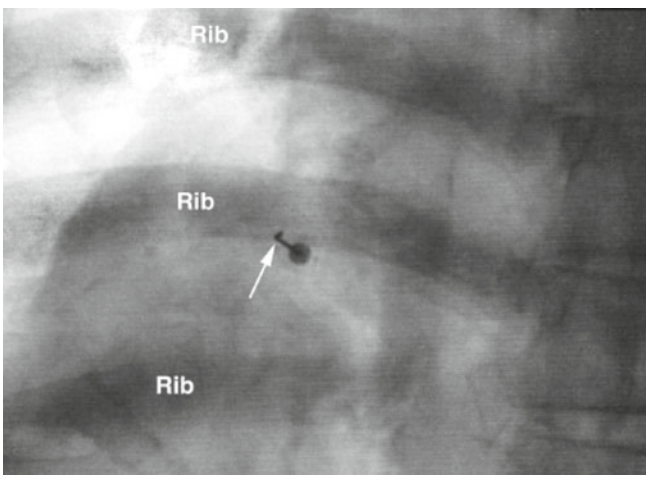


Fig. 29.10 Fluoroscopic standard intercostal nerve injection; the needle is perpendicular to the nerve (Image courtesy of Miles Day, MD)

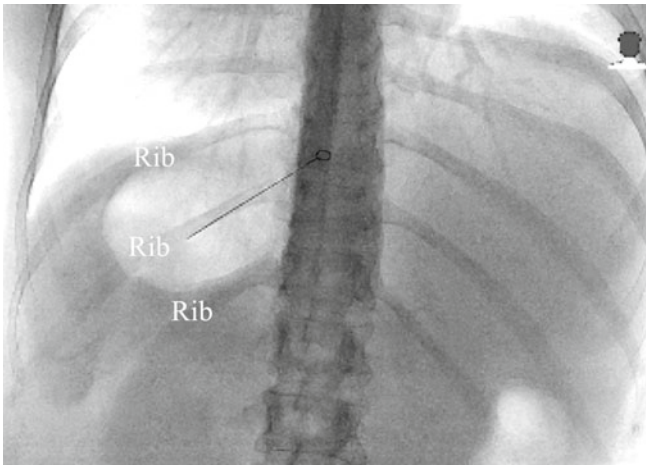


Fig. 29.11 Fluoroscopic intercostal nerve injection; the needle is parallel to the inferior edge of the rib and then is slid up under the rib (Image courtesy of Andrea Trescot, MD)

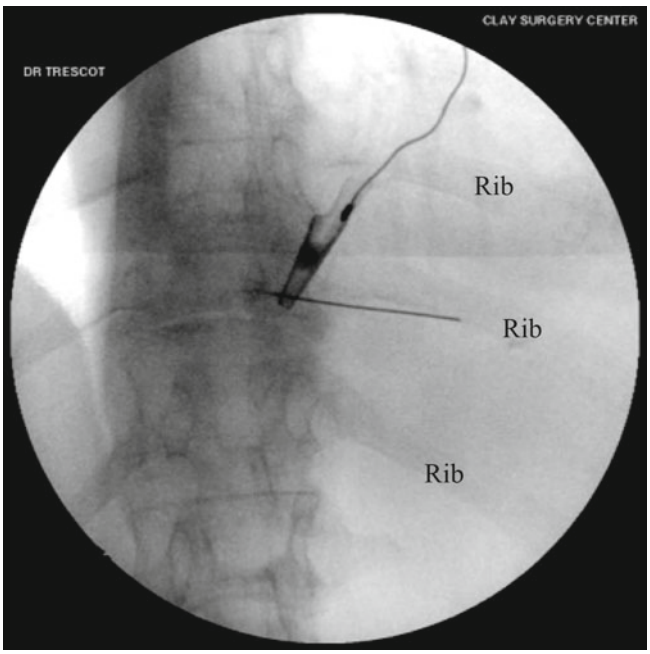


Fig. 29.12 Fluoroscopic intercostal nerve injection; the needle is parallel to the inferior edge of the rib and then is slid up under the rib using a peripheral nerve stimulator (Image courtesy of Andrea Trescot, MD)

(Fig. 29.10), the needle can be placed obliquely at the inferior edge of the rib, sliding up underneath the edge of the rib (Fig. 29.11). A peripheral nerve stimulator will help to identify the nerve (Fig. 29.12), and contrast will confirm the perivascular spread of medication (Fig. 29.13).

Ultrasound-Guided Technique

Use of ultrasound has dramatically decreased the risk of pneumothorax by allowing clear identification of the rib at the bedside. The patient can be positioned prone, lateral, or sitting. With the US probe placed vertically (Fig. 29.14a), the

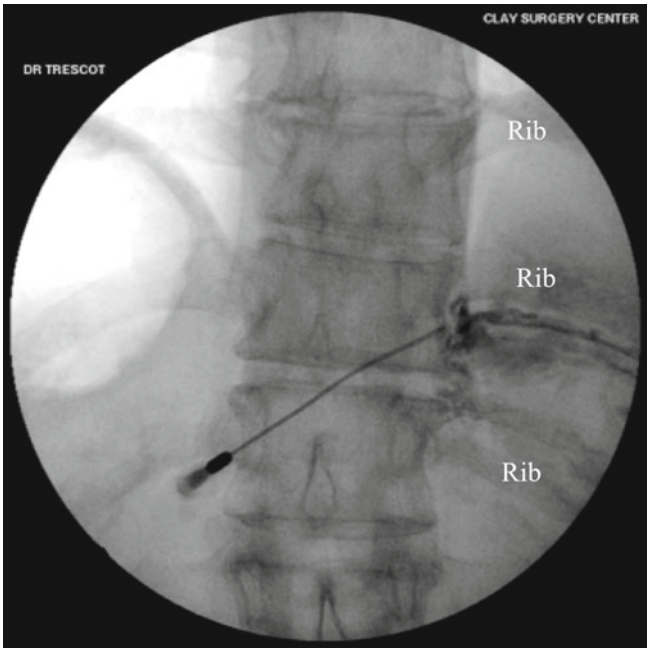


Fig. 29.13 Fluoroscopic intercostal nerve injection; the needle is parallel to the inferior edge of the rib and then is slid up under the rib using a peripheral nerve stimulator, with position confirmed by contrast (Image courtesy of Andrea Trescot, MD)

US images demonstrate three layers of the *intercostal muscles* (*external*, *internal*, and *innermost*) covering the pleural line (Fig. 29.14b) [13]. The neurovascular bundle lies between the internal and innermost intercostal muscles. The US probe is then placed horizontally (Fig. 29.15) or kept vertically (Fig. 29.16), and the needle is introduced in-plane.

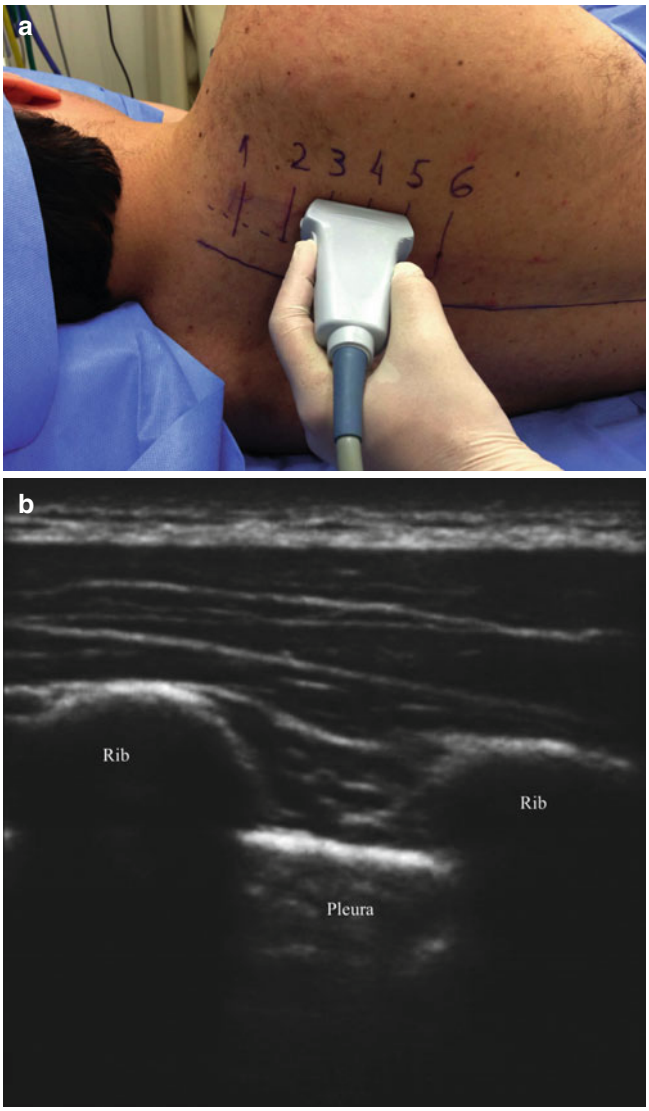


Fig. 29.14 Ultrasound identification of the intercostal space, with the US probe placed vertically. (**a**) Location of probe; (**b**) US image showing the rib, intercostal space, and pleura (Images courtesy of Thialgo Nouer Frederico, MD)

Neurolytic/Surgical Technique

Intercostal nerve blocks can predict how a patient will respond to neurolytic techniques. A good response usually means the patient will benefit from neurolytic procedures as well. Fluoroscopic or ultrasound guidance improves the accuracy of these blocks and minimizes complications.

Cryoneuroablation

One of the most commonly described uses of cryoneuroablation has been for intercostal neuralgia. A 14-gauge Angiocath is introduced at the inferior border of the rib and advanced laterally; the stylet is then removed and the probe introduced underneath the inferior edge of the rib (Figs. 29.17 and 29.18).

Byas-Smith and Gulati [14] reported on the use of cryoneuroablation of the intercostal nerves under US to treat post-thoracotomy pain. They were able to see the tip of the probe and the pleura, as well as the ice ball itself (Fig. 29.19).

Green and colleagues [15] retrospectively looked at 43 patients with chronic chest pain treated with cryoneuroablation. The mean duration of pain prior to cryoneurolysis was 31 months (range 0.5 months to 24 years). Sixty percent of the patients reported a decrease in their pain immediately after the procedure. Three months after the cryoneurolysis, 50 % of the patients still reported significant pain relief.

Radio-frequency Lesioning

A case series by Engel [16] reports efficacy of conventional radio-frequency neurolysis in refractory intercostal neuralgia due to blunt chest wall trauma. Garcia Cosamalòn et al. [17] describe the use of CT-guided dorsal percutaneous

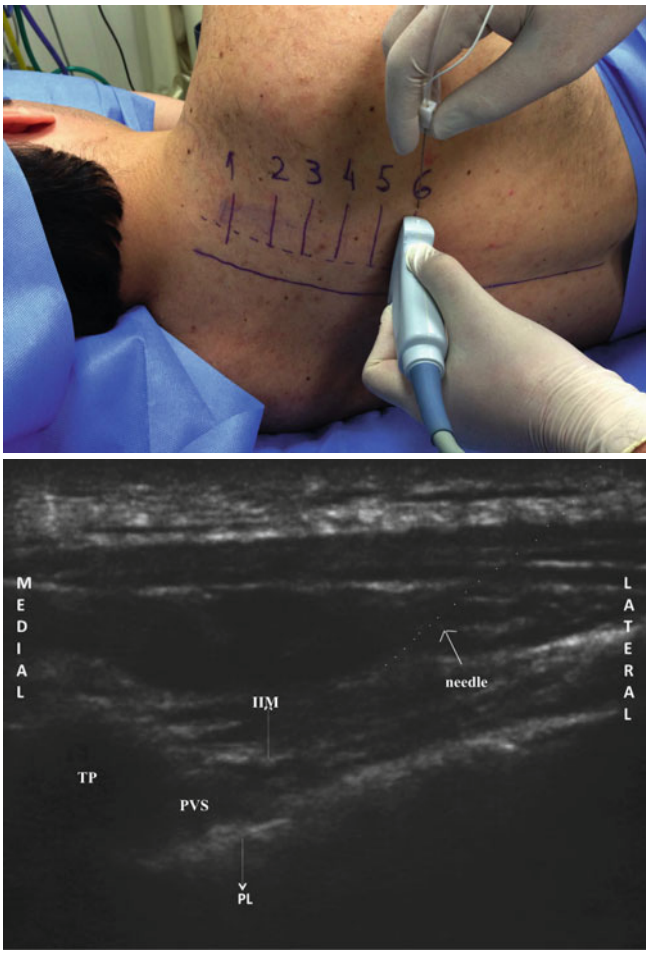


Fig. 29.15 Ultrasound injection of the intercostal space, from vertical orientation. *PL* pleural space, *TP* transverse process, *PVS* paravertebral space, *IIM* internal intercostal membrane (Images courtesy of Thialgo Nouer Frederico, MD)

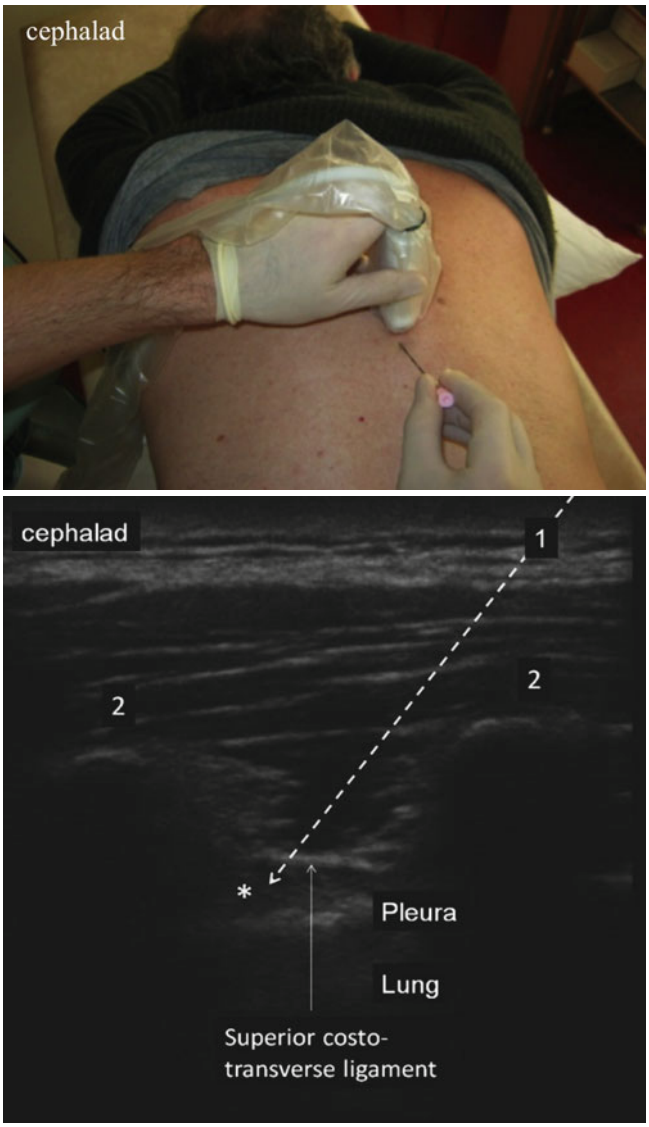


Fig. 29.16 Ultrasound injection of the intercostal space, from horizontal orientation. *1* needle, *2* rib (Images courtesy of Róbert Rapčan, MD)

radio-frequency rhizotomy for intercostal neuralgia. Akkaya and Ozkan [18] described good relief treating three intercostal neuralgia patients using pulsed radio frequency under US guidance.

Chemical Neurolysis

Alcohol and phenol are the preferred agents for neurolytic procedures because they cause axonal degeneration within minutes and effectively interrupt the central transmission of pain impulses. Chemical neurolysis can result in significant pain relief in selected patients but should only be considered for refractory neuralgic pain due to intercostal nerve injury from rib fracture or post-thoracotomy and for refractory postherpetic neuralgia or cancer invasion, since complications can include unintended spread of alcohol or phenol to the root cuff, epidural space, or cerebrospinal fluid (which could cause paralysis).

Neuromodulation

Spinal Cord Stimulation

Graybill et al. [19] describe successful pain relief from post-thoracotomy pain with spinal cord stimulation in a single case report, with a single lead placed midline up to T3.

Peripheral Nerve Stimulation

Peyravi et al. [20] report the use of subcutaneous peripheral neurostimulation for the treatment of severe chronic post-sternotomy neuralgia.

Peripheral Nerve Field Stimulation

McJunkin et al. [21] described a case of field stimulation used successfully to treat pain after a thoracotomy, with 80 % relief and dramatic improvement in mobility.

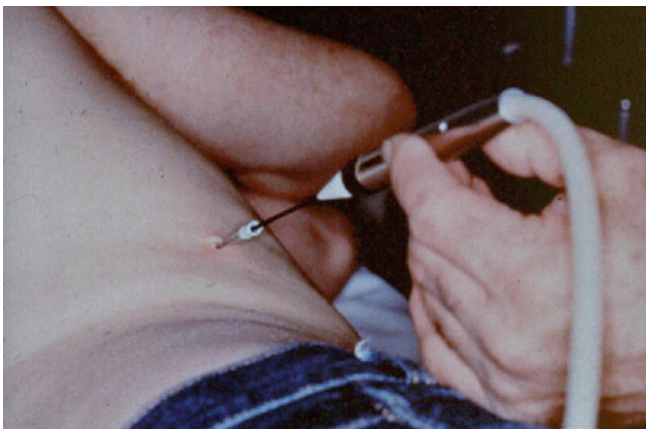


Fig. 29.17 Intercostal cryoneuroablation (Image courtesy of Andrea Trescot, MD)

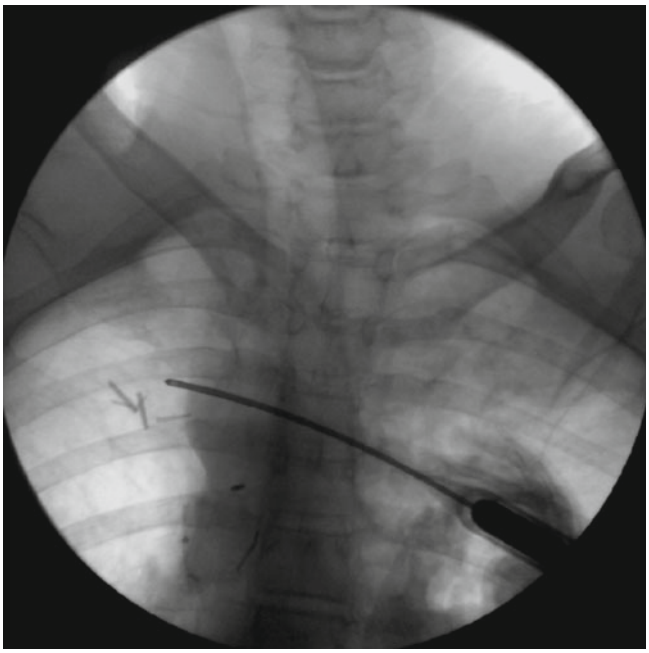


Fig. 29.18 Location of the cryoprobe for intercostal neuralgia (Image courtesy of Andrea Trescot, MD)

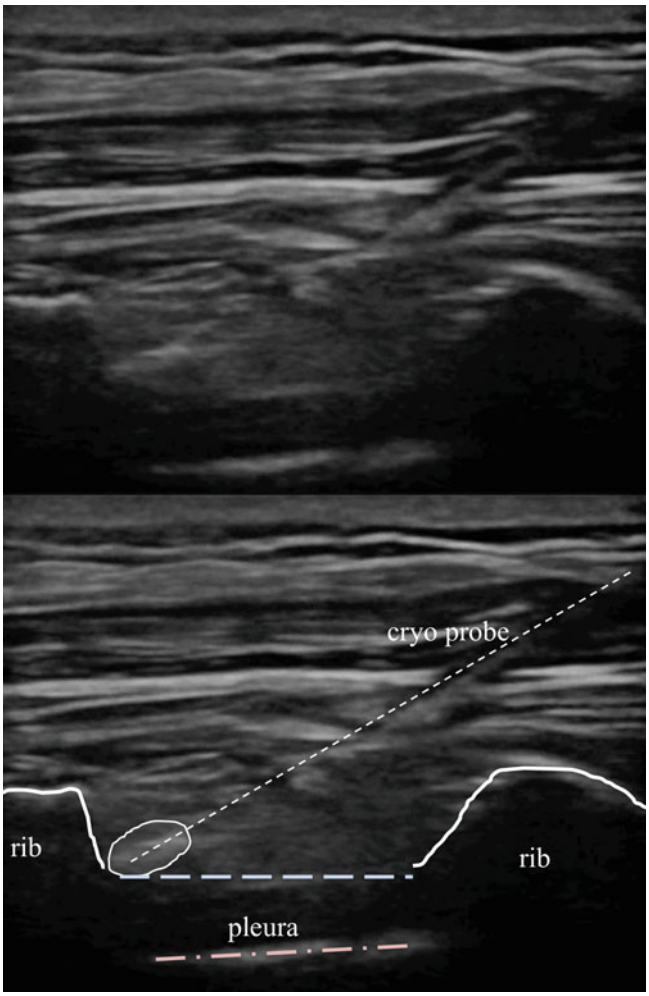


Fig. 29.19 Cryoneuroablation probe placed under ultrasound direction (Image courtesy of Christ Declerck, MD)

Long-Term Epidural Nerve Catheter

Samlaska and Dews [22] report successful pain relief of pregnancy-induced intercostal neuralgia using long-term indwelling epidural analgesia.

Surgical Decompression/Neurectomy

Oesch et al. [5] reported results of a retrospective study of 14 patients with intercostal nerve impingement at the level of the anterior rectus sheath, called anterior cutaneous nerve entrapment (ACNE) (see Chap. 42). All patients were treated with neurectomy (surgical resection) at the point of exit of the nerve from the anterior rectus sheath. Minimal side effects were noted, but follow-up was limited. In a double-blind, randomized, controlled trial, Boelens et al. [23] studied 44 patients with anterior cutaneous nerve entrapment from 2008 to 2010. Half of the patients underwent neurectomy at the level of the abdominal wall. The other half underwent a sham surgical procedure. Of the 22 neurectomy patients, 16 reported a successful pain response at 6 weeks postoperatively. The authors concluded that neurectomy is an effective surgical procedure for pain reduction in anterior cutaneous nerve entrapment syndrome.

Williams et al. [24] studied five consecutive patients who underwent neurectomy of one or more intercostal nerves and implantation of the cut nerve(s) into the latissimus dorsi or into the rib. Average follow-up after surgery was 8.8 months. Preoperatively, mean average pain level was 8 (range 7–9). Mean average pain level postoperatively was 2.2 (range 0–7). The authors concluded that neurectomy and implantation of the cut intercostal nerve into the latissimus dorsi or

into the rib was an efficacious treatment for this small group of patients.

Concerns still exist regarding the overall efficacy of neurectomy as a treatment for intercostal nerve impingement. Neurectomy complications include neuroma formation, which may result in worsened pain. According to Shapiro et al. [25], nerve transections of rat sciatic nerves performed by use of a scalpel or a CO2 laser resulted in neuroma formation in all cases.

Complications

Pneumothorax or penetration of peritoneum and abdominal viscera are potential complications. Absorption of local anesthetic from the intercostal space is rapid; toxicity is always a concern with multiple or continuous intercostal injections. Because the dural sheath can extend up to 8 cm laterally, there is a chance of spinal anesthesia and unexpected spread of chemical neurolytics into the spinal canal.

Summary

Intercostal neuralgia can cause severe, debilitating pain that may be difficult to treat. Causes include chest wall injury, pregnancy, abdominal impingement, and shingles. The syndrome may also occur iatrogenically following thoracotomy procedures, axillary node dissection, and breast implant surgery. The condition is also implicated in complex regional pain syndrome. When conservative treatments fail, the first line of more aggressive management consists of a series of anesthetic and corticosteroid nerve blocks. In patients who do not respond to repeat nerve blocks, cryoneuroablation or radio-frequency neurolysis may provide pain relief. Additional measures described in the literature include spinal cord stimulation, long-term epidural block, and neurectomy.

References

1. Trejo-Gabriel-Galan JM, Macarron-Vicente JL, Lazaro L, Rodriguez-Pascual L, Calvo I. Intercostal neuropathy and pain due to pleuritis. Pain Med. 2013;14(5):769–70.
2. Nguyen JT, Buchanan IA, Patel PP, Aljinovic N, Lee BT. Intercostal neuroma as a source of pain after aesthetic and reconstructive breast implant surgery. J Plast Reconstr Aesthet Surg. 2012;65(9):1199–203.
3. Davies F, Gladstone RJ, Stibbe EP. The anatomy of the intercostal nerves. J Anat. 1932;66(Pt 3):323–33.
4. Trescot AM. Shingles and interventional pain treatment. ASIPP News. 2006;(Fall):22–3.
5. Oesch A, Kupfer K, Bodoky A. Intercostal nerve syndrome. Swiss Surg Schweiz Chir Chir Suisse Chir Svizzera. 2001;7(2):82–5.
6. Rasmussen JW, Grothusen JR, Rosso AL, Schwartzman RJ. Atypical chest pain: evidence of intercostobrachial nerve sensitization in complex regional pain syndrome. Pain Phys. 2009;12(5):E329–34.
7. Rogers ML, Henderson L, Mahajan RP, Duffy JP. Preliminary findings in the neurophysiological assessment of intercostal nerve injury during thoracotomy. Eur J Cardiothorac Surg Off J Eur Assoc Cardiothorc Surg. 2002;21(2):298–301.
8. Wildgaard K, Ravn J, Kehlet H. Chronic post-thoracotomy pain: a critical review of pathogenic mechanisms and strategies for prevention. Eur J Cardiothorac Surg Off J Eur Assoc Cardiothorc Surg. 2009;36(1):170–80.
9. Gimenez-Mila M, Busquets C, Ojeda A, Fauli A, Moreno LA, Videla S. Neuropathic pain with features of complex regional syndrome in the upper extremity after herpes zoster. Pain Pract. 2014;14(2):158–61.
10. Chen SM, Chen JT, Kuan TS, Hong CZ. Myofascial trigger points in intercostal muscles secondary to herpes zoster infection of the intercostal nerve. Arch Phys Med Rehabil. 1998;79(3):336–8.
11. Johnson ER, Powell J, Caldwell J, Crane C. Intercostal nerve conduction and posterior rhizotomy in the diagnosis and treatment of thoracic radiculopathy. J Neurol Neurosurg Psychiatry. 1974; 37(3):330–2.
12. Pradhan S, Taly A. Intercostal nerve conduction study in man. J Neurol Neurosurg Psychiatry. 1989;52(6):763–6.
13. Sakai F, Sone S, Kiyono K, Imai S, Izuno I, Oguchi M, et al. High resolution ultrasound of the chest wall. RoFo: Fortschr Gebiete Rontgenstrahlen Nuklearmedizin. 1990;153(4):390–4.
14. Byas-Smith MG, Gulati A. Ultrasound-guided intercostal nerve cryoablation. Anesth Analg. 2006;103(4):1033–5.
15. Green CR, de Rosayro AM, Tait AR. The role of cryoanalgesia for chronic thoracic pain: results of a long-term follow up. J Natl Med Assoc. 2002;94(8):716–20.
16. Engel AJ. Utility of intercostal nerve conventional thermal radiofrequency ablations in the injured worker after blunt trauma. Pain Phys. 2012;15(5):E711–8.
17. Garcia Cosamalon PJ, Mostaza A, Fernandez J, Vinuela J, Mazabel M, Costilla S, et al. Dorsal percutaneous radiofrequency rhizotomy guided with CT scan in intercostal neuralgias. Technical note. Acta Neurochir (Wien). 1991;109(3–4):140–1.
18. Akkaya T, Ozkan D. Ultrasound-guided pulsed radiofrequency treatment of the intercostal nerve: three cases. J Anesth. 2013;27(6):968–9.
19. Graybill J, Conermann T, Kabazie AJ, Chandy S. Spinal cord stimulation for treatment of pain in a patient with post thoracotomy pain syndrome. Pain Phys. 2011;14(5):441–5.
20. Peyravi M, Capelle HH, Fischer S, Haverich A, Krauss JK. Subcutaneous peripheral neurostimulation for the treatment of severe chronic poststernotomy neuralgia. Stereotact Funct Neurosurg. 2011;89(4):253–7.
21. McJunkin TL, Berardoni N, Lynch PJ, Amrani J. An innovative case report detailing the successful treatment of post-thoracotomy syndrome with peripheral nerve field stimulation. Neuromodulation. 2010;13(4):311–4.
22. Samlaska S, Dews TE. Long-term epidural analgesia for pregnancy-induced intercostal neuralgia. Pain. 1995;62(2):245–8.
23. Boelens OB, van Assen T, Houterman S, Scheltinga MR, Roumen RM. A double-blind, randomized, controlled trial on surgery for chronic abdominal pain due to anterior cutaneous nerve entrapment syndrome. Ann Surg. 2013;257(5):845–9.
24. Williams EH, Williams CG, Rosson GD, Heitmiller RF, Dellon AL. Neurectomy for treatment of intercostal neuralgia. Ann Thorac Surg. 2008;85(5):1766–70.
25. Shapiro S, Woodall B, Muller J. Comparative study of neuroma formation in the rat sciatic nerve after CO2 laser and scalpel neurectomy in combination with milliwatt CO2 laser “sealing”. Surg Neurol. 1989;32(4):281–4.

Long Thoracic Nerve Entrapment

30

Sola Olamikan and Helen W. Karl

Introduction

Long thoracic nerve (LTN) entrapment can occur at several sites along its course. The usual injury is mechanical in nature – by direct compression or excessive traction; the LTN can also be anesthetized or injured during brachial plexus blocks, especially from a posterior approach with a catheter [1]. In a significant number of cases, no specific cause can be identified. *Medial scapular winging* (prominence of the scapula) is the hallmark of LTN injury. *Serratus anterior* palsy, due to damage of the LTN, is the most diagnosed form of scapular winging [2]. Patients with LTN dysfunction usually present with shoulder and scapula pain and weakness when lifting objects away from the body or with overhead activity. They may be uncomfortable when sitting against the back of a chair or when supine. Sometimes patients will have an insidious onset of weakness. The scapula resembles a "wing" because its medial border sticks out from the back, and the medial and inferior borders are closer to the spine and elevated when compared to the normal side. Scapular winging is a major disability with substantial interference with arm function [3–5]. The LTN has also been called the *external respiratory nerve of Bell* or the *long thoracic nerve of Bell* [6, 7].

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Clinical Presentation (Table 30.1)

Patients with LTN dysfunction often have a nonspecific deep ache in the shoulder radiating "through the neck to the scapula" [7] (Fig. 30.1), or it may be near the inferior scapula and lateral chest wall (Fig. 30.2) [3]. There may be weakness with forward elevation and overhead activities. Some authors think that pain is a less important symptom than decreased performance and progressive scapular winging [7]. However, in one series, pain was the primary complaint (3/18 = 17 %) [8], and in another, immediate burning pain at the lower pole of the scapula was a "striking feature, present in each patient" [15]. Raising the ipsilateral arm and tilting or turning the head to the opposite side may aggravate the discomfort [7]. These patients are often young (average age: 30 years old) males [3, 7]. Other series are comprised of slightly older patients without male predominance [19]. Their symptoms are often on the right, even when the patient is left-handed [19].

Although many injuries to the LTN come from athletic activities, it can also be injured during breast surgery (specifically mastectomy) or even carrying a heavy bag over the shoulder for a long time.

A study from the Mayo Clinic identified 50 patients with LTN pathology; 17 were traumatic (3 surgical, 5 blunt trauma, and 9 stretch injuries), 20 were inflammatory, and 13 were idiopathic [16]. The dominant arm was involved in 86 % of the patients. Similarly, Nath et al. [20] described 47 consecutive patients with LTN entrapment; 3 cases were bilateral, 31 patients (62 %) had a history of weight lifting, 2 patients (4 %) noted winging immediately after deep massage in the supraclavicular fossa, and 1 patient was a postal worker who had been reaching overhead at work for several years. In this study, there was no identifiable cause in 9 patients (18 %). Interestingly, only about half of the patients presented with pain.

Scapular winging is an underappreciated cause of decreased function and disability, due to muscle spasms,

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Table 30.1 Occupation/exercise/trauma history relevant to long thoracic nerve entrapment

Muscular exertion 35 % [8]	Sports involving extreme shoulder abduction [9] (volleyball, wrestling, weight lifting, ballet, archery, tennis, bowling, soccer, golf, hockey, riflery, gymnastics) [4, 7, 10, 20]
Repetitive microtrauma	Heavy backpacks [7]
	Abnormal sleep position or reading in bed with head propped up on the hand [7, 8]
	Childbirth [8]
	Chiropractic neck manipulation [11]
	Use of a single axillary crutch [12]
	Instrumental musicians [13]
	Postal worker [20]
Direct trauma 26 % [8]	Fall or collision leading to closed chest or scapular injury [3, 14]. Acute depression of the shoulder by a direct blow or by sudden arm traction such as when a heavy object falls unexpectedly [15] or landing on their side with the arm outstretched [10]
	Blunt trauma (such as a hockey stick) to the neck or shoulder [10]
Locally invasive procedures 11 % [8]	Particularly breast [9] or cardiac surgery [14] and transaxillary resection of the first rib [9, 14]; associated with poor outcome [16, 17]
General anesthesia 5 % [8]	Operating room positioning with extreme arm abduction [14]
Illnesses (viral, brachial neuritis), toxins, and vitamin deficiencies [7, 8, 18]	30/136 (22 %) patients with *Parsonage–Turner brachial neuritis* (also known as *neuralgic amyotrophy*) had unilateral LTN involvement [18]
Surgical trauma	Radical neck dissection, carotid endarterectomy, excision of cervical subcutaneous mass, cervical lymph node biopsy, radical mastectomy [10]
	Cardiac surgery (CABG, port-access mitral valve surgery), resection of the first rib, transaxillary thoracic sympathectomy [13]

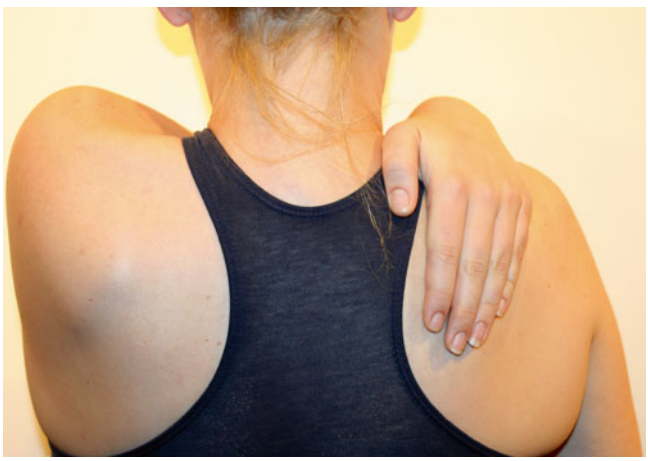

Fig. 30.1 Posterior scapular pattern of long thoracic neuralgia (Image courtesy of Andrea Trescot, MD)

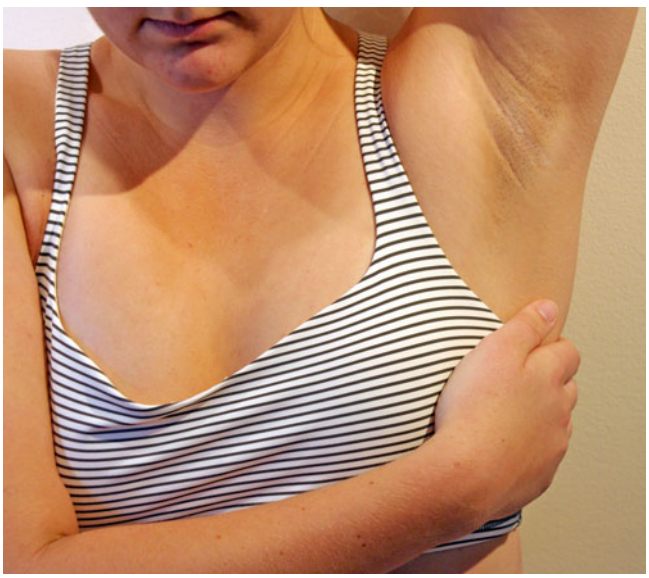

Fig. 30.2 Inferior scapular and lateral chest wall pattern of long thoracic neuralgia (Image courtesy of Andrea Trescot, MD)

tendonitis, and decreased range of motion, leading to adhesive capsulitis, subacromial impingement, and brachial plexus radiculitis [20].

Anatomy (Table 30.2)

True to its name, the LTN is long (27 ± 4 cm in one study) and superficial for most of its course, making it vulnerable to a variety of injuries [6]. It is derived from the ventral rami of the C5, C6, C7, and occasionally C8 (in 8 % of patients) nerve roots. The C5 to C6 portions usually traverse the middle scalene muscle, whereas the C7 contribution passes between the anterior and middle scalenes. These nerves unite distal to the scalene muscles to form the LTN (Fig. 30.3), which descends behind the brachial plexus and the axillary vessels and over the first and second ribs. It runs between the *subscapularis* and the *serratus anterior muscle* (SAM) and inferiorly along the chest wall in the midaxillary line on the outer surface of the SAM, which it innervates (Fig. 30.4) [21].

Table 30.2 Long thoracic nerve anatomy

Origin	Anterior branches of C5-C7 spinal nerves
General route	The C5 and C6 spinal nerves join in or near the middle scalene muscle
	The C7 contribution comes in 4–6 cm more distally, 33 % at the level of the first rib, 61 % at the second and is usually anterior to the middle scalene [6, 21]
	The nerve travels from behind the clavicle in the axillary sheath (part of the fascia surrounding the brachial plexus) and moves with movement of the arm
	It penetrates the sheath where the upper edge of the first rib crosses the midaxillary line to head down along the lateral thorax to the serratus anterior muscle [9]
	The axillary sheath is likely the "fascial sling," described by other authors [22]
	The nerve is minimally mobile and protected by the scapula and pectoral muscles until the fourth–fifth rib, where it is covered only by skin and subcutaneous tissue
	As it extends more distally, the nerve is increasingly mobile as the arm moves; the lower angle of the scapula rotates about 60° with arm movement [23]
Sensory distribution	No cutaneous distribution
Motor innervation	Serratus anterior muscle (SAM) stabilizes the scapula on the chest wall and is important for arm abduction and elevation. It rotates (as when the arm is lifted) and protracts (slides from medial to lateral as when the shoulder is pushed forward) the scapula. Also an accessory muscle of respiration
Anatomic variability	The number of spinal nerve branches: 84 % have all three, 8 % C5-C6 only, and 8 % C8 also [21]
	Level of the connections between the spinal nerves [6, 21]: 65 % at lateral border middle scalene, 22 % 2–5 cm distal to the lateral border, and 13 % at or below the clavicle [21]
	Relationship of the C5 and C6 portions to the middle and posterior scalene muscles [21]: 10/18 (56 %) between the middle and posterior scalene muscles, 6/18 (33 %) through the middle scalene, and 2/18 (11 %) between the middle scalene and brachial plexus [6]
	Distance between the LTN and the inferior angle of the scapula 0.5–5 cm [23]
Other relevant structures	Axillary sheath [6, 9]
	Thoracodorsal artery 11/18 (61 %) had a short branch crossing the LTN [3]
	Serratus anterior muscle (SAM) [24]: origin on ribs 1–9, inserts on the superior angle, medial border, and inferior angle of the scapula. The lower portions also may receive innervation from the intercostal nerves [6, 21]
	Bursae [21]

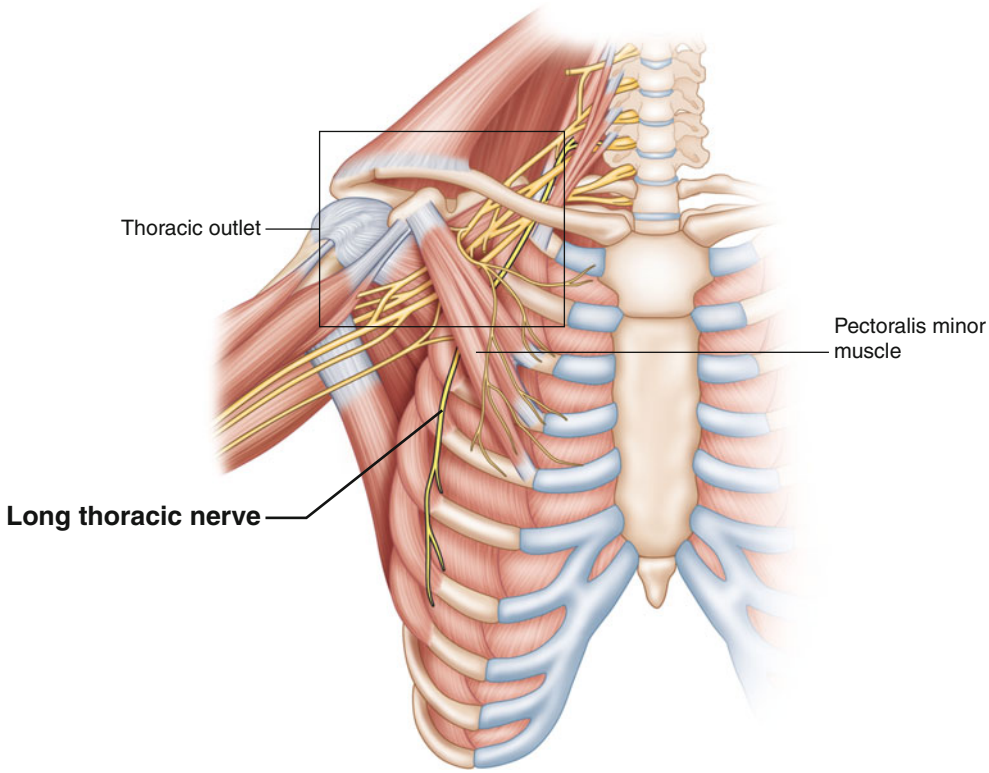

Fig. 30.3 Long thoracic nerve anatomy (Image by Springer)

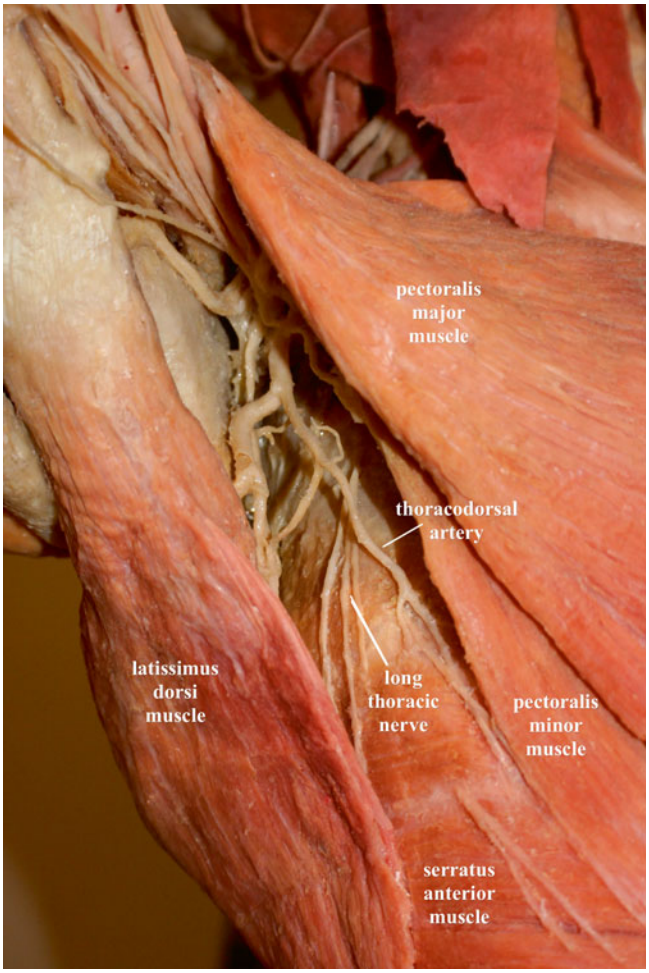

Fig. 30.4 Anatomy of the axilla and long thoracic nerve, modified from an image from *Bodies*, *The Exhibition*, with permission (Image courtesy of Andrea Trescot, MD)

Entrapment (Table 30.3)

Compression or entrapment can occur at any of a number of sites along the length of the LTN. The intricate anatomy of the cervicoaxillary regions and the complex actions of the SAM provide challenges for those evaluating and treating patients with shoulder pain and scapular winging. Areas of potential entrapment include between the middle and posterior scalene muscles, the first rib, between the clavicle and the second rib (under the *subclavicularis muscle*), between the second rib and the coracoid (under the pectoralis minor muscle), the inferior angle of the scapula, or by inflamed bursa along the course of the LTN [10]. The nerve can also be injured by penetrating trauma or surgical procedures; the pathology may be evident immediately or delayed for several years as a cicatrix forms around the nerve.

Successful surgical nerve releases have been described in both cervical and thoracic regions (see below). Accurate diagnosis of both the fact and the site of LTN entrapment is key to providing successful therapy.

Physical Exam

The upward medial displacement and prominence of the scapula seen in patients with LTN dysfunction is subtle while at rest. The scapula is slightly higher and closer to the midline (medial winging) when compared to the unaffected side, and it can be amplified by a "wall push-up" (Fig. 30.5) [4, 5, 7]. Patients will demonstrate difficulty in forward flexing the arm above shoulder level and will have decreased shoulder range of motion and strength. Point tenderness and paresthesias may

Table 30.3 Reported sites of potential LTN Entrapment (from proximal to distal)

Anatomic site	Comment
In the middle scalene muscle or between the middle and posterior scalenes [6, 21, 25, 26]	However, the dorsal scapular nerve that innervates the rhomboids also frequently traverses the middle scalene, and the C7 contribution to the LTN almost always is anterior to it. These facts make this a less likely cause of isolated LTN dysfunction [5, 21, 24]
Between the clavicle and second rib [27]	However, one author tried to push on cadaver arms and was not able to compress the nerve at this site without simultaneous injury to other axillary contents [7]
Between the coracoid process and second rib [21]	A subsequent anatomist found no evidence for this [23]
Between the under surface of the scapula and the second rib [15]	
Raising the arm bends and stretches the LTN where it comes out from the axillary sheath or fascial sling, since the axillary sheath moves with the arm and both ends are fixed [9, 22]	
Compression by a branch of the thoracodorsal artery where it crosses the LTN [3, 28]	
Chest tube placement in the midaxillary line [29]	
Anterior to the lower angle of the scapula: passive arm adduction and extension may lead to traction and compression of the LTN possibly compounded by relative ischemia if this movement also decreases flow in the accompanying blood vessels [23]	

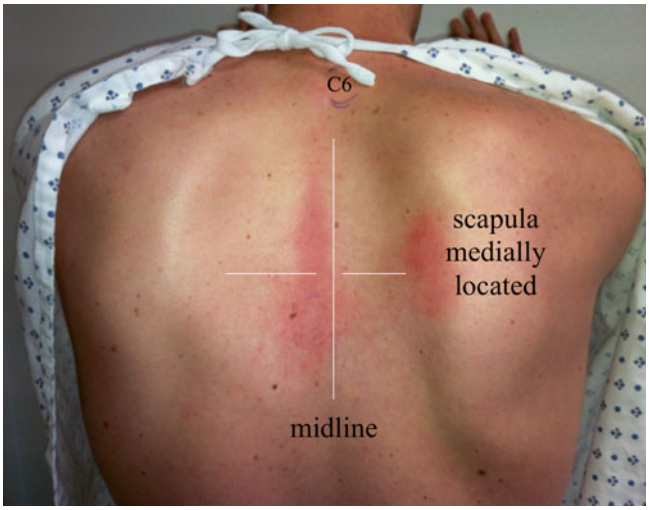

Fig. 30.5 Winged scapula from long thoracic nerve injury. Note the right scapula has prominent winging during a "wall push-up," with the medial border of the scapula more medially located than the left (Image courtesy of Heath McAnally, MD)

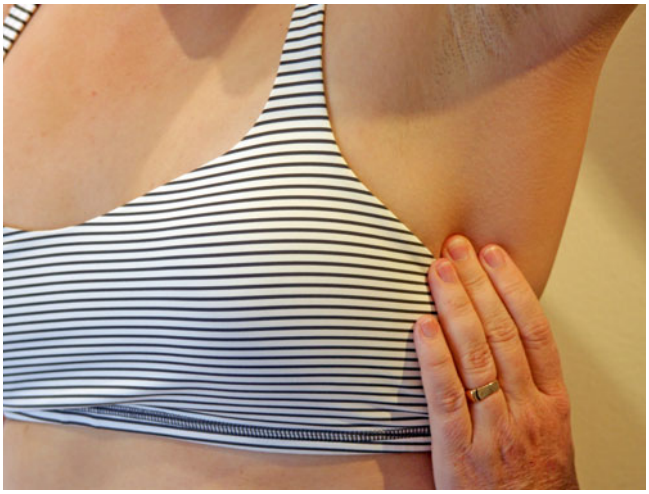

Fig. 30.6 Axillary examination of the long thoracic nerve (Image courtesy of Andrea Trescot, MD)

be elicited by palpation in the axilla and lateral chest wall in the midaxillary line along the course of the LTN (Video 30.1) that will radiate vertically into the axilla (Fig. 30.6) [3].

Medial scapular winging must be differentiated from *lateral scapular winging* caused by the trapezius from spinal accessory nerve pathology (see Chap. 26) or rhomboid paralysis from dorsal scapular neuralgia (see Chap. 32). Lateral winging is elicited with the arms abducted, not with forward elevation; typically, the affected shoulder droops, and the patient is not able to shrug [4, 5, 10]. Table 30.4 compares the types of winging.

Table 30.4 Comparison of winging from long thoracic, spinal accessory, and dorsal scapular nerve pathology

Nerve	Muscles involved	Type of winging	Provocative maneuvers
Long thoracic	Serratus anterior	Medial winging	Forward elevation and pushing with outstretched arms (wall push-up)
Spinal accessory	Trapezius	Lateral winging with drooping shoulder	Resisted arm abduction or external rotation
Dorsal scapular (a rare cause of subtle winging)	Rhomboid and levator scapulae	Scapula shifted laterally and dorsally	Slowly lowering arm from forward elevation

Differential Diagnosis (Table 30.5)

There are a variety of conditions that can mimic LTN dysfunction (see Table 30.5). Spinal accessory nerve injury presents with lateral winging, unlike the medial winging of LTN injury. Pathology of the dorsal scapular nerve leads to rhomboid paralysis, but does not usually result in winging. Usually, pain is not a major complaint with LTN entrapment. Severe pain should raise suspicion of *brachial neuritis* (also called *Parsonage–Turner syndrome*), an inflammation of the brachial plexus. MRI imaging showing edema of the SAM can also help to confirm the diagnosis function (See Tables 30.5 and 30.6).

Table 30.7 lists some of the diagnostic tests for long thoracic nerve entrapment. MRI of the shoulder may offer diagnostic clues, since edema (on T2-weighted images) or atrophy of the muscles of the shoulder can be related to the corresponding nerve pathology (Fig. 30.7 and Table 30.6). Wait at least 2–4 weeks after initial injury before performing electromyography (EMG) of the LTN. It can take that long to see the abnormal spontaneous potentials indicative of denervation (positive sharp waves and fibrillations), which can confirm the diagnosis. Both EMG and nerve conduction studies can help identify the location and severity of the injury.

Identification and Treatment of Contributing Factors

Three stages of LTN injury have been characterized [31]. In the acute stage, the LTN injury causes SAM pain, and the goals of treatment include pain reduction, range of motion (ROM) exercises, and activity modification to limit further injury to the shoulder. Once the nerve begins to heal, passive stretching of the rhomboids, levator scapulae, and pectoralis minor is used to prevent contracture of these muscles due to

Table 30.5 Differential diagnosis of scapular and shoulder pain

	Potential distinguishing features
Spinal accessory nerve injury leading to trapezius paralysis [7] (see Chap. 26)	Lateral winging [4, 5] and the shoulder are depressed (unable to shrug) with the scapula lower and further from the midline when compared to the unaffected side. It is increased by external rotation against resistance. Patients' primary difficulty is with arm abduction
Dorsal scapular nerve injury leading to rhomboid paralysis [4, 5] (see Chap. 32)	Pain and atrophy near the medial border of the scapula. Minimal winging at rest. With hands on hips, limited ability to push elbow back against resistance
Glenohumeral joint disorders [4] (Acromial fracture [7], aseptic necrosis of the humeral head [7], rotator cuff tendinitis or tear, adhesive capsulitis)	Normal electrodiagnostic studies
	X-rays and MRI, Abnormal X-rays and MRI
Parsonage–Turner brachial neuritis [3, 18, 19]	Severe pain for several weeks before palsy
	Often involves other shoulder muscles
	Usually resolves spontaneously
Thoracic outlet syndrome (TOS) [3] (see Chap. 33)	Scalene spasm
	Decreased radial pulses
C7 radiculopathy or cervical disk disease [24]	Numbness, weakness, and decreased reflexes
Facioscapulohumeral muscular dystrophy [3]	Facial muscle weakness as well as shoulder girdle weakness
	Spreads to other muscles
Other potential sources of periscapular pain	SAM dysfunction leads to imbalance of the other shoulder girdle muscles and painful tonic contraction of the trapezius, levator scapulae, and rhomboid muscles [3]

Table 30.6 Relationship between muscle pathology and nerve entrapment

Denervated muscle	Suspected nerve entrapment
Serratus anterior muscle	Long thoracic nerve
Supraspinatus and infraspinatus muscles	Suprascapular nerve at suprascapular notch (Chap. 28)
Trapezius muscle	Spinal accessory nerve (Chap. 27)
Isolated infraspinatus muscle	Suprascapular nerve at spinoglenoid (Chap. 28)
Teres minor muscle	Axillary nerve at quadrilateral space (Chap. 31)
Rhomboid and/or levator muscle	Dorsal scapular nerve at scalene muscle (Chap. 32)

Table 30.7 Diagnostic tests for long thoracic nerve entrapment

	Potential distinguishing features
Physical exam	Scapular winging
Provocative maneuvers [7]	Both arms stretched forward 90°; pushing on a wall will elicit winging
Diagnostic injection	At middle scalene muscle (Fig. 30.8) or axilla (Fig. 30.9)
Ultrasound	Nerve can be identified at scalene, infraclavicular, or axillary sites
MRI	To rule out a neoplasm in patients without a history of trauma or brachial plexitis May show edema of the SAM
Arteriography	Not useful
X-ray	To evaluate integrity of bony structures
Electrodiagnostic studies	Conflicting opinions
	Does not predict outcome, even with severe injury [16]
	EMG "is a definitive part of the evaluation" [3, 7]
	Nerve conduction: both needle and surface electrode methods should be used [30]

the loss of SAM activity. In the third stage, the SAM becomes progressively stronger and shoulder mechanics improve, so strengthening exercises of all shoulder girdle muscles, including the trapezius, should be implemented. Continue to avoid overstretching the SAM. Some have recommended the use of a custom brace to help support the scapula; however, success with this device is inconsistent [10].

Frequently, there is spontaneous improvement with "benign neglect": avoiding activities that put excess traction on the nerve as well as stretching and strengthening associated muscles [7]. It is important for patients to keep their shoulders moving so as to avoid adhesive capsulitis [4, 10]. Overall, this approach has led to good outcomes in patients with entrapments and closed, athletic, or incomplete injuries; most patients recover within 1 year [4, 7]. If there is insufficient improvement with these measures or evidence of complete LTN transection, consider referral for surgery to avoid permanent damage to the LTN and the SAM [3].

Interestingly, according to Friendenberg et al., electrodiagnostic studies did not predict the functional outcome [16]. Idiopathic or inflammatory LTN pathologies were associated with a poor prognosis.

Injection Technique

Landmark-Guided Injection

Because the LTN passes through the middle scalene muscle, a proximal injection into the muscle can anesthetize the nerve for diagnostic and potentially therapeutic effects (especially if the nerve is entrapped by the middle scalene muscle). After a sterile prep, a local anesthetic wheal is

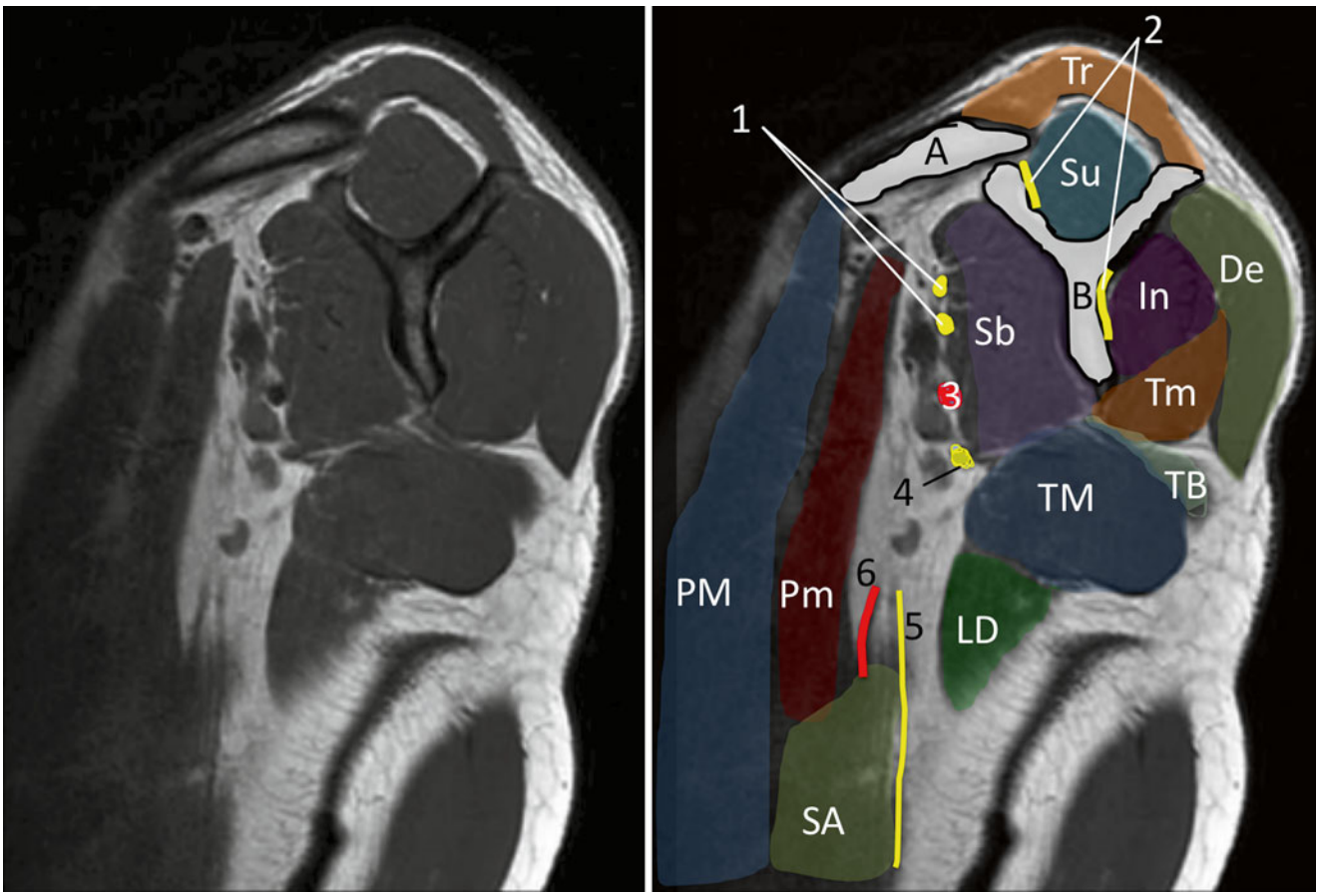

Fig. 30.7 Sagittal MRI image of the scapular structures. *A* clavicle, *B* scapula, *De* deltoid muscle, *In* infraspinatus muscle, *LD* latissimus dorsi muscle, *PM* pectoralis major muscle, *Pm* pectoralis minor muscle, *Sb* subscapularis muscle, *Su* suprascapular muscle, *TM* teres major muscle, *Tm* teres minor muscle, *Tr* trapezius muscle, *TB* triceps brachii muscle, *1* brachial plexus, *2* suprascapular nerve, *3* axillary artery, *4* suprascapular nerve, *5* long thoracic nerve, *6* long thoracic artery (Image courtesy of Andrea Trescot, MD)

placed at the apex of the posterior triangle of the neck, between the SCM and the trapezius, and the needle is advanced into the body of the middle scalene muscle (Fig. 30.8).

Ramamurthy et al. reported nerve stimulator-guided LTN injections to treat lateral chest wall pain thought to be due to SAM spasm [32]. They identified the middle scalene muscle by palpation, entered it at the level of C6, and directed the needle caudally, watching for maximum SAM contractions in response to stimulation. A successful block should result in SAM motor block and may block other nearby branches of the superficial cervical plexus.

The LTN can be approached distally at the axilla, which can be particularly useful for LTN injuries from chest wall injuries. The fingers of the non-injecting hand secure the nerve, while the needle is directed cephalad between the fingers (Video 30.2) (Fig. 30.9). Caution should be used when performing this injection because the lung is just deep to the injection site.

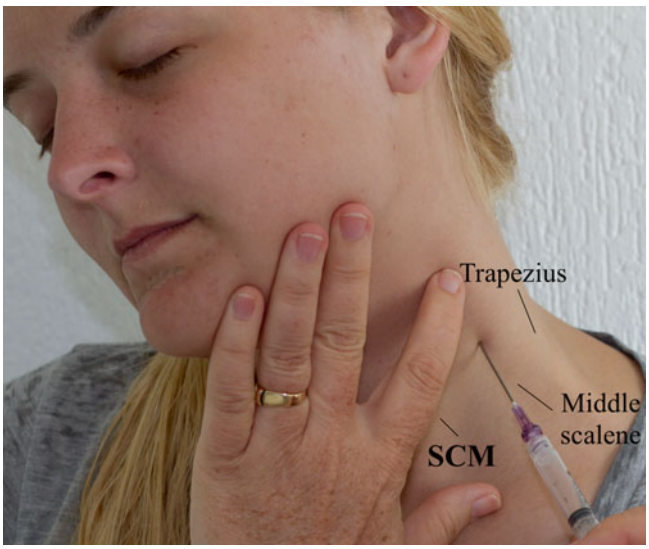

Fig. 30.8 Landmark-guided injection of the proximal long thoracic nerve at the middle scalene muscle (Image courtesy of Andrea Trescot, MD)

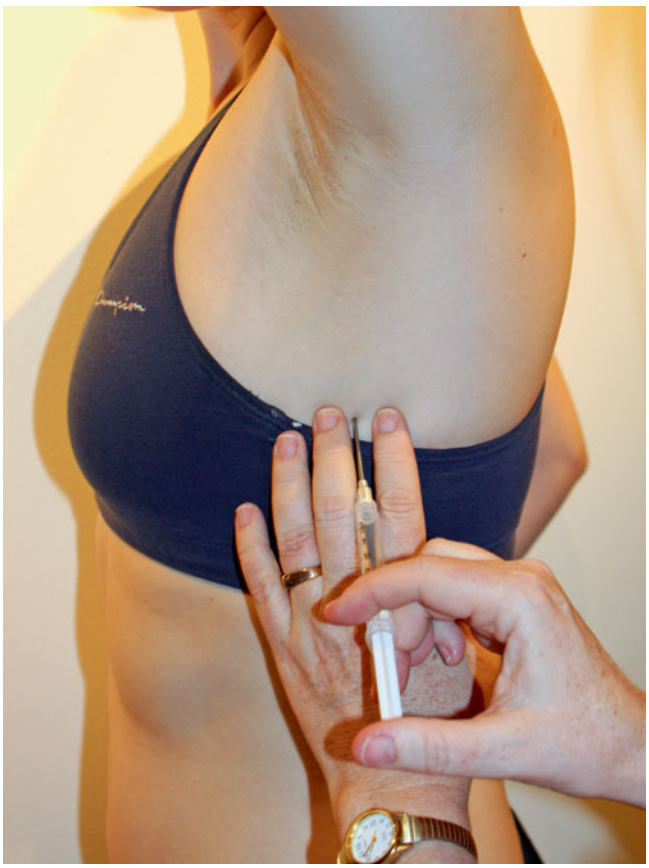

Fig. 30.9 Landmark-guided injection of the long thoracic nerve at the axilla (Image courtesy of Andrea Trescot, MD)

Fluoroscopic-Guided Injections

There are no described fluoroscopic-guided techniques for LTN injection.

Ultrasound-Guided Injections

Because of the substantial variability in the anatomy of the nerves and muscles in this highly vascular area of the neck (Table 30.2), ultrasound (US) guidance confirmed with nerve stimulation is particularly useful for LTN injections.

In the neck, the LTN is usually a flat structure within the body of the middle scalene muscle (Fig. 30.10), and it becomes round after it leaves the muscle. It travels under the clavicle and pectoralis major and minor muscles (Fig. 30.11) and then into the axilla (Fig. 30.12).

Hanson and Auyong [33] described US identification of the dorsal scapular nerve and LTN during interscalene blocks in 50 patients scheduled for shoulder surgery. They defined the nerves as hyperechoic structures with hypoechoic centers within or superficial to the middle scalene muscle; 90 % of patients had one or both nerves visible. A stimulating needle was inserted at a site more posterior than has been traditionally taught and used to confirm nerve identity. Palpation of a SAM contraction indicated stimulation of the LTN (23 %), while contractions of the rhomboids and levator scapulae showed dorsal scapular nerve stimulation (77 %).

The ultrasound-guided injection can be performed in plane or out of plane, with or without a peripheral nerve stimulator, in the neck (Fig. 30.13), subclavicular region (Fig. 30.14), or axilla (Fig. 30.15).

Blanco et al. [34] described the early results of a "serratus plane block," injecting a large volume of local anesthetic superficial or deep to the SAM at T4 and T5 with the US probe in the midaxillary line to provide anesthesia for lateral chest wall surgery. This block anesthetizes the lateral cutaneous branches of the intercostal nerves and presumably the LTN.

Neurolytic Techniques

Cryoneuroablation/Radio-frequency Lesioning

There are no reported techniques of cryoneuroablation or radio-frequency lesioning of the LTN. However, Trescot has used cryoneuroablation to treat LTN dysfunction, directing the cryoprobe from inferior to superior, parallel to the nerve (personal communication) (Fig. 30.16).

Alcohol/Phenol

There are no published reports of the use of phenol or alcohol for LTN ablation.

Surgical Treatment

Successful surgical neurolysis has been described at several different anatomic levels. When entrapment was deemed to be within or near the middle scalene muscle, a supraclavicular approach has been used [25, 26]. Many of the patients in one small series probably also had *thoracic outlet syndrome* (Chap. 33) [25]. In another report, Nath and Melcher observed LTN compression within the middle scalene muscle at surgery with narrowing of the nerve and epineural neovascularization [26], though it was unclear how patients were diagnosed and selected for surgery.

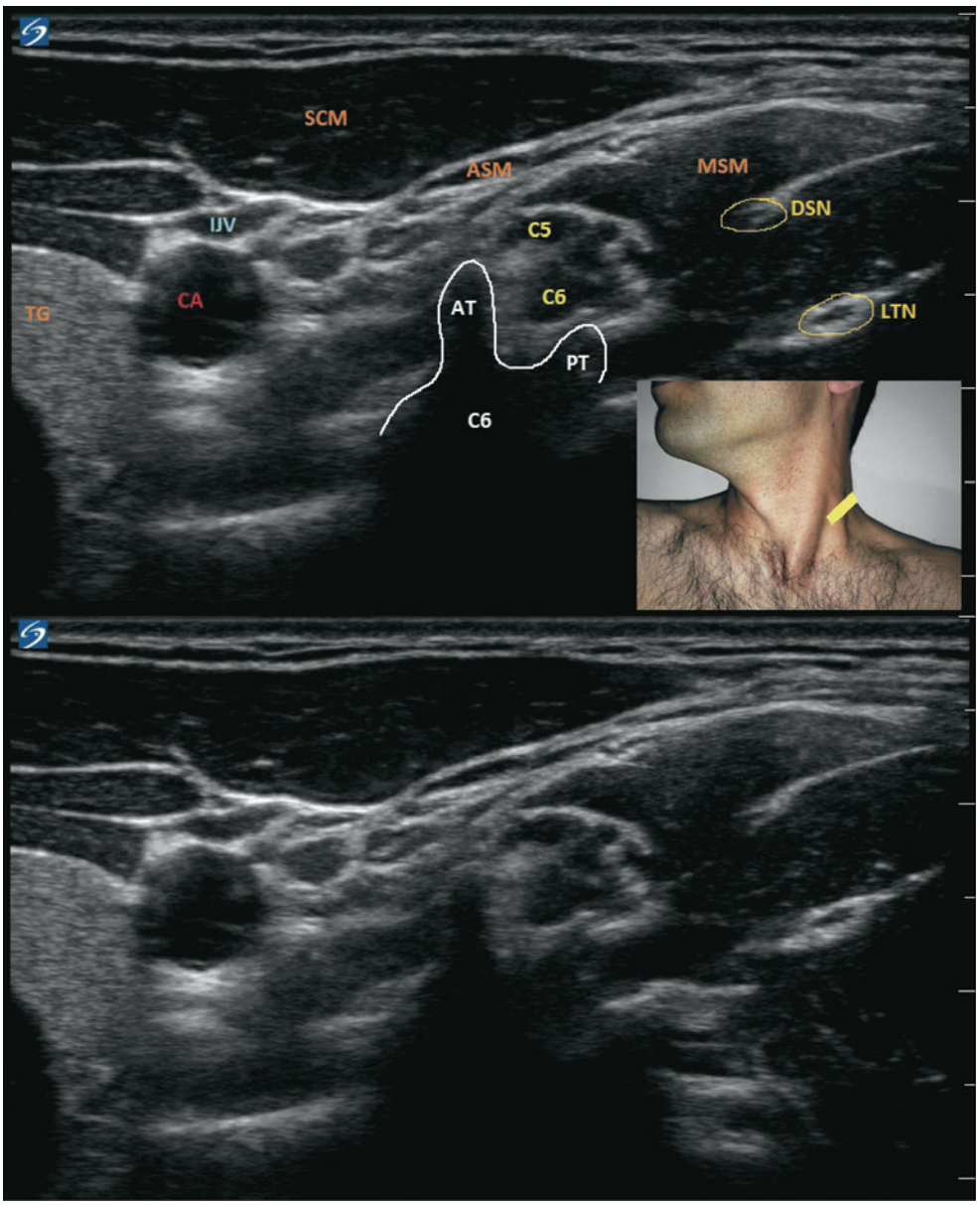

Fig. 30.10 Ultrasound identification of the long thoracic nerve at the middle scalene muscle. *SCM* sternocleidomastoid muscle, *MSM* middle scalene muscle, *ASM* anterior scalene muscle, *TG* thyroid gland, *CA* carotid artery, *AT* anterior tubercle of C6 vertebra, *PT* Posterior tubercle of C6 vertebra, *DSN* dorsal scapular nerve, *LTN* long thoracic nerve (Image courtesy of Agnes Stogicza, MD)

Nath also worked with another group [20], reporting the results of supraclavicular decompression and neurolysis of the LTN in 50 cases (47 patients, 3 of whom were bilateral). They noted the importance of identifying the suprascapular nerve in this region, as it often traverses the middle layers of the scalene fat pad (see Chap. 28). Seventy-three percent of the patients who had pain from the LTN entrapment noted relief of their pain postoperatively, and all had improved range of motion.

More distal neurolysis has been described in a larger number of patients. Laulan et al. [3] reported on 18 consecutive patients who underwent surgical neurolysis in the thoracic region; patients had isolated SAM paralysis for more than 3 months, tenderness along the thoracic portion of the LTN, and abnormal EMG on at least two occasions. At surgery, the authors saw fibrosis and vascular or muscular compression along the thoracic portion of the nerve. Pain relief was rapid and usually preceded the return of SA

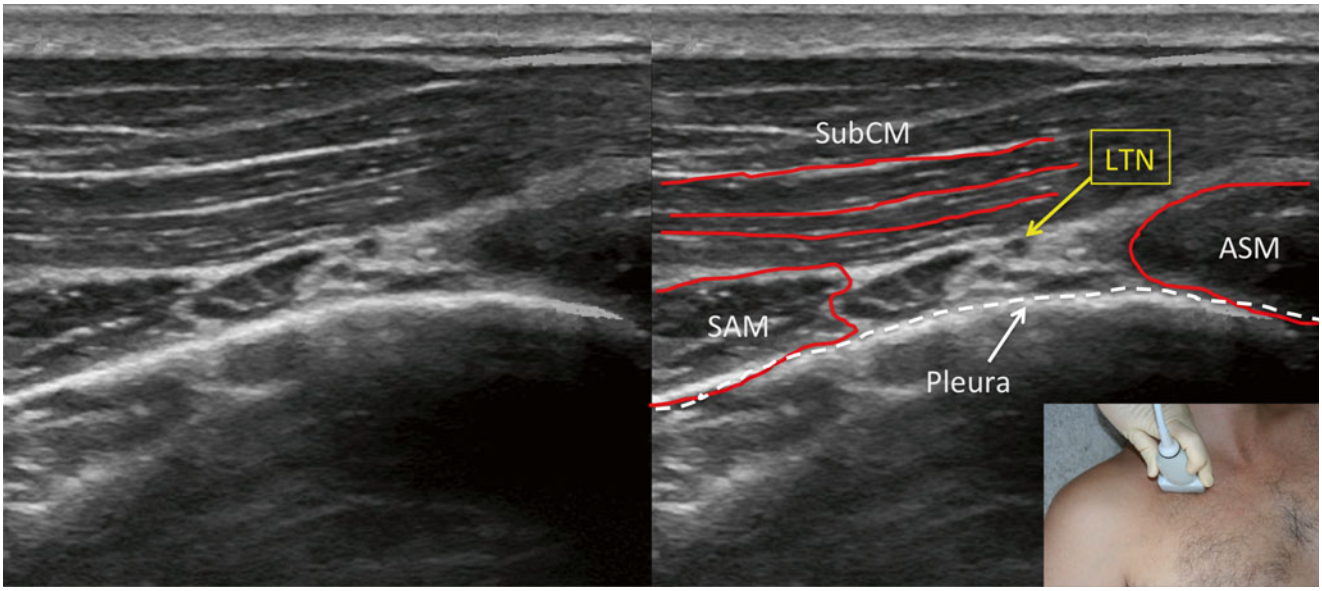

Fig. 30.11 Ultrasound identification of the long thoracic nerve at the subclavicular region. *LTN* long thoracic nerve, *SAM* serratus anterior muscle, *SubCM* subclavicularis muscle, *ASM* anterior scalene muscle (Image courtesy of Andrea Trescot, MD)

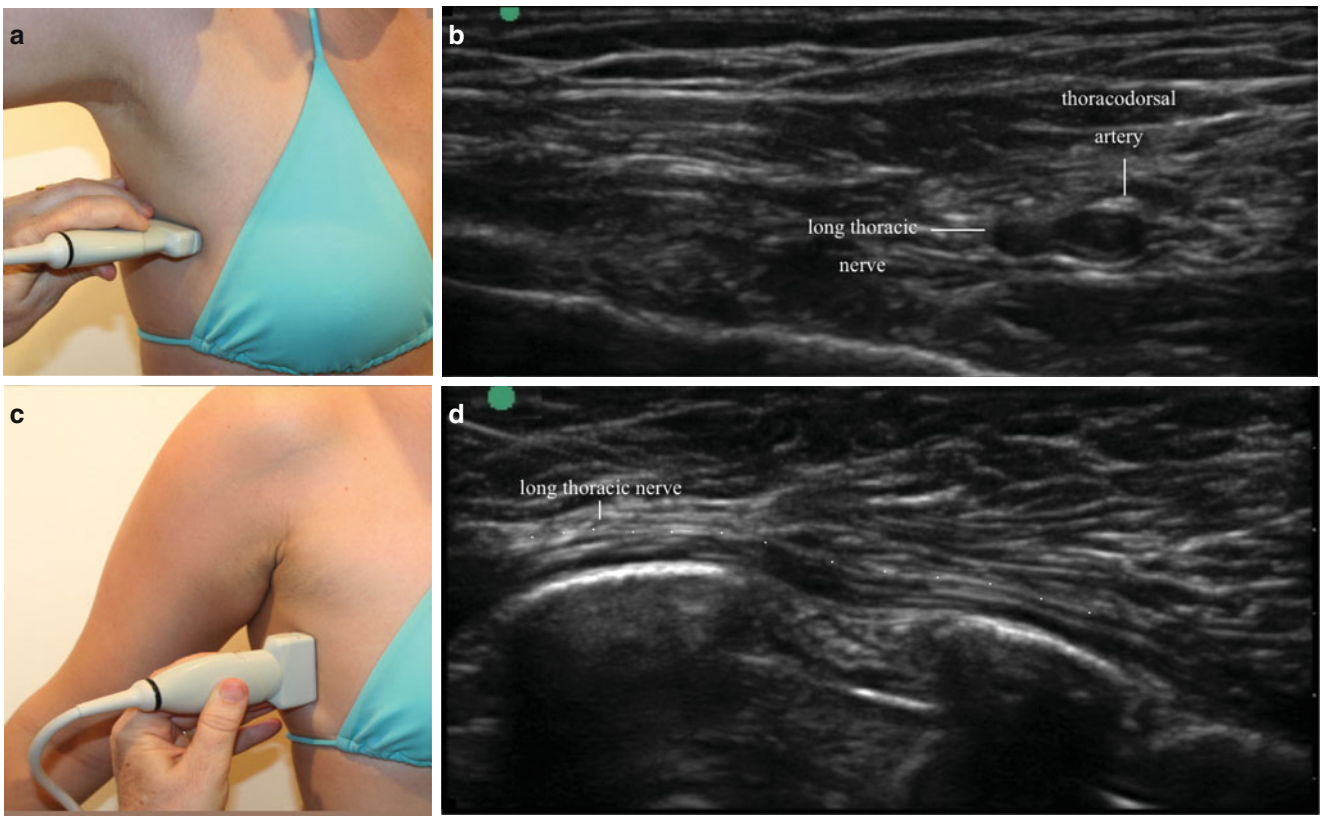

Fig. 30.12 Ultrasound of long thoracic nerve at the axilla. The nerve is first imaged with the probe horizontal (**a**, **b**), and then the probe is rotated vertically to image the ribs more clearly (**c**, **d**) (Image courtesy of Andrea Trescot, MD)

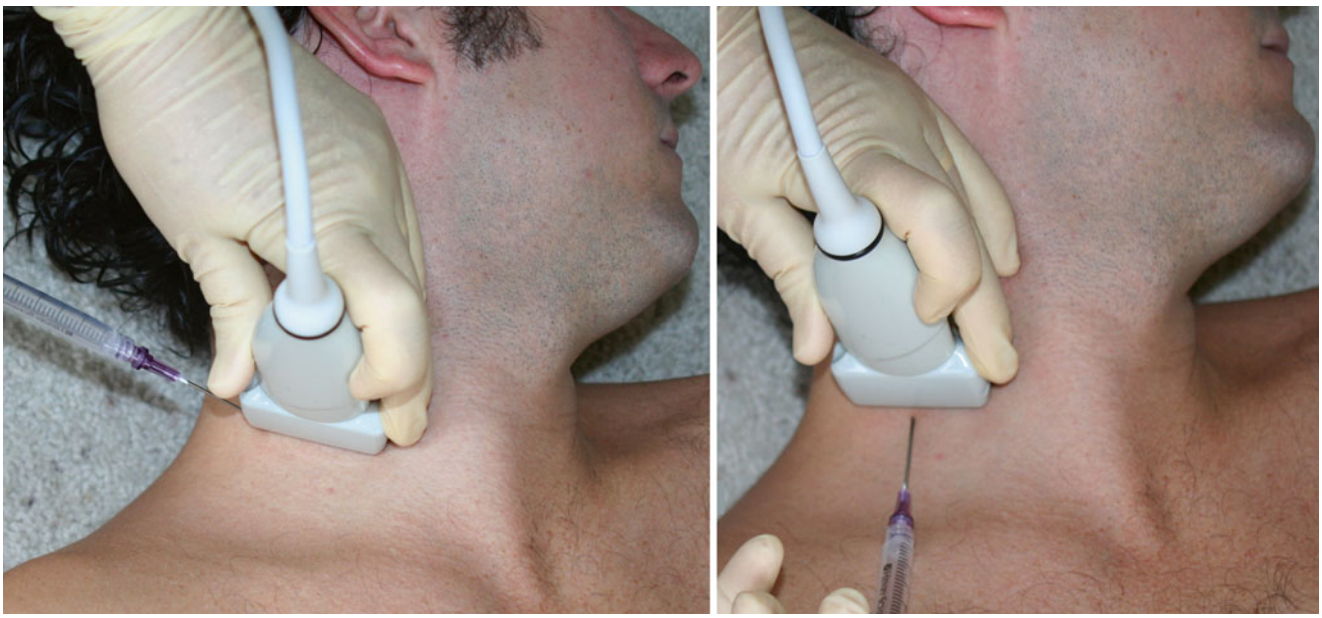

Fig. 30.13 Ultrasound-guided injection of the long thoracic nerve at the middle scalene muscle, showing in-plane (image **a**) and out-of-plane (image **b**) approach (Image courtesy of Andrea Trescot, MD)

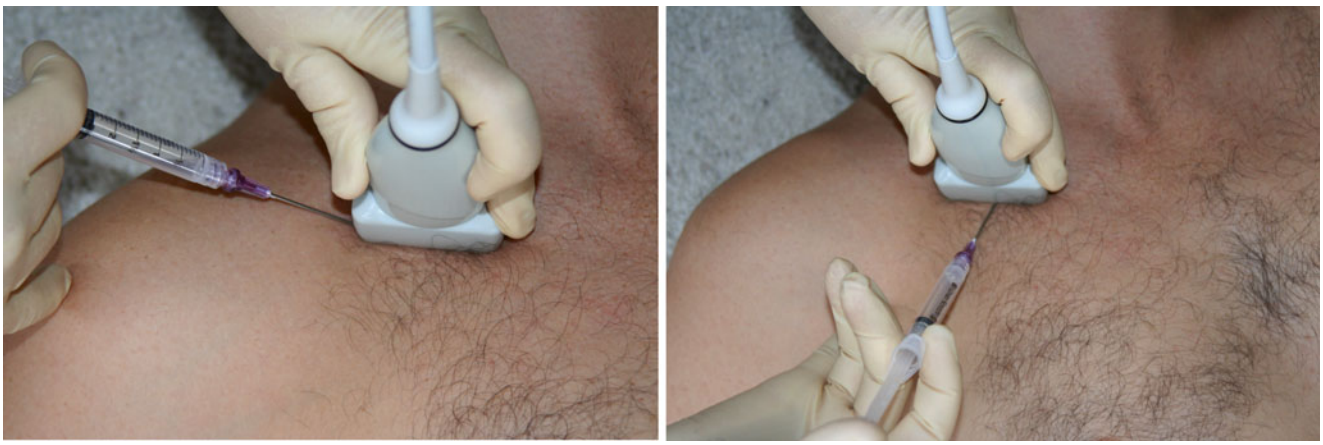

Fig. 30.14 Ultrasound-guided injection of the long thoracic nerve at the subclavicular region, showing in-plane and out-of-plane approach (Image courtesy of Andrea Trescot, MD)

strength. All of the patients in this study were greatly improved or recovered: all were pain free at rest and during daily activities, and all returned to their former occupation, though results were best when surgery was performed within 6 months of the initial paralysis [3]. Most recently, Le Nail et al. [28] described 52 consecutive cases of LTN entrapment treated surgically; 36 patients had pain preoperatively, and all had EMG abnormalities before surgery. After an average of 2.5 years, 45 patients reported good to excellent responses, and none was worse after surgery.

If symptoms persist beyond a year or 2 despite nonoperative management, and there are no signs of reinnervation on EMG, the patient may be a candidate for reconstructive surgery. Traditional surgical options include muscle transfers, scapulopexy, and scapulothoracic fusion, although none of these procedures will allow return to most competitive sports that require arm strength and motion [5, 10].

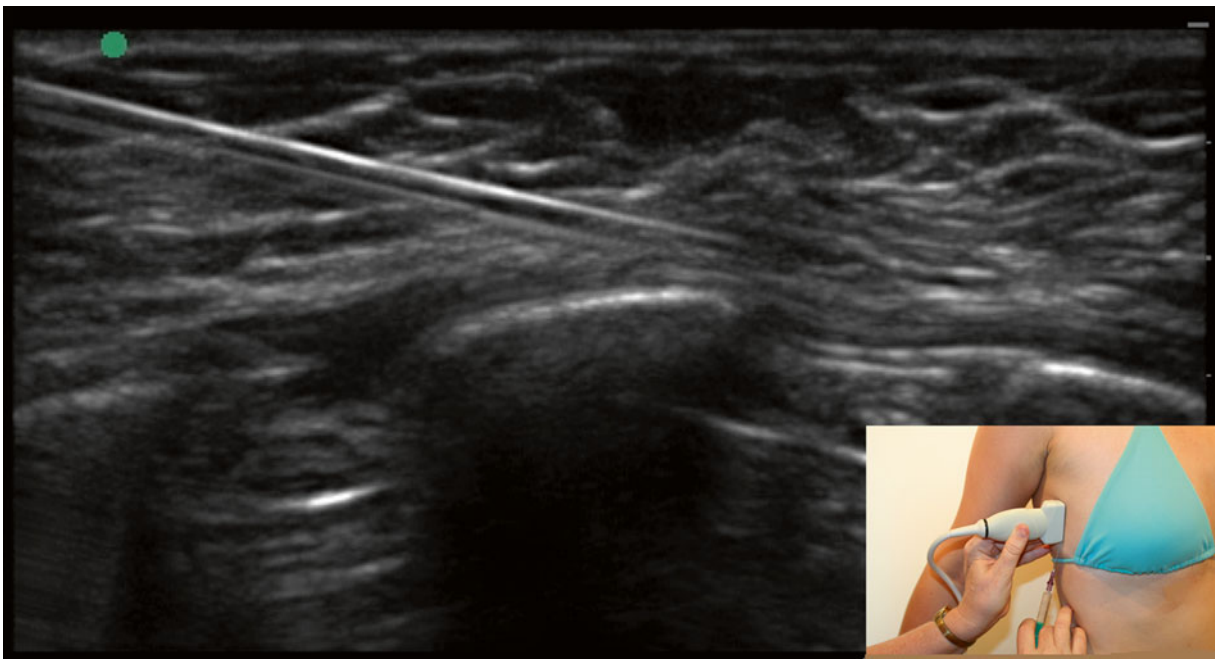

Fig. 30.15 Ultrasound-guided injection of the long thoracic nerve at the axilla using an in-plane technique (Image courtesy of Andrea Trescot, MD)

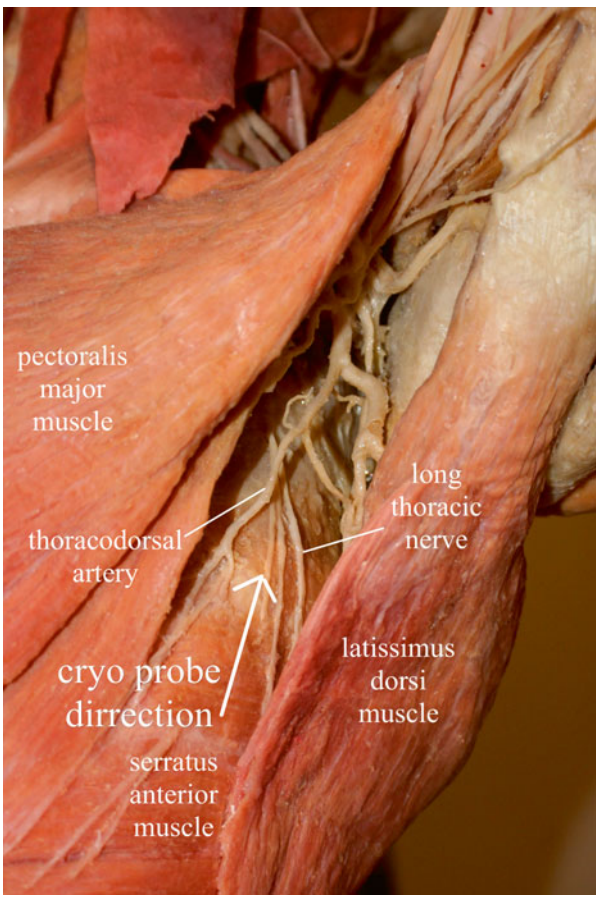

Fig. 30.16 Simulated site of cryoprobe placement (Image courtesy of Andrea Trescot, MD)

Summary

Scapular winging is a rare, disabling condition that can seriously impair athletic performance and negatively impact activities of daily living. It contributes to loss of power and range of motion of the upper extremity and can be a source of considerable pain [4, 5]. Injuries to several different nerves can cause scapular winging, and partial or complete LTN injury can occur at multiple levels. These facts underline the importance of careful diagnosis as the foundation for successful treatment of LTN entrapment.

References

1. Thomas SE, Winchester JB, Hickman G, DeBusk E. A confirmed case of injury to the long thoracic nerve following a posterior approach to an interscalene nerve block. Reg Anesth Pain Med. 2013;38(4):370.
2. Srikumaran U, Wells JH, Freehill MT, Tan EW, Higgins LD, Warner JJ. Scapular winging: a great masquerader of shoulder disorders: AAOS exhibit selection. J Bone Joint Surg Am. 2014; 96(14):e122.
3. Laulan J, Lascar T, Saint-Cast Y, Chammas M, Le Nen D. Isolated paralysis of the serratus anterior muscle successfully treated by surgical release of the distal portion of the long thoracic nerve. Chirurgie de la main. 2011;30(2):90–6.
4. Kuhn JE, Plancher KD, Hawkins RJ. Scapular winging. J Am Acad Orthop Surg. 1995;3(6):319–25.
5. Martin RM, Fish DE. Scapular winging: anatomical review, diagnosis, and treatments. Curr Rev Musculoskelet Med. 2008;1(1): 1–11.

6. Tubbs RS, Salter EG, Custis JW, Wellons 3rd JC, Blount JP, Oakes WJ. Surgical anatomy of the cervical and infraclavicular parts of the long thoracic nerve. J Neurosurg. 2006;104(5):792–5.
7. Gregg JR, Labosky D, Harty M, Lotke P, Ecker M, DiStefano V, et al. Serratus anterior paralysis in the young athlete. J Bone Joint Surg Am. 1979;61(6a):825–32.
8. Vastamaki M, Kauppila LI. Etiologic factors in isolated paralysis of the serratus anterior muscle: a report of 197 cases. J Shoulder Elbow Surg. 1993;2(5):240–3.
9. Ebraheim NA, Lu J, Porshinsky B, Heck BE, Yeasting RA. Vulnerability of long thoracic nerve: an anatomic study. J Shoulder Elbow Surg. 1998;7(5):458–61.
10. Safran MR. Nerve injury about the shoulder in athletes, part 2: long thoracic nerve, spinal accessory nerve, burners/stingers, thoracic outlet syndrome. Am J Sports Med. 2004;32(4):1063–76.
11. Oware A, Herskovitz S, Berger AR. Long thoracic nerve palsy following cervical chiropractic manipulation. Muscle Nerve. 1995;18(11):1351.
12. Murphy MT, Journeaux SF. Case reports: long thoracic nerve palsy after using a single axillary crutch. Clin Orthop Relat Res. 2006; 447:267–9.
13. Lederman RJ. Focal peripheral neuropathies in instrumental musicians. Phys Med Rehabil Clin N Am. 2006;17(4):761–79.
14. Bizzarri F, Davoli G, Bouklas D, Oncchio L, Frati G, Neri E. Iatrogenic injury to the long thoracic nerve: an underestimated cause of morbidity after cardiac surgery. Tex Heart Inst J. 2001;28(4):315–7.
15. Gozna ER, Harris WR. Traumatic winging of the scapula. J Bone Joint Surg Am. 1979;61(8):1230–3.
16. Friedenberg SM, Zimprich T, Harper CM. The natural history of long thoracic and spinal accessory neuropathies. Muscle Nerve. 2002;25(4):535–9.
17. Mondelli M, Aretini A, Ginanneschi F. Predictive factors of recovery in long thoracic mononeuropathy. Orthopedics. 2013;36(6): e707–14.
18. Parsonage MJ, Turner JW. Neuralgic amyotrophy; the shoulder-girdle syndrome. Lancet. 1948;1(6513):973–8.
19. Foo CL, Swann M. Isolated paralysis of the serratus anterior. A report of 20 cases. J Bone Joint Surg (Br). 1983;65(5):552–6.
20. Nath RK, Lyons AB, Bietz G. Microneurolysis and decompression of long thoracic nerve injury are effective in reversing scapular winging: long-term results in 50 cases. BMC Musculoskelet Disord. 2007;8:25–32.
21. Horwitz MT, Tocantins LM. An anatomical study of the role of the long thoracic nerve and the related scapular bursae in the pathogenesis of local paralysis of the serratus anterior muscle. Anat Rec. 1938;71(4):375–85.
22. Hester P, Caborn DN, Nyland J. Cause of long thoracic nerve palsy: a possible dynamic fascial sling cause. J Shoulder Elbow Surg. 2000;9(1):31–5.
23. Kauppila LI. The long thoracic nerve: possible mechanisms of injury based on autopsy study. J Shoulder Elbow Surg. 1993;2(5):244–8.
24. Hamada J, Igarashi E, Akita K, Mochizuki T. A cadaveric study of the serratus anterior muscle and the long thoracic nerve. J Shoulder Elbow Surg. 2008;17(5):790–4.
25. Disa JJ, Wang B, Dellon AL. Correction of scapular winging by supraclavicular neurolysis of the long thoracic nerve. J Reconstr Microsurg. 2001;17(2):79–84.
26. Nath RK, Melcher SE. Rapid recovery of serratus anterior muscle function after microneurolysis of long thoracic nerve injury. J Brachial Plex Peripher Nerve Inj. 2007;2:4.
27. Wiater JM, Flatow EL. Long thoracic nerve injury. Clin Orthop Relat Res. 1999;368:17–27.
28. Le Nail LR, Bacle G, Marteau E, Corcia P, Favard L, Laulan J. Isolated paralysis of the serratus anterior muscle: surgical release of the distal segment of the long thoracic nerve in 52 patients. Orthop Traumatol Surg Res. 2014;100(4s):S243–s8.
29. Hassan WU, Keaney NP. Winging of the scapula: an unusual complication of chest tube placement. J Accid Emerg Med. 1995; 12(2):156–7.
30. Seror P. The long thoracic nerve conduction study revisited in 2006. Clin Neurophysiol. 2006;117(11):2446–50.
31. Watson CJ, Schenkman M. Physical therapy management of isolated serratus anterior muscle paralysis. Phys Ther. 1995;75(3):194–202.
32. Ramamurthy S, Hickey R, Maytorena A, Hoffman J, Kalantri A. Long thoracic nerve block. Anesth Analg. 1990;71(2):197–9.
33. Hanson NA, Auyong DB. Systematic ultrasound identification of the dorsal scapular and long thoracic nerves during interscalene block. Reg Anesth Pain Med. 2013;38(1):54–7.
34. Blanco R, Parras T, McDonnell JG, Prats-Galino A. Serratus plane block: a novel ultrasound-guided thoracic wall nerve block. Anaesthesia. 2013;68(11):1107–13.

Axillary Nerve Entrapment

31

Christopher J. Burnett and Helen W. Karl

Introduction

The *axillary nerve* (AN) is a mixed sensory and motor nerve, and is one of the terminal branches of the brachial plexus. Its primary area for potential entrapment is in the *quadrilateral space* (also known as the *quadrangular space*), hence, the name *quadrilateral space syndrome*, but it can be injured in a multitude of ways. Regardless of the mechanism, injury of the AN results in a subtle but distinctive kind of shoulder pain, first described by Cahill in 1983 [1]. It is an uncommon (or poorly recognized) condition, found in just 0.8 % of patients referred for shoulder MRI, and it is usually associated with other pathologies [2].

Clinical Presentation (Table 31.1)

Patients suffering from AN entrapment or injury are mostly young adults presenting with symptoms in the dominant arm. Athletes involved in overhead sports requiring repetitive movements are at particular risk [5]. There can be a work-related component, as seen with the arm in abduction for window cleaning [9]. Patients have diffuse, poorly localized shoulder and upper arm pain (Fig. 31.1) that may have an insidious onset. Those affected often complain of arm fatigue with overhead activity or throwing, and many report a non-dermatomal pattern of dull ache, burning, or paresthesias of the lateral and posterior upper arm (Fig. 31.2), especially in the deltoid region, that may wake them from sleep [1, 3, 5, 10]. The pain is aggravated by sustained arm flexion, abduction and external rotation, or overhead activities [5]. Trauma and stretching of the nerve occur with dislocation of the shoulder (between 19 and 55 % of patients with an anterior shoulder dislocation have AN entrapment) and proximal humeral fractures (up to 58 % of patients with a proximal humeral fracture will have AN dysfunction) [5].

Table 31.1 Occupation/exercise/trauma history relevant to axillary nerve entrapment

Repetitive overhead movements (overuse)	Baseball pitching, tennis, volleyball [3]
Direct pressure	Improper crutch use [3], prolonged side birthing position [4]
Shoulder dislocation	19–55 % of anterior shoulder dislocations. The AN is stretched over the humeral head. Injury more likely when age >40 and/or >12 h before relocation [3, 5, 6]. Shoulder dislocation + rotator cuff tear + peripheral nerve (usually AN) injury = "unhappy triad" [7]
Fracture of proximal humerus	>50 % of proximal humeral fractures [5]
Direct contusion, from sports such as football and hockey	Helmet to anterolateral shoulder compresses nerve against humerus [5, 6]. The more superficial posterior branch is particularly vulnerable [7]
Shoulder surgery [3, 5]	
Fall on an outstretched hand [5]	
Paraplegia	Hypertrophy of teres major and minor decreases the size of the quadrilateral space [8]

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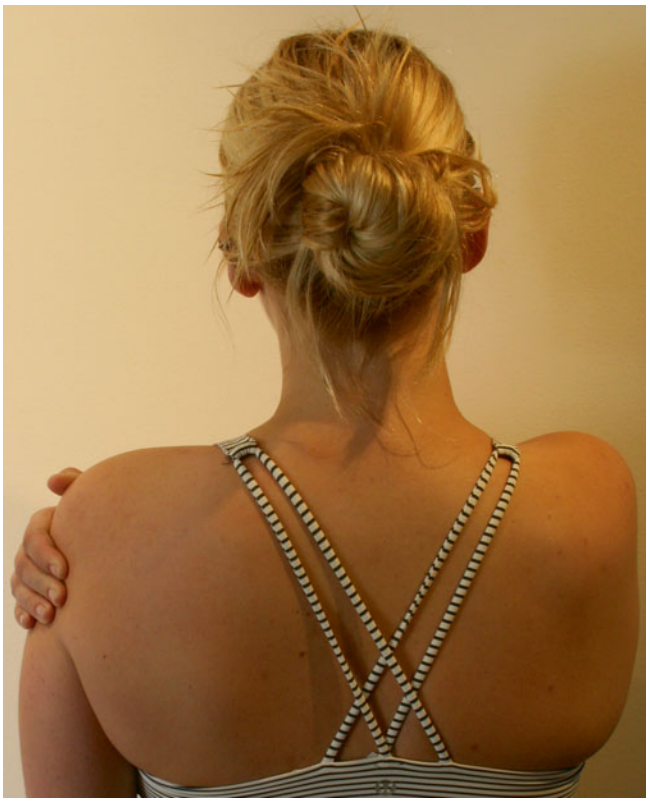

Fig. 31.1 Patient identification of pain location for axillary nerve entrapment (Image courtesy of Andrea Trescot, MD)

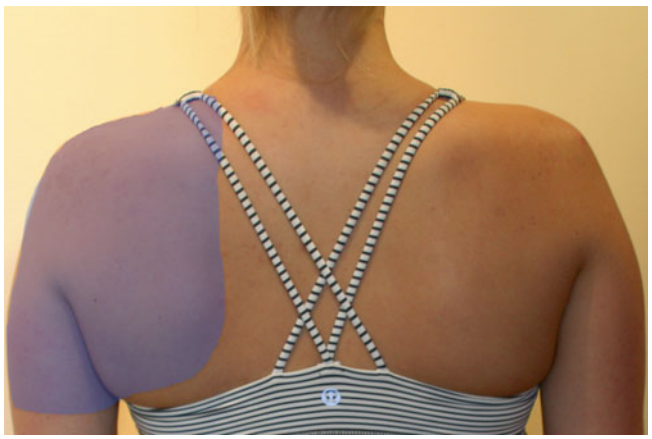

Fig. 31.2 Pain pattern associated with axillary nerve entrapment (Image courtesy of Andrea Trescot, MD)

Table 31.2 Axillary nerve anatomy

Origin	C5–C6 (with infrequent contributions from C4) nerve roots to the posterior cord of brachial plexus
General route	Under coracoid and across subscapularis, through the superior portion of the quadrilateral space (lateral axillary foramen) between teres major, teres minor, long head of the triceps, humerus. Accompanied by the posterior humeral circumflex artery (PHCA)
Sensory distribution	Anterior shoulder joint capsule
	Superior lateral brachial cutaneous nerve (SLBCN), the terminal portion of the posterior AN branch: skin over lateral deltoid ("regimental badge" area) [11]
Motor innervation	*Deltoid*: the acromial and clavicular parts are consistently innervated by the anterior AN branch; the spinal (posterior) part may be innervated by either or both branches [7]
	Teres minor: innervated by the posterior branch of the AN and contributes to external rotation (~45 %) and adduction of humerus along with infraspinatus (external rotation) and some portions of the deltoid (external rotation, adduction)
Anatomic variability [7, 8, 12, 13]	Location of division into anterior and posterior branches from the main axillary nerve trunk, most commonly (65–87 %) in the quadrilateral space, though sometimes more proximally
	Source of articular branches is very variable
	Muscular branches, except those to the posterior deltoid, are more consistent
Other relevant structures	PHCA (posterior humeral circumflex artery) is also called posterior circumflex humeral artery (PCHA) [14]
	Acromion and coracoid: relationships to bony landmarks change with the position of the arm and after surgery or trauma [7]

Anatomy (Table 31.2)

The terminal branches of the posterior cord of the brachial plexus are the axillary and the radial nerves. The AN carries fibers from the C5 and C6 nerve roots and arises from the posterior cord of the brachial plexus. It runs below the coracoid process and obliquely across the anterior surface of the subscapularis muscle. It then proceeds near the glenoid labrum under the shoulder joint, through the *quadrilateral* (or *quadrangular*) space to the back of the shoulder. The quadrilateral space is bounded superiorly by the *teres minor*, inferiorly by the *teres major*, medially by the long head of the *triceps*, and laterally by the proximal *humerus*. The quadrilateral space also contains the *posterior circumflex humeral artery* (PCHA) and *posterior circumflex humeral vein* (PCHV) (Fig. 31.3).

The AN usually divides into anterior and posterior branches in the quadrilateral space [5, 7, 12]. The anterior

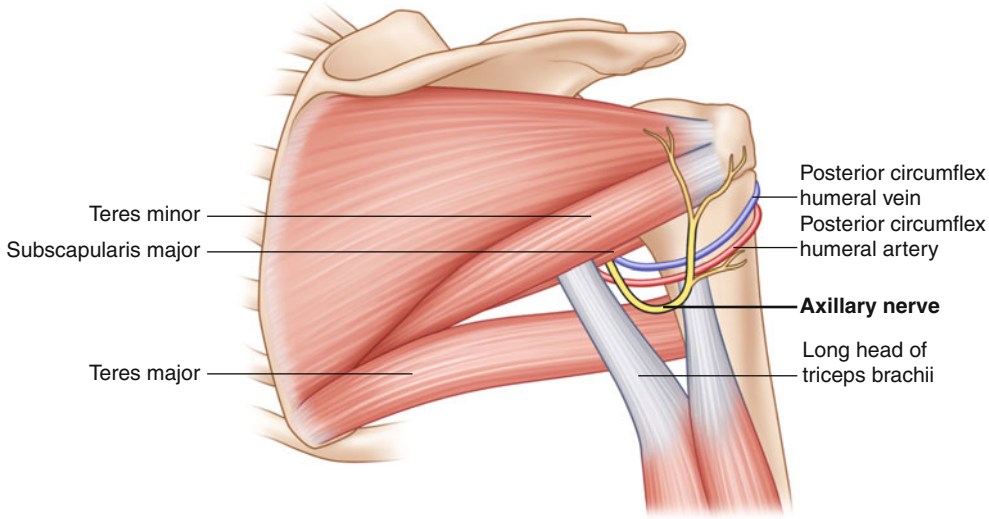

Fig. 31.3 Anatomy of the quadrilateral space (Image courtesy of Springer)

branch winds around the surgical neck of the humerus, then under the *deltoid muscle* to provide motor innervation to its acromial and clavicular portions. The posterior branch supplies the *teres minor* and *deltoid* muscle and then continues as the *superior lateral brachial cutaneous nerve* (SLBCN).

The motor function of the AN results in glenohumeral flexion and extension, horizontal abduction, as well as medial and lateral rotation of the humerus. It also provides sensation to the glenohumeral joint [12].

Entrapment

Most AN injuries occur in the setting of multiple nerve injuries following an injury to the brachial plexus, while isolated AN injury more commonly follows open surgical procedures that require splitting of the deltoid muscle. The AN can be compromised by mass lesions (such as tumors), dilated veins [5], and posteroinferior paralabral cysts [2]. Large osteophytes due to osteoarthritis of the glenohumeral joint or scapular fracture can also impinge on the AN. Post-traumatic nerve injury can occur in as many as 45 % of anterior shoulder dislocations where the nerve is stretched over the dislocated humeral head, just proximal to the quadrilateral space.

The most common site of AN entrapment is the quadrilateral space. Of note is the decreased size of this space with arm abduction, making compression of the nerve more likely [5] (Fig. 31.4). Fibrous bands, particularly between the long head of the triceps and the teres major, also decrease the area of this space with shoulder rotation [1, 11, 15]. The existence of these bands was initially controversial, but more recent surgical [16] and anatomic [11] reports confirm their existence. Entrapment in this space can occur during overhead sports in which the arm is repetitively abducted and externally rotated (e.g., swimming, baseball, tennis, racquetball),

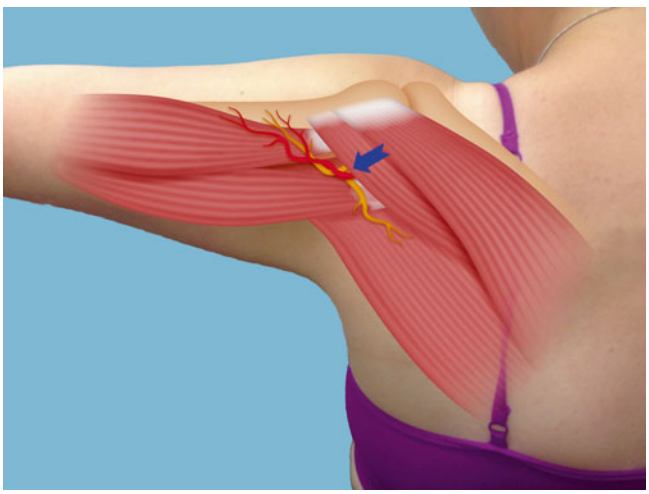

Fig. 31.4 A posterior view of the quadrilateral space with the arm in abduction, showing entrapment of the axillary nerve and artery (*arrow*) by the teres major and teres minor (Image courtesy of David Trescot and Steve McGuire)

especially if they lead to hypertrophy or spasm of the muscles that frame the quadrilateral space (teres major, teres minor, or triceps). Prolonged abduction and external rotation that can occur during sleep may also lead to entrapment. Rotator cuff tears can trigger spasm of the affected muscles, resulting in narrowing of the quadrilateral space [10], so the two conditions can coexist.

Cadaveric studies of subjects with no known shoulder pathology have shown that the long head of the triceps often develops a thick fascial layer as it approaches the *infraglenoid tubercle*, which becomes a distinct fibrous band that runs across to the teres major. The resulting sling can become tight with external rotation of the shoulder and 90° abduction and can provoke symptoms of quadrilateral space syndrome [11].

Physical Exam

Physical examination should begin with inspection for any physical deformity, obvious shoulder dislocation, atrophy of the deltoid or teres minor muscles [5, 16], or scar tissue indicative of prior surgical procedures in the area.

Point tenderness in the quadrilateral space is very commonly found in patients with AN entrapment [1, 15, 16]. The clinician should evaluate sensation over the inferolateral deltoid muscle and then palpate the quadrilateral space for tenderness (Video 31.1). Position the examining hand on top of the patient's shoulder from behind; then place the thumb below the scapular spine to identify the teres minor, which runs horizontally, angling cephalad. The clinician's thumb should then drop further inferolaterally, between the long head of the triceps and the lateral edge of the scapula, pushing inward to elicit a paresthesia [17] (Fig. 31.5).

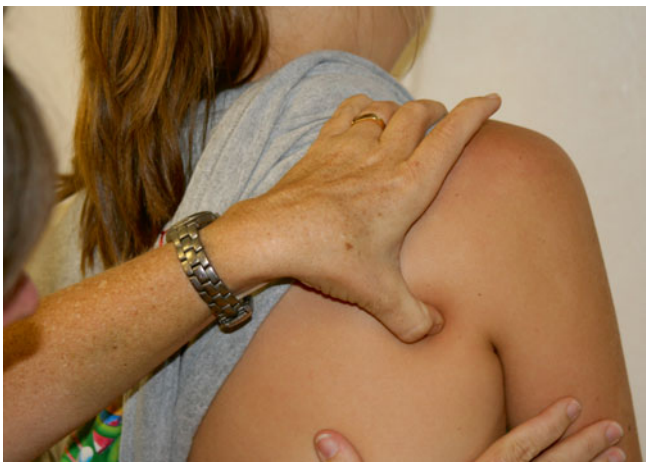

Fig. 31.5 Physical exam of the axillary nerve and space (Image courtesy of Andrea Trescot, MD)

Muscle strength testing reveals weakness of arm abduction and external rotation [3]. In addition, the *deltoid extension lag sign* reveals a discrepancy between passive and active ROM of the shoulder (Fig. 31.6). It provides objective evidence of deltoid muscle dysfunction and is a way to identify and grade the severity of AN injury. The clinician stands behind the patient and holds both wrists to elevate the extended arms posteriorly at approximately 30°. The patient is asked to actively maintain maximum shoulder extension; the difference between the affected and unaffected sides is compared. Only the posterior portion of the deltoid muscle (innervated by variable portions of the AN) extends the shoulder, allowing for accurate assessment of those structures [18]. Unfortunately, capsular adhesions invalidate this test.

Flexion, abduction, and external rotation of the upper arm for 60 s can reproduce entrapment symptoms [1, 15]. The radial pulse may be diminished, while the arm is held in this position, due to simultaneous compression of the PCHA within the quadrilateral space.

Differential Diagnosis quadrilateral (Table 31.3)

The differential diagnosis of shoulder pain includes *rotator cuff pathology* or other joint-related abnormalities [10, 17, 19], *shoulder impingement syndrome* [17], *labral tear*, *adhesive capsulitis* [10], *Parsonage-Turner syndrome* [2, 10], *thoracic outlet syndrome* (Chap. 33), and pain referred from cervical structures [17]. Parsonage-Turner syndrome almost always affects the suprascapular nerve but may involve the axillary nerve in 50 % of the cases [10]. AN entrapment may be difficult to diagnose clinically [2] (Table 31.4), and its identification may be aided by electrodiagnostic studies, magnetic resonance imaging (Figs. 31.7 and 31.8), or high-resolution ultrasonography [10, 19].

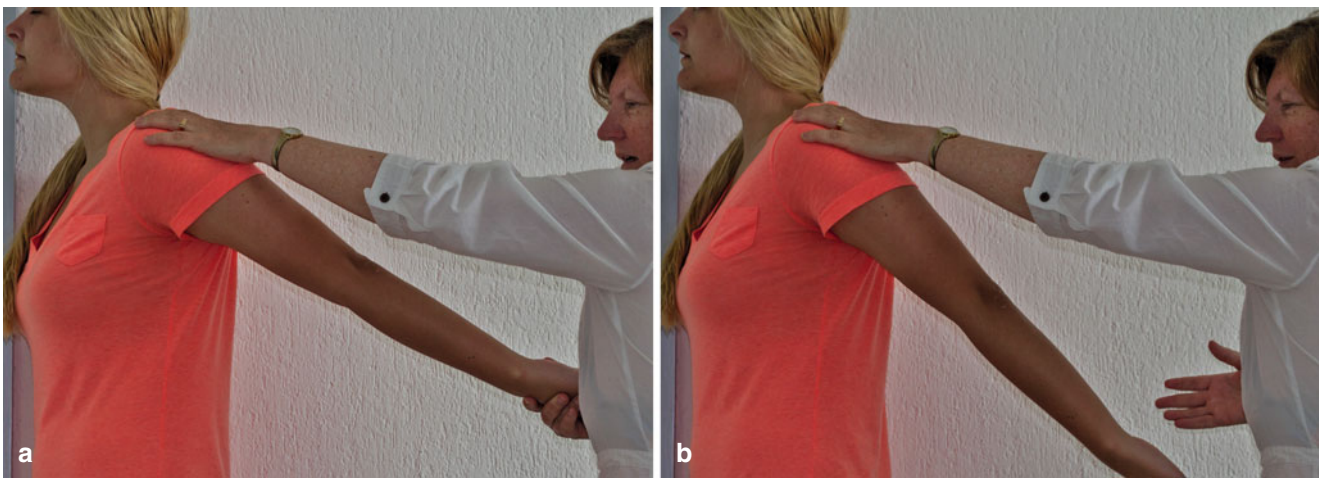

Fig. 31.6 Deltoid extension lag sign. When the patient's arm is passively extended posteriorly, the patient with axillary nerve dysfunction is unable to maintain the arm in that position. (**a**) Arm held in posterior extension; (**b**) arm released (Image courtesy of Andrea Trescot, MD)

Table 31.3 Differential diagnosis of shoulder pain

	Potential distinguishing features
Rotator cuff pathology, labral tear	Seen on MRI (AN entrapment may coexist with rotator cuff tears)
DJD shoulder	X-rays show degeneration
Adhesive capsulitis	Will invalidate the deltoid extension lag sign
Parsonage-Turner brachial plexitis	Will affect the entire brachial plexus
Thoracic outlet syndrome	See Chap. 33
Cervical radiculopathy	Decreased strength and reflexes
Impingement syndrome	Supraspinatus tendon catches under the acromion and encroaches on the subacromial bursa between 60° and 120° of abduction, can be seen on MRI
Biceps tendinitis	Tenderness over the biceps tendon in the bicipital groove

Table 31.4 Relationship between muscle pathology and nerve entrapment

Denervated muscle	Suspected nerve entrapment
Teres minor muscle	Axillary nerve at quadrilateral space
Supraspinatus and infraspinatus muscles	Suprascapular nerve at suprascapular notch (Chap. 28)
Trapezius muscle	Spinal accessory nerve (Chap. 27)
Isolated infraspinatus muscle	Suprascapular nerve at spinoglenoid (Chap. 28)
Serratus anterior muscle	Long thoracic nerve (Chap. 30)
Rhomboid and/or levator muscle	Dorsal scapular nerve at scalene muscle (Chap. 32)

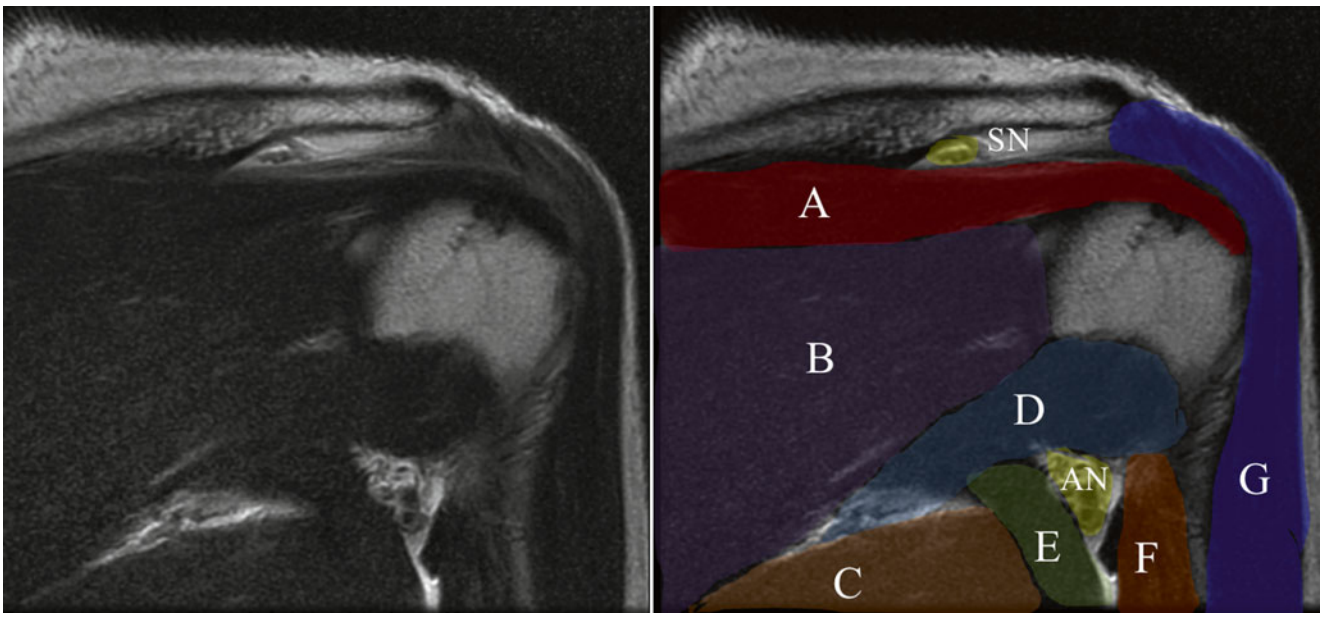

Fig. 31.7 Labeled MRI of the shoulder. *SN* suprascapular nerve, *AN* axillary nerve and posterior circumflex humeral vessels, *A* supraspinatus muscle, *B* infraspinatus muscle, *C* teres major, *D* teres minor, *E* long head of the triceps muscle, *F* short head of the triceps, *G* deltoid muscle (Image courtesy of Andrea Trescot, MD)

Diagnostic Tests (Table 31.5)

The diagnosis of AN entrapment is dependent on a high level of suspicion from the clinician. AN or suprascapular entrapment (Chap. 28) should be considered when there appear to be several shoulder muscles involved [10]. Physical exam and diagnostic injections provide significant diagnostic clues, which can be confirmed by MRI, US, and electrodiagnostic studies. Because of potential vascular compromise from entrapment, arteriography may be useful.

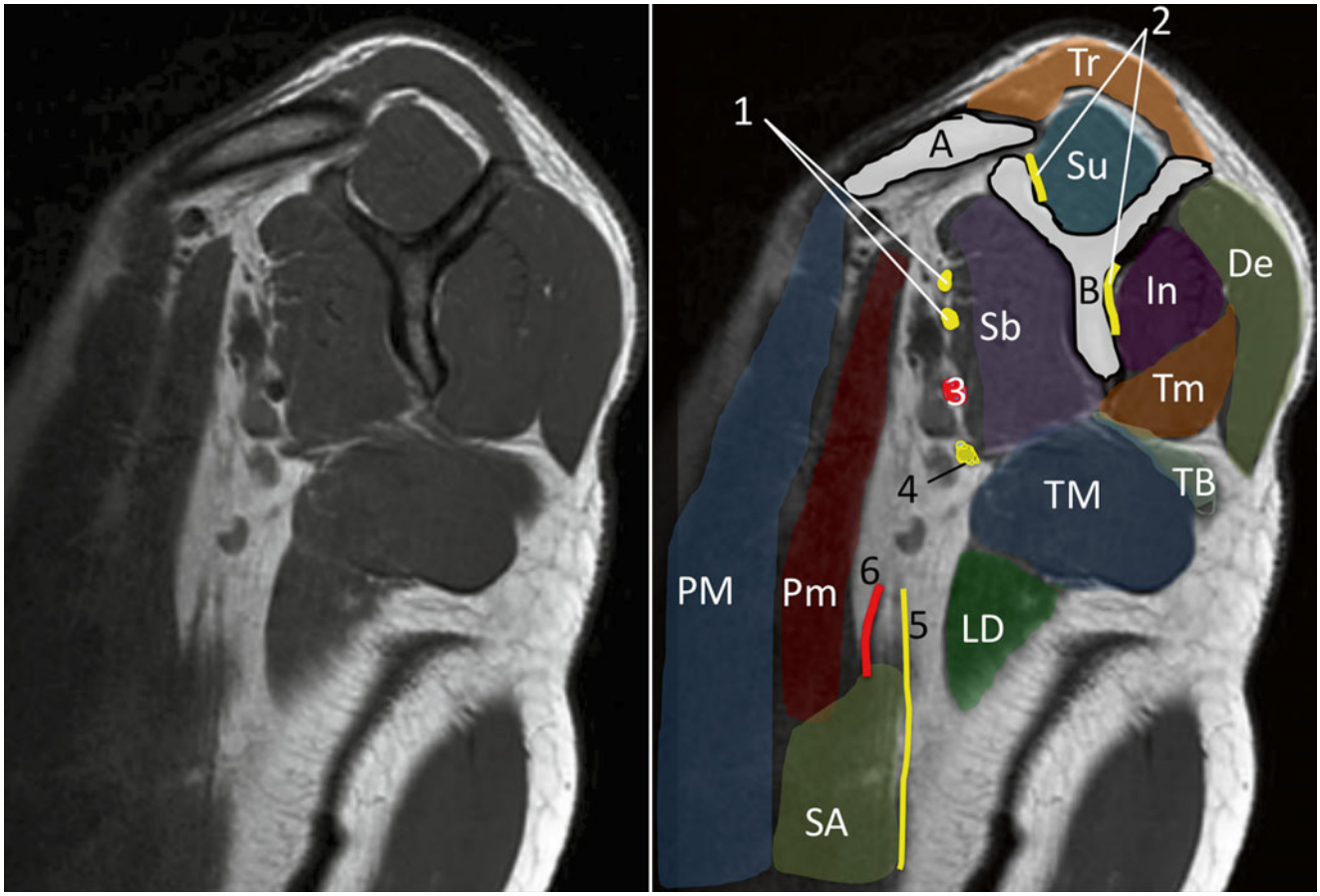

Fig. 31.8 Sagittal MRI image scapular muscles. *A* clavicle, *B* scapula, *De* deltoid muscle, *In* infraspinatus muscle, *LD* latissimus dorsi muscle, *PM* pectoralis major muscle, *Pm* pectoralis minor muscle, *Sb* subscapularis muscle, *Su* suprascapular muscle, *TM* teres major muscle, *Tm* teres minor muscle, *Tr* trapezius muscle, *TB* triceps brachii muscle, *1* brachial plexus, *2* suprascapular nerve, *3* axillary artery, *4* suprascapular nerve, *5* long thoracic nerve, *6* long thoracic artery (Image courtesy of Andrea Trescot, MD)

Table 31.5 Diagnostic tests for axillary nerve entrapment

	Potential distinguishing features
Physical exam	Tenderness in the quadrilateral space
Provocative maneuvers	Deltoid extension lag sign [18]
Diagnostic injection [8]	Relief of symptoms after local anesthetic injection into the quadrilateral space
MRI [10]	Selective atrophy of teres minor muscle [20]
	Look for mass lesions or soft tissue injuries
Ultrasound	Compression of the PHCA with arm extension
Arteriography [1, 15]	Compression of PHCA with arm hyperextension and external rotation; was initially required for diagnosis
	Less used now since PHCA occlusion has been demonstrated with manipulation of asymptomatic shoulders [21]
Electrodiagnostic studies	Not helpful early in the course [5]
	EMG of deltoid muscle will be abnormal [16]
	AN conduction study shows decreased amplitude and slowing; may be useful [16]

Identification and Treatment of Contributing Factors

AN entrapment is often seen after trauma or injury to the axilla. Examples of this include overaggressive stretching, falling on the shoulder, pressure from casts or splints, and improper use of crutches. AN injury can also be caused iatrogenically during arthroscopy, anterior shoulder operations, or rotator cuff repair. Deep infections of the axilla may also result in damage to the AN [3, 10, 19, 22].

Relative rest, stretching, and evaluation of an athlete's biomechanics may provide relief [5].

Injection Technique

Landmark-Guided Injection

There are several landmark-guided approaches to block the AN. Three of the most common are described below.

Straddle the long and lateral heads of the triceps with the index and middle finger of the non-injecting hand, and then slide the fingertips cephalad to identify the horizontal teres minor (Video 31.2). A 25-gauge 2 inch needle is advanced perpendicular to the skin to pierce the quadrilateral space (Fig. 31.9). A nerve stimulator can be used to elicit twitching of the anterior deltoid muscle and confirm appropriate needle placement near the AN. Once the needle is in position, inject 1–2 cc of local anesthetic and steroid.

The Price technique [14] targets the AN immediately after it passes through the quadrilateral space, using landmarks readily palpable in most patients. The goal is to find the nerve as it emerges from the quadrilateral space so that the needle is as far as possible from the joint capsule. The patient is seated with the arm hanging at the side. The anterior aspect of the acromion and the inferior angle of the scapula are marked, and a line is drawn between the two. The midpoint of the line is identified, and a second line is drawn horizontally from that point toward the humerus. The quadrilateral space should lie at the level of this horizontal line. The posterolateral aspect of the acromion is marked, and a third line is drawn down from this point parallel to the plane of the humerus. The point where this vertical line crosses the previously drawn horizontal line is the skin entry site. A nerve stimulator may be used to assist with the block. The needle is advanced perpendicular to the long axis of the humerus. Twitches may be seen in the posterior deltoid, due to direct stimulation of the muscle tissue. At a depth of 6–8 cm, twitches should be seen in the anterior deltoid with the current at 0.5 mA or less, indicating proximity to the AN. If twitches are not seen in the anterior deltoid, the needle should be walked inferiorly or superiorly in the vertical plane along the posterior surface of the humerus until anterior deltoid twitches are observed. Ten to 15 mL of local anesthetic is injected for blockade to control postoperative pain, in combination with a suprascapular nerve block. This method has been criticized as requiring too much needle movement [23].

According to Checcucci [24], the AN can be located by drawing a line between the posterolateral angle of the acromion and the olecranon. A second line starting from the axillary fold is drawn perpendicular to the first. The needle is placed approximately 2 cm superior to the intersection of these lines along the axis of the first. A nerve stimulator can be used to confirm proximity to the AN. A surgical block using this technique uses 15 mL local anesthetic. Kim states that the landmarks for this block are inconsistent, since they can change depending on the position of the shoulder [23].

Fluoroscopy

Fluoroscopy can facilitate the AN block. To start, the patient is positioned prone. An AP fluoroscopic view over the targeted shoulder shows the medial aspect of the humerus, which is the lateral edge of the quadrilateral space. A stimulating needle can then be placed in a coaxial view, just medial to the medial edge of the humerus at the level of the surgical neck (Fig. 31.10a). This allows access to the AN in the quadrilateral space. Alternatively, a more lateral AP approach is at the midpoint of the humerus at the level of the

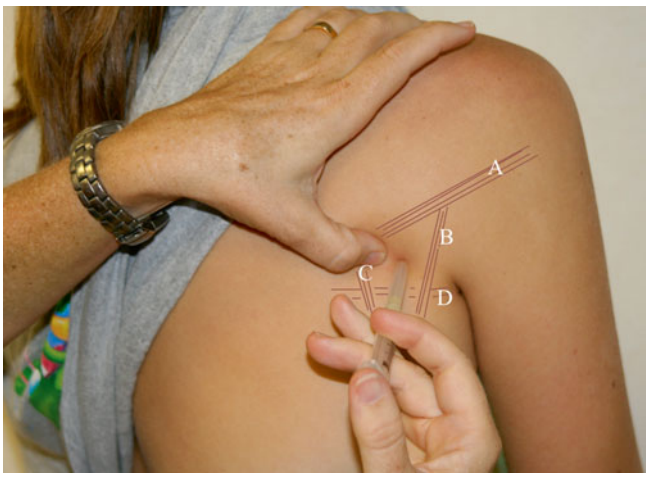

Fig. 31.9 Axillary nerve injection in the quadrilateral space: *A* teres minor, *B* short head of the biceps, *C* long head of the biceps, *D* teres major (Image courtesy of Andrea Trescot, MD)

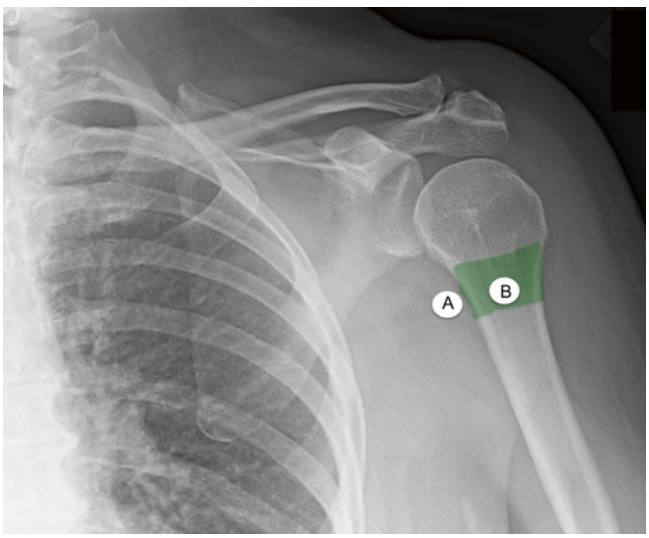

Fig. 31.10 Fluoroscopic location of axillary nerve. *Green* surgical neck of the humerus; *A* axillary nerve injection site at medial edge of the humerus, *B* axillary nerve injection site at the midpoint of the surgical neck of the humerus (Image courtesy of Andrea Trescot, MD)

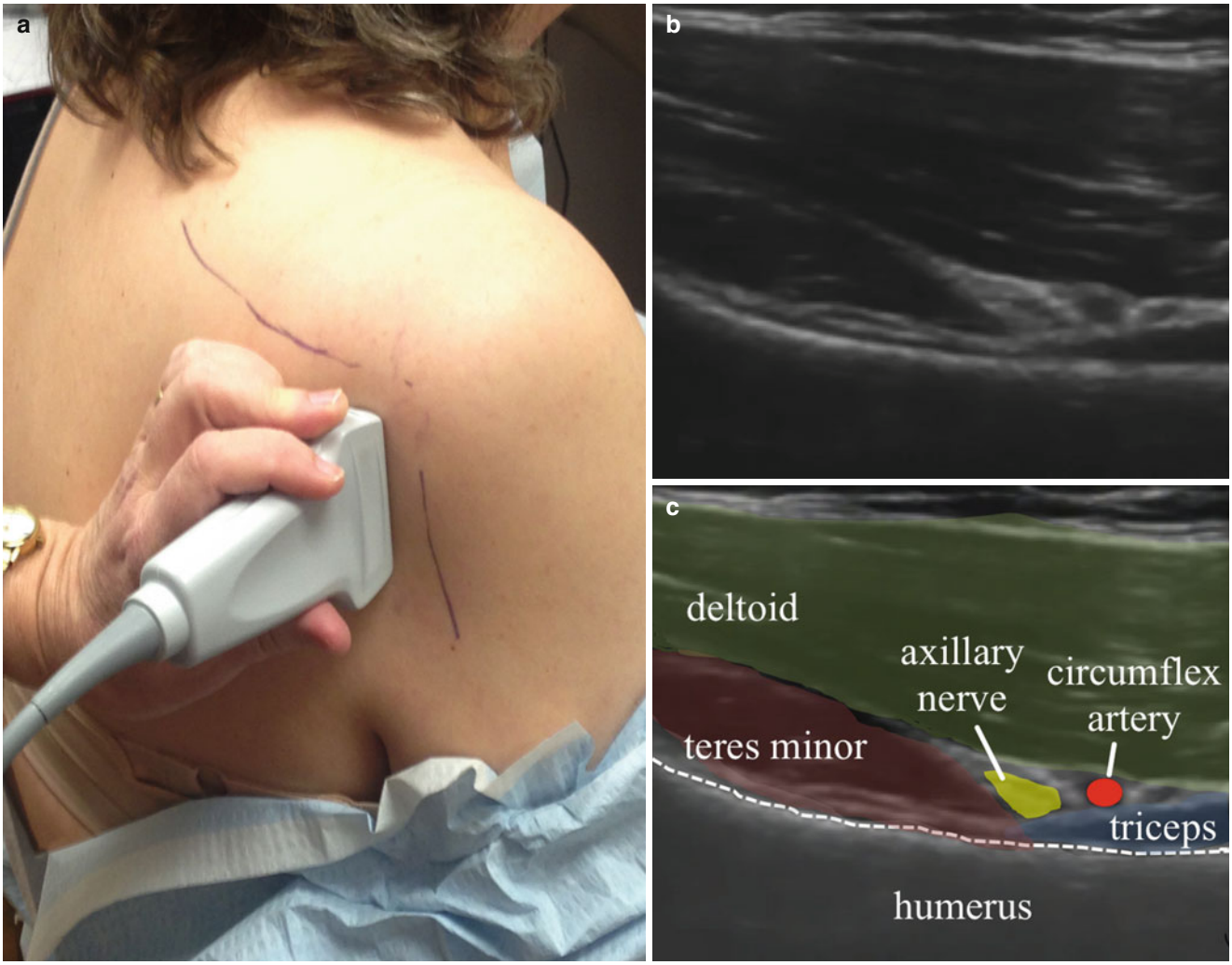

Fig. 31.11 Ultrasound image of the axillary nerve. (**a**) Location of probe and needle direction, (**b**) ultrasound image of axillary nerve, (**c**) labeled ultrasound image (Image courtesy of Andrea Trescot, MD)

surgical neck of the humerus. This midpoint is the skin entry site, and the needle is advanced under fluoroscopic guidance in a coaxial view to contact periosteum (Fig. 31.10b). A peripheral nerve stimulator (PNS) can be utilized to confirm proximity to the AN as described above. Contrast can be injected with the local anesthetic to show the space beneath the posterior deltoid and the more medial quadrilateral space [25].

Ultrasound-Guided Injection

With ultrasound, the posterior surface of the humerus can be seen using an in-plane technique parallel to the long axis of the humerus, resulting in a short axis view of the PHCA and AN. The patient is positioned sitting with the hand on their knee, slightly rotating the shoulder inward. The US probe is placed parallel to the humerus. The artery is usually easy to identify with ultrasound Doppler and is a reliable landmark to ensure local anesthetic administration near the nerve (Fig. 31.11).

Injection of the AN is performed using a similar technique. The probe is placed parallel to the humerus, and the landmarks are identified. The needle is directed cephalad to caudad in plane, toward the artery/nerve complex (Fig. 31.12). If the artery cannot be seen clearly, the humerus can be traced cephalad until it merges with the joint capsule. Placing the injection 1 cm caudal to this will result in blockade of the AN. It may be difficult to see the AN itself, but it becomes more evident once local anesthetic is injected. Blockade for management of postoperative pain is performed with 8–10 mL of local anesthetic, though smaller volumes of local anesthetic would be used for diagnostic injections. A peripheral nerve stimulator can be utilized in conjunction with ultrasonography to confirm proximity to the AN, with deltoid twitching noted [26].

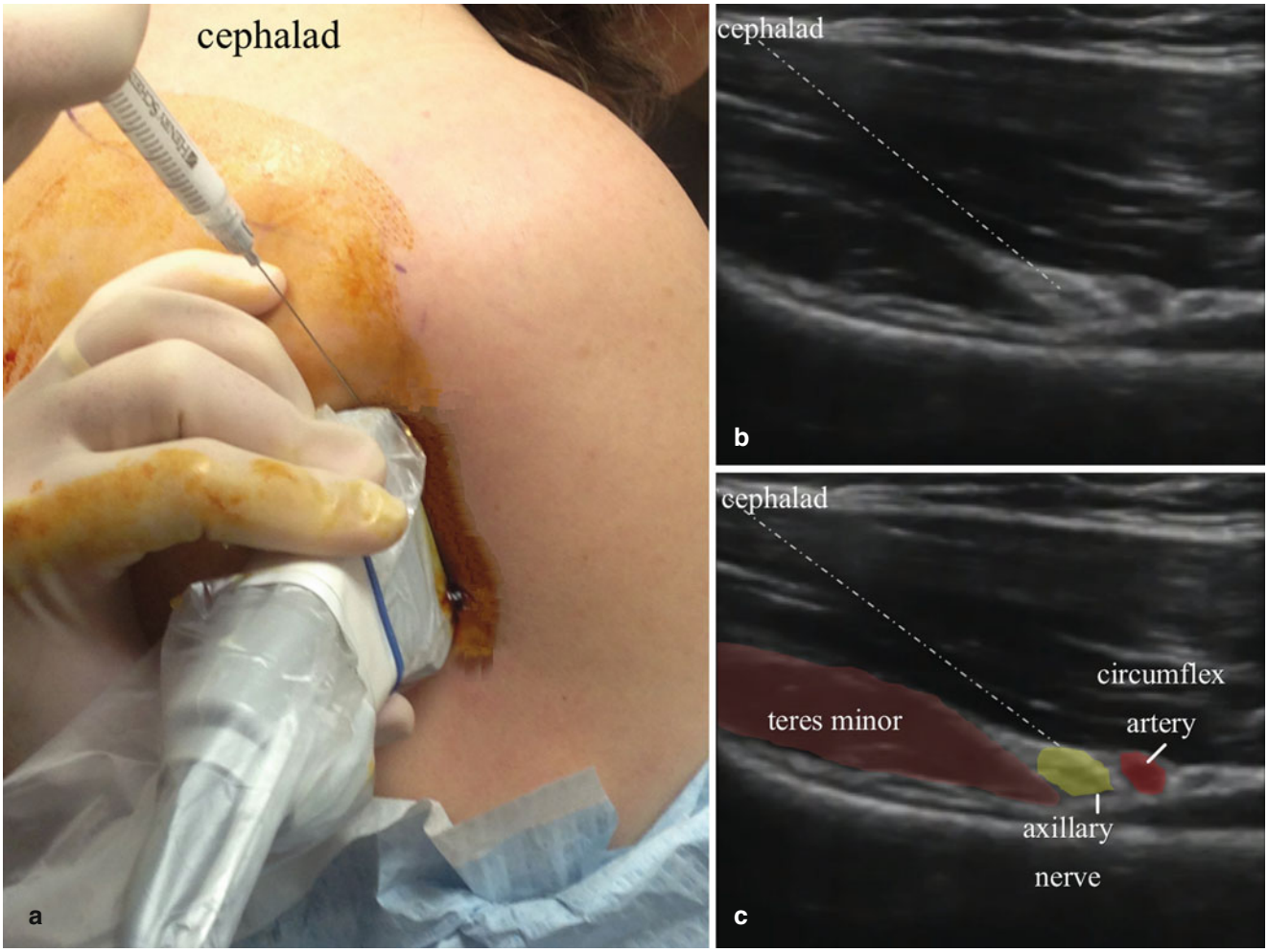

Fig. 31.12 Ultrasound-directed injection of the axillary nerve. (**a**) Location of probe and needle direction, (**b**) ultrasound image of axillary nerve with simulated needle path, (**c**) labeled ultrasound image with simulated needle path (Image courtesy of Andrea Trescot, MD)

Neurolytic Technique

Cryoneuroablation

For patients who respond well to diagnostic AN block, cryoneuroablation or pulsed radio-frequency ablation could be performed to provide a prolonged sensory block. Although not described in the literature, the techniques would be the same as used for similar nerve entrapments such as the suprascapular nerve. Patients must be aware that they may experience weakness with shoulder abduction and external rotation following these procedures.

Neurostimulation

Although it has not been reported in the literature, peripheral nerve stimulation can be performed for the AN (personal communication – Porter McRoberts, MD) (Fig. 31.13).

Surgical Technique

Surgery is rarely indicated in AN entrapment [3]. When symptoms persist for more than 6 months and there is clear local tenderness in the quadrilateral space (and perhaps a positive arteriogram), surgical intervention is targeted at releasing the AN and the PHCA by lysing the fibrous bands in the quadrilateral space. In rare cases where entrapment results from osteophyte formation inferior to the glenohumeral joint, surgical resection of the osteophyte may be indicated [15]. Early postoperative mobilization is very important [16]. Initial results using this approach showed that 8 out of 18 (44 %) were "dramatically improved"; in 8 out of 18, night symptoms continue; and 2 out of 18 (11 %) were not improved [1].

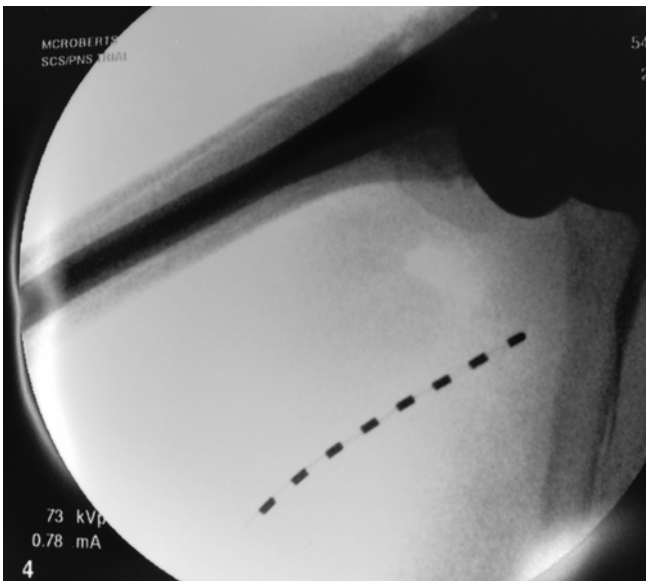

Fig. 31.13 Peripheral nerve stimulation of the axillary nerve (Image courtesy of W. Porter McRoberts, MD)

Complications

Complications after AN blocks include nerve trauma, hematoma, direct needle trauma to the neurovascular bundle, and infection.

Summary

Axillary nerve entrapment is an uncommonly reported clinical condition, most likely because it goes unrecognized. The physical exam is simple, and the diagnostic injections are relatively simple and safe; like many of the nerves discussed in this book, a high index of suspicion is necessary for appropriate diagnosis and treatment.

References

1. Cahill BR, Palmer RE. Quadrilateral space syndrome. J Hand Surg [Am]. 1983;8(1):65–9.
2. Cothran Jr RL, Helms C. Quadrilateral space syndrome: incidence of imaging findings in a population referred for MRI of the shoulder. AJR Am J Roentgenol. 2005;184(3):989–92.
3. Neal S, Fields KB. Peripheral nerve entrapment and injury in the upper extremity. Am Fam Physician. 2010;81(2):147–55.
4. Ouchi N, Suzuki S. Lateral axillary nerve palsy as a complication of labor. J Matern Fetal Neonatal Med. 2008;21(3):217–8.
5. Safran MR. Nerve injury about the shoulder in athletes, part 1: suprascapular nerve and axillary nerve. Am J Sports Med. 2004;32(3):803–19.
6. Perlmutter GS, Leffert RD, Zarins B. Direct injury to the axillary nerve in athletes playing contact sports. Am J Sports Med. 1997;25(1):65–8.
7. Apaydin N, Tubbs RS, Loukas M, Duparc F. Review of the surgical anatomy of the axillary nerve and the anatomic basis of its iatrogenic and traumatic injury. Surg Radiol Anat. 2010;32(3):193–201.
8. Tubbs R, Tyler-Kabara E, Aikens A, Martin J, Weed L, Salter E, et al. Surgical anatomy of the axillary nerve within the quadrangular space. J Neurosurg. 2005;102(5):912–4.
9. Chautems RC, Glauser T, Waeber-Fey MC, Rostan O, Barraud GE. Quadrilateral space syndrome: case report and review of the literature. Ann Vasc Surg. 2000;14(6):673–6.
10. Budzik JF, Wavreille G, Pansini V, Moraux A, Demondion X, Cotten A. Entrapment neuropathies of the shoulder. Magn Reson Imaging Clin N Am. 2012;20(2):373–91, xii.
11. McClelland D, Paxinos A. The anatomy of the quadrilateral space with reference to quadrilateral space syndrome. J Shoulder Elbow Surg. 2008;17(1):162–4.
12. Uz A, Apaydin N, Bozkurt M, Elhan A. The anatomic branch pattern of the axillary nerve. J Shoulder Elbow Surg. 2007;16(2):240–4.
13. Apaydin N, Uz A, Bozkurt M, Elhan A. The anatomic relationships of the axillary nerve and surgical landmarks for its localization from the anterior aspect of the shoulder. Clin Anat. 2007;20(3):273–7.
14. Price DJ. The shoulder block: a new alternative to interscalene brachial plexus blockade for the control of postoperative shoulder pain. Anaesth Intensive Care. 2007;35(4):575–81.
15. Lester B, Jeong GK, Weiland AJ, Wickiewicz TL. Quadrilateral space syndrome: diagnosis, pathology, and treatment. Am J Orthop (Belle Mead NJ). 1999;28(12):718–22, 25.
16. Francel TJ, Dellon AL, Campbell JN. Quadrilateral space syndrome: diagnosis and operative decompression technique. Plast Reconstr Surg. 1991;87(5):911–6.
17. Hoskins WT, Pollard HP, McDonald AJ. Quadrilateral space syndrome: a case study and review of the literature. Br J Sports Med. 2005;39(2), e9.
18. Hertel R, Lambert SM, Ballmer FT. The deltoid extension lag sign for diagnosis and grading of axillary nerve palsy. J Shoulder Elbow Surg. 1998;7(2):97–9.
19. Dong Q, Jacobson JA, Jamadar DA, Gandikota G, Brandon C, Morag Y, et al. Entrapment neuropathies in the upper and lower limbs: anatomy and MRI features. Radiol Res Pract. 2012;2012:230679.
20. Linker CS, Helms CA, Fritz RC. Quadrilateral space syndrome: findings at MR imaging. Radiology. 1993;188(3):675–6.
21. Mochizuki T, Isoda H, Masui T, Ohkawa Y, Takahashi M, Takehara Y, et al. Occlusion of the posterior humeral circumflex artery: detection with MR angiography in healthy volunteers and in a patient with quadrilateral space syndrome. AJR Am J Roentgenol. 1994;163(3):625–7.
22. Linda DD, Harish S, Stewart BG, Finlay K, Parasu N, Rebello RP. Multimodality imaging of peripheral neuropathies of the upper limb and brachial plexus. Radiographics. 2010;30(5):1373–400.
23. Kim YA, Yoon KB, Kwon TD, Kim DH, Yoon DM. Evaluation of anatomic landmarks for axillary nerve block in the quadrilateral space. Acta Anaesthesiol Scand. 2014;58(5):567–71.
24. Checcucci G, Allegra A, Bigazzi P, Gianesello L, Ceruso M, Gritti G. A new technique for regional anesthesia for arthroscopic shoulder surgery based on a suprascapular nerve block and an axillary nerve block: an evaluation of the first results. Arthroscopy. 2008;24(6):689–96.
25. Price D. Blockade of the axillary nerve. Acta Anaesthesiol Scand. 2011;55(8):1029–30.
26. Rothe C, Asghar S, Andersen HL, Christensen JK, Lange KH. Ultrasound-guided block of the axillary nerve: a volunteer study of a new method. Acta Anaesthesiol Scand. 2011;55(5):565–70.

Dorsal Scapular Nerve Entrapment

32

Andrea M. Trescot

Introduction

There are many causes of neck and shoulder pain. One of the under-recognized causes is entrapment of the *dorsal scapular nerve* (DSN), which can also be called the *dorsoscapular nerve*. Another name for this interscapular pain sometimes used is *notalgia paresthetica* [1]. Originally described in 1994 [2], the DSN is a primarily motor nerve that may share a common path with the long thoracic nerve (see Chap. 30).

Clinical Presentation (Table 32.1)

Patients with DSN entrapment present with interscapular pain, shoulder and arm pain, weakness of arm abduction, and/or winged scapula [1, 12]. They may complain of sharp, stabbing, burning, or knife-like medial scapular pain (Fig. 32.1), lateral arm and forearm pain (Fig. 32.2), neck and back dull ache, and a sense of "traction" within the shoulder [13]. There may be an itching sensation in the ipsilateral interscapular region [1]. This author has also noted a concomitant anterior chest wall pain and tenderness over the T4 sternocostal border (Fig. 32.3), often with poorly localized ipsilateral arm pain that may be much more prominent than the interscapular pain, leading to hospitalization for a possible heart attack. The scapular winging often goes undiagnosed, and the subsequent shoulder pain can result in unnecessary or unsuccessful shoulder surgeries [13]. If DSN dysfunction is chronic, there may be rhomboid or levator atrophy [7]. The onset of pain can be sudden or develop slowly over time. There may be a history of overhead work or overhead sports (see Table 32.1). Though DSN entrapment is considered quite rare, it may be greatly underrecognized, and it may be confused with or included with *long thoracic nerve entrapment*. Because the DSN passes through the middle scalene muscle, it can be injured during *interscalene brachial plexus injections* [3]. It can be traumatized at the same time as the long thoracic [5] or *suprascapular nerves* [6], and DSN entrapment can present as an "atypical" *thoracic outlet syndrome* [14]. Patients with *scapular winging* have posterior shoulder pain that may radiate down the arm or up to the neck. Haim and Urban [15] described a patient with DSN with shoulder/arm dystonia and rhythmic, involuntary jerking of the shoulder. The conditions associated with DSN entrapment are listed in Table 32.1.

Cheng et al. [14] described 36 patients (28 females and 8 males), all between 28 and 40 years old (average age of 34). The major complaint was neck, back, and shoulder discomfort; four patients had anterior chest discomfort. Pain was aggravated by weather changes, winter, and overexertion. The patients usually couldn't sleep well because of the pain, but they couldn't localize it. Seventeen patients (47 %) had occasional arm pain, and 23 (64 %) had sensory changes over the medial forearm, while 2 (0.6 %) had sensory changes over the fifth digit and radial palm.

Sultan and Younis El-Tantawi [1] described 55 consecutive patients with unilateral interscapular pain, which were compared to 30 healthy controls. Winged scapula was noted

Table 32.1 Occupation/exercise/trauma history relevant to dorsal scapular nerve entrapment

Trauma	Interscalene brachial plexus injection [3]
	Heavy lifting [4]
	Concomitant injury to the long thoracic [5] or suprascapular nerve [6]
	Stretching of scalene during "whiplash" [7]
	Anterior shoulder dislocation [8]
Entrapment	Hypertrophy of middle scalene muscle [4]
	Abnormally long transverse process of C7 [9]
	Body building [8]
	Volleyball [1, 10, 11], basketball [1]
Occupational history	Extended overhead work, such as teachers [1], painters, and electricians [12]

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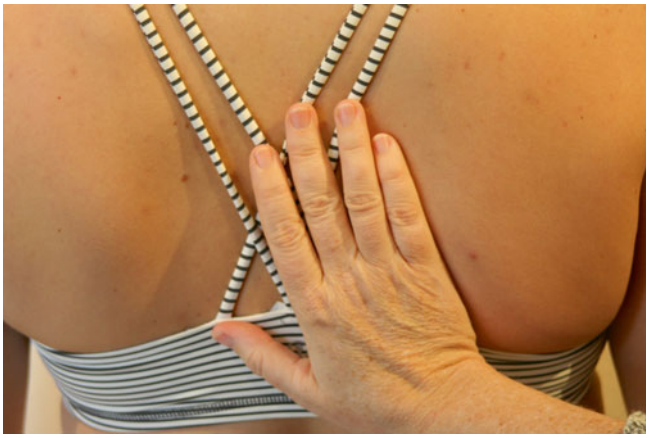

Fig. 32.1 The patient-identified scapular pain associated with dorsal scapular nerve entrapment (Image courtesy of Andrea Trescot, MD)

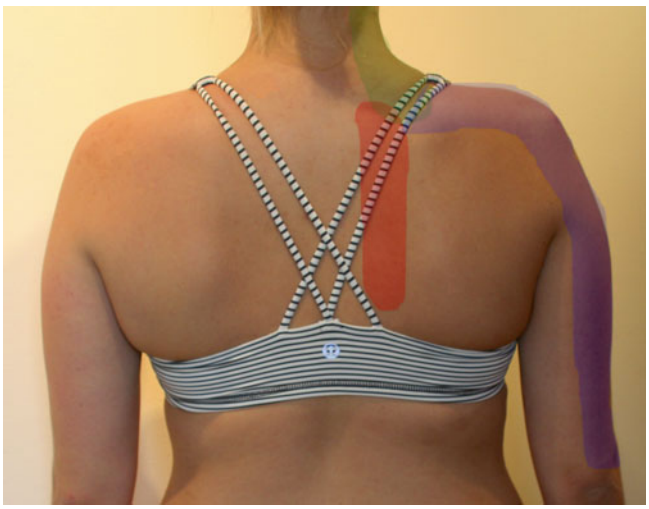

Fig. 32.2 Pain pattern associated with dorsal scapular nerve entrapment. *Red* interscapular pain, *green* posterior neck and "levator" pain, *purple* lateral arm pain (Image courtesy of Andrea Trescot, MD)

in 9 patients (16.4 %), while EMG abnormalities of the rhomboids were seen in 29 patients (52.7 %). Patients had increased pain with neck rotation and extension.

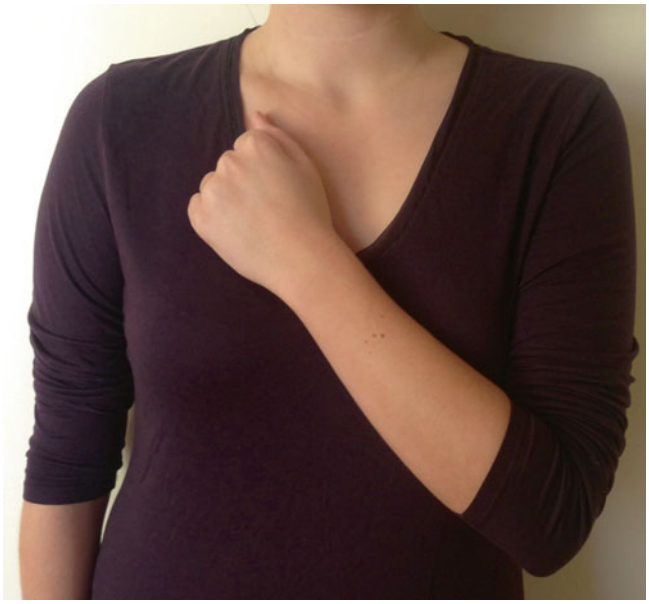

Fig. 32.3 Anterior chest wall pain and arm pain from dorsal scapular nerve entrapment (Image courtesy of Andrea Trescot, MD)

Table 32.2 Dorsal scapular nerve anatomy

Origin	The C5 ventral ramus (frequently also with contributions from C4)
General route	Often shares a trunk with the long thoracic nerve. Pierces the middle scalene or its posterior surface, passes posterior to the levator to the inferior part of the levator and the serratus posterior superior, then parallel to the medial margin of the scapula on the inner surface of the rhomboid muscles. Joins the dorsal scapular artery (from the transverse cervical artery)
Sensory distribution	Skin sensation along the medial border of the scapula
Motor innervation	The rhomboid (major and minor) and occasionally the levator scapulae muscles
Anatomic variability	Variable contributions from C4 to T1, may share a trunk with the long thoracic nerve and may innervate the levator scapulae
Other relevant structures	The dorsal scapular artery, serratus posterior superior muscle

Anatomy (Table 32.2)

The DSN arises from C5 with occasional contributions from C4 to T1, and it is usually considered part of the *brachial plexus*, sharing a common trunk with the *long thoracic nerve* [16]. The DSN arises within the *posterior cervical triangle* deep to the prevertebral fascia (Fig. 32.4) and pierces the *middle scalene muscle* to innervate the *rhomboid muscles* (Figs. 32.4 and 32.5). It then travels posteriorly over the ribs between the *posterior scalene* and the *serratus posterior superior muscles* and provides some of the motor innervation of the *levator scapulae muscle* (see Table 32.2). The nerve passes under the levator and then becomes more superficial between the rhomboid major and minor, as it travels caudally along the medial border of the scapula. The *dorsal scapular artery* goes between the middle and inferior trunks of the brachial plexus before the joining the DSN as it leaves the middle scalene; the artery accompanies the DSN to provide blood flow to the inferior trapezius muscle [3].

Frank et al. [11] dissected 35 neck specimens and found the DSN innervating the levator in 11 of the 35 specimens. Cheng et al. [14] dissected ten formalin and two fresh cadav-

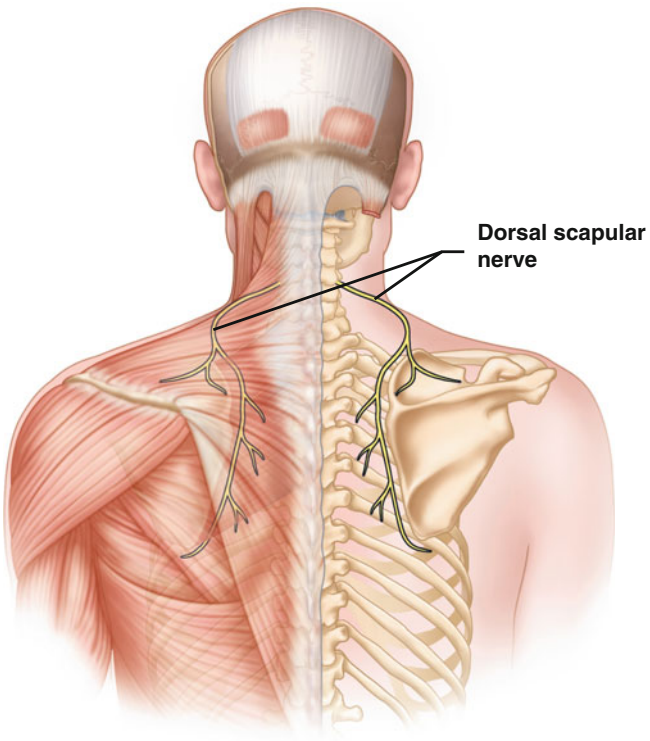

Fig. 32.4 Anatomy of the dorsal scapular nerve (Image courtesy of Springer)

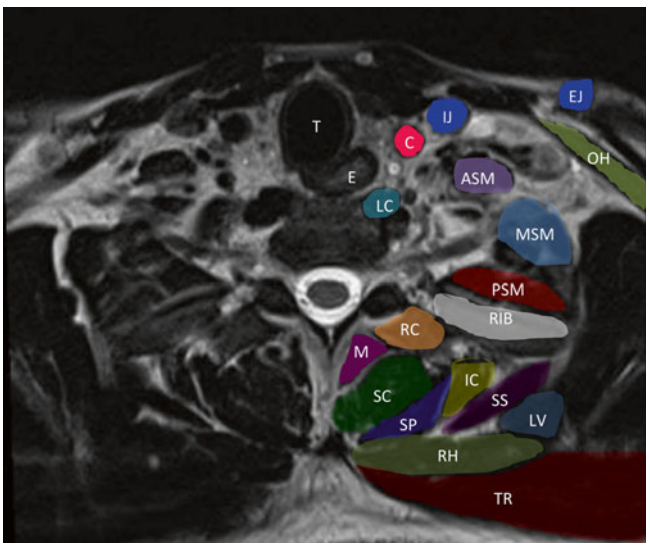

Fig. 32.5 Axial MRI of the lower neck. *ASM* anterior scalene muscle, *C* carotid artery, *E* esophagus, *EJ* external jugular vein, *IC* iliocostalis muscle, *LC* longus coli muscle, *LV* levator scapula muscle, *M* multifidus muscle, *MSM* medial scalene muscle, *OH* omohyoid muscle, *RC* rotator cervicis muscle, *PSM* posterior scalene muscle, *RH* rhomboid muscle, *RIB* rib, *SC* semispinalis cervicis muscle, *SP* splenius capitis muscle, *SS* serratus posterior muscle, *T* trachea, *TR* trapezius muscle (Image courtesy of Andrea Trescot, MD)

ers, looking at the DSN and long thoracic nerves. In 14 of the sides dissected, the DSN and long thoracic nerves shared a common trunk. In two sides, the DSN received contributions from C4. Tubbs et al. [16] dissected ten cadavers (20 sides); the DSN was identified on the anterior surface of the rhomboid and then traced cephalad to the scalenes. They found that the DSN pierced the middle scalene a mean distance of 3 cm (1.8–4.5 cm) fromv the origin of the exiting nerve root and 1.5 cm (1–3.2 cm) medial to the vertebral border of the scapula between the *serratus posterior superior muscle* and the *levator scapulae muscle*; it was also found a mean distance of 2.5 cm (1.2–3.8 cm) medial to the *spinal accessory nerve* (Chap. 26). In all the specimens, the DSN was intertwined with the DSN along the anterior border of the rhomboid muscles. The *serratus posterior superior muscle* was not innervated by the DSN in any specimen.

Entrapment

The DSN has two apparent sites of entrapment – at the scalenes and at the rhomboids. Hypertrophy of the middle scalene muscle is the most commonly described entrapment [1, 10, 14, 16, 18]. Pan et al. [19] noted "tendinous fibrous tissues" at the inferior edge of the middle scalene in 32 dissections, which they proposed was the main factor responsible for DSN entrapment.

Identification and Treatment of Contributing Factors

Cervical spine stabilization and strengthening of the trapezius muscle can help to compensate for the scapular dysfunction [13]. Postural changes as well as anatomic variations can trigger entrapment of the DSN. Unilateral shoulder postural changes, such as seen with leg length discrepancies or scoliosis, can contribute to DSN entrapment at the level of the scalene or the rhomboid muscles. Alternatively, one case report identified an accessory tendon of the levator scapulae that attached unilaterally onto the rhomboid and serratus posterior superior muscles, innervated by a branch of the DSN [20]. The authors felt that this asymmetric muscle function could lead to unilateral traction on the vertebrae, resulting in scoliosis and myofascial pain. Boehnke [17] described the use of myofascial release of the scalene muscle to treat DSN entrapment.

Muscles that stabilize the scapula include the deltoid, trapezius, rhomboid (major and minor), supraspinatus, infraspinatus, subscapularis, teres minor, latissimus dorsi (a small slip), triceps (the long head), biceps (the long head), coracobrachialis, pectoralis minor, and omohyoid (Figs. 32.5, 32.6, and 32.7) [13]. Shoulder pain can be com-

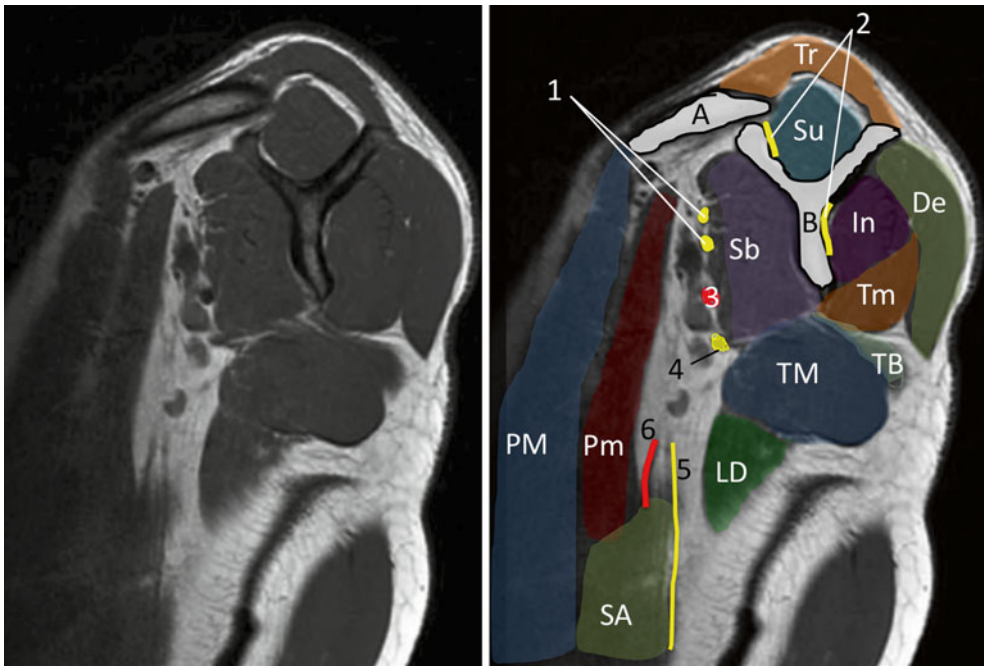

Fig. 32.6 Sagittal MRI image of the scapular structures. *A* clavicle, *B* scapula, *De* deltoid muscle, *In* infraspinatus muscle, *LD* latissimus dorsi muscle, *PM* pectoralis major muscle, *Pm* pectoralis minor muscle, *Sb* subscapularis muscle, *Su* suprascapular muscle, *TM* teres major muscle, *Tm* teres minor muscle, *Tr* trapezius muscle, *TB* triceps brachii muscle, *1* brachial plexus, *2* suprascapular nerve, *3* axillary artery, *4* suprascapular nerve, *5* long thoracic nerve, *6* long thoracic artery (Image courtesy of Andrea Trescot, MD)

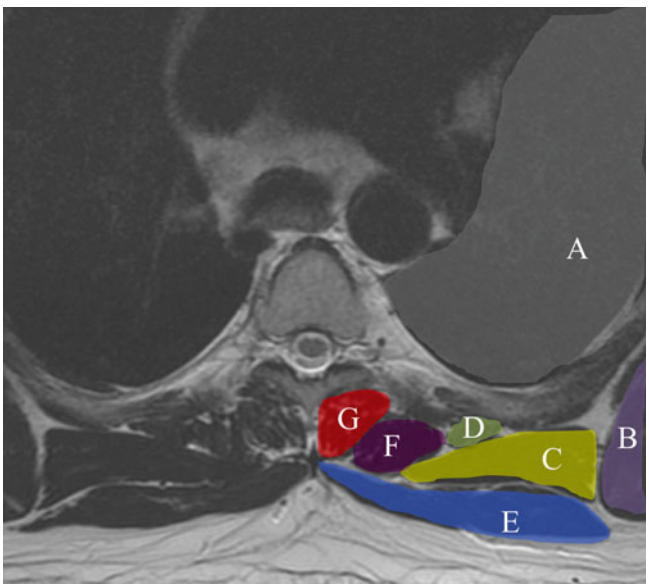

Fig. 32.7 Axial MRI image of the upper thorax. *A* lung, *B* scapula, *C* rhomboid major muscle, *D* serratus posterior superior muscle, *E* trapezius muscle, *F* erector spinae muscle, *G* multifidus muscle (Image courtesy of Andrea Trescot, MD)

plicated; for instance, an unstable scapula caused by weak rhomboids (from DSN entrapment) or anterior serratus (with a long thoracic palsy) causes the scapula to move excessively, resulting in stretching or entrapment of the *suprascapular nerve* (see Chap. 28). Thus, correcting DSN or long thoracic nerve pathology (Chap. 30) can treat suprascapular pathology [17].

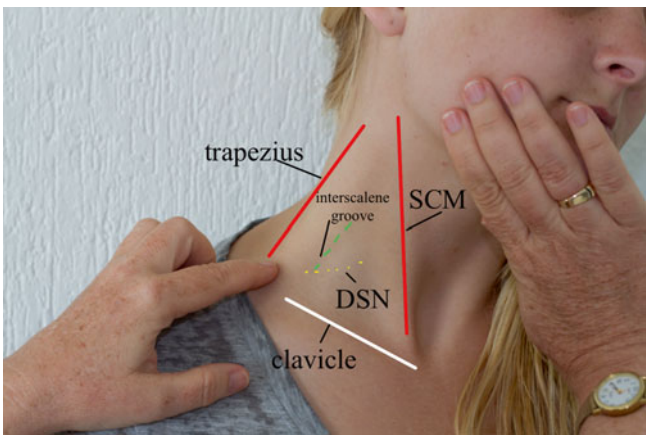

Fig. 32.8 Surface landmarks of the posterior cervical triangle (Image courtesy of Andrea Trescot, MD)

Physical Exam

The physical exam for the DSN dysfunction can be very nonspecific. The DSN is found between the middle and anterior scalene in the interscalene groove (Fig. 32.8). There may be tenderness to palpation medial to the scapula, especially with the shoulder rotated forward (Fig. 32.9). Winged scapula is probably the most specific finding, but winging is subtle and usually attributed to pathology of the long thoracic nerve (see Chap. 30) or the spinal accessory nerve (see Chap. 26). The winged scapula associated with the DSN shows a prominent lower medial border and inferior angle of the scapula (Fig. 32.10). In this image, note the prominence of the laterally displaced inferior

border of the scapula without the sloping of the shoulder that would be seen with *spinal accessory nerve* pathology (see Chap. 26) [13]. The comparison of the winging from the long thoracic, spinal accessory and DSN is seen in Table 32.3. The pain associated with winging is usually due to spasm of the antagonistic muscles. Patients will have decreased ROM of the shoulder, often noting clicking or popping with abduction as the scapula changes its glide path across the posterior chest wall. Because the nerve is entrapped at the lower two-thirds of the middle scalenes, rotation or extension of the neck may replicate or exacerbate the symptoms [4]. Pain will increase with provocative movements (see Table 32.3) and decrease with manual compression of the scapula onto the chest wall.

The 36 patients diagnosed with DSN entrapment described by Chen et al. [14] had normal neck and shoulder exams, but they had a tender point 3 cm lateral to the spinous process of the T3–T4 vertebrae at the medial inferior scapular tip. The lower two-thirds of the scalene muscle was also significantly tender, and palpation at that point may replicate the arm pain. Patients will often have a head-forward posture, perhaps with lateral flexion and rotation of the neck (which decreases the tension on the scalene muscles). Radial artery pulsations disappeared with suprascapular pressure in 31 of the 36 patients, and EMG was normal.

Sultan and Younis El-Tantawi [1] evaluated 55 patients with interscapular pain; there were myofascial rhomboid spasms in 12 patients, hypertrophy of the splenius medius in 4 patients, and an elongated transverse process of C7 in 2 patients. One patient (who described an itchy interscapular sensation) had an area of decreased pinprick sensation just medial to the scapular border. The scapula was more medially located on the symptomatic side in nine patients, associated with rhomboid wasting.

Boehnke [17] also reported on ten patients with unilateral scapular pain. He described placing his thumbs on the inferior angle of the scapulae bilaterally and having the patients abduct the arms as high as possible; on the symptomatic side, the scapula will travel further laterally.

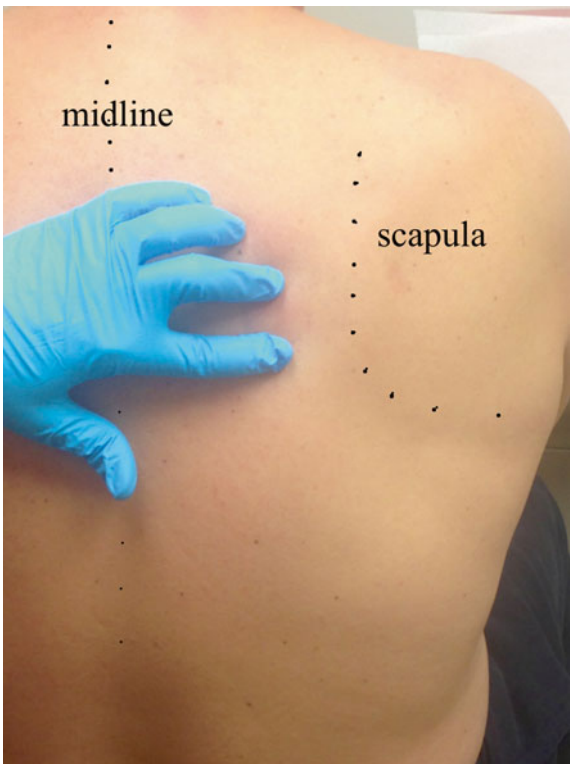

Fig. 32.9 Medial scapular examination of the dorsal scapular nerve, with the shoulder rotated anteriorly (Image courtesy of Andrea Trescot, MD)

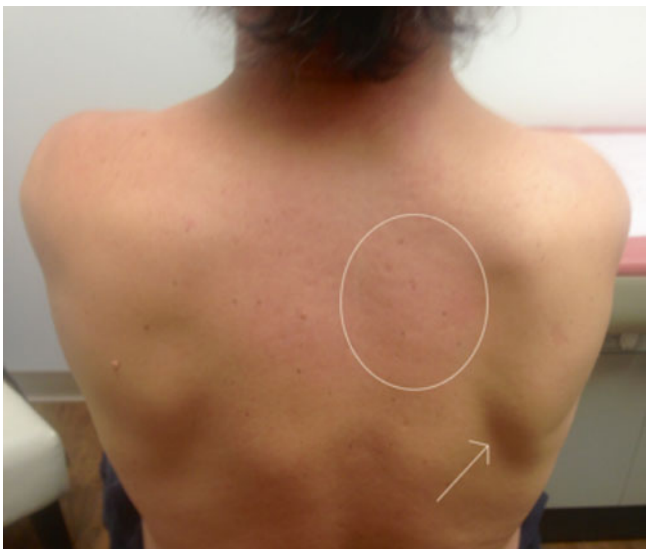

Fig. 32.10 This photograph shows an example of subtle winging with prominence of the medial and inferior scapula (*white arrow*) without sloping of the shoulder. The *white circle* represents the site of tenderness to palpation (Image courtesy of Andrea Trescot, MD)

Table 32.3 Comparison of winging from long thoracic, spinal accessory, and dorsal scapular nerve pathology

Nerve	Muscles involved	Type of winging	Provocative maneuvers
Long thoracic	Serratus anterior	Medial winging	Forward elevation and pushing with outstretched arms (wall push-up)
Spinal accessory	Trapezius	Lateral winging with drooping shoulder	Resisted arm abduction or external rotation
Dorsal scapular (a rare cause of subtle winging)	Rhomboid and levator scapulae	Scapula shifted laterally and dorsally	Slowly lowering arm from forward elevation

Differential Diagnosis (Table 32.4)

Because the DSN is often overlooked as a diagnosis, most patients with DSN entrapment have been misdiagnosed prior to the accurate diagnosis. The major symptoms seen in the 36 patients reported by Chen et al. [14] included discomfort of the neck, back, and shoulder, with four patients also complaining of anterior chest pain. Seventeen complained of occasional hand numbness (three on the radial side, two on the ulnar side); 23 patients also had sensory changes over the medial forearm. Adson's sign (loss of radial pulse with ipsilateral head rotation) was positive in five patients; Wright's test (loss of radial pulse decreased with shoulder abduction and external rotation) was positive in 30 patients. Patients were unable to point to the specific site of pain. Symptoms were aggravated by weather changes (especially rainy days or winter storms) and increased activity, and patients complained of difficulty sleeping due to pain.

Diagnostic Tests (Table 32.5)

Chen et al. [14] described six diagnostic criteria for DSN pathology:

1. Commonly seen in women 30–40 years old
2. No shoulder arthritis or cervical spondylosis
3. No X-ray changes except possible elongated transverse process of the lower cervical vertebrae
4. Obvious tenderness at the middle of the SCM posterior border and the paravertebral T3/T4, which is temporarily relieved with an injection of local anesthetic
5. Subtle brachial plexus symptoms, such as sensory changes on the ulnar forearm
6. Normal electrodiagnostics, except possible evidence of lower trunk compression

In addition, the thoracic MRI should be closely reviewed for rhomboid edema, atrophy, or signal changes (Figs. 32.10 and 32.11). The differential diagnosis of muscle atrophy sites is described in Table 32.6. According to Akgun [12], increased rhomboid signal on MRI corresponds closely with spontaneous activity on EMG, with a relative sensitivity of 84 % and specificity of 100 % for detecting denervation.

Sultan and Younis El-Tantawi [1] described 55 cases of unilateral scapular pain compared to 30 healthy controls; 52.7 % had electrodiagnostic studies consistent with DSN

Table 32.4 Differential diagnosis of dorsal scapular nerve

	Potential distinguishing features
Shoulder impingement	Shoulder X-ray with arm abducted
Adhesive capsulitis	Shoulder arthrogram
AC joint pathology	X-ray showing AC degeneration, AC joint injection
Rotator cuff disease	MRI showing rotator pathology
Glenohumeral instability	X-rays showing glenohumeral instability
Cervical radiculopathy	Dermatomal pain pattern, weakness/sensory changes, reflex changes
Brachial plexopathy	EMG
Rhomboid myofascial pain	Taut bands or myofascial nodules
Thoracic facet pathology	Tenderness more medially over the paravertebral region, spondylosis
Thoracic disk	Paresthesias in a dermatomal pattern, increased with coughing

Table 32.5 Diagnostic tests of dorsal scapular nerve

	Potential distinguishing features
Physical exam	Tenderness over the middle scalene and medial scapula; winged scapula (lateral displacement) and no trapezius weakness
Provocative maneuvers	Slowly lowering arm from forward arm elevation
Diagnostic injection	At scalene or interscapular site
X-rays	Elongated C7 transverse process
MRI	Atrophy and possible abnormal signal of the rhomboid muscle
Ultrasound	Flattening of the nerve at the middle scalene
Arteriography	Not useful
Electrodiagnostic studies	Needle stimulation of the rhomboid at Erb's point shows prolonged distal latency. No abnormality of the supraspinatus, infraspinatus, or deltoid. Needle EMG rhomboid may show long duration, polyphasic MUP with spontaneous activity

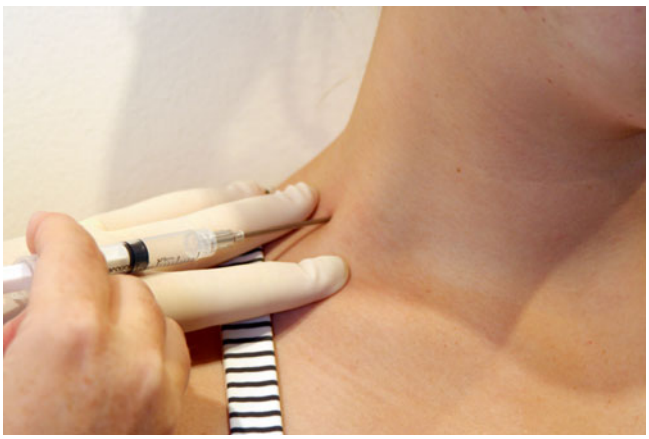

Fig. 32.11 Anterior injection of the dorsal scapular nerve at the level of the middle scalene (Image courtesy of Andrea Trescot, MD)

Table 32.6 Relationship between muscle pathology and nerve entrapment

Denervated muscle	Suspected nerve entrapment
Rhomboid and/or levator muscle	Dorsal scapular nerve at the scalene
Trapezius muscle	Spinal accessory nerve (Chap. 27)
Supraspinatus and infraspinatus muscle	Suprascapular nerve at the suprascapular notch (Chap. 28)
Isolated infraspinatus muscle	Suprascapular nerve at the spinoglenoid (Chap. 28)
Serratus anterior muscle	Long thoracic nerve (Chap. 30)
Teres minor muscle	Axillary nerve at the quadrilateral space (Chap. 31)

pathology, and 16.4 % had scapular winging. They described the electrodiagnostic criteria to be:

1. Delayed potentials exceeding the cutoff value of the controls or side-to-side latency difference exceeding the cutoff point obtained from the controls
2. EMG abnormalities recorded from the rhomboid major and levator scapula

Identification and Treatment of Contributing Factors

Overhead work, especially lifting overhead, has been implicated as a cause of DSN entrapment [12]. Imbalance between external and internal rotator muscles, unstable acromioclavicular joint, a slouched position, and even sacroiliac dysfunction can cause DSN entrapment [17].

Treatment of the scalene entrapment (with myofascial release, trigger point injections, or postural reeducation) would be expected to improve the DSN entrapment.

Injection Technique

Landmark-Guided Injection

There are two possible approaches to the DSN: an anterior approach within the scalene and a posterior approach close to the levator before descending below the rhomboids [15]. Because of the lack of "firm landmarks," the use of a peripheral nerve stimulator (PNS) can aid in location of the nerve [15]. A third approach by this author identifies the nerve at the medial scapula.

For the anterior approach, after a sterile prep, a local anesthetic wheal is placed at the apex of the posterior triangle of the neck, between the SCM and the trapezius, and the PNS is used to stimulate a rhomboid contraction (Fig. 32.11) [15].

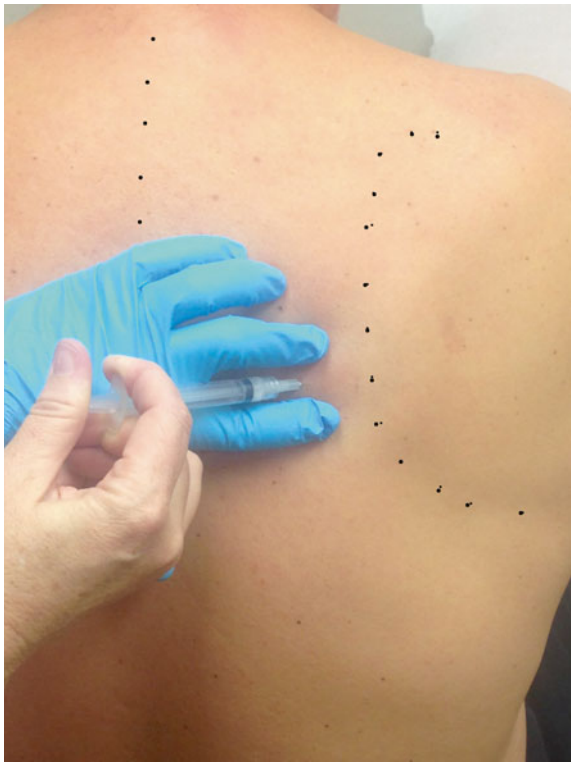

Fig. 32.12 Subscapular injection of the dorsal scapular nerve, with the shoulder rotated anteriorly ("Trescot approach") (Image courtesy of Andrea Trescot, MD)

For the posterior approach, the rhomboid is palpated, and the groove identified between the splenius captious and the levator scapulae. After a sterile prep, a local anesthetic wheal is placed, and an insulated needle advanced with a PNS through the trapezius in a "slightly outward direction" toward the levator [15]; contraction of the rhomboid muscles confirms location.

For the "Trescot approach," the shoulder is rotated anteriorly to move the scapula laterally. A reproducible area of tenderness just below the inferior edge of the scapula can be palpated. Straddling this region with the non-injecting hand, with the fingers parallel to the rib, the needle is introduced under the rib at an acute angle to avoid the lung (Fig. 32.12), and the medication is injected. Occasionally, a nerve stimulator will be used if the initial injection gave only temporary relief.

Liu et al. [21] described 128 patients with DSN entrapment treated with injections of the "pain points" at the neck and the T3–T4 parascapular region. Patients were contacted 6 months to a year after the injections (having received one to six injections, depending on response). Most patients noted relief of the back, neck, and shoulder pain almost instantly; "excellent" relief at 6–12 months was noted in 87 cases, "good" relief in 28 cases, "poor" relief in 3 cases, and an "ineffective" response in 2 cases, with 7 cases lost to follow-up. They noted that

90.9 % of the patients requiring four to six injections had symptoms for more than 6 months, suggesting that earlier treatment is associated with improved outcome.

Fluoroscopy-Guided Injections

There are no reported fluoroscopy-guided injections of the DSN.

Ultrasound-Guided Injections

Because of the substantial variability in the anatomy of the nerves and muscles in this highly vascular area of the neck (Table 32.2), ultrasound (US) guidance confirmed with nerve stimulation is particularly useful for DSN injections. The DSN can be approached with US-guided injections at both the neck and the posterior scapular regions. In the neck, the DSN is seen under US in the body of the middle scalene muscle (Fig. 32.13); it is usually a flat structure within the body of the scalene muscle but becomes round after it leaves the muscle [3]. Hanson and Auyong [22] described US identification of the DSN and LTN during interscalene blocks in 50 patients scheduled for shoulder surgery. They defined the nerves as hyperechoic structures with hypoechoic centers within, or superficial to, the middle scalene muscle (Fig. 32.13); 90 % of patients had one or both nerves visible. A stimulating needle was inserted at a site more posterior than has been traditionally taught and was used to confirm nerve identity. Rhomboid and levator scapulae contractions confirmed DSN stimulation (77 %), while contraction of the serratus anterior muscle indicated stimulation of the LTN (23 %).

Restrepo-Garces et al. [23] described an US-guided injection of the DSN at the middle scalene, using an in-plane approach (Fig. 32.14) and a peripheral nerve stimulator, looking for rhomboid stimulation. They injected 2 cc of 0.5 % bupivacaine and noted a 70 % decrease in pain for 24 h.

Using US, the DSN can also be injected more distally, tracing the nerve under the levator and then inferiorly to the rhomboids. With the probe held horizontally, the nerve is seen under the rhomboid (Fig. 32.15) and injected from a medial in-plane approach.

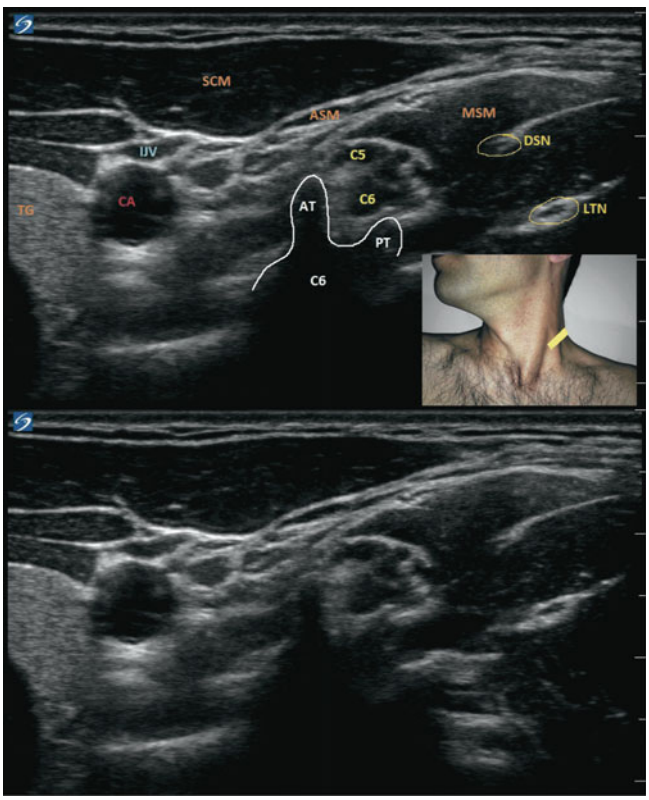

Fig. 32.13 Ultrasound identification of the dorsal scapular nerve at the middle scalene muscle. *SCM* sternocleidomastoid muscle, *MSM* middle scalene muscle, *ASM* anterior scalene muscle, *TG* thyroid gland, *CA* carotid artery, *AT* anterior tubercle of C6 vertebra, *PT* posterior tubercle of C6 vertebra, *DSN* dorsal scapular nerve, *LTN* long thoracic nerve (Image courtesy of Agnes Stogicza, MD)

Neurolytic Technique

Cryoneuroablation

There are no cryoneuroablation techniques described in the literature.

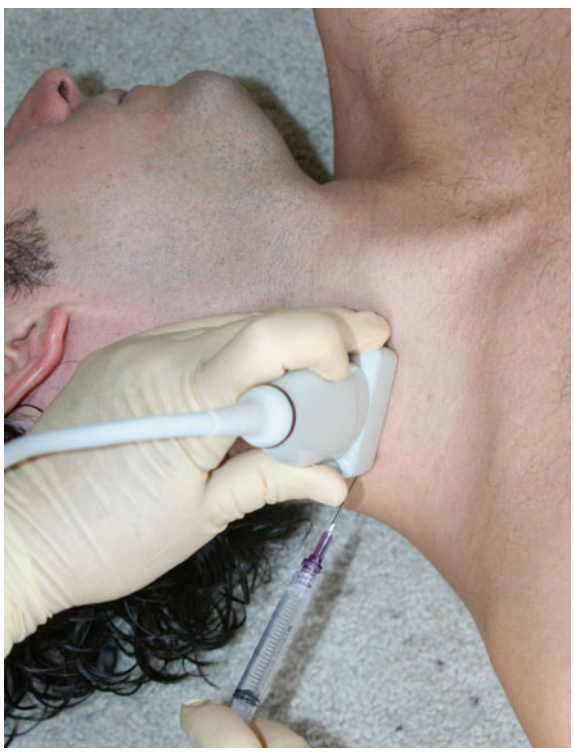

Fig. 32.14 Ultrasound injection of the dorsal medial nerve at the middle scalene (Image courtesy of Andrea Trescot, MD)

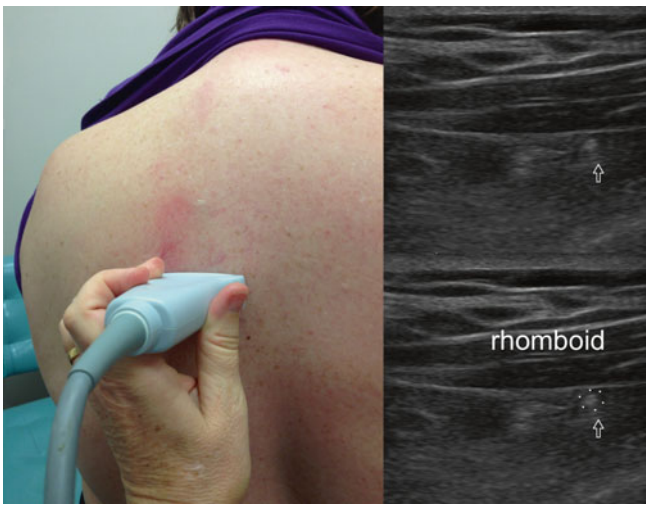

Fig. 32.15 Probe location and US imaging of the dorsal scapular nerve at the rhomboid (Image courtesy of Andrea Trescot, MD)

Radiofrequency Lesioning

Restrepo-Garces et al. [23] used US at the middle scalene in an in-plane approach to perform pulsed RF of the DSN. After sensory and motor confirmation, they made two lesions of 42 °C for 120 s. The patient noted 60 % reduction in her pain.

Alcohol/Phenol

There is no literature regarding the use of alcohol or phenol to treat DSN pathology.

Botulinum Toxin

Haim and Urban [15] described the use of 100 units of botulinum toxin in the rhomboid muscles to treat the dystonia associated with DSN entrapment.

Surgery

Of the 36 patients diagnosed with DSN entrapment, Chen et al. [14] reported on 22 patients who underwent middle scalene resection for DSN entrapment after good, but only temporary, relief of their arm and neck pain with DSN injections in the neck; most had been diagnosed with "thoracic outlet syndrome." All 22 noted that their symptoms were "mostly relieved" after surgery.

Stimulation

There are no peripheral stimulation techniques reported in the literature.

Complications

Because of the proximity of the brachial plexus, supraclavicular artery, and lung, both scalene and subscapular DSN injections have the potential for serious complications, though none have been described in the literature. The use of US for the neck injections, and positioning the needle parallel to the scapula for the chest wall injections, should dramatically decrease the risk of complications.

Summary

Dorsal scapular nerve entrapment is a very under-recognized cause of neck and shoulder pain. The scapular winging may be subtle, and the symptoms are easily mistaken for other problems. Careful history and physical exam, along with a high index of suspicion, are necessary for accurate diagnosis and therefore appropriate treatment.

References

1. Sultan HE, Younis El-Tantawi GA. Role of dorsal scapular nerve entrapment in unilateral interscapular pain. Arch Phys Med Rehabil. 2013;94(6):1118–25.
2. Benoit P, Deplante F. Paralysis of the dorsal nerve of the scapula: an unusual cause of scapular displacement. Presse Med (Paris, France: 1983). 1994;23(7):348.
3. Gray AT. Dorsal sapular nerve imaging. In: Gray AT, editor. Atlas of ultrasound-guided regional anesthesia: expert consult – online. 2013. 2nd ed. Philadelphia: Elsevier; 2013. p. 84–5.
4. Aker PD, Gross AR, Goldsmith CH, Peloso P. Conservative management of mechanical neck pain: systematic overview and meta-analysis. BMJ. 1996;313(7068):1291–6.
5. Jerosch J, Castro WH, Geske B. Damage of the long thoracic and dorsal scapular nerve after traumatic shoulder dislocation: case report and review of the literature. Acta Orthop Belg. 1990;56(3-4):625–7.
6. Ravindran M. Two cases of suprascapular neuropathy in a family. Br J Sports Med. 2003;37(6):539–41.
7. Walther DS. Selected papers of the international college of applied kinesiology. Detroit: International College of Applied Kinesiology; 1982.
8. Mondelli M, Cioni R, Federico A. Rare mononeuropathies of the upper limb in bodybuilders. Muscle Nerve. 1998;21(6):809–12.
9. Wood VE, Twito R, Verska JM. Thoracic outlet syndrome. The results of first rib resection in 100 patients. Orthop Clin North Am. 1988;19(1):131–46.
10. Pecina MM, Krmpotic-Nemanic J, Markiewitz AD. Dorsal scapular nerve syndrome. In: Pecina MM, Krmpotic-Nemanic J, Markiewitz AD, editors. Tunnel syndromes: peripheral nerve compression syndromes. 3rd ed. Boca Raton: CRC Press; 2001. p. 69–70.

11. Frank DK, Wenk E, Stern JC, Gottlieb RD, Moscatello AL. A cadaveric study of the motor nerves to the levator scapulae muscle. Otolaryngol Head Neck Surg. 1997;117(6):671–80.
12. Akgun K, Aktas I, Terzi Y. Winged scapula caused by a dorsal scapular nerve lesion: a case report. Arch Phys Med Rehabil. 2008;89(10):2017–20.
13. Srikumaran U, Wells JH, Freehill MT, Tan EW, Higgins LD, Warner JJ. Scapular winging: a great masquerader of shoulder disorders: AAOS exhibit selection. J Bone Joint Surg Am. 2014; 96(14):e122.
14. Chen D, Gu Y, Lao J, Chen L. Dorsal scapular nerve compression. Atypical thoracic outlet syndrome. Chin Med J (Engl). 1995;108(8):582–5.
15. Haim K, Urban BJ. Dorsal scapular nerve block: description of technique and report of a case. Anesthesiology. 1993;78(2):361–3.
16. Tubbs RS, Tyler-Kabara EC, Aikens AC, Martin JP, Weed LL, Salter EG, et al. Surgical anatomy of the dorsal scapular nerve. J Neurosurg. 2005;102(5):910–1.
17. Boehnke H. Dorsal scapular nerve syndrome [10/14/14]. Available from: http://balancedstructure.com/files/Dorsal_Scapular_Nerve_Syndrome.pdf.
18. Martin RM, Fish DE. Scapular winging: anatomical review, diagnosis, and treatments. Curr Rev Musculoskelet Med. 2008;1(1):1–11.
19. Pan Z, Liu W, Zhang Y, He Z. [Anatomical study of dorsal scapular nerve compression] [Chinese]. Chinese J Hand Surgery. 1996;1:17–9.
20. Loukas M, Louis Jr RG, Merbs W. A case of atypical insertion of the levator scapulae. Folia Morphol (Warsz). 2006;65(3):232–5.
21. Liu Q-j, Zhang X-p, Xia P, Zhu J, Li J. Clinical analysis of 128 patients with dorsal scapular nerve compression treated with pain-point injections. Pain Clin J. 2010;1:10–2.
22. Hanson NA, Auyong DB. Systematic ultrasound identification of the dorsal scapular and long thoracic nerves during interscalene block. Reg Anesth Pain Med. 2013;38(1):54–7.
23. Restrepo-Garces CE, Gomez CM, Jaramillo S, Ramirez L, Vargas F. Dorsal scapular nerve block under ultrasound guidance. Poster presentation ASRA 11th Annual meeting. Miami. 2012

Part V

Upper Extremity Peripheral Nerve Entrapments

Virtaj Singh

Introduction

In 2008, Huisstede et al. [1] looked at the prevalence and characteristics of "complaints of arm, neck and/or shoulder" (CANS) pain and found that 36.8 % of the nearly 4,000 people surveyed complained of acute or chronic arm, neck, and/or shoulder pain. Paresthesia and weakness of the upper extremity due to a neural injury and entrapment can occur in a variety of locations. The spinal nerves that supply motor function and sensation to the upper extremity emanate from the spinal cord at five levels: C5, C6, C7, C8, and T1. Before finally dividing into the various peripheral nerves, these nerves begin as cervical roots and then combine in an intricate fashion to form the brachial plexus. The focus of this section is on the entrapments of the peripheral nerves.

Nerve entrapments can occur in a number of locations throughout the body. Our focus in this section will be on upper extremity peripheral nerve entrapments, but it is worthwhile to briefly highlight three other common entrapments. The classic cervical radiculopathy occurs at the level of the cervical nerve roots. Entrapments at this level will present in characteristic "radicular" patterns, and providers must consider this possibility in the differential diagnosis of any of the entrapments discussed in this section. Similarly, brachial plexopathies are another common location for neural injury of which providers need to be aware. Indeed, neurogenic thoracic outlet syndrome represents one such entrapment of the lower trunk of the brachial plexus to which we have devoted more detailed discussion (Chap. 33) because it is often subject to misdiagnosis.

Overall, we will discuss each of the most common upper extremity peripheral nerve entrapments, from the most proximal to the most distal. We will begin with the most common peripheral nerve entrapments of the shoulder girdle, the suprascapular nerve, and the axillary nerve. We will then discuss the radial nerve, followed by discussions of the median and ulnar nerves. For each nerve, we will describe the anatomy, with particular focus on the various sites of potential entrapment. Next, we will highlight the clinical entrapment syndromes and their associated physical examination findings. Finally, we will outline the various treatment approaches with an emphasis on injection techniques.

Reference

1. Huisstede BM, Wijnhoven HA, Bierma-Zeinstra SM, Koes BW, Verhaar JA, Picavet S. Prevalence and characteristics of complaints of the arm, neck, and/or shoulder (CANS) in the open population. Clin J Pain. 2008;24(3):253–9.

Thoracic Outlet Syndrome (Neurogenic)

33

Virtaj Singh, Christopher J. Burnett,
and Richard E. Seroussi

Introduction

Thoracic outlet syndrome (TOS) refers to a diverse group of disorders that result from entrapment of one or more of the elements of the neurovascular bundle as they leave the cervical spine and travel to the axilla. Due to the complex anatomy of the region, there are multiple potential sites of entrapment of the brachial plexus or subclavian vessels. Depending on the site of entrapment, this condition is also called *costoclavicular syndrome*, *scalenus anticus syndrome* [1–3], *cervical rib syndrome*, *first thoracic rib syndrome*, *pectoralis minor syndrome* [1], *hyperabduction syndrome*, and *neurogenic pectoralis minor syndrome (NPMS)* [4]. Although both forms typically exist concomitantly, *neurogenic TOS* (>90 % of TOS and the focus of this chapter) should be distinguished from *arterial TOS* (ATOS) (<1 %) and *venous TOS* (VTOS) (3 %) [3]. VTOS is sometimes called *effort thrombosis* or *Paget-Schroetter disease*.

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Clinical Presentation (Table 33.1)

Paget first described what is now called *thoracic outlet syndrome* (TOS) in 1875 [7], though it was not named TOS until 1956. TOS is defined as "upper extremity symptoms due to compression of the neurovascular bundle by various structures in the area just above the first rib and behind the clavicle" [3]. There are several relatively common causes of TOS (Table 33.1). The brachial plexus can be compressed at two locations, namely, above and below the clavicle. Often (75 % of the time) [5], the compression is at both locations, leading to a "double crush" syndrome (see Chap. 1).

The patient with TOS will present with neck, shoulder, and arm pain and paresthesias (Fig. 33.1), as well as occipital headaches [3]. Patients typically present with symptoms of TOS between the ages of 20 and 40, though the condition has been reported in patients as young as 11 years old. The syndrome is more commonly seen in women than men [6, 8]. The clinical presentation will vary depending on the structures that are being compressed. Most have a history of some kind of trauma [3], although patients with cervical ribs may have no trauma history.

Neurogenic TOS (NTOS) comprises 90–97 % of cases (Table 33.2) [6]. This form of TOS is caused largely by compression of the distal C8–T1 nerve roots or proximal fibers of the lower trunk of the brachial plexus, primarily in the distribution of the ulnar nerve. These patients may present with arm weakness, fatigability, numbness, and tingling in the distribution of the posterior and medial cords of the brachial

Table 33.1 Occupation/exercise/trauma history relevant to thoracic outlet syndrome

Neck trauma, especially a hyperextension injury	Motor vehicle accidents are common [5]
Sports with repetitive arm movements	Swimming is the most common sport affected [5]
Overhead activities	Sleeping with arm over the head [6]
Work-related injuries	Repetitive arm maneuvers, lifting heavy objects [3]

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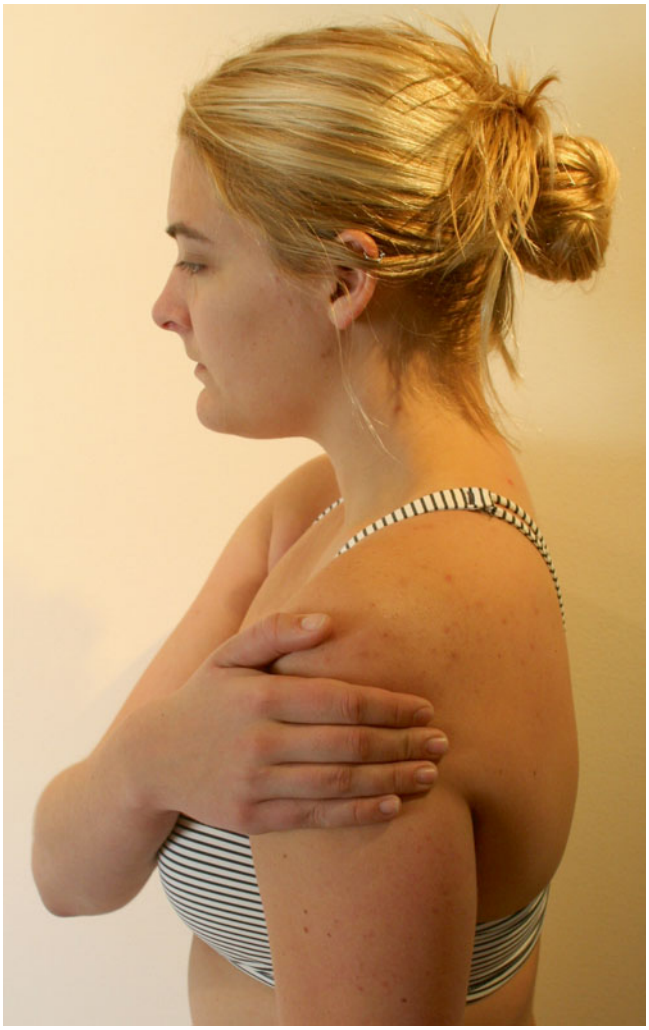

Fig. 33.1 Patient pain complaints from thoracic outlet syndrome (Image courtesy of Andrea Trescot, MD)

Table 33.2 Types of thoracic outlet syndrome

Types of TOS	Characteristics
Neurogenic TOS	Weakness, tingling, numbness, ulnar distribution
Venous TOS	Edema and heaviness; vascular thrombosis causes cyanosis and chest wall/upper arm venous distension
Arterial TOS	Arm appears cool and pale, with decreased pulses

plexus. This distribution is primarily in the lower trunk or medial cord of the brachial plexus, mainly in the distribution of the ulnar nerve [6], though there may be numbness of all five fingers [3]. Atrophy of the thenar, hypothenar, or interossei muscles of the hand may be present, although this presentation is fairly rare in clinical practice.

These patients may complain of an aching sensation in the cervical region, shoulder, or arm, as well as occipital

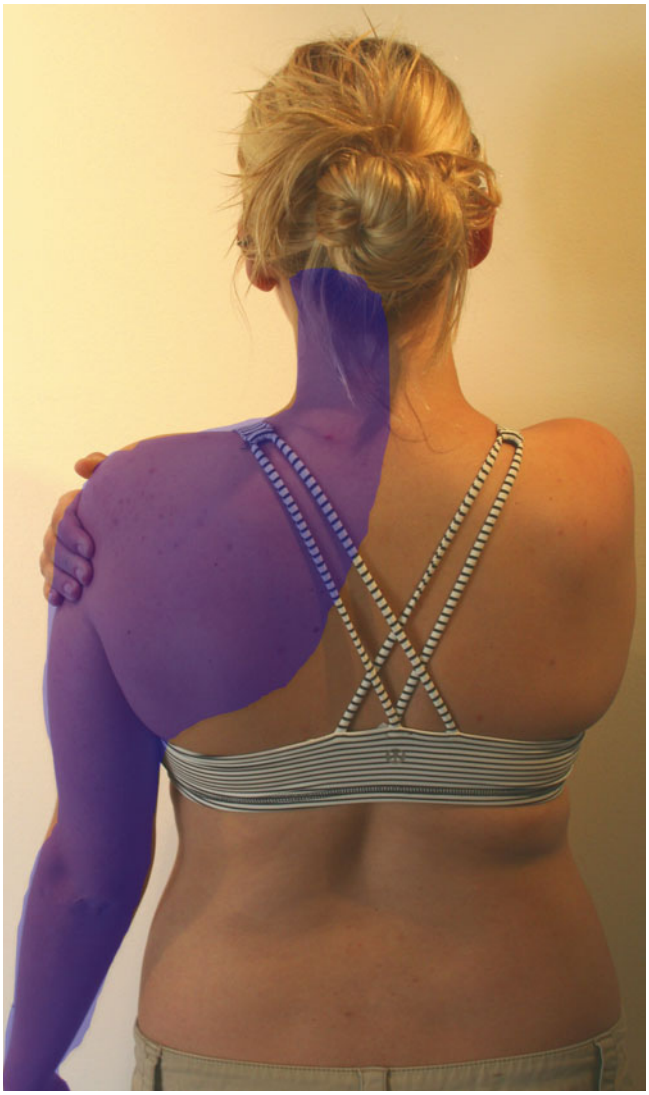

Fig. 33.2 Pattern of pain from thoracic outlet syndrome (Image courtesy of Andrea Trescot, MD)

headaches (Fig. 33.2). Symptoms may be exacerbated by combing the hair, sleeping with the arm above the head, carrying heavy objects, repetitive overhead arm maneuvers, or lifting heavy objects overhead [3, 6, 9, 10]. On physical exam (see section "Physical exam" below), there is often subtle C8–T1 distributed motor weakness and more notable sensory changes in the affected extremity. *Neurogenic pectoralis minor syndrome (*NPMS), which is a more distal entrapment, results in symptoms that are more in the shoulder and arm, with tenderness over the pectoralis minor (see section "Physical exam" below).

Venous TOS (VTOS) is caused by a discreet compression of the subclavian and/or axillary vein in the costoclavicular space, seen in 2–4 % of patients [3, 6, 8]. This condition can result from positional compression of the vein between the clavicle and the first thoracic or a cervical rib. Positional

compression may present as edema and heaviness of the affected arm with repetitive overhead movements. Alternatively, repeated friction from the same structures can result in development of a vascular thrombosis in the subclavian vein, thus the name "*effort thrombosis*"; it is also sometimes called *Paget-Schroetter disease*. Patients with thrombosis may present with cyanosis or edema of the involved upper extremity with concurrent distension of shoulder and chest wall collateral veins. Patients with the thrombotic form are at risk for serious complications such as *pulmonary emboli* [6, 8, 10].

Arterial TOS is the most rare, but potentially most serious, form of the condition. It results from compression of the subclavian artery in the interscalene triangle, perhaps partially by compression from a bony anomaly such as a cervical or aberrant first thoracic rib. These patients often present with intermittent pain, paresthesias, and fatigability with extended use or overhead positioning of the arm. The extremity may appear cool and pale, with weakness and diminished pulses. In advanced cases, patients may develop advanced ischemic damage to the distal upper extremity secondary to emboli to the hand or digits [6, 8, 10]. It is interesting to note that, while cervical ribs are seen in less than 1 % of the population, 70 % are found in women. It may be that a cervical rib predisposes development of TOS after flexion-extension injuries [3].

It should be understood that some authors—primarily neurologists and physiatrists who have a focus on electrodiagnostics—believe that "neurogenic thoracic outlet syndrome" is an extremely rare disorder with classic findings of marked intrinsic muscle atrophy in the hand, electrodiagnostic changes of axon loss on needle EMG exam of these muscles, and nerve conduction findings of decreased sensory amplitude of the ulnar nerve and decreased motor amplitude of the median-innervated *abductor pollicis brevis* (APB) muscle. Thus, this group of authors requires "neurogenic thoracic outlet syndrome" to show clear electrophysiologic evidence of a C8–T1 lower trunk-distributed brachial plexopathy [1, 11].

This same group of specialists would consider the most common forms of TOS as "nonspecific" rather than neurogenic. In clinical practice, as physiatrists with expertise in electrodiagnosis, these authors have not found such electrophysiologic criteria to be useful. The vast majority of patients presenting with TOS, without notable vascular abnormalities, also have normal electrodiagnostic studies. In these authors' experience, to require electrodiagnostic abnormalities is to employ a screening tool that is unacceptably insensitive. In other words, the authors of this chapter believe that, were electrodiagnostic abnormalities required for diagnosis, an unacceptable number of patients would remain undiagnosed and without adequate care. As discussed below, the use of a scalene muscle injection is recommended to confirm the diagnosis.

Anatomy (Table 33.3)

The cervical nerves exit their neural foramen and join to form the brachial plexus and then pass through the *scalene muscles* (the *anterior*, *middle*, and *posterior scalene* muscles). The thoracic outlet is the region of the shoulder and thorax through which the subclavian vessels exit the chest and join the brachial plexus, traversing through the *interscalene triangle* over the first rib and under the clavicle to enter the axilla. The anatomic boundaries of the thoracic outlet are the superior surface of the first rib and the anterior and middle scalene muscles, which both insert onto the *first rib* (Fig. 33.3).

The *subclavian artery* originates from the brachiocephalic artery on the right and directly from the aorta on the left. Conversely, the *subclavian vein* carries venous blood from the distal upper extremity toward the thorax, joining the internal jugular veins and ultimately combining to form the *superior vena cava* [12–14].

The *brachial plexus* is formed from the anterior primary rami of the cervical nerve roots of C5, C6, C7, and C8 and the greater part of the first thoracic nerve (Fig. 33.3). After emerging from their respective intervertebral foramen, the nerves course posterior to the vertebral artery, traveling horizontally and laterally. At the lateral border of the middle scalene muscle, the roots combine to form the *superior*, *middle*, and *inferior trunks* of the brachial plexus (Fig. 33.4). The subclavian artery and the three trunks of the plexus pass over the first rib, between the anterior scalene muscle anteriorly and the middle scalene muscle posteriorly. This space is termed the *interscalene triangle*. At this level, the brachial plexus lies posteriorly and laterally to the artery.

Table 33.3 Anatomy of the thoracic outlet syndrome

Origin	C6–T1 brachial plexus roots (anterior rami of spinal nerves) travel between the anterior and middle scalene muscles, above the clavicle
General route	Converge into upper, middle, and lower brachial plexus trunks above the clavicle and lateral to the scalene muscles
	Divide into three anterior (flexors) and three posterior (extensors) divisions behind the clavicle
	Converge into lateral, medial, and posterior cords in the axilla, behind the pectoralis minor muscle
	Elements from the cords converge to form the infraclavicular brachial plexus nerves
Sensory distribution	Medial antebrachial nerve [2]; all the nerves of the distal brachial plexus, especially ulnar distribution
	Second intercostal brachial nerve (cutaneous branch of second intercostal)
Motor innervation	All the muscles of the distal brachial plexus

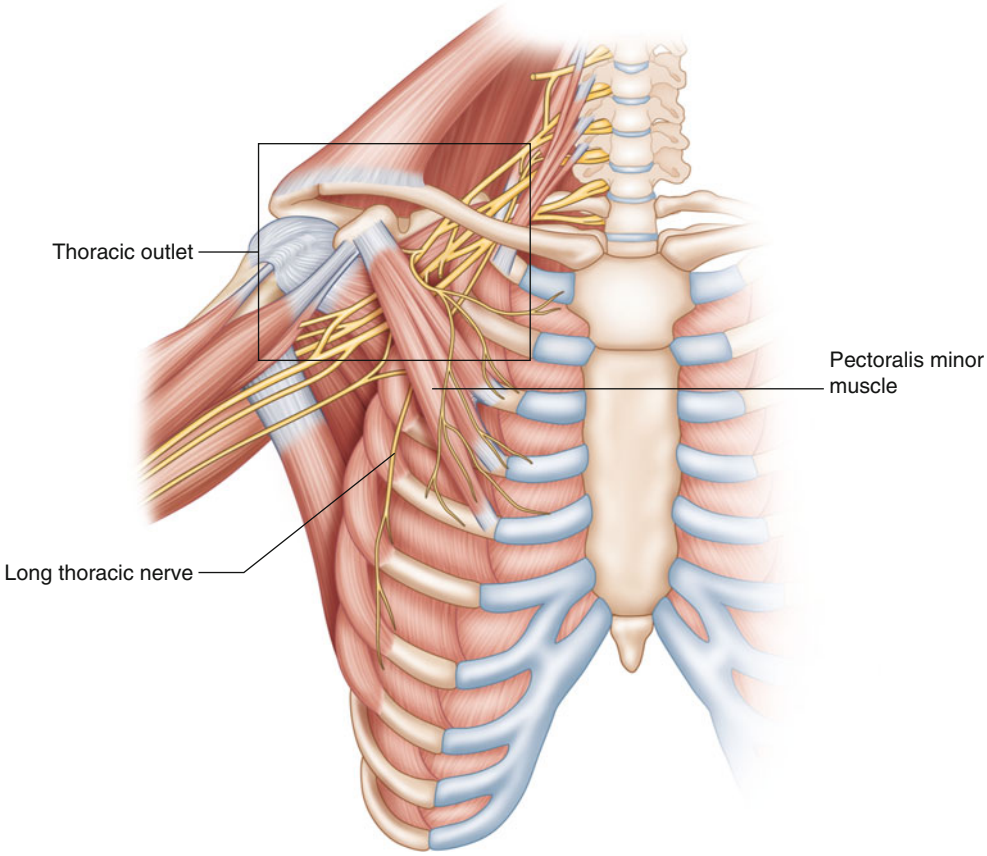

Fig. 33.3 Anatomy of the thoracic outlet (Image by Springer)

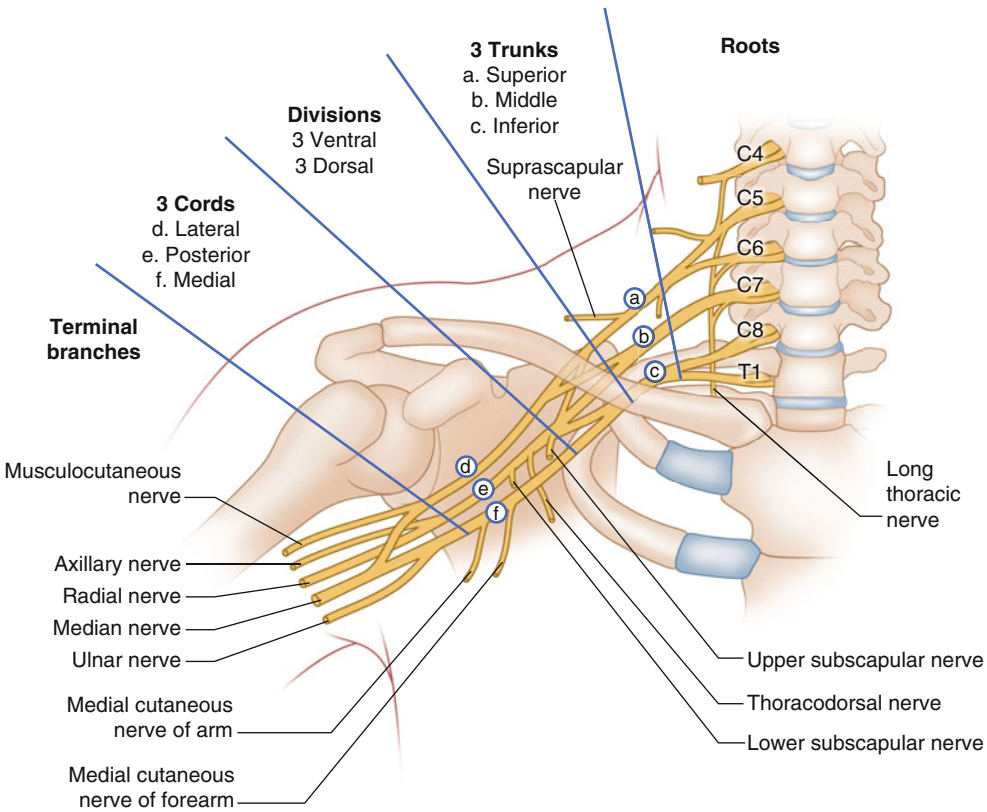

Fig. 33.4 Schematic showing the relationship of the nerves of the brachial plexus (Image by Springer)

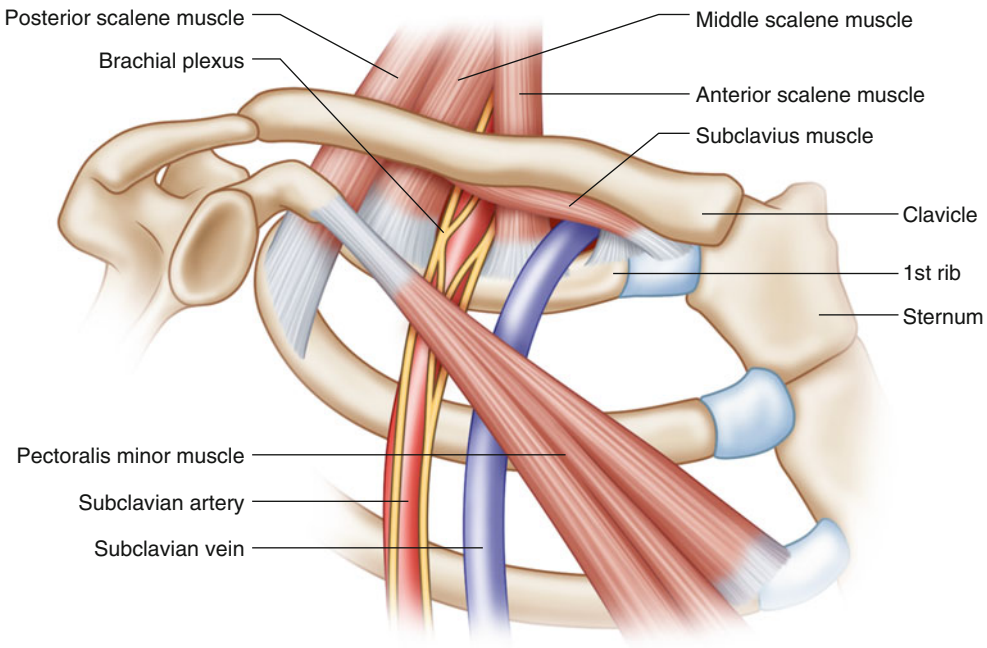

Fig. 33.5 Schematic showing the relationship between the nerves, vessels, and bony structures (Image by Springer)

The subclavian vein lies anterior to the anterior scalene muscle (Fig. 33.5) [12, 14, 15].

At the lateral border of the first rib, slightly above or behind the middle third of the clavicle, the trunks of the brachial plexus divide into anterior and posterior divisions (Fig. 33.4). Here, the subclavian artery also transitions into the *axillary artery*. The divisions of the brachial plexus continue to descend under the clavicle toward the axilla in the costoclavicular space, along with the axillary artery. While passing below the clavicle, the divisions join again to form a *posterior*, *medial*, and *lateral cord* (Fig. 33.4). These cords are named according to their spatial relationship to the axillary artery, which lies in the center [12, 14, 15].

The triangular-shaped *pectoralis minor muscle* (Fig. 33.5) originates at the third to fifth rib and attaches to the coracoid process of the scapula. This muscle creates a passage, under which the neurovascular structures of the arm must travel. Once the nerves of the brachial plexus reach the lateral border of the pectoralis minor muscle, the three cords divide to form the terminal branches of the *brachial plexus* [12, 14].

Entrapment

There are three common locations for entrapment of the neurovascular bundle that can result in TOS: the *interscalene triangle*, the *costoclavicular space*, and the *pectoralis minor* space or *subcoracoid* space [6, 8, 9].

In clinical practice, by far the most common source of entrapment in thoracic outlet syndrome is the interscalene triangle. The most common cause for neurogenic thoracic outlet syndrome occurs from *motor vehicle crashes* (MVCs). In these authors' clinical experience, the patient who has sustained a motor vehicle crash more commonly has left-sided or left-worse-than-right TOS, presumably due to biomechanical factors of having the shoulder restraint seatbelt tethering the left upper chest wall while the head and neck are subjected to sudden acceleration-deceleration forces.

Patients with neurogenic TOS from a MVC are more commonly women, in a ratio of over 2:1 as compared to men [14]. This ratio is similar to that observed among chronic whiplash patients, who are also twice as likely to be women. In these authors' clinical experience, women with generalized hypermobility are particularly susceptible to both *chronic whiplash syndrome* and *post-whiplash neurogenic TOS*.

Entrapment at the interscalene triangle can occur from the presence of a *cervical rib* (Fig. 33.6), which is an incidental finding on 0.5–0.7 % of all chest radiographs. It is a bilateral finding in 70 % of those affected and is seen twice as commonly in women. These structures are often incomplete with a fibrous attachment to the scalene tubercle of the true first rib. Entrapment can occur from the cervical rib inferiorly, anterior scalene muscle anteriorly, and middle scalene muscle posteriorly. As it passes through the interscalene triangle, an anomalous insertion site of the anterior scalene muscle can also entrap the neurovascular bundle [6, 8, 9]. Given that the lower nerve roots are draped across the first rib (or the cervical rib if present), it should not be surprising that early symptoms may be limited to an ulnar distribution.

Although a cervical rib is *associated* with thoracic outlet syndrome, the presence of a cervical rib does not clinch the

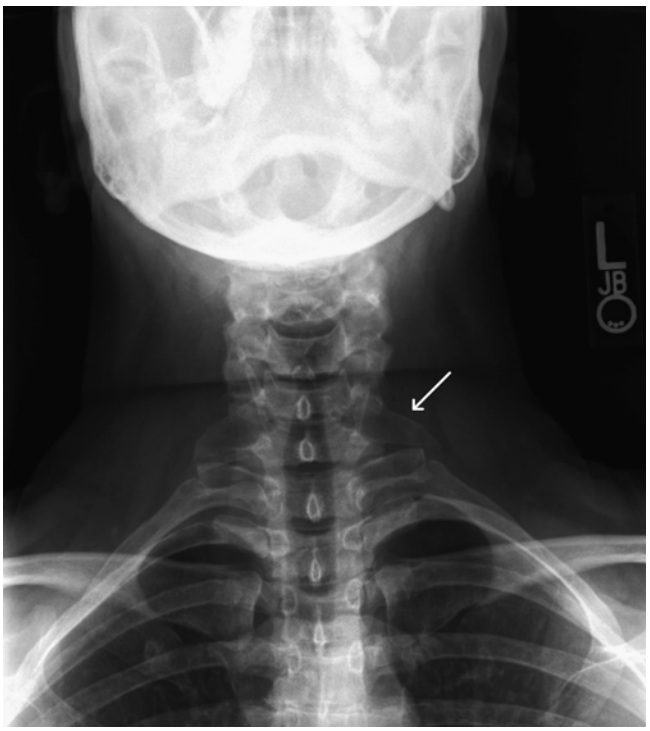

Fig. 33.6 Cervical X-ray showing cervical rib (*white arrow*) (Image courtesy of Andrea Trescot, MD)

diagnosis. The presence of a cervical rib is linked to more serious forms of this disorder, including arterial TOS and the rare neurogenic form of TOS that shows marked electrodiagnostic abnormalities consistent with muscle denervation in a C8–T1 distribution. Thoracic outlet syndrome with present medical diagnostics is mostly a clinical diagnosis without pathognomonic imaging or electrophysiologic findings. However, if symptoms are severe without response to conservative treatment, one should consider surgical consultation, cervical X-rays to examine for the presence of a cervical rib, and performing electrodiagnostic studies—partly to address competing diagnoses such as *cubital tunnel syndrome* (see Chap. 38), *carpal tunnel syndrome* (see Chap. 37) with concomitant neck or shoulder problems, and cervical radiculopathy.

Entrapment at the costoclavicular space is also termed *costoclavicular syndrome*. It occurs as a result of a posterior downward displacement of the shoulders, which may be caused by abnormalities of the first rib, prior clavicular fractures, or hypertrophy of the subclavius muscle. Similarly, descent of the shoulder girdle seen with aging or deconditioning can produce an analogous clinical syndrome [6, 8].

Entrapment in the pectoralis minor space results from compression associated with arm abduction, also known as *hyperabduction syndrome*. It results from placement of the upper extremities in the extreme abducted position. Entrapment occurs anterior to the first rib in the retroclavicular space as the neurovascular bundle passes under the coracoid process beneath the pectoralis minor muscle [6, 8].

In these authors' clinical experience, entrapment at the pectoralis minor insertion at the coracoid process is a distant second-most common cause for TOS (after scalene entrapment). It may be relatively more common in the occupational or non-whiplash trauma setting, such as a case of a patient who has had abrupt manipulation of the shoulder or who needs to perform repetitive forward reaching or overhead activities in the worksite. The literature on involvement of the pectoralis minor muscle and tendon in producing TOS is fairly scant, with a number of articles concentrated from Richard Sanders, MD [3, 4, 16], a vascular surgeon who has helped pioneer the understanding of TOS and its clinical features.

Physical Exam

Physical examination for thoracic outlet syndrome should include an examination of the cervical spine and shoulder and a neuromuscular examination. There are tests that are more relevant for vascular thoracic outlet syndrome such as an *Adson's maneuver*, looking for a decreased radial pulse with rotation of the head. Specific to neurogenic thoracic outlet syndrome, one can check an *upper limb tension test (ULTT)*. For this maneuver, the arms are abducted to 90°, and the wrist should be extended 90° on both sides. The cervical spine is then bent laterally with the contralateral ear getting as close as possible to the contralateral shoulder (Fig. 33.7). If positive, these movements should recreate and worsen the dysesthesias the patient typically feels. Alternatively, the *90° abduction in an external rotation stress test* (*90° AER*) or *elevated arm stress test* (*EAST*) (Fig. 33.8) and *EAST with contralateral head rotation* (Fig. 33.9) can be useful provocative tests for NTOS. Although originally described to evaluate a decrease in radial pulse, indicative of arterial compression, Sanders and colleagues reported that 94 % of NTOS patients had reproduction of symptoms with the EAST, but only 24 % had a decreased pulse [3].

Other key findings in neurogenic thoracic outlet syndrome include pain and spasm with palpation over the anterior scalenes (Video 33.1) (Fig. 33.10) and/or the pectoralis minor (Fig. 33.11). In addition, there is often weakness in a pattern typical of compression of the lower trunk of the brachial plexus, such as weakness of the abductor pollicis brevis (APB), abductor digiti minimi (ADM), interossei, finger flexors, and finger extensors. *Neurogenic pectoralis minor syndrome* (NPMS), which is a more distal entrapment, can result in symptoms that are more in the shoulder and arm, with tenderness over the pectoralis minor.

In these authors' clinical experience, weakness is usually only detected in the intrinsic muscles of the hand and is not

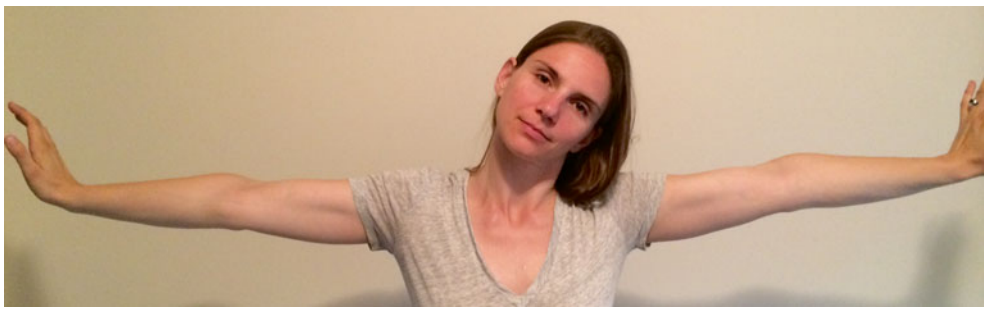

Fig. 33.7 Upper limb tension test (ULTT) (Image courtesy of Virtaj Singh, MD)

Fig. 33.8 Elevated arm stress test (EAST) (Image courtesy of Virtaj Singh, MD)

Fig. 33.9 Elevated arm stress test (EAST) with contralateral head rotation (Image courtesy of Andrea Trescot, MD)

Fig. 33.10 Palpation of the anterior scalene muscle with head in resisted rotation (Image courtesy of Andrea Trescot, MD)

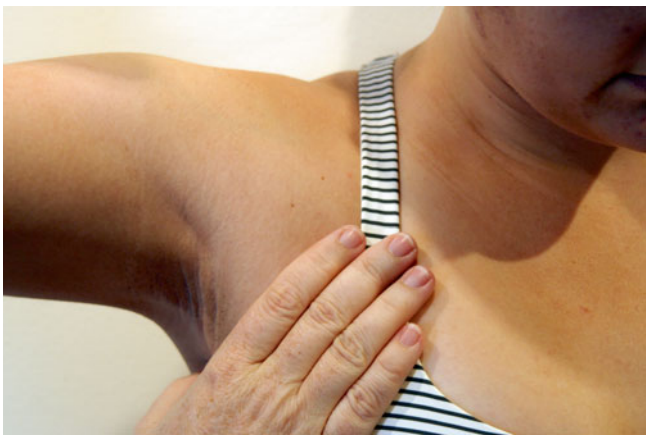

Fig. 33.11 Palpation of the pectoralis minor muscle (Image courtesy of Andrea Trescot, MD)

notable in the forearm muscles, such as the finger flexors and extensors. When subjected to dynamometer testing, the weakness in grip strength may be detectable, especially in more severe cases, but the difference in handgrip measurements between the affected and unaffected side in TOS is often negligible. When there is a baseline difference in handgrip strength, this difference can be tracked over time and used as a gauge of clinical improvement.

Differential Diagnosis (Table 33.4)

Diagnosis of TOS is based on history and physical examination. The differential diagnosis includes: cervical radiculopathy, cervical facet arthritis, cervical tumors, complex regional pain syndrome, shoulder bursitis, glenohumeral joint instability, rotator cuff injury, peripheral nerve

Table 33.4 Differential diagnosis of pain and parasthesias in the upper extremity

	Potential distinguishing features
ATOS [3]	Onset is spontaneous and almost always associated with a cervical rib. Pain is due to emboli and is often in the hand. Absent radial pulse is likely
VTOS [3]	May have a history of excessive arm movement. PE is distinctive for arm swelling, distended superficial veins, cyanosis. Pain and/or ache may or may not be a prominent feature
Cervical radiculopathy	Dermatomal pain pattern, hypoesthesia, weakness, decreased reflexes
Cervical facet pathology	Tenderness over the cervical facets, response from facet injections
Myofascial pain (such as scalene or trapezius)	Muscle spasm, palpable trigger points, improvement with myofascial release

compression (cubital and carpal tunnel syndrome), rheumatologic conditions, neoplasm of peripheral nerves, apical pulmonary neoplasm, multiple sclerosis, diabetes, and vascular disorders [7–9].

Among trauma patients, including those with whiplash, the differential diagnosis can be challenging. The diagnostician often needs to tease out whether the patient has a primary shoulder problem, neck problem with concomitant carpal tunnel or cubital tunnel syndrome, TOS, or some combination of these problems.

Diagnostic Tests for Thoracic Outlet Syndrome (Table 33.5)

To identify the patient's primary problem, the provider often needs to use diagnostic injections with local anesthetic addressing competing disorders, for example, the scalene motor block for TOS (described below), subacromial shoulder injection, carpal tunnel injection, and/or cervical epidural injection. Cervical MRI, especially coronal views (Fig. 33.12), may offer information on the differential diagnosis.

As described in the "Physical exam" section, in the upper limb tension test (ULTT), the arms are abducted to 90° and the wrist should be extended 90° on both sides. The cervical spine is then bent laterally with the contralateral ear getting as close as possible to the contralateral shoulder. In 2015, a group of investigators placed markers within the foramen of the C5, C6, C7, and C8 nerve roots of 11 cadavers and then performed the ULTT under fluoroscopy, measuring the amount of movement with this maneuver [17]. They showed a significant movement of the nerve roots and greater than 6 % strain on the nerves; 6 % strain has been shown to reduce nerve action potential propagation, and 8 % strain can significantly decrease nerve blood flow.

Table 33.5 Diagnostic tests for thoracic outlet syndrome

	Potential distinguishing features
MRI	Expensive; not useful for diagnosis
Diagnostic blocks of tender muscle	If both are tender, first block the PMM; if still symptomatic, then block the scalene
Arteriography	Only if in S&S there is ischemia. Used to plan arterial reconstruction
Plain X-rays	If WNL, rule out ATOS. Look for the presence of cervical ribs and anomalous first ribs
Electrodiagnostic studies [2, 11]	Usual EMG and NCV are usually WNL. Changes in latency and/or amplitude of the medial antebrachial cutaneous N (lowest branch of the brachial plexus) are very useful, but cannot distinguish between compression above and below the clavicle. Use asymptomatic side as control
C8 root stimulation [2]	Abnormal values further confirm NTOS. Difficult technique. Use asymptomatic side as control

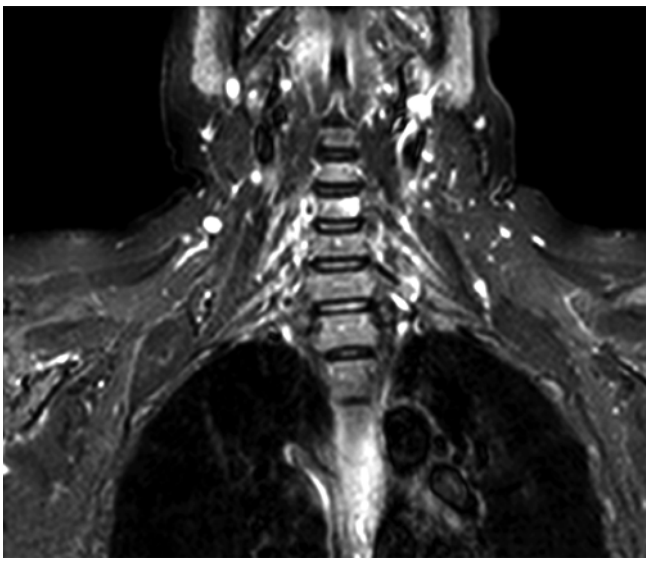

Fig. 33.12 MRI coronal view of the brachial plexus (Image courtesy of Andrea Trescot, MD)

Identification and Treatment of Contributing Factors

Factors that contribute to TOS may include presence of a cervical rib, aberrant insertion site of the anterior or middle scalene muscles, hypertrophy or spasm of the anterior or middle scalene muscles, or the presence of a high first thoracic rib. They also include decent of the shoulder girdle seen with aging, deconditioning, or large pendulous breasts, presence of fibrous bands in any of the potential entrapment spaces, or the presence of tumors near the neurovascular bundle [6–9].

Physical therapy and/or occupational therapy are the primary means to treat NTOS from a conservative standpoint. The focus of any therapy program should be developing a home exercise program focused on scapular stabilization, scalene/pectoralis minor stretches, postural retraining, and nerve glide exercises. Massage therapy can also be useful to help mobilize the tissues, which may provide compression of the brachial plexus in the thoracic outlet.

Injection Technique

Although there is no gold standard for the diagnosis of neurogenic TOS, the use of diagnostic anterior scalene injections has been validated [18]. This procedure involves the injection of a small amount of anesthetic into the anterior scalene muscle to weaken the muscle and presumably reduce compression of the brachial plexus in the thoracic outlet. If the block is effective using anesthetic, a similar procedure as discussed below can be used to chemodenervate the scalene muscle using *botulinum toxin*.

Landmark-Guided Technique

The anterior scalene muscle can be identified by having the patient lift or turn his/her head against resistance (Fig. 33.10). The index and middle finger straddle either side of the muscle to keep it from rolling to one side and to prevent the needle from slipping into the interscalene groove between the anterior and middle scalene muscles and the location of the brachial plexus. The needle is angled slightly cephalad so as not to enter the cervical foramen and advanced only through the fascia into the body of the muscle (Video 33.2) (Fig. 33.13). Care must be taken to avoid piercing through the muscle because of the nerves and vessels that lie deep to the scalene muscles. After negative aspiration, 1–2 cc of local anesthetic is injected in divided doses. There should be a "sausage-shaped" filling of the muscle body, easily felt between the fingers. However, given the potential for complications listed below, the authors of this chapter do not advise doing a scalene injection without guidance, using either EMG, fluoroscopy, or ultrasound.

Fluoroscopic-Guided Technique

Since the anterior scalene muscle is not visible under fluoroscopy, it is usually not injected under fluoroscopy. However, it attaches to the transverse processes of C3–C6 superiorly and the first rib inferiorly (Fig. 33.3), both of which are visible on a fluoroscopic image (Fig. 33.14). The needle entry should be from anterior to posterior, on to the bone, so as to avoid entry into the cervical foramen or vertebral artery. Injection of contrast can confirm the location as well as the lack of vascular uptake. Figure 33.15 shows the 3D CT image of contrast in the anterior scalene muscle after an injection of local anesthetic and contrast onto the muscle.

Ultrasound-Guided Technique

The use of ultrasound guidance has been validated as safe and well tolerated for injection into the anterior scalene muscle [19]. For this technique, the patient lies supine and rotates his/her cervical spine to the contralateral side in order to open up the interscalene triangle on the affected side. After a sterile skin preparation, the ultrasound probe is placed in the supraclavicular region in a transverse fashion. The subclavian artery is identified. Superior lateral to this,

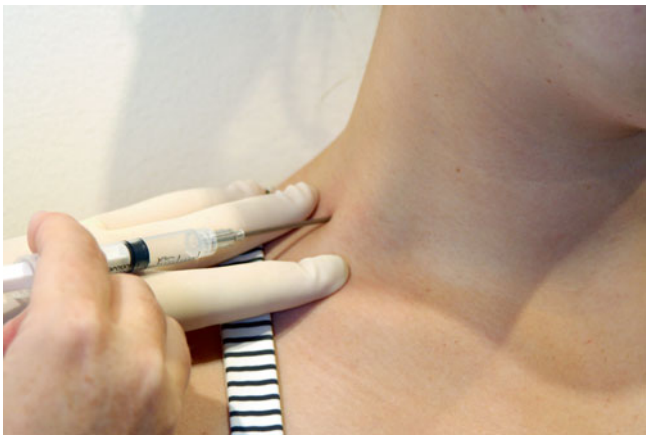

Fig. 33.13 Landmark-guided scalene injection (Image courtesy of Andrea Trescot, MD)

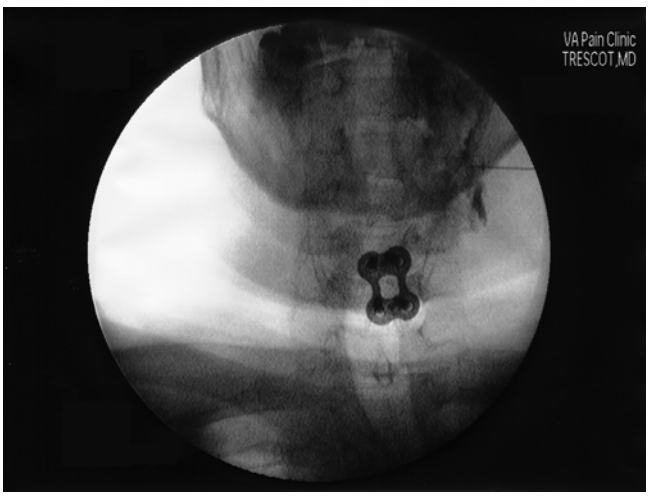

Fig. 33.14 Fluoroscopic image of scalene injection at the transverse process (Image courtesy of Andrea Trescot, MD)

the anterior scalene muscle can be identified along with the adjoining brachial plexus (Fig. 33.16). Being sure to avoid injection too close to the brachial plexus, a 27-gauge, 1.5-in. needle can be used to inject 1–2 cc of local anesthetic directly into the anterior scalene muscle (Fig. 33.17). Immediately after the procedure, patients should be monitored for any complications and then monitored for response to the injection itself.

Benzon and colleagues [20] studied 12 patients with neurogenic thoracic outlet syndrome. Each underwent an anterior scalene muscle injection with 2–5 cc 0.25 % bupivacaine under US guidance. All patients had pain relief; only 6 of the 12 patients developed dermatomal numbness, and there was no relationship between numbness and length of relief. They concluded that the relief of NTOS with anterior scalene injections is not related to brachial plexus blockade.

Fig. 33.15 3D image of the anterior scalene muscle injection of contrast (Image courtesy of Andrea Trescot, MD)

Neurostimulation

Neuromodulation for the brachial plexus and TOC is still in its infancy. Dr. Christ DeClerck described placement of a peripheral nerve stimulator onto the brachial plexus under US (personal communication) (Fig. 33.18).

Neurolytic/Surgical Technique

The use of botulinum toxin for chemodenervation of the anterior scalene is becoming more commonplace as a treatment for neurogenic thoracic outlet syndrome [21]. When this treatment is ineffective or only temporarily effective, one can consider surgical intervention. There are multiple surgical approaches with the common theme of trying to open up space within the thoracic outlet. Common approaches

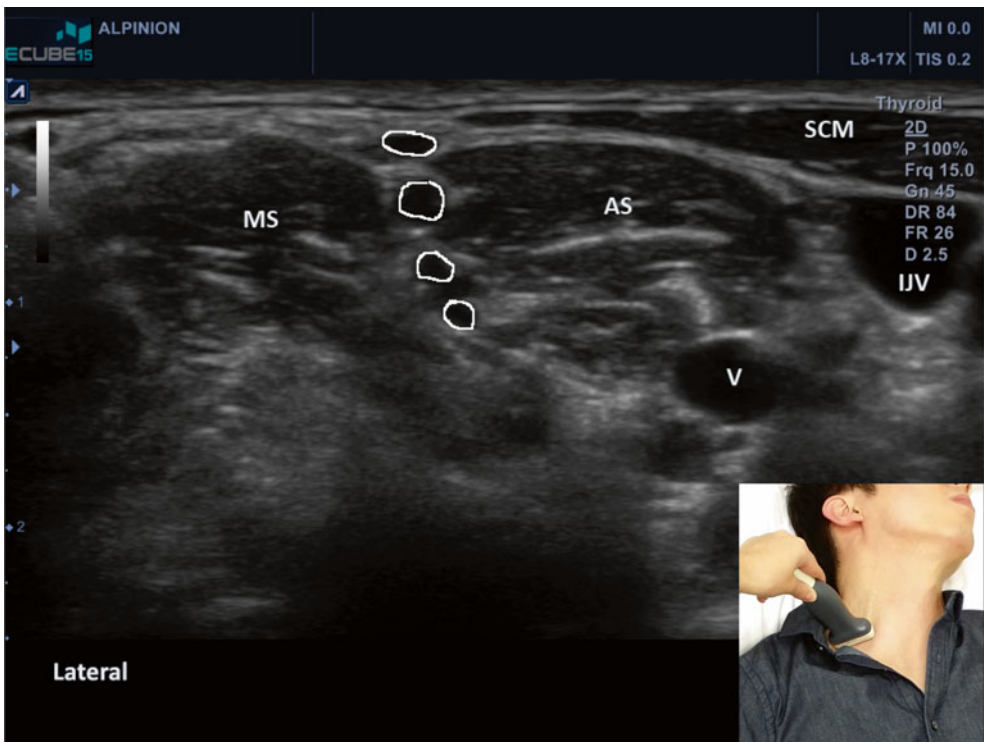

Fig. 33.16 Ultrasound image of the thoracic outlet. Horizontal ultrasound probe with the head turned to the contralateral side. *SCM* sternocleidomastoid muscle, *AS* anterior scalene muscle, *MS* middle scalene muscle, *circles* brachial plexus. *IJV* internal jugular vein, *V* vein (Image courtesy of Virtaj Singh, MD)

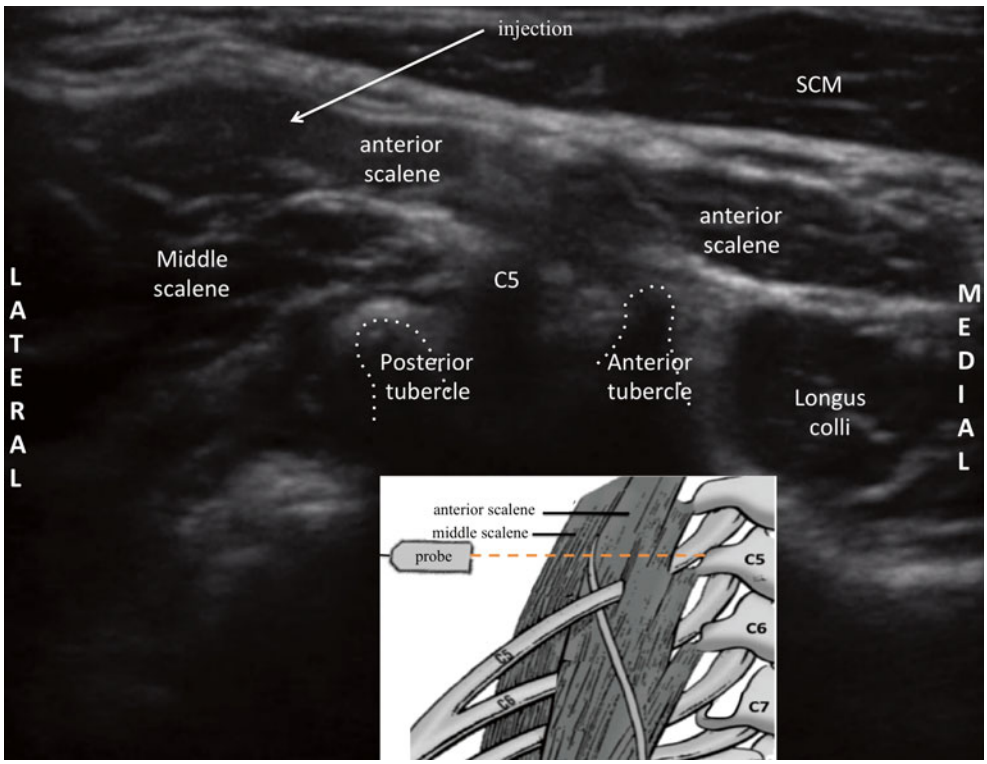

Fig. 33.17 Simulated injection of the anterior scalene muscle under ultrasound guidance. *SCM* sternocleidomastoid muscle (Image courtesy of Thiago Nouer Frederico, MD)

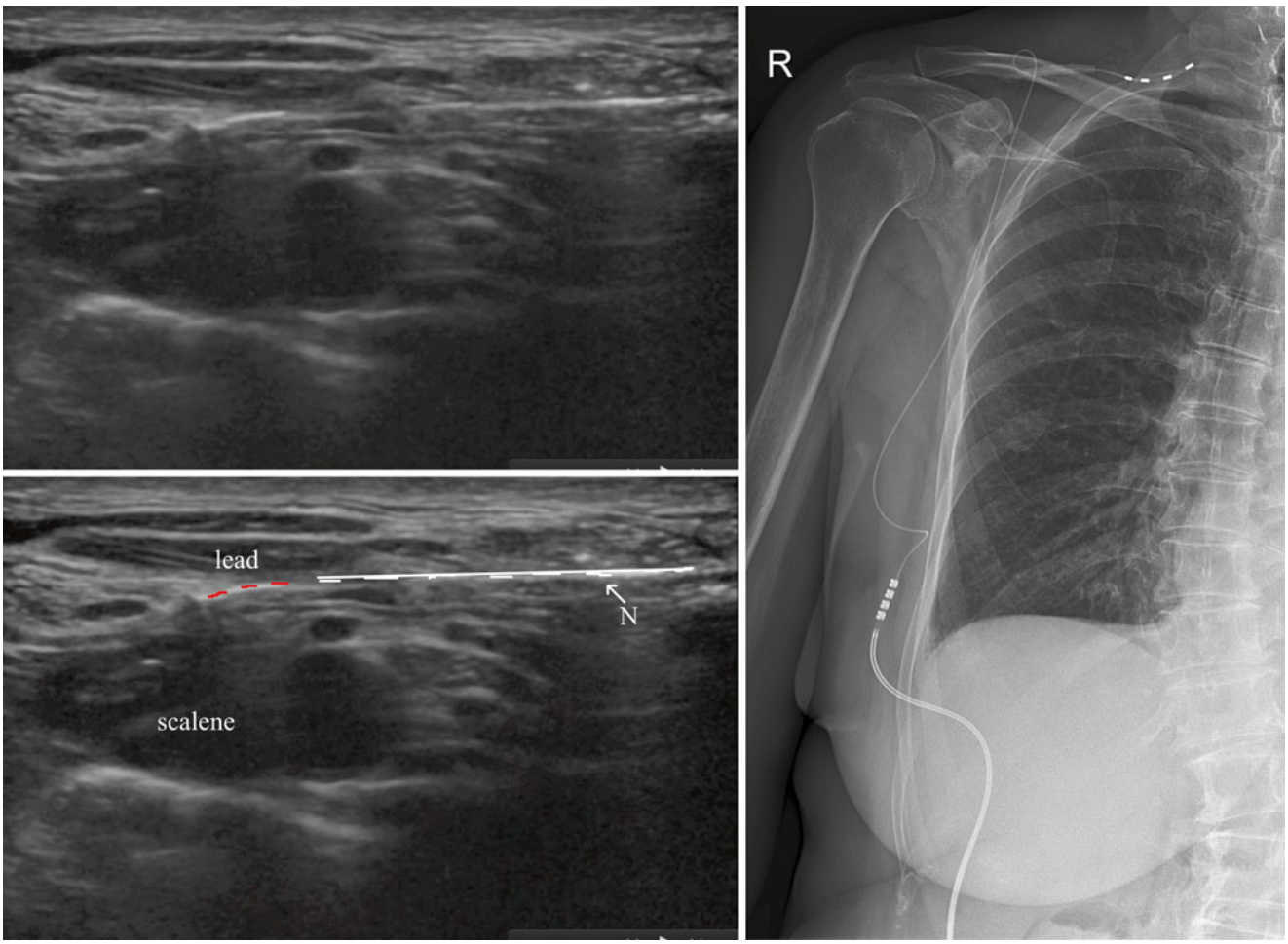

Fig. 33.18 Peripheral nerve stimulation of the brachial plexus placed under ultrasound guidance (Image courtesy of Christ DeClerck, MD)

include resecting the scalene muscle(s), performing tenotomy of the pectoralis minor, and/or resection of the first rib.

Complications

With regard to scalene chemodenervation, risks include bleeding, infection, neural injury, and pneumothorax, among others. Further, the use of botulinum toxin has additional risks, such as weakness and swallowing difficulties. With regard to surgical intervention, NTOS carries up to a 30 % (others suggest closer to 5 %) complication rate [22]. Complications are similar to those discussed above, including bleeding, infection, and pneumothorax. Other risks include diaphragmatic hemiparalysis due to phrenic nerve injury.

Summary

The thoracic outlet entrapment syndrome results in a wide variety of symptoms and presentations, based on the site of entrapment. A high index of suspicion is necessary to avoid misdiagnosis.

References

1. Ferrante MA. The thoracic outlet syndromes. Muscle Nerve. 2012;45(6):780–95.
2. Machanic BI, Sanders RJ. Medial antebrachial cutaneous nerve measurements to diagnose neurogenic thoracic outlet syndrome. Ann Vasc Surg. 2008;22(2):248–54.
3. Sanders RJ, Hammond SL, Rao NM. Diagnosis of thoracic outlet syndrome. J Vasc Surg. 2007;46:601–4.
4. Sanders RJ, Rao NM. The forgotten pectoralis minor syndrome: 100 operations for pectoralis minor syndrome alone or accompanied by neurogenic thoracic outlet syndrome. Ann Vasc Surg. 2010;24(6):701–8.
5. Sanders RJ, Annest SJ, Goldson E. Neurogenic thoracic outlet and pectoralis minor syndromes in children. Vasc Endovasc Surg. 2013;47(5):335–41.
6. Ozoa G, Alves D, Fish DE. Thoracic outlet syndrome. Phys Med Rehabil Clin N Am. 2011;22(3):473–83, viii–ix.
7. Abdollahi K, Wood VE. Thoracic outlet syndrome. In: DeLee JC, Drez DJ, Miller MD, editors. DeLee and Drez's orthopaedic sports medicine, vol. 1. 3rd ed. Philadelphia: Saunders Elsevier; 2010. p. 1127–36.
8. Aufderheide TP. Peripheral arteriovascular disease. In: Marx JA, Hockberger RS, Walls RM, Adams J, Rosen P, editors. Rosen's emergency medicine: concepts and clinical practice. 7th ed. Philadelphia: Mosby/Elsevier; 2010. p. 1103–23.
9. McGann SA, Flores RH, Nashel DJ. Entrapment neuropathies and compartment syndromes. In: Hochberg MC, editor. Rheumatology. 5th ed. Philadelphia: Mosby/Elsevier; 2011. p. 783–96.
10. Lindgren K-A. Thoracic outlet syndrome. In: Frontera WR, Silver JK, editors. Essentials of physical medicine and rehabilitation. Philadelphia: Hanley & Belfus; 2002. p. 497–503.
11. Tsao BE, Ferrante MA, Wilbourn AJ, Shields RW. Electrodiagnostic features of true neurogenic thoracic outlet syndrome. Muscle Nerve. 2014;49(5):724–7.
12. Bollini CA, Wikinski JA. Anatomical review of the brachial plexus. Tech Reg Anesth Pain Manag. 2006;10:69–78.
13. Dubuisson A, Lamotte C, Foidart-Dessalle M, Nguyen Khac M, Racaru T, Scholtes F, et al. Post-traumatic thoracic outlet syndrome. Acta Neurochir (Wien). 2012;154(3):517–26.
14. Boezaart AP, Haller A, Laduzenski S, Koyyalamudi VB, Ihnatsenka B, Wright T. Neurogenic thoracic outlet syndrome: a case report and review of the literature. Int J Should Surg. 2010;4(2):27–35.
15. Dubuisson AS. The thoracic outlet syndrome. http://www.medschool.lsuhsc.edu/neurosurgery/nervecenter/TOS.html [Internet]. 2013 May 27, 2013 [cited 2013 May 27, 2013].
16. Sanders RJ. Recurrent neurogenic thoracic outlet syndrome stressing the importance of pectoralis minor syndrome. Vasc Endovasc Surg. 2011;45(1):33–8.
17. Lohman CM, Gilbert KK, Sobczak S, Brismee JM, James CR, Day M, et al. Cervical nerve root displacement and strain during upper limb neural tension testing: part 1: a minimally invasive assessment in unembalmed cadavers. Spine. 2015;40(11):793–800.
18. Jordan SE, Ahn SS, Gelabert HA. Differentiation of thoracic outlet syndrome from treatment-resistant cervical brachial pain syndromes: development and utilization of a questionnaire, clinical examination and ultrasound evaluation. Pain Physician. 2007;10(3):441–52.
19. Torriani M, Gupta R, Donahue DM. Sonographically guided anesthetic injection of anterior scalene muscle for investigation of neurogenic thoracic outlet syndrome. Skeletal Radiol. 2009;38(11):1083–7.
20. Benzon HT, Rodes ME, Chekka K, Malik K, Pearce WH. Scalene muscle injections for neurogenic thoracic outlet syndrome: case series. Pain Pract. 2012;12(1):66–70.
21. Foley JM, Finlayson H, Travlos A. A review of thoracic outlet syndrome and the possible role of botulinum toxin in the treatment of this syndrome. Toxins. 2012;4(11):1223–35.
22. Franklin GM, Fulton-Kehoe D, Bradley C, Smith-Weller T. Outcome of surgery for thoracic outlet syndrome in Washington state worker's compensation. Neurology. 2000;54(6):1252–7.

Suprascapular Nerve Entrapment: Upper Extremity

34

Christopher J. Burnett and Helen W. Karl

Introduction

The *suprascapular nerve* (SN) is a mixed sensory and motor nerve that originates from the upper trunk of the *brachial plexus* that can be a cause of shoulder pain and weakness. There are two primary sites for entrapment of the SN; the clinical presentation varies depending on the site of entrapment. Patients with proximal SN entrapment at the suprascapular notch primarily complain of poorly localized posterolateral shoulder pain and weakness. Entrapment at this location is discussed in Chapter 28. Diagnosis is made by injection at the suprascapular notch, using a peripheral nerve stimulator (PNS), fluoroscopy, ultrasound (US), or CT scan. Treatment includes cryoneuroablation, pulsed radiofrequency (PRF), surgery, or peripheral stimulation. Entrapment of the distal SN at the spinoglenoid notch causes much less pain and is the subject of this chapter.

Clinical Presentation (Table 34.1)

Signs and symptoms of SN entrapment depend on the location of nerve compression. Entrapment at the *suprascapular notch* results in significant shoulder pain due to compression of the deep sensory fibers innervating the *glenohumeral* and *acromioclavicular joints* [2, 10]. The pain is described as a dull ache in the posterolateral aspect of the shoulder and scapular regions (Fig. 34.1) that may radiate into the ipsilateral shoulder, arm, or neck (Fig. 34.2). This condition is seen primarily in athletes or people performing repetitive overhead motions (e.g., weight lifting, baseball, tennis, swimming, carpentry). It can be triggered by a traumatic or acute event, such as a lifting injury with the arm internally rotated (like carrying a heavy suitcase), but the onset is typically insidious, involving the dominant arm in patients (usually male) from 20 to 50 years of age. The patient may complain of shoulder weakness or fatigue, particularly in abduction and external rotation of the arm. Any forward movement of the scapula can elicit pain, including movements as simple as reaching across the chest. Patients can also develop a "*frozen shoulder*," (adhesive capsulitis), one cause of which is unwillingness to move the shoulder joint due to pain [1, 2, 10].

If the entrapment occurs more distally, at the *spinoglenoid notch*, the patient will have isolated atrophy and weakness of the infraspinatus muscle. In this situation, pain is largely absent because the deep sensory fibers to the shoulder joint exit proximally to this entrapment site [2, 3]. Liveson et al. [8] reviewed 13 reported cases of entrapment at the spinoglenoid notch, most of whom did not complain of pain or weakness, but rather came to medical attention because of insidious infraspinatus atrophy. They felt that the lack of weakness complaints was due to compensation of other muscles.

Brachial neuritis can also cause suprascapular neuropathy, though, in this case, weakness is not confined to the supraspinatus and infraspinatus muscles [2].

Anatomy (Table 34.2)

The suprascapular nerve originates from the fibers of the fifth and sixth cervical nerve roots, with frequent contributions (15–22 % of cases) [4] from the fourth cervical nerve root. It branches from the upper trunk of the brachial plexus and runs across the posterior cervical triangle, then under the *coracoclavicular ligament* and the *trapezius muscle* [4], and then posteriorly to pass through the *suprascapular notch* (also known as the *supraspinatus notch* or *incisura scapulae*)

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Table 34.1 Occupation/exercise/trauma history relevant to suprascapular nerve entrapment

Repetitive shoulder movements, especially external rotation and abduction, which usually cause entrapment at the spinoglenoid fossa [1]	Sports: baseball [2, 3]; weightlifting [2]; swimming; dancing [2]; tennis [4]; volleyball [2, 3]
	Carpentry [5]
Surgical positioning	Knee-chest with the scapula protracted [2]
Carrying heavy objects [3]	Meat packers [3]
	Newsreel cameramen [3]
	Roofers [6]
Stretch and direct trauma	Fracture of the scapula, humerus, clavicle [3]
	Anterior shoulder dislocation [3]
	Shoulder surgery [7]
	Skeet shooting [8]
Space-occupying lesions are often associated with a trauma history	Ganglion cyst [3, 5]; lipoma [2]; hematoma [3]
Other	Tumor [3]
	Insertion of the spinoglenoid ligament onto the scapulohumeral joint, causing tension on the ligament (trapping the nerve) with arm movements [9]

Fig. 34.1 Patient pain pattern with distal suprascapular entrapment (Image courtesy of Andrea Trescot, MD)

(Fig. 34.3). Anatomical [11] and radiologic [4] studies show that the suprascapular notch can have a wide variety of shapes. The *transverse scapular ligament* encloses the superior portion of the suprascapular notch. This ligament is of variable thickness and can become partially or completely

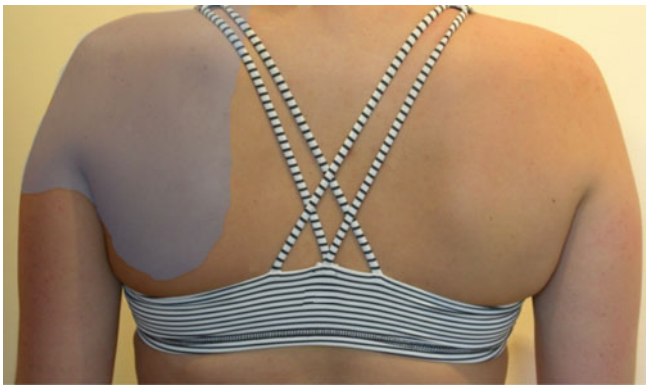

Fig. 34.2 Pain pattern associated with suprascapular nerve entrapment (Image courtesy of Andrea Trescot, MD)

Table 34.2 Suprascapular nerve anatomy

Origin	A direct branch of the upper trunk of the brachial plexus (C4–C6)
General route	Crosses the posterior neck, under the trapezius, toward the suprascapular foramen, through the suprascapular notch/foramen, and then through the spinoglenoid notch
Sensory distribution [7]	Glenohumeral joint (shoulder joint)
	Acromioclavicular joint (shoulder joint)
	Subacromial bursa
	A patch of skin on the lateral upper shoulder
Motor innervation: 2 of the 4 rotator cuff muscles	Supraspinatus: abducts the humerus, especially the first 20–30°
	Infraspinatus: externally rotates the humerus. The posterior deltoid and teres minor muscles also perform this function, so isolated infraspinatus muscle dysfunction may be asymptomatic [4]

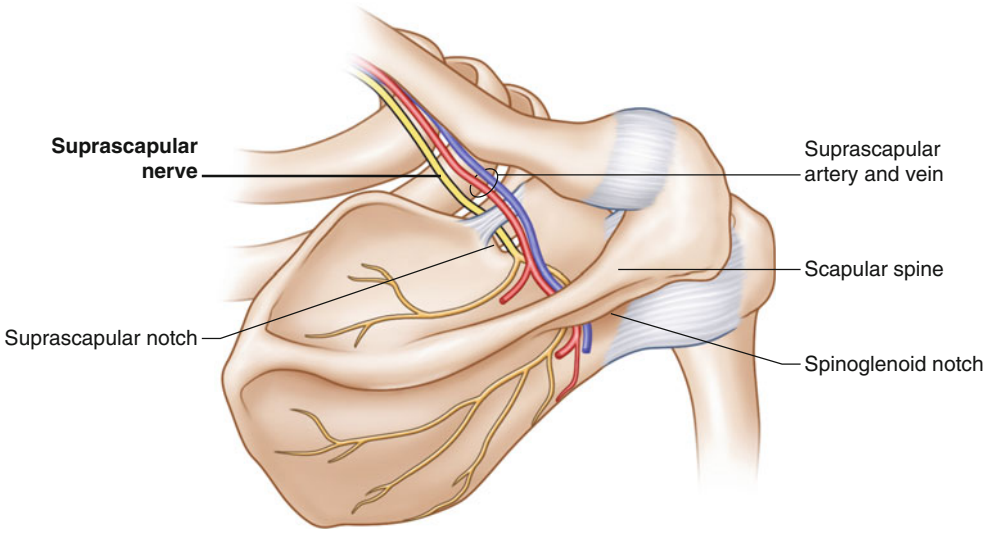

Fig. 34.3 Anatomy of the suprascapular nerve. Note the suprascapular nerve (in *yellow*) as it passes underneath the transverse scapular ligament in the suprascapular notch. Note also that the suprascapular artery and vein lie above the transverse scapular ligament. Sensory branches from the shoulder joint not shown (Image courtesy of Springer)

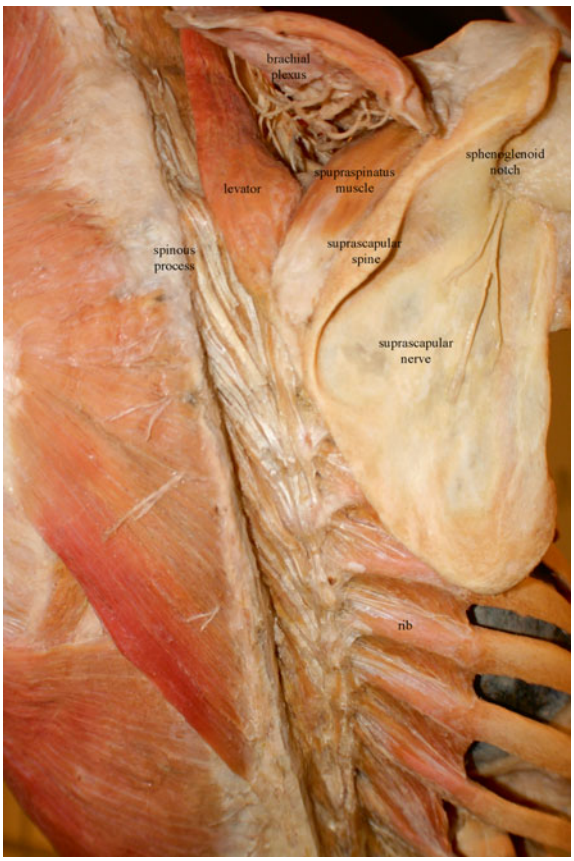

Fig. 34.4 Suprascapular anatomy, modified from an image from *Bodies*, *The Exhibition*, with permission (Image courtesy of Andrea Trescot, MD)

ossified, which can contribute to entrapment neuropathy. The position of the nerve in the notch is variable, making injection using anatomic landmarks alone difficult. Both the *suprascapular artery* and the *suprascapular vein* usually cross the upper edge of the scapula above the suprascapular ligament (Fig. 34.3) [1, 3, 10, 12], though a recurrent branch of the suprascapular artery may accompany the nerve through the notch [4].

After passing through the suprascapular notch, the nerve travels underneath the *supraspinatus muscle* and provides its motor innervation. The SN, in combination with the *lateral pectoral nerve* [7], also supplies sensory branches to the shoulder capsule, the *glenohumeral joint*, the *acromioclavicular (AC) joint*, the *coracoclavicular ligament*, and the *subacromial bursa* [4]. It has been estimated that the suprascapular nerve provides up to 70 % of the sensory innervation of the shoulder joint [10]. After innervating the supraspinatus muscle, the suprascapular nerve then travels inferolaterally toward the rim of the *glenoid fossa*; it makes a tight turn around the lateral edge of the scapular spine, beneath the *spinoglenoid ligament* (also known as the *inferior transverse scapular ligament*) [3], which creates a fibro-osseous tunnel called the *spinoglenoid notch* (or *infraspinatus notch*), through which the suprascapular nerve courses toward the infraspinatus fossa (Fig. 34.4). Here, the nerve provides motor innervation to the *infraspinatus muscle* [2, 3, 10, 13]. The deep sensory fibers to the shoulder joint exit proximal to the spinoglenoid ligament [2, 3]. There is anatomic and clinical evidence for a small cutaneous branch, which in some people provides sensory innervation to a patch of the skin on the lateral aspect of the upper shoulder [6, 14].

Entrapment

The anatomical course of the SN presents three distinct potential sites of entrapment: cervical origin, suprascapular notch, and spinoglenoid notch [10]. The most common site of its entrapment is at the suprascapular notch (see Chapt 28). Kopell and Thompson [15] first described entrapment of

the suprascapular nerve at the suprascapular notch in 1959. The nerve has little freedom of movement there, while the shoulder and scapula are extremely mobile.

Spinoglenoid notch entrapment, first described in 1981 [16], also occurs because the notch is a relatively fixed tunnel through which the nerve passes, limiting its mobility and predisposing it to entrapment [1, 2, 8, 10, 13]. Liveson and colleagues [8] suggest that the spinoglenoid entrapment is more common than the entrapment at the suprascapular notch, and it needs to be recognized to avoid treatment of the wrong condition.

Mass lesions can also contribute to SN entrapment, specifically a lipoma or ganglion cyst near the nerve. The latter is often associated with labral tears, and has a tendency to form near the spinoglenoid notch [17].

Physical Examination

Begin the physical examination of the patient with suspected SN entrapment by inspecting for atrophy of the supraspinatus or infraspinatus muscles. Atrophy is most easily visualized in the infraspinatus muscle, because the trapezius muscle can obscure the supraspinatus (Fig. 34.5). The clinician should evaluate the active range of motion of the shoulder, comparing it to the contralateral side and looking specifically for limitation of abduction in the plane of the scapula or limitation of external rotation with the arm held in 90° of abduction. Specific provocative tests may reveal supraspinatus and/or infraspinatus weakness (see below) [18].

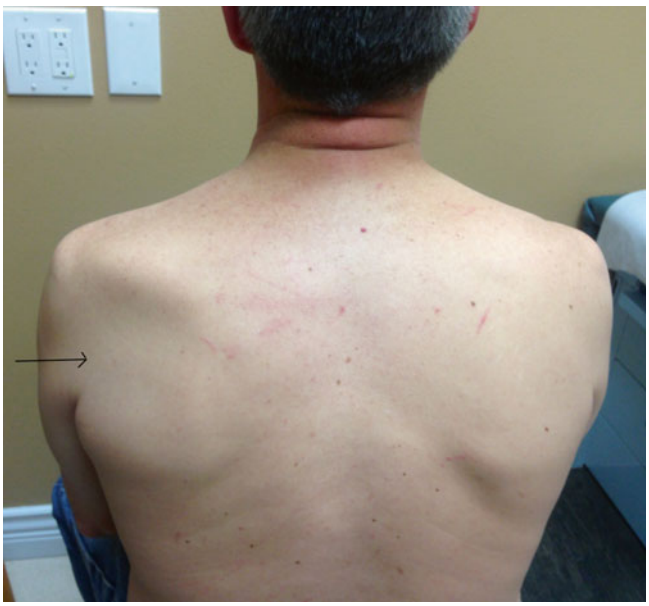

Fig. 34.5 *Arrow* shows left infraspinatus atrophy (Image courtesy of Andrea Trescot, MD)

The clinician should then palpate the suprascapular notch to elicit pain. The notch can be found by placing a hand on the patient's affected shoulder with the fingertips on the clavicle. The clinician then presses their thumb along the distal third of the scapular spine ("the Vulcan death grip") (Video 34.1) (Fig. 34.6). The examiner should then move their thumb inferiorly and laterally to palpate the spinoglenoid notch and evaluate for potential entrapment at that site [1, 2, 10, 13, 17]. Weakness in the initial 20–30° of arm abduction and forearm external rotation in comparison to the non-affected side may be noted. The *cross-body adduction test* described by Kopell and Thompson [15] (Fig. 34.7), which is performed by having the patient elevate the arm to 90° and forcibly adduct the arm across their chest, can tighten the spinoglenoid ligament and compress the nerve to reproduce the patient's symptoms.

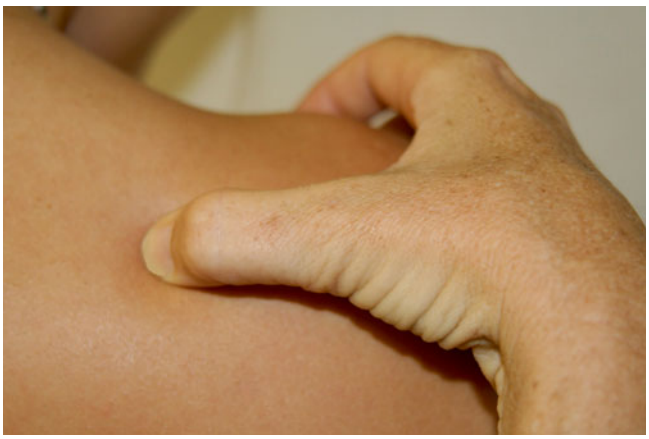

Fig. 34.6 Palpation of the suprascapular notch region to elicit pain (Image courtesy of Andrea Trescot, MD)

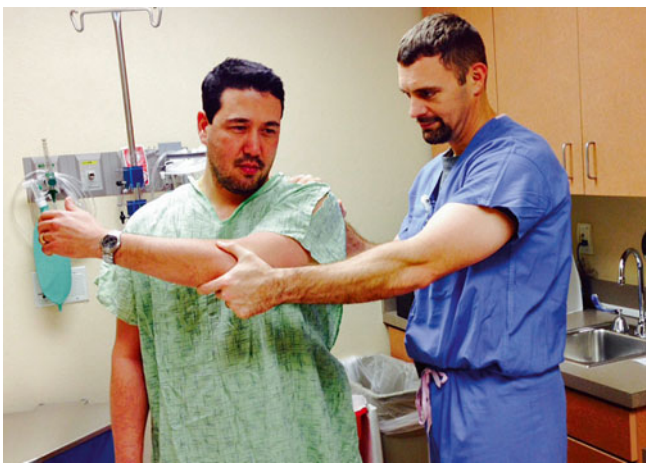

Fig. 34.7 Cross-body adduction test (Image courtesy of Christopher Burnett, MD)

Differential Diagnosis (Table 34.3)

Diagnosis may be aided by electrodiagnostic studies, magnetic resonance imaging, or high-resolution ultrasonography. X-rays may show an unusual suprascapular notch. EMG/NCV can show denervation changes, such as an increase in distal motor latency and signs of denervation (increased spontaneous activity, positive sharp waves, fibrillation, polyphasic activity, decreased amplitude evoked potentials, and single-unit recruitment of normal motor unit potentials) [4]. EMG evaluation of the supraspinatus and infraspinatus muscles can distinguish between entrapment sites: if the supraspinatus is normal and the infraspinatus denervated, the lesion must be at the spinoglenoid notch [8]. MRI (Fig. 34.8) may show infraspinatus atrophy (Fig. 34.9), tumors, or cysts.

Table 34.4 lists some of the diagnostic tests for distal SN entrapment.

Table 34.3 Relationship between muscle pathology and nerve entrapment

Denervated muscle	Suspected nerve entrapment
Supraspinatus and infraspinatus muscle	Suprascapular nerve at suprascapular notch
Trapezius muscle	Spinal accessory nerve (Chap. 27)
Isolated infraspinatus muscle	Suprascapular nerve at spinoglenoid (Chap. 28)
Serratus anterior muscle	Long thoracic nerve (Chap. 30)
Teres minor muscle	Axillary nerve at quadrilateral space (Chap. 31)
Rhomboid and/or levator muscle	Dorsal scapular nerve at scalene muscle (Chap. 32)

Identification and Treatment of Contributing Factors

A common contributing factor of SN entrapment arises from overuse of the muscles of the rotator cuff. Because this nerve supplies the muscles that allow for the abduction and external rotation of the arm, overuse of these muscles, especially the supraspinatus, can lead to muscle spasms. These spasms can potentially trap the suprascapular nerve as it enters the scapular notch.

Range of motion and stretching exercises (including supine internal rotation and axial extension) can help to relieve the entrapment; strengthening exercises can include scapular depression/adduction and isomeric external rotation.

Injection Techniques

Landmark-Guided Technique

There are multiple approaches for landmark-guided injection of the suprascapular nerve. We will describe two different approaches, one with and one without the use of a peripheral nerve stimulator. To perform the first landmark-guided technique, the patient is placed in a seated or prone position with their arms to their sides. The clinician begins by inserting a 25-gauge, 2-in. needle at the distal third of the scapula and directing it vertically down to the scapular spine. The needle is then advanced anteriorly and superiorly, maintaining contact with the scapula until it is felt to advance into the scapular notch (Fig. 34.10). After negative aspiration for the blood or air, the planned injectate can be administered. Pneumothorax is a significant clinical concern with this technique, as the cupola of the lung lies in close proximity to the nerve at this location [23].

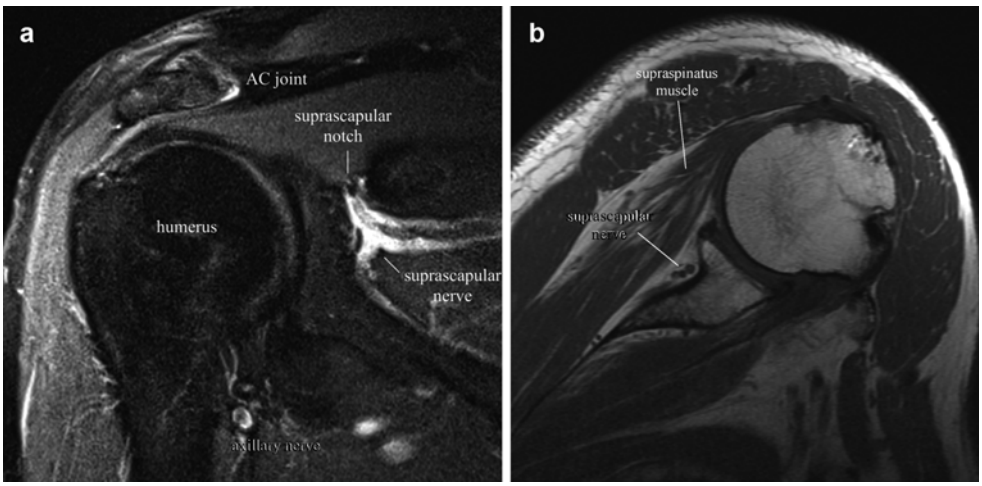

Fig. 34.8 MRI anatomy of the suprascapular notch: (**a**) T2-weighted coronal view; (**b**) T1-weighted axial view (Image courtesy of Andrea Trescot, MD)

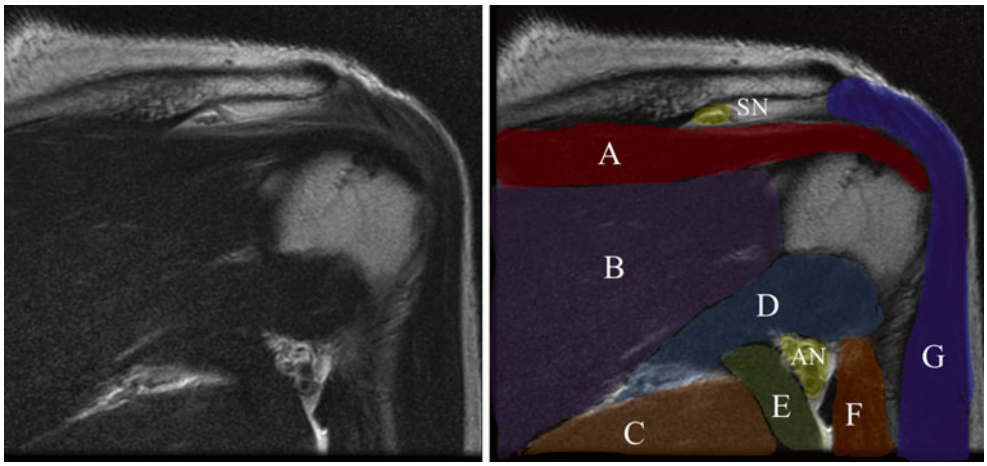

Fig. 34.9 Labeled MRI of the shoulder. *SN* suprascapular nerve, *AN* axillary nerve and axillary vessels, *A* supraspinatus muscle, *B* infraspinatus muscle, *C* teres major, *D* teres minor, *E* long head of the triceps muscle, *F* short head of the triceps, *G* deltoid muscle (Image courtesy of Andrea Trescot, MD)

Table 34.4 Diagnostic tests for distal suprascapular nerve entrapment

	Potential distinguishing features
Physical exam	Atrophy of infraspinatus only; weakness abduction/external rotation; cross-body adduction test
Ultrasound	May show edema of the infraspinatus muscle
Electrodiagnostic studies	Needle EMG should show axonal loss limited to SN distribution. Motor NCSs may show reduced amplitude
MRI	Edema, atrophy, or fatty infiltration of infraspinatus muscle

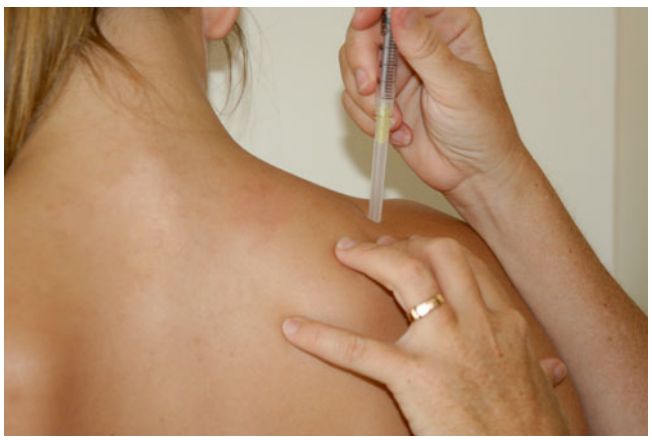

Fig. 34.10 Vertical approach for the landmark-guided injection of the suprascapular nerve (Image courtesy of Andrea Trescot, MD)

The second landmark-guided technique uses a horizontal instead of vertical needle position. In this approach, the patient is positioned in the seated or prone position with their arms relaxed at their sides (Video 34.2). The distal third of the scapular spine is palpated, and a 25-gauge, 2-in. needle is advanced just above the scapular spine, perpendicular to the skin (Fig. 34.11). This technique is easily modified to add a peripheral nerve stimulator (Fig. 34.12). Initial target needle position is the posterior wall of the lateral border of the scapular spine, parallel to the floor and lateral to the scapular notch. The needle is advanced medially in the plane of the scapular spine with the use of continuous motor stimulation until the needle tip is in proximity to the nerve, as evidenced by external rotation of the shoulder, indicating contraction of the infraspinatus and supraspinatus muscles. After negative aspiration for the blood or air, the planned injectate can be administered. By utilizing the bone as a backdrop, the risk of pneumothorax is negligible. Having the patient place their hand on the opposite shoulder further decreases the risk of pneumothorax by moving the scapula away from the chest wall and underlying pleura [17].

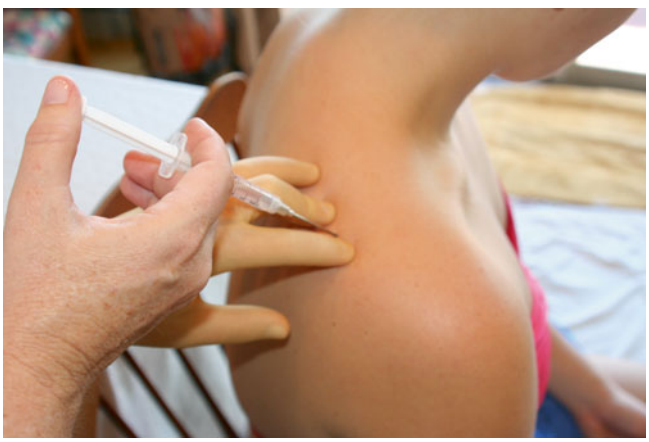

Fig. 34.11 Horizontal approach for landmark-guided injection of the suprascapular nerve (Image courtesy of Andrea Trescot, MD)

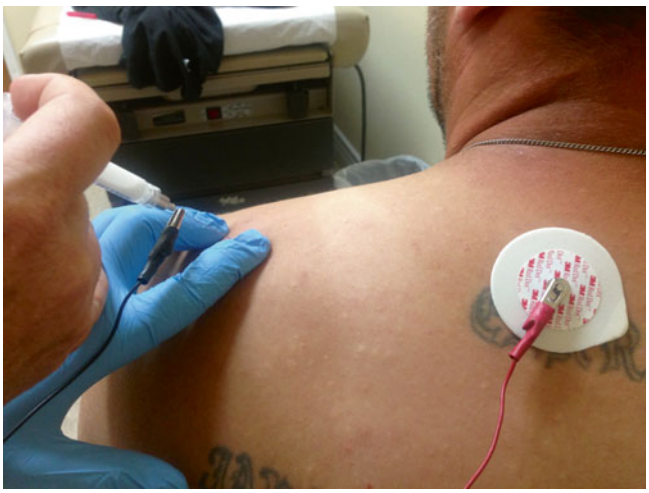

Fig. 34.12 Horizontal suprascapular landmark-guided injection with peripheral nerve stimulator (Image courtesy of Andrea Trescot, MD)

Fluoroscopic-Guided Injection Technique

Using the fluoroscopic-guided technique, the patient is placed prone on the fluoroscopy table, with the arms at the sides and the head turned to the contralateral side. Initially, straight AP fluoroscopic imaging is used to identify the T2 and T3 levels. Cephalocaudal tilt of the fluoroscope will help optimize the view of the suprascapular notch (Fig. 34.13). The skin and subcutaneous tissue at the inferior aspect of the suprascapular notch are anesthetized, and a 22- or 25-gauge, 2-in. needle is advanced toward this target in a coaxial fluoroscopic view until contact is made with the scapula. The needle is then withdrawn slightly and advanced in the cephalad direction no more than 1 cm toward the suprascapular notch. A paresthesia may be encountered, either from direct needle contact with the nerve or as a result of muscle spasm compressing the nerve. Many advocate the use of a nerve stimulator for improved safety and efficacy. Careful aspiration should be performed for the blood or air. Contrast can be injected to further verify needle placement, and the planned injectate can be administered [12].

Ultrasonography (US): Guided Technique

Harmon and Hearty first described US injections of the SN in 2007 [24]. The patient is placed either in a seated position with their ipsilateral hand resting on their contralateral shoulder or supine with arms by their side (Fig. 34.14). The initial US scan is performed in the sagittal plane at the

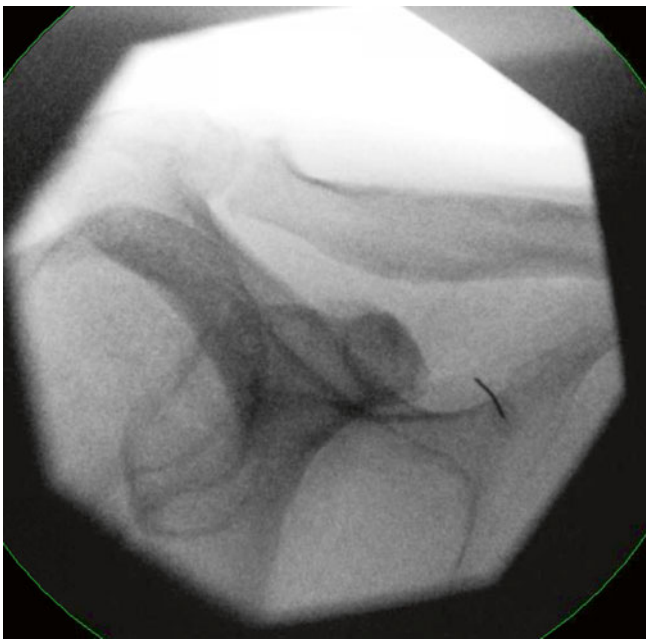

Fig. 34.13 Fluoroscopic image of suprascapular notch located just above the needle (Image courtesy of Christopher Burnett, MD)

superior border of the scapula to identify the pleura at a depth of approximately 4 cm. US scanning can then proceed with the same transducer orientation moving laterally, parallel to the scapular spine. The transducer is then moved cephalad to visualize the suprascapular fossa. The transducer is moved in small increments laterally and/or superiorly until the suprascapular notch is visualized. The suprascapular nerve will appear as a round, hyperechoic structure beneath the transverse scapular ligament (Fig. 34.15). A 22-guage, 2-in. needle is inserted in the longitudinal axis of the ultrasound beam and advanced under full US visualization until the needle tip is in proximity to the suprascapular nerve. After negative aspiration for the blood or air, the planned injectate can be administered. Real-time imaging can be used to watch local anesthetic spread around the nerve [24].

In 2012, Siegenthaler et al. [25] described an ultrasound technique for the SN targeting the nerve in the supraclavicular region underneath the omohyoid muscle. They scanned 60 volunteers and were able to identify the nerve in 81 %.

CT-Guided Injection

Shanahan and colleagues [23] randomized 77 shoulders to either anatomy-guided (blind) suprascapular procedures (using 11 cc of local anesthetic and steroid) or CT-guided

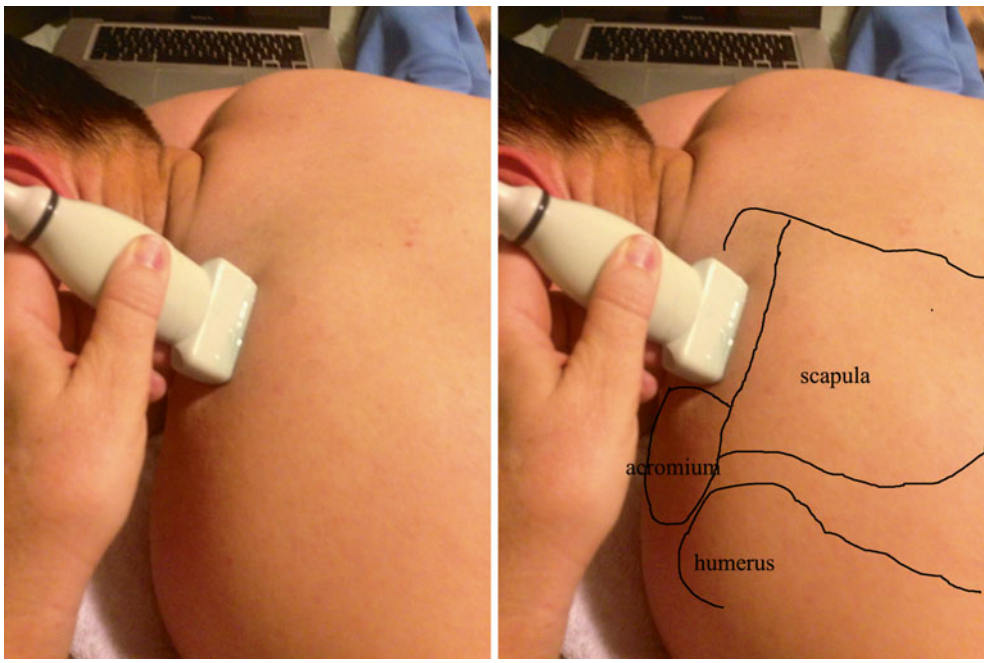

Fig. 34.14 Composite view of the ultrasound probe placement for visualization of the suprascapular nerve (Image courtesy of Andrea Trescot, MD)

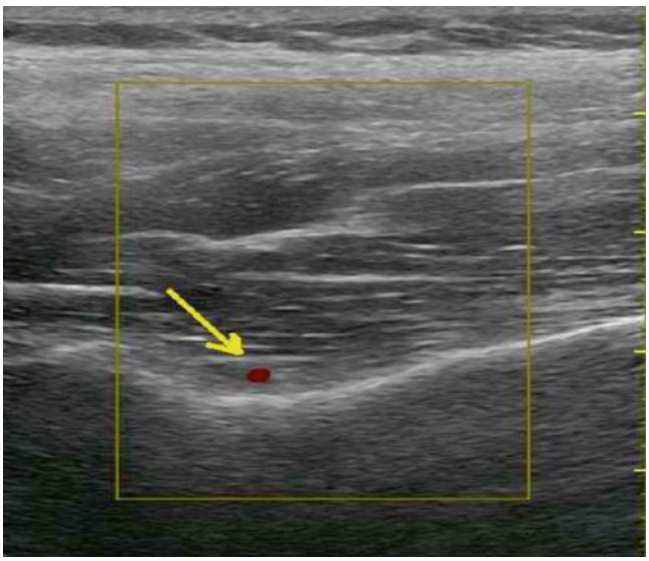

Fig. 34.15 Ultrasound image of suprascapular notch; *yellow arrow* shows the transverse scapular ligament (From Elahi and Reddy [28], with permission from the American Society of Interventional Pain Physicians)

suprascapular injections (using 3 cc of local anesthetic and steroid) and found similar efficacy; neither group had any complications.

Neurolytic Technique

Cryoneuroablation

Trescot [26] described a cryoneuroablation technique for the suprascapular nerve. The patient is positioned seated (as for landmark or US-directed procedures) (Fig. 34.16) or prone

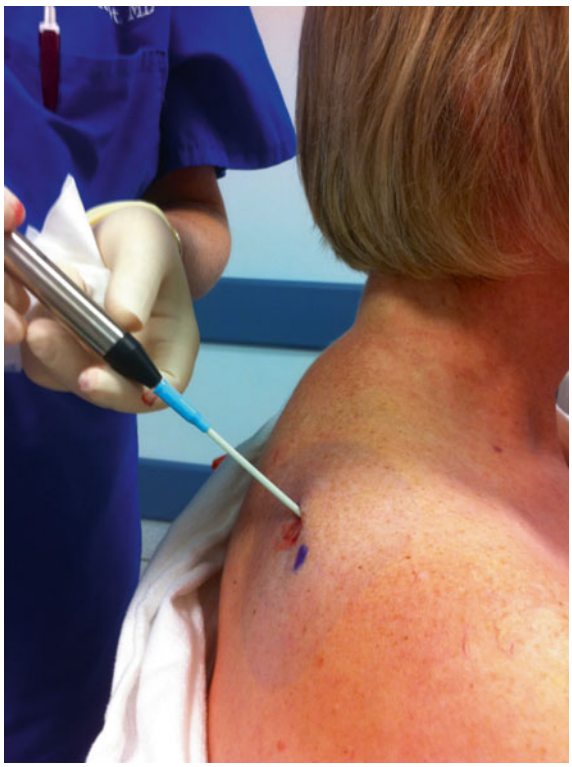

Fig. 34.16 Cryoneuroablation of the suprascapular nerve (Image courtesy of Andrea Trescot, MD)

(for fluoroscopic directed procedures). A 12-guage introducer is advanced into the suprascapular notch, parallel to the direction of the nerve and perpendicular to the scapula. Consider using fluoroscopy if the superior border of the scapula is not easily palpated. The 2.0-mm probe is then advanced through the catheter, and the nerve is identified using sensory or motor stimulation, this being one of the few mostly motor nerves amenable to cryoneuroablation.

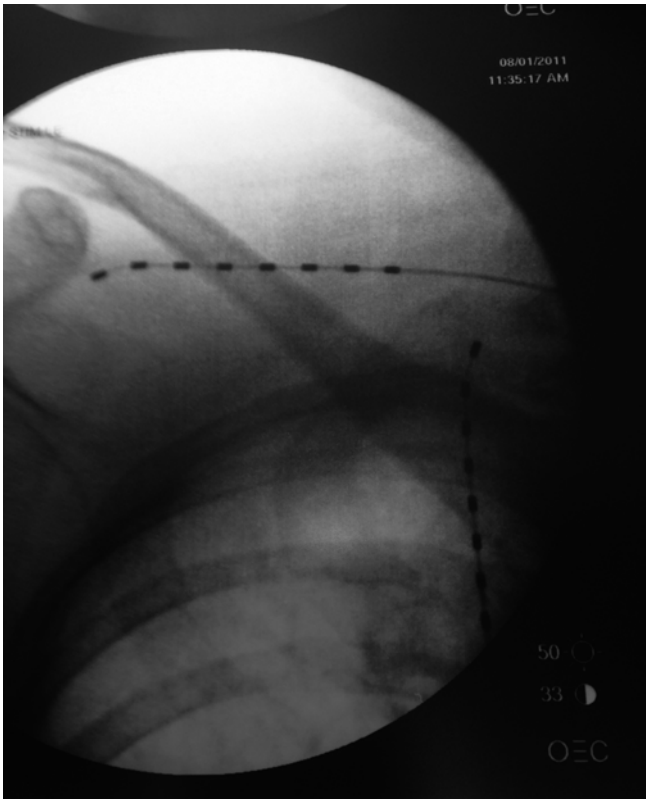

Fig. 34.17 Peripheral stimulation of the suprascapular nerve (Image courtesy of George Arcos, DO)

Radiofrequency Lesioning

The use of either pulsed radiofrequency ablation or thermal radiofrequency ablation of the suprascapular nerve has been described and found to be a safe and effective way to obtain longer-term pain resolution [12, 27].

Neurostimulation

Elahi and Reddy [28] described a patient with persistent intractable shoulder pain after seven shoulder surgeries; they placed a peripheral nerve stimulator on the suprascapular nerve under US guidance (Figs. 34.17 and 34.18) with excellent relief.

Surgical Technique

Surgical decompression of the suprascapular nerve may be considered in patients who have significant muscle wasting, weakness, or intractable pain and who have failed conservative therapy. Traditionally, an open surgical approach was used to decompress the suprascapular or spinoglenoid notch. More recently, arthroscopic surgical approaches have been described for suprascapular nerve decompression [2, 19, 29]. In 2010, Boykin et al. [30] published a review of the surgical techniques for SN entrapment.

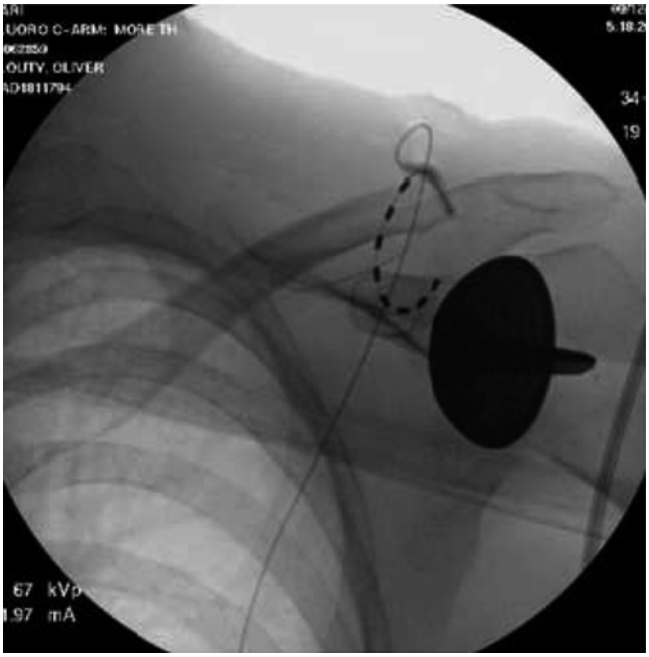

Fig. 34.18 Fluoroscopic imaging of a peripheral suprascapular stimulation lead placed under ultrasound guidance (From Elahi and Reddy [28], with permission from the American Society of Interventional Pain Physicians)

Complications

Complications for suprascapular nerve block include pneumothorax, direct needle trauma to the neurovascular bundle, intravascular injection, or infection [23].

Summary

Suprascapular entrapment should be considered in patients with poorly localized shoulder and upper arm pain and weakness, especially in those patients with scapular atrophy. Knowledge of the condition will help to answer many of the causes of shoulder, neck, and arm pain.

References

1. Linda DD, Harish S, Stewart BG, Finlay K, Parasu N, Rebello RP. Multimodality imaging of peripheral neuropathies of the upper limb and brachial plexus. Radiographics. 2010;30(5):1373–400.
2. Shapiro BE, Preston DC. Entrapment and compressive neuropathies. Med Clin North Am. 2009;93(2):285–315. vii.
3. Kim DH, Murovic JA, Tiel RL, Kline DG. Management and outcomes of 42 surgical suprascapular nerve injuries and entrapments. Neurosurgery. 2005;57(1):120–7. discussion -7.
4. Blum A, Lecocq S, Louis M, Wassel J, Moisei A, Teixeira P. The nerves around the shoulder. Eur J Radiol. 2013;82(1):2–16.
5. Iannotti JP, Ramsey ML. Arthroscopic decompression of a ganglion cyst causing suprascapular nerve compression. Arthroscopy. 1996;12(6):739–45.

6. Harbaugh KS, Swenson R, Saunders RL. Shoulder numbness in a patient with suprascapular nerve entrapment syndrome: cutaneous branch of the suprascapular nerve: case report. Neurosurgery. 2000;47(6):1452–5. discussion 5–6.
7. Ebraheim NA, Whitehead JL, Alla SR, Moral MZ, Castillo S, McCollough AL, et al. The suprascapular nerve and its articular branch to the acromioclavicular joint: an anatomic study. J Shoulder Elbow Surg. 2011;20(2):e13–7.
8. Liveson JA, Bronson MJ, Pollack MA. Suprascapular nerve lesions at the spinoglenoid notch: report of three cases and review of the literature. J Neurol Neurosurg Psychiatry. 1991;54(3):241–3.
9. Aiello I, Serra G, Traina GC, Tugnoli V. Entrapment of the suprascapular nerve at the spinoglenoid notch. Ann Neurol. 1982;12(3):314–6.
10. Budzik JF, Wavreille G, Pansini V, Moraux A, Demondion X, Cotten A. Entrapment neuropathies of the shoulder. Magn Reson Imaging Clin N Am. 2012;20(2):373–91. xii.
11. Rengachary SS, Burr D, Lucas S, Brackett CE. Suprascapular entrapment neuropathy: a clinical, anatomical, and comparative study. Part 3: comparative study. Neurosurgery. 1979;5(4):452–5.
12. Simopoulos TT, Nagda J, Aner MM. Percutaneous radiofrequency lesioning of the suprascapular nerve for the management of chronic shoulder pain: a case series. J Pain Res. 2012;5:91–7.
13. Dong Q, Jacobson JA, Jamadar DA, Gandikota G, Brandon C, Morag Y, et al. Entrapment neuropathies in the upper and lower limbs: anatomy and MRI features. Radiol Res Pract. 2012; 2012:230679.
14. Safran MR. Nerve injury about the shoulder in athletes, part 1: suprascapular nerve and axillary nerve. Am J Sports Med. 2004;32(3):803–19.
15. Kopell HP, Thompson WA. Pain and the frozen shoulder. Surg Gynecol Obstet. 1959;109(1):92–6.
16. Ganzhorn RW, Hocker JT, Horowitz M, Switzer HE. Suprascapular-nerve entrapment. J Bone Joint Surg Am. 1981;63(3):492–4.
17. Burbank KM, Stevenson JH, Czarnecki GR, Dorfman J. Chronic shoulder pain: part I. Evaluation and diagnosis. Am Fam Physician. 2008;77(4):453–60.
18. Neal S, Fields KB. Peripheral nerve entrapment and injury in the upper extremity. Am Fam Physician. 2010;81(2):147–55.
19. Toussaint CP, Zager EL. What's new in common upper extremity entrapment neuropathies. Neurosurg Clin N Am. 2008;19(4): 573–81. vi.
20. Carroll KW, Helms CA, Otte MT, Moellken SM, Fritz R. Enlarged spinoglenoid notch veins causing suprascapular nerve compression. Skeletal Radiol. 2003;32(2):72–7.
21. Burbank KM, Stevenson JH, Czarnecki GR, Dorfman J. Chronic shoulder pain: part II. Treatment. Am Fam Physician. 2008;77(4):493–7.
22. Hill LJ, Jelsing EJ, Terry MJ, Strommen JA. Evaluation, treatment, and outcomes of suprascapular neuropathy: a 5-year review. PM R. 2014;6(9):774–80.
23. Shanahan EM, Smith MD, Wetherall M, Lott CW, Slavotinek J, FitzGerald O, et al. Suprascapular nerve block in chronic shoulder pain: are the radiologists better? Ann Rheum Dis. 2004; 63(9):1035–40.
24. Harmon D, Hearty C. Ultrasound-guided suprascapular nerve block technique. Pain Physician. 2007;10(6):743–6.
25. Siegenthaler A, Moriggl B, Mlekusch S, Schliessbach J, Haug M, Curatolo M, et al. Ultrasound-guided suprascapular nerve block, description of a novel supraclavicular approach. Reg Anesth Pain Med. 2012;37(3):325–8.
26. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
27. Gofeld M, Restrepo-Garces CE, Theodore BR, Faclier G. Pulsed radiofrequency of suprascapular nerve for chronic shoulder pain: a randomized double-blind active placebo-controlled study. Pain Pract. 2013;13(2):96–103.
28. Elahi F, Reddy CG. Neuromodulation of the suprascapular nerve. Pain Physician. 2014;17(6):E769–73.
29. Lafosse L, Tomasi A, Corbett S, Baier G, Willems K, Gobezie R. Arthroscopic release of suprascapular nerve entrapment at the suprascapular notch: technique and preliminary results. Arthroscopy. 2007;23(1):34–42.
30. Boykin RE, Friedman DJ, Higgins LD, Warner JJ. Suprascapular neuropathy. J Bone Joint Surg Am. 2010;92(13):2348–64.

Deep Branch of the Radial Nerve Entrapment

35

Richard E. Seroussi, Virtaj Singh, and Helen W. Karl

Introduction

Entrapment of the *deep branch of the radial nerve* (DBRN) results in two distinct clinical syndromes. *Posterior interosseous nerve syndrome (PINS)* is a well-defined but rare nerve palsy involving some of the wrist extensors and all of the finger extensors [1, 2]. By contrast, *radial tunnel syndrome (RTS)* is a controversial, painful repetitive stress type of injury without "hard" neurologic symptoms [3–7].

Of note, there is a fair amount of ambiguity in the literature as to what constitutes the radial tunnel, as well as the definitions of the DBRN and the *posterior interosseous nerve (PIN)*. Further confusion stems from RTS coexisting with other diagnoses such as *lateral epicondylitis* ("tennis elbow"), milder forms of *carpal tunnel* or *cubital tunnel syndrome* [8], proximal radial nerve entrapment [9], or tendinitis in the forearm or wrist. Other names for DBRN entrapment include *supinator syndrome* [10, 11] and *treatment-resistant tennis elbow* [3].

Also, of note, some authors and clinicians merge RTS and PINS, using the terms interchangeably. For the purposes of this review, RTS is considered distinct from PINS—involving more pain than palsy and implying a more proximal entrapment. PINS involves much more palsy than pain and should be considered as a distinct nerve disorder, often caused by abrupt trauma or space-occupying lesions such as lipomas or ganglion cysts.

Most of the focus of this chapter will be on RTS—a more commonly diagnosed disorder in an outpatient musculoskeletal practice than PINS. To put into perspective the controversy behind RTS, the authors offer the following quote from one recent neurology review of radial nerve disorders: "The radial tunnel and its associated syndrome seem to be orthopedic concepts that engender much skepticism from neurologists" [12].

Clinical Presentation (Table 35.1)

Numerous authors have reviewed the causes for DBRN entrapment, often without distinguishing between RTS and PINS. The most common causes are listed in Table 35.1.

In the absence of discrete trauma, patients with PINS usually have a gradual onset of weakness in PIN-innervated muscles, with finger extension more affected than wrist extension [12]. They may have pain, but motor symptoms are more prominent [2, 15]. Patients with PINS presenting with

Table 35.1 Occupation/exercise/trauma history relevant to DBRN entrapment

Cause	Comments
Benign tumors, cysts	Lipomas are most common [1]
Repetitive stress injuries, involving repeated forearm supination-pronation with elbow extended	Manual labor [4, 13]
	Writer's cramp [13]
	Violin player or music director [14, 15]
	Athletic activities [14], especially throwing sports [9], swimming activities [15], and tennis [16]
Trauma	Proximal radial fracture or dislocation [12, 17]
	Ulnar fracture [12]
Inflammatory conditions	Rheumatoid arthritis [12]

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weakness have compression to the point where large myelinated motor fiber function is compromised [1].

Patients with RTS present with a deep ache in the proximal lateral forearm extensor muscles, just distal to the lateral epicondyle (Fig. 35.1) [4]. Initial symptoms are usually fatigue and skill decrement, rather than pain. Note that for patients with repetitive stress injuries and neurologic symptoms, compressions of the ulnar (see Chap. 38) and median nerve (see Chap. 37) are much more common than radial nerve involvement [7, 12, 15].

Pain onset is usually insidious [18]. It is often in the dominant arm, may extend proximally or distally [13], and is often troublesome at night [9]. It generally worsens with increased use of the lateral extensor muscles of the arm, such as with keyboarding, other fine motor activities, or lifting. Tasks involving handgrip combined with elbow pronation and wrist extension, such as picking up a luggage, may be particularly painful. Generally, there are no significant paresthesias or numbness unless there is concomitant carpal tunnel syndrome, which is not unusual in the setting of treating injured workers. Patients with RTS may also have weakness, but this is often due to pain with use of the forearm, rather than a primary neurologic deficit [4].

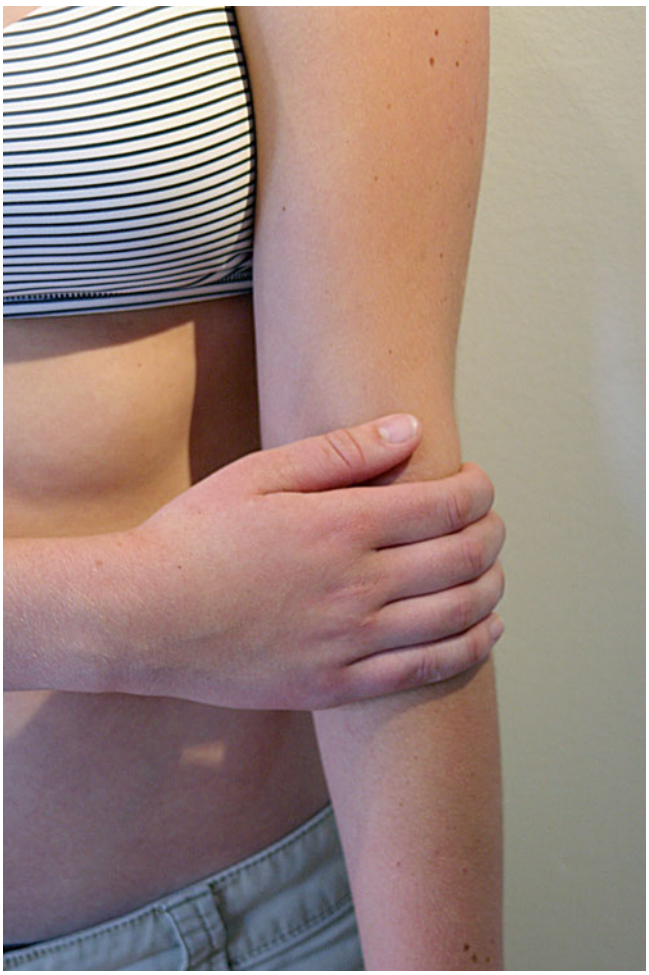

Fig. 35.1 Patient identification of elbow pain, consistent with radial tunnel entrapment (Image courtesy of Andrea Trescot, MD)

Anatomy (Table 35.2)

The radial nerve arises from the posterior cord of the brachial plexus, with contributions from C5 to T1, wrapping around the humerus and then traveling across the radial-humeral (RH) joint near the lateral epicondyle to descend into the forearm. Figure 35.2 illustrates the major cutaneous nerves of the upper extremity. Note that patients with RTS do not have involvement of the superficial radial nerve. The main trunk of the radial nerve then divides into the superficial sensory branch (SRN) and the DBRN (Fig. 35.3). Most authors define the proximal edge of the *radial tunnel* as the point at which the DBRN crosses the RH joint, just distal to the bifurcation, although some characterize the radial tunnel as beginning proximal to the RH joint and including the SRN [13].

The distal terminus of the radial tunnel has a more variable definition. Some authors include the proximal portion of the *supinator muscle*, known as the *arcade of Frohse* (AF), but not the entire length of the supinator muscle [4, 12, 24]. Others add the entire span of the supinator muscle, including the muscle's distal edge [23, 25, 29]. The supinator muscle itself has a complicated anatomy, arising from the humerus, lateral collateral ligament of the elbow, and the ulna and then attaching to the radius in both a deep and superficial layer.

Further complicating nomenclature is the variable labeling of the DBRN. Most authors consider that the PIN begins at the bifurcation of the main trunk of the radial nerve (synonymous with the DBRN) [1, 11, 15, 22, 24], while some state that the PIN does not strictly begin until the nerve emerges from the distal edge of the supinator [25]. These anatomic distinctions likely do not have much clinical importance, but they certainly add to the confusion in the literature on radial nerve entrapment near the elbow.

The cadaveric study by Hazani [25] and others mapped out the proximal and distal borders of the supinator muscle, using the radial head as a reference point. The DBRN dives under the AF, at an average of 3.5 cm distal to the radial head, and emerges as the PIN 7.5 cm distal to the radial head. These are fairly straightforward landmarks, helpful when planning a diagnostic injection for a patient with RTS.

Similarly, Berton et al. [28] dissected 28 embalmed upper limbs and traced the radial nerve from the bicipital groove to the distal edge of the supinator. They found superficial and deep layers of the supinator in all the specimens, and the motor innervation of the supinator was from the DBRN in each case; 20 specimens had a clear arcade of Frohse, and all the DBRNs were slightly flattened at proximal and distal radial tunnel.

Thirty limbs dissected by Clavert et al. [23] showed that the AF was not the only compression site. These authors

Table 35.2 Radial (deep) nerve anatomy

Origin	A direct continuation of the posterior cord of the brachial plexus, C5–T1
General route	Spirals around the posterior humerus, gives off branches to the triceps and anconeus muscles, then through the lateral intermuscular septum into the anterior compartment ~ 10 cm proximal to the elbow [19]
	Gives off a branch to the *extensor carpi radialis longus* (*ECRL*), then divides near the lateral epicondyle into the deep and superficial branches [16]
	Deep branch of the radial nerve (DBRN) passes through the radial tunnel underneath the proximal edge of the supinator muscle and then between the superficial and deep layers of the supinator
Sensory distribution	Wrist joint [15, 16, 20, 21]
	Occasionally elbow joint [4, 16]
Motor innervation	All the wrist and finger extensors with variable numbers of branches to each [22]
	RN: triceps, brachioradialis (BR), brachialis (shared innervation with musculocutaneous nerve), ECRL
	PIN: *extensor carpi radialis brevis* (*ECRB*), supinator, finger extensors, extensor pollicis longus (EPL), extensor pollicis brevis (EPB), abductor pollicis longus (APL), extensor carpi ulnaris (ECU)
Anatomic variability	Site of radial nerve bifurcation into deep and superficial branches. This may occur distal to the *radiohumeral* (*RH*) joint [4, 22, 23]
	Order of muscle innervation by a web of deep radial nerve branches [22]
	Structure of the proximal edge of the supinator muscle, more commonly known as the *arcade of Frohse* (*AF*) [23–25]. The degree of fibrosis seems to be use related, since this structure is entirely muscular in full-term fetuses [26]
	Structure of the distal edge of the supinator muscle [25]
	Number and position of recurrent branches of the radial artery [24]
	Position of the PIN changes with forearm pronation and supination [15, 23] though not with elbow flexion [27]
Other relevant structures	Supinator muscle [28]
	"Mobile wad": the proximal forearm compartment containing BR, ECRL, ECRB

believe that repeated supination and pronation promote gradual DBRN compression in adults.

One may wonder how a nerve often described as having "pure motor function" can cause a painful condition. This was nicely reviewed in a paper by Naam and Nemani [6]. They note that the DBRN includes unmyelinated group IV afferent nerve fibers—also called C fibers—which carry nociceptive inputs to the spinal cord from structures in the forearm [4, 15].

Entrapment

"*Radial tunnel syndrome*," caused by compression of the DBRN, actually has several potential etiologies. The most common site of compression is the proximal edge of the superficial portion of the *supinator muscle* (*the arcade of Frohse*), especially when the forearm is pronated [4, 14, 15, 28, 30]. The DBRN is seen adjacent to the supinator muscle in a proximal forearm MRI cross-section in Fig. 35.4. Changes in position increase the pressure in the radial tunnel, believed to occur when the supinator edge is fibrous. According to one study, passive pronation increases the pressure to 46 ± 21 mmHg, while tetanic contraction of the supinator dramatically escalates it to 195 ± 65mmHg [31]. Proposed mechanisms of compression include the stress of the activity itself—for example, throwing—or secondary anatomic changes such as muscular hypertrophy or injury and repetitive traction stresses [9].

Multiple other potential sites of compression have been described, including, from proximal to distal:

- Above the elbow, the radial nerve (RN) is compressible by a fibrous arcade within the lateral or long head of the triceps [9, 13].
- Fibrous bands from the annular ligament and radial head [25].
- Recurrent branches of the radial vessels ("l*eash of Henry*") may compress the PIN at the radial neck [14, 25, 30].
- The edge of the ECRB [4, 14].
- Distal edge of the supinator muscle [4, 14, 28].

Rarely, compression and entrapment can be caused by a ganglion or lipoma, which would likely cause the well-defined, but rare, PINS, with clear weakness and electrodiagnostic abnormalities [4, 6].

Physical Exam

Radial Tunnel Syndrome

The history and physical examination for RTS are fairly nonspecific, although its hallmark is pain and tenderness approximately 3–6 cm or three fingerbreadths distal to the radial head (Fig. 35.5). One may attempt to palpate the proximal radius in this area. However, in practice, it is easiest for

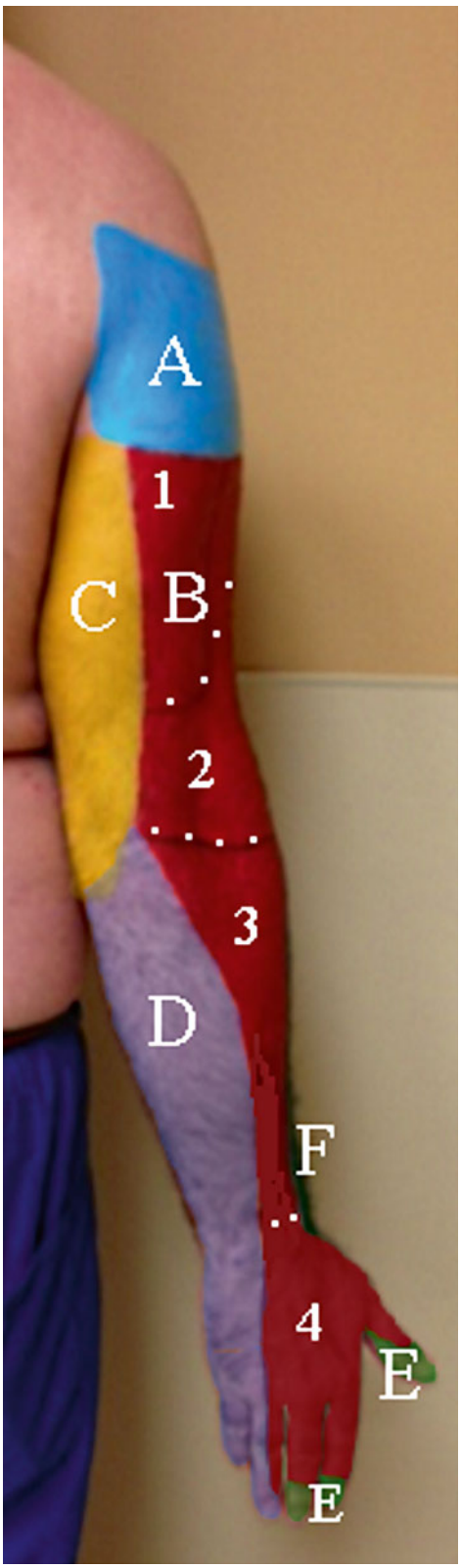

Fig. 35.2 Distribution of arm nerves: *A* axillary nerve, *B* radial nerve (*1* posterior cutaneous nerve of the arm, *2* inferior lateral cutaneous nerve, *3* posterior cutaneous nerve of the forearm, *4* superficial radial nerve), *C* intercostal brachial nerve, *D* medial cutaneous nerve of the forearm, *E* median nerve, *F* lateral cutaneous nerve of the forearm (Image courtesy of Terri Dallas-Prunskis, MD)

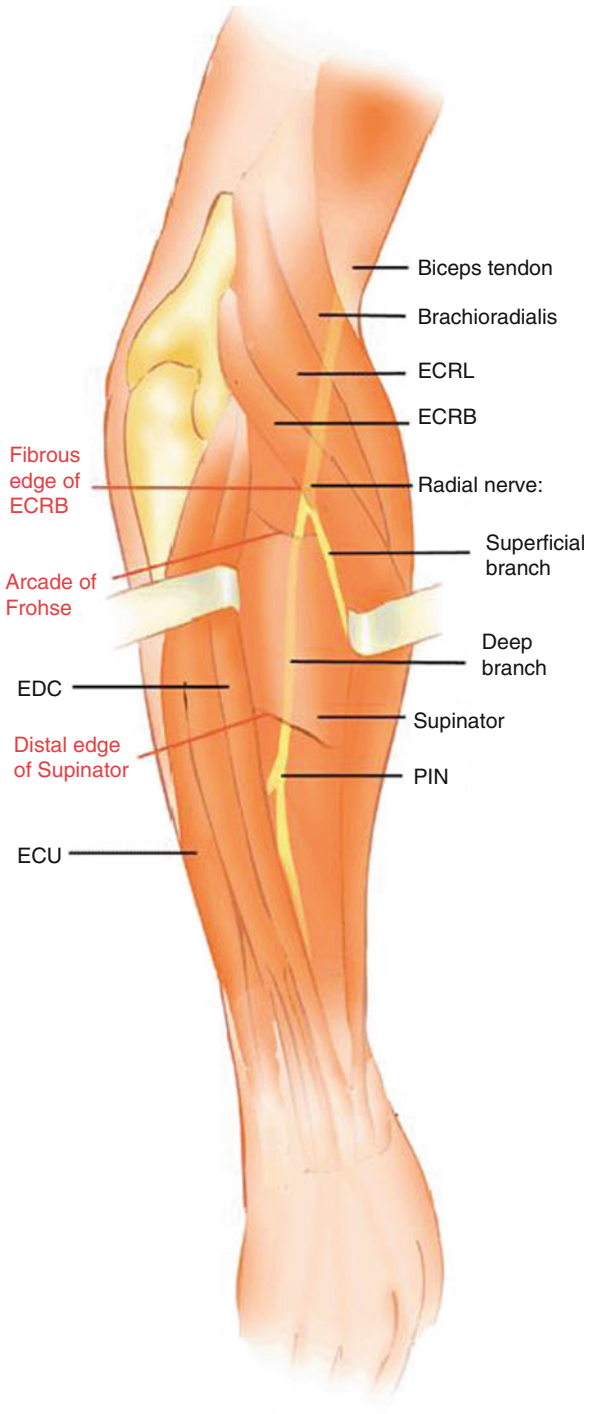

Fig. 35.3 Relevant anatomy for radial tunnel syndrome. Illustration of the radial tunnel content through a dorsal approach demonstrating the bifurcation of the radial nerve into superficial and deep branches. The deep branch becomes the posterior interosseous nerve as it exits the distal edge of the supinator. Points of compression are marked in *red*. *ECRB* extensor carpi radialis brevis, *ECRL* extensor carpi radialis longus, *ECU* extensor carpi ulnaris, *EDC* extensor digitorum communis, *PIN* posterior interosseous nerve (Reproduced with permission from Hazani et al. [25])

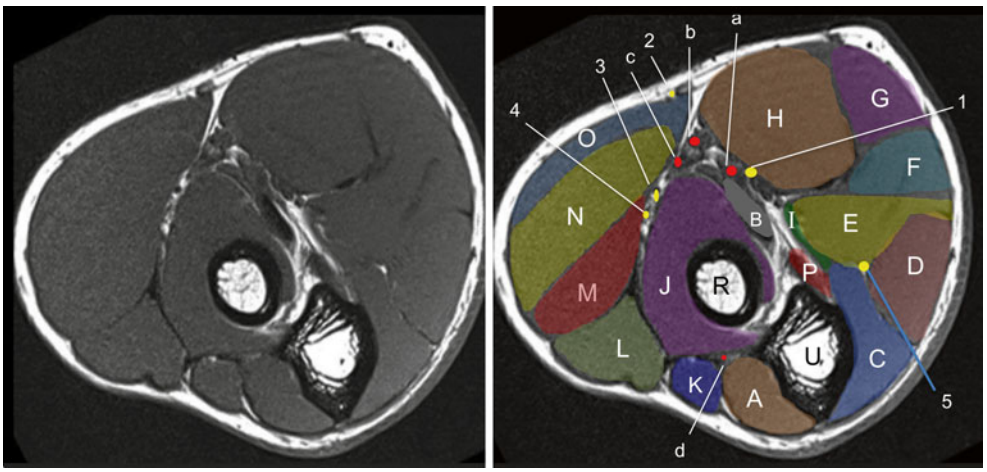

Fig. 35.4 MRI axial image of the proximal forearm. *A* anconeus muscle, *B* biceps tendon, *C* flexor digitorum profundus muscle, *D* flexor carpi ulnaris muscle, *E* flexor digitorum superficialis muscle, *F* palmaris longus muscle, *G* flexor carpi radialis muscle, *H* pronator teres muscle (humeral head), *I* pronator teres muscle (ulnar head), *J* supinator muscle, *K* extensor digiti minimi muscle, *L* extensor digitorum muscle, *M* extensor carpi radialis brevis muscle, *N* extensor carpi radialis longus muscle, *O* brachioradialis muscle, *P* brachialis muscle; *a* ulnar artery, *b* radial artery, *c* radial recurrent artery, *d* recurrent interosseus artery, *1* median nerve, *2* lateral cutaneous nerve of the forearm, *3* superficial branch radial nerve, *4* deep branch radial nerve, *5* ulnar nerve (Image courtesy of Andrea Trescot, MD)

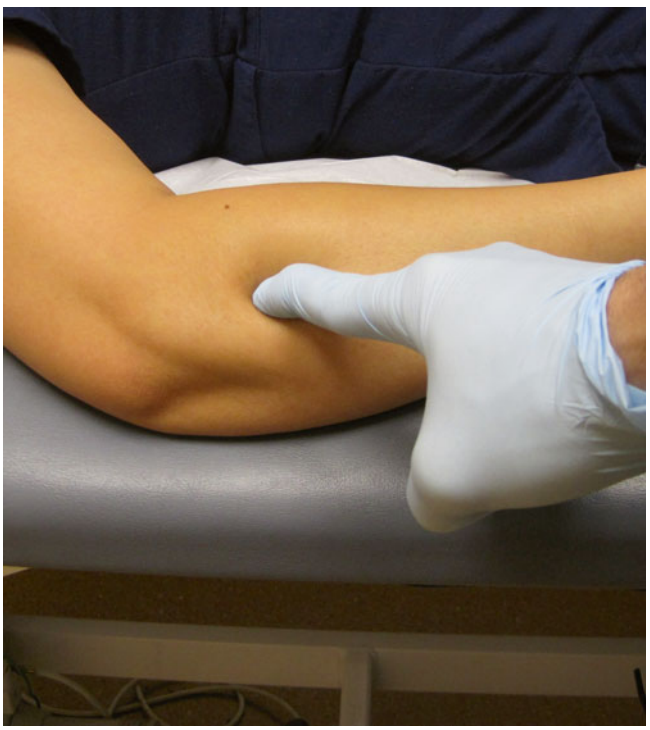

Fig. 35.5 Physical exam of radial nerve entrapment, showing point of maximum tenderness. (Image courtesy of Richard Seroussi, MD)

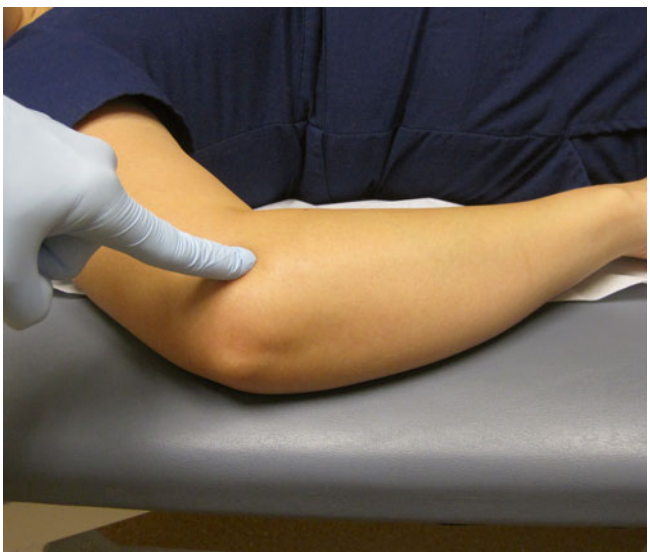

Fig. 35.6 Point of maximum tenderness for lateral epicondylitis, proximal to RTS tenderness. (Image courtesy of Richard Seroussi, MD)

the examiner to press the patient's pronated forearm at a junction between the dorsal proximal ulna and the extensor mass of muscles—often termed the "*mobile" wad*—which includes the brachioradialis and the wrist extensors (Video 35.1). This area can be mildly tender in asymptomatic persons, so the examiner should compare tenderness between the affected and unaffected sides to gauge the clinical relevance of this finding.

Also, the patient may have lateral epicondylitis (tennis elbow) (Fig. 35.6) as a competing or additional diagnosis, and comparison of palpation findings between the lateral epicondyle and the radial tunnel should be made. Indeed, RTS has been called "treatment-resistant tennis elbow," given ambiguity between the two disorders on history and physical exam.

Positions that increase traction on the radial nerve (forearm pronation with elbow extended and wrist flexed)

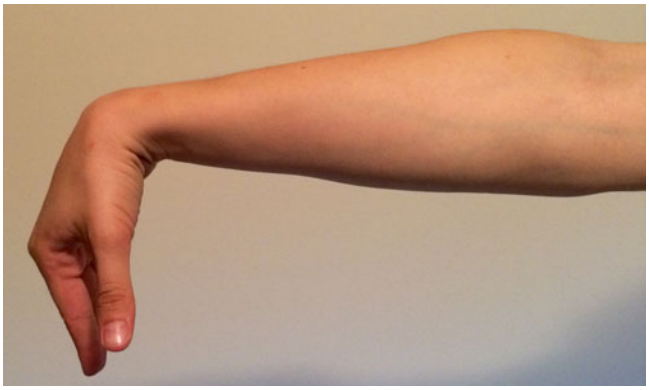

Fig. 35.7 Provocation of radial nerve tension by pronation of the forearm with the elbow extended and wrist flexed (Image courtesy of Virtaj Singh, MD)

(Fig. 35.7) can be used as provocative tests to enhance symptoms [1]. However, this position will also aggravate lateral epicondylitis symptoms.

Resisted wrist extension in patients with RTS is generally only mildly painful, in contrast to the usual sharp pain elicited in those with lateral epicondylitis. Some authors describe the "*middle finger test*," where resisted middle finger extension reproduces concordant pain for the RTS patient, but in these authors' experience, this is also a fairly nonspecific finding [32].

RTS may be part of a "*double crush*" situation with an associated more proximal injury [9]. We recommend screening evaluation of the neck and shoulders and a myotomal screen of upper extremity strength, even in the absence of symptoms in these regions, to rule out competing or additional diagnoses. Neck examination includes passive or active assisted range of motion of the neck with paraspinal palpation and the use of What is this?. Shoulder examination should include passive overhead forward flexion and crossed adduction of the shoulder, as well as forward shoulder flexion with internal rotation (*Hawkins test*) (Fig. 35.8) [33].

Posterior Interosseous Nerve Syndrome

The wrist extensor weakness of patients with PINS is more subtle than the wrist drop of patients with proximal RN palsies, since the *extensor carpi radialis* longus muscle (ECRL) remains intact. Careful neurologic exam is therefore essential for clinical detection of PINS. The classic findings are decreased finger extension—especially fourth and fifth fingers—and drift of the hand in a radial direction when wrist extension is attempted due to *extensor carpi ulnaris* (ECU) weakness [15]. Again, careful neurologic exam reveals well-defined and painless finger extensor weakness for PINS.

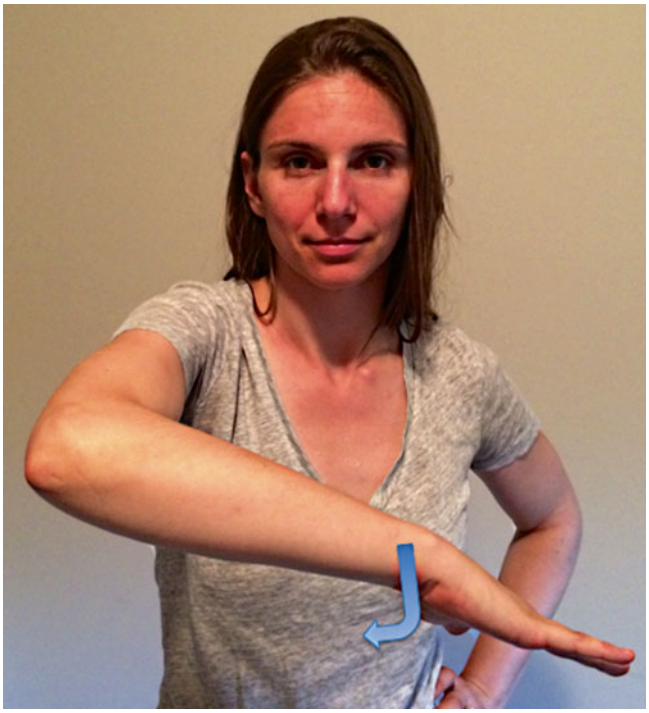

Fig. 35.8 Hawkins test—passive overhead forward flexion and cross adduction (Image courtesy Virtaj Singh, MD)

A more proximal radial nerve lesion can be excluded with the following manual muscle tests during neurologic exam: (1) intact strength of triceps: the examiner's ability to detect weakness is maximized if the patient attempts elbow extension from an initially flexed position; (2) sparing of brachioradialis, tested with the patient attempting elbow flexion while simulating holding a glass of water to maximize the muscle action of brachioradialis and minimize contribution from biceps; and (3) detection of partial wrist extensor strength, confirming sparing of ECRL.

Differential Diagnosis (Table 35.3)

There are several causes of forearm and elbow pain (Table 35.3). The most closely related disorder is lateral epicondylitis, and patients may present with a combination of both disorders [9, 32]. Additional conditions such as cervical radiculopathy, shoulder impingement syndrome, thoracic outlet syndrome, and CTS should be addressed by history. For example, if pain extends proximally toward the shoulder and neck and is worse with overhead activities or movement of the neck, consider cervical and/or shoulder problems in the differential diagnosis. If the patient wakes at night "flicking" their wrist because it has "fallen asleep," this is highly suggestive of CTS (see Chap. 37). If the patient has increased symptoms with flexion of the elbow such as with holding a telephone, *cubital tunnel syndrome* (see Chap. 38) should be considered as well.

Table 35.3 Differential diagnosis of forearm and elbow pain

Diagnosis	Potential distinguishing features
Lateral epicondylitis (tennis elbow) [4, 9, 32]	Lateral elbow pain accompanied by tenderness at the lateral epicondyle at the ECRB insertion (Fig. 35.4), decreased grip strength with the elbow extended. Notable pain with isometric wrist extension against resistance
	May coexist with RTS
Extensor tendinitis [4]	Pain with isometric muscle activation or passive stretch of the affected tendon, without radial tunnel tenderness
Joint pathology [4]	Pain and crepitance over the elbow joint, including with passive range of motion
More proximal radial nerve, brachial plexus, or cervical spine problem [4, 12]	Weakness of more proximally innervated muscles, decreased reflexes, pain worse with overhead activities
Other peripheral nerve entrapments [8]	Carpal tunnel syndrome followed by cubital tunnel syndrome (ulnar neuropathy at the elbow) are much more common peripheral nerve entrapments and present with specific sensorimotor abnormalities on neurologic exam

There are few reliable objective data for the diagnosis of RTS, which has a primarily clinical basis (Table 35.4) [4–6, 36, 37].

Table 35.4 Diagnostic tests for deep radial nerve (PIN) entrapment

	Potential distinguishing features
Physical exam	Tenderness over the "mobile wad," 3–6 cm distal to the radial head
Provocative test(s)	Proximal wrist pain with resisted wrist extension [1, 3]
	Extend elbow and pronate arm—resisted supination will lead to RTS symptoms [1, 4, 13]
	Resisted middle finger extension compresses the PIN against the edge of ECRB, causing pain [1, 4, 13]
Diagnostic injection	Radial tunnel injection results in pain relief [4]—this is particularly useful because there are few objective tests for RTS
Ultrasound	May be useful in clarifying the anatomic cause of neuropathy [34, 35] in PINS, but generally not useful for RTS
MRI	MRI microscopy can demonstrate PIN swelling and its cause (e.g., mass lesion) [34, 35]
	Routine MRI shows early and late signs of muscle denervation [18]
	Only 4/25 of patients with RTS (no weakness) had normal MRI in one retrospective study, but MRI abnormalities were nonspecific [18]
X-ray	To rule out other bony pathologies [4, 13]
Electrodiagnostic studies	RTS: usually normal [1, 9]
	Electrodiagnosis is viewed on the spectrum from "very helpful" [14] to "necessary to confirm the diagnosis" of PINS [12, 28]

Identification and Treatment of Contributing Factors

Repeated pronation-supination of the forearm while lifting, especially when combined with elbow extension, appears to be an occupational risk factor for RTS [7, 38]. RTS is also frequent among office workers with repetitive wrist extension and pronation activities [13].

Relative rest from activities that aggravate upper extremity symptoms; ergonomic intervention including, increasingly, the use of wireless headsets and voice recognition technology; medications including some of the newer topical gels that provide analgesia locally; sleep restoration if applicable, and physical or occupational therapy, including hand therapy [4, 9], may help alleviate symptoms. Splinting with the wrist extended and forearm pronated [4, 9] and anti-inflammatory medication may also be useful [4, 9]. Workers with jobs that require sustained reaching and lifting, especially with repetitive forearm rotation, should be evaluated for any possible remedies from these work site tasks. Unfortunately, a number of patients, especially those in the building trades, do not have much in the way of "light duty" or ergonomic hope for modifying occupational tasks.

Myofascial release techniques, muscle energy techniques, eccentric strengthening, and gradual lengthening of the extensor muscles can all be considered as part of a noninvasive treatment program. Treatment of associated conditions such as poor posture with weakness of the scapular muscles and anterior head carriage may also provide some relief. In these authors' experience, a proximal forearm band often is helpful for lateral epicondylitis, but not generally for RTS.

If the patient is not responding to the above regimen, it is advised to move forward with an injection to the area of maximal tenderness within the radial tunnel, partly to establish the diagnosis and partly to provide pain relief, if needed.

Injection Technique

Landmark-Guided Technique

The patient is positioned either supine or sitting supported, with the forearm pronated. In these authors' experience, it is most comfortable to have the patient on a raised exam table in the

supine position with the elbow extended and the forearm pronated. The proximal and distal borders of the supinator muscle are mapped, using the radial head as a reference point. The point of maximal tenderness is palpated as described above (Video 35.2). After sterile skin preparation, a 27-gauge, 1.5-in. needle is directed anteromedially in a transverse plane without image guidance toward the point of maximal tenderness (Fig. 35.9). It is counterproductive to anesthetize the skin first, as this may obscure the patient's response to reaching the point of maximal tenderness, and involves a second needle procedure. A reasonable injectate composition is 1 cc of 1 % or 2 % lidocaine mixed with 1 cc of a corticosteroid, such as triamcinolone.

The role of an injection is mostly diagnostic, although some patients can have benefit from an injection for up to months at a time. For better diagnostic information, provide a post-procedure symptom diary for patients to return on follow-up. Patients are advised to use their forearm, including aggravating activities, but not to push themselves too hard while the anesthetic is still in effect. RTS patients usually have at least 50 % acute improvement in symptoms, including activities that would normally aggravate their condition. Patients usually experience acute palsy of the finger extensors with this procedure and should be informed *before* the block that this is an expected outcome that implies injection accuracy. Depending on the duration of nerve palsy, patients are advised against driving and may need a driver to accompany them for the procedure. If the physician does not use a large volume of concentrated local anesthetic, most patients can be discharged independently 10–40 min after the procedure.

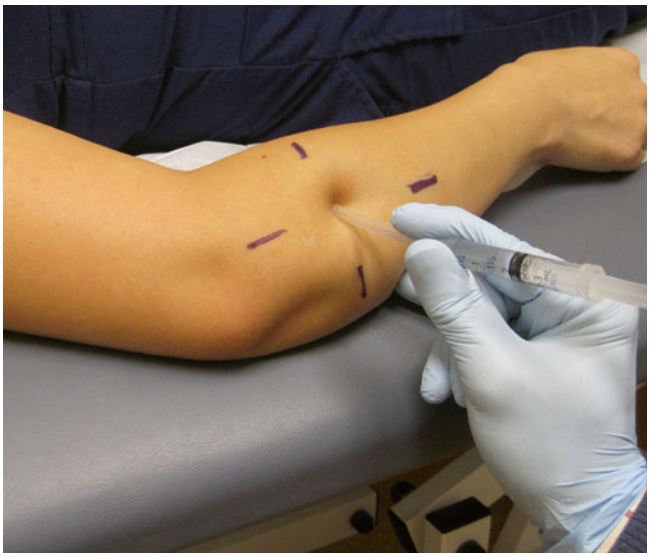

Fig. 35.9 Landmark-guided injection of deep branch of radial nerve at the radial tunnel (Image courtesy of Richard Seroussi, MD)

Fluoroscopic-Guided Technique

There is no role for fluoroscopic guidance for this procedure.

Ultrasound (US)-Guided Technique

Ultrasound-guided DBRN injections can be quite helpful. Martinoli and others highlight the US anatomy of the transverse and the longitudinal anatomy of the proximal radial tunnel under ultrasound; the DBRN is sandwiched between the superficial and deep bellies of supinator muscle [10, 11]. The transducer is held in a transverse plane over the brachioradialis muscle at the area of maximal tenderness (Fig. 35.10) for a long axis view of the injecting needle. Injection just adjacent to the nerve is generally fairly straightforward; however, it can be difficult to discern in cross section, so shifting the

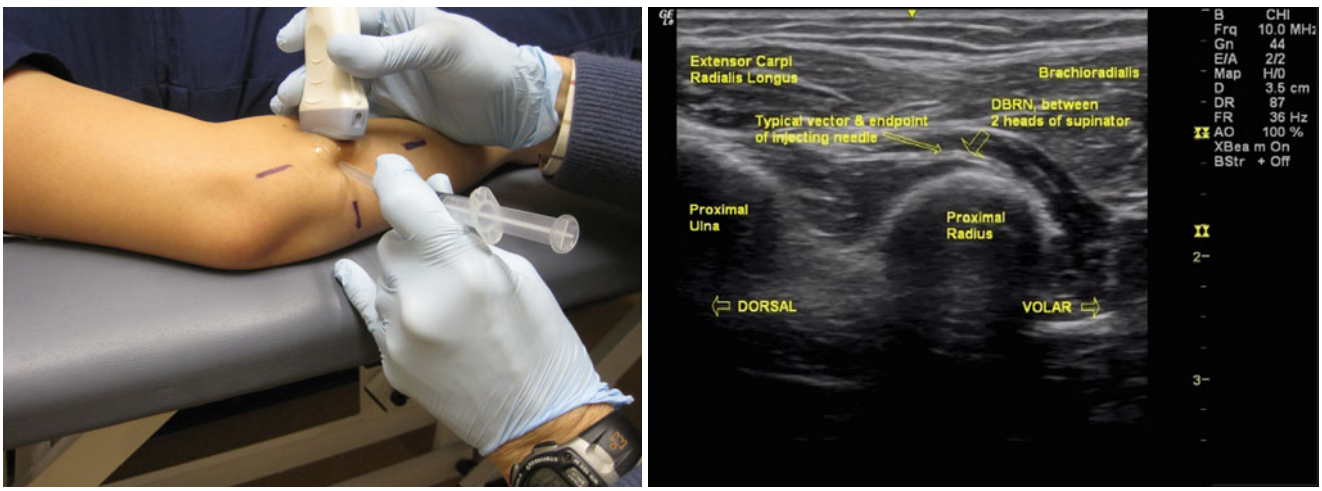

Fig. 35.10 Ultrasound-guided injection of deep branch of radial nerve at the radial tunnel (Image courtesy of Richard Seroussi, MD)

probe proximally and distally to track the nerve as it courses radially toward the wrist can be helpful.

Surgical Treatment

There is a body of surgical literature regarding PINS and RTS, mostly outside the scope of this discussion. To date, there are no reports of controlled trials of operative or nonoperative interventions to treat either syndrome. Systematic reviews of observational case series revealed only a few reports of sufficient quality for analysis: all involved surgical treatment of RTS ($n=6$) [5] or PINS ($n=2$) [2]. The authors concluded that "surgical decompression of the radial tunnel might be effective in patients with RTS" [5] and "there is a tendency for the effectiveness of surgical decompression of the PIN in patients with PINS" [2].

If the patient has a fairly good response to diagnostic injection, referral to a surgical specialist for radial tunnel surgery is likely indicated [4]. Since PINs may lead to permanent nerve injury, patients should have surgical release if not improved by 3 months [9]. Delay is usually not a problem in patients with RTS, because of the pain [4]. It is most important to have the correct diagnosis (especially with respect to possible lateral epicondylitis) [4] and to release all sites of compression [4, 13].

When referring a patient for possible surgery, the provider should have confidence that the surgical consultant has extensive experience in radial tunnel release, since it is not a common procedure.

Complications

Complications of the injection technique are similar to those of most peripheral nerve injections, including the chance of excessive bleeding, nerve injury, and/or infection. Inducing increased pain is possible; acute nerve palsy is an expected outcome, as discussed above. Generally, however, the procedure is fairly well tolerated without significant long-term morbidity.

References

1. Dang AC, Rodner CM. Unusual compression neuropathies of the forearm. part I: radial nerve. J Hand Surg [Am]. 2009;34(10):1906–14.
2. Huisstede BM, Miedema HS, van Opstal T, de Ronde MT, Kuiper JI, Verhaar JA, et al. Interventions for treating the posterior interosseus nerve syndrome: a systematic review of observational studies. J Peripher Nerv Syst. 2006;11(2):101–10.
3. Roles NC, Maudsley RH. Radial tunnel syndrome. J Bone Joint Surg Am. 1972;4B:784–90.
4. Sarris IK, Papadimitriou NG, Sotereanos DG. Radial tunnel syndrome. Tech Hand Up Extrem Surg. 2002;6(4):209–12.
5. Huisstede B, Miedema HS, van Opstal T, de Ronde MT, Verhaar JA, Koes BW. Interventions for treating the radial tunnel syndrome: a systematic review of observational studies. J Hand Surg [Am]. 2008;33(1):72–8.
6. Naam NH, Nemani S. Radial tunnel syndrome. Orthop Clin N Am. 2012;43(4):529–36.
7. Latinovic R, Gulliford MC, Hughes RA. Incidence of common compressive neuropathies in primary care. J Neurol Neurosurg Psychiatry. 2006;77(2):263–5.
8. Roquelaure Y, Raimbeau G, Dano C, Martin YH, Pelier-Cady MC, Mechali S, et al. Occupational risk factors for radial tunnel syndrome in industrial workers. Scand J Work Environ Health. 2000;26(6):507–13.
9. Keefe DT, Lintner DM. Nerve injuries in the throwing elbow. Clin Sports Med. 2004;23(4):723–42.
10. Martinoli C, Bianchi S, Pugliese F, Bacigalupo L, Gauglio C, Valle M, et al. Sonography of entrapment neuropathies in the upper limb (wrist excluded). J Clin Ultrasound. 2004;32(9):438–50.
11. Bodner G, Harpf C, Meirer R, Gardetto A, Kovacs P, Gruber H. Ultrasonographic appearance of supinator syndrome. J Ultrasound Med. 2002;21(11):1289–93.
12. Wang LH, Weiss MD. Anatomical, clinical, and electrodiagnostic features of radial neuropathies. Phys Med Rehabil Clin N Am. 2013;24(1):33–47.
13. Lawrence T, Mobbs P, Fortems Y, Stanley JK. Radial tunnel syndrome. A retrospective review of 30 decompressions of the radial nerve. J Hand Surg (Br). 1995;20(4):454–9.
14. Riffaud L, Morandi X, Godey B, Brassier G, Guegan Y, Darnault P, et al. Anatomic bases for the compression and neurolysis of the deep branch of the radial nerve in the radial tunnel. Surg Radiol Anat. 1999;21(4):229–33.
15. Cravens G, Kline DG. Posterior interosseous nerve palsies. Neurosurgery. 1990;27(3):397–402.
16. Tubbs RS, Salter EG, Wellons 3rd JC, Blount JP, Oakes WJ. Superficial surgical landmarks for identifying the posterior interosseous nerve. J Neurosurg. 2006;104(5):796–9.
17. Khan R, Birch R. Iatropathic injuries of peripheral nerves. J Bone Joint Surg (Br). 2001;83(8):1145–8.
18. Ferdinand BD, Rosenberg ZS, Schweitzer ME, Stuchin SA, Jazrawi LM, Lenzo SR, et al. MR imaging features of radial tunnel syndrome: initial experience. Radiology. 2006;240(1):161–8.
19. Tubbs RS, Apaydin N, Uz A, Sullivan A, Loukas M, Shoja MM, et al. Anatomy of the lateral intermuscular septum of the arm and its relationships to the radial nerve and its proximal branches. Laboratory investigation. J Neurosurg. 2009;111(2):336–9.
20. Van de Pol GJ, Koudstaal MJ, Schuurman AH. Bleys RL Innervation of the wrist joint and surgical perspectives of denervation. J Hand Surg [Am]. 2006;31(1):28–34.
21. Dellon AL, Seif SS. Anatomic dissections relating the posterior interosseous nerve to the carpus, and the etiology of dorsal wrist ganglion pain. J Hand Surg [Am]. 1978;3(4):326–32.
22. Abrams RA, Ziets RJ, Lieber RL, Botte MJ. Anatomy of the radial nerve motor branches in the forearm. J Hand Surg [Am]. 1997;22(2):232–7.
23. Clavert P, Lutz JC, Adam P, Wolfram-Gabel R, Liverneaux P, Kahn JL. Frohse's arcade is not the exclusive compression site of the radial nerve in its tunnel. Orthop Traumatol Surg Res. 2009;95(2):114–8.
24. Ozkan M, Bacakoglu AK, Gul O, Ekin A, Magden O. Anatomic study of posterior interosseous nerve in the arcade of Frohse. J Shoulder Elbow Surg. 1999;8(6):617–20.
25. Hazani R, Engineer NJ, Mowlavi A, Neumeister M, Lee WP, Wilhelmi BJ. Anatomic landmarks for the radial tunnel. Eplasty. 2008;8, e37.
26. Spinner M. The arcade of Frohse and its relationship to posterior interosseous nerve paralysis. J Bone Joint Surg (Br). 1968;50(4):809–12.

27. Jones JA, Jones CM, Grossman MG. Effect of elbow flexion on the proximity of the PIN during 2-incision distal biceps repair. Orthopedics. 2013;36(7):e931–5.
28. Berton C, Wavreille G, Lecomte F, Miletic B, Kim HJ. Fontaine C The supinator muscle: anatomical bases for deep branch of the radial nerve entrapment. Surg Radiol Anat. 2013;35(3):217–24.
29. Prasartritha T, Liupolvanish P, Rojanakit A. A study of the posterior interosseous nerve (PIN) and the radial tunnel in 30 Thai cadavers. J Hand Surg [Am]. 1993;18(1):107–12.
30. Brabston EW, Genuario JW, Bell JE. Anatomy and physical examination of the elbow. Oper Tech Orthop. 2009;19(4):190–8.
31. Werner CO, Haeffner F, Rosen I. Direct recording of local pressure in the radial tunnel during passive stretch and active contraction of the supinator muscle. Arch Orthop Trauma Surg. 1980; 96(4):299–301.
32. Rose NE, Forman SK, Dellon AL. Denervation of the lateral humeral epicondyle for treatment of chronic lateral epicondylitis. J Hand Surg [Am]. 2013;38(2):344–9.
33. Woodward TW, Best TM. The painful shoulder: part I. Clinical evaluation. Am Fam Physician. 2000;61(10):3079–88.
34. Chien AJ, Jamadar DA, Jacobson JA, Hayes CW, Louis DS. Sonography and MR imaging of posterior interosseous nerve syndrome with surgical correlation. AJR Am J Roentgenol. 2003;181(1):219–21.
35. Furuta T, et al. Magnetic resonance microscopy imaging of posterior interosseous nerve palsy. Jpn J Radiol. 2009;27(1):41–4.
36. Rinkel WD, Schreuders TA, Koes BW, Huisstede BM. Current evidence for effectiveness of interventions for cubital tunnel syndrome, radial tunnel syndrome, instability, or bursitis of the elbow: a systematic review. Clin J Pain. 2013;29(12):1087–96.
37. van den Ende KI, Steinmann SP. Radial tunnel syndrome. J Hand Surg [Am]. 2010;35(6):1004–6.
38. van Rijn RM, Huisstede BM, Koes BW, Burdorf A. Associations between work-related factors and specific disorders at the elbow: a systematic literature review. Rheumatology (Oxford). 2009;48(5): 528–36.

Superficial Radial Nerve Entrapment

36

Andrea M. Trescot and Helen W. Karl

Introduction

Superficial radial nerve (SRN) entrapment is an often-overlooked cause of forearm and thumb pain, mimicking de *Quervain's tenosynovitis, DJD* of the thumb, carpal tunnel syndrome (Chap. 37), and Complex Regional Pain Syndrome (CRPS). It is also called *cheiralgia paresthetica*, *Wartenberg's syndrome*, and *handcuff* or *wristwatch neuropathy*.

Clinical Presentation (Table 36.1)

Entrapment of the SRN was first described by Stopford in 1922 [11]. Ten years later, Wartenberg described an isolated entrapment of the SRN in five patients who complained of radial wrist and thumb pain (Fig. 36.1) [12]. Because it seemed similar to the entrapment of the lateral femoral cutaneous nerve (known as *meralgia paresthetica* – see Chap. 60), he named the entrapment *cheiralgia paresthetica*. According to Dang, SRN entrapment is relatively rare, with an annual incidence of 0.003 % [13].

Patients with superficial radial neuralgia usually have pain and/or abnormal sensation (numbness, hyperalgesia, and hyperesthesia) over the back of the hand, thumb, and index fingers (Fig. 36.2) [14]. This pain may lead to weakness of the thumb and decreased grip strength. Wrist movements [14], especially ulnar-volar flexion [15] and forearm

Table 36.1 Occupation/exercise/trauma history relevant to superficial radial nerve entrapment

Brachioradialis entrapment	Hammering
	Computer mouse use
	Repetitive pronation/supination (as in throwing sports [1])
Forearm trauma	IV infiltration
	Steroid injection for wrist tendonitis [2,3]
	Distal radius (e.g., Colles') fracture
	Forearm lacerations [2]
	Handcuffs [4]
	Digital fracture [2]
Surgical	Post trapeziectomy [2]
	Treatment with an external fixation device [5] or Kirschner wires [6]
Inflammation	de Quervain's tenosynovitis with or without surgical release [3, 7–9]
Compression	Watchband [10]
Stretch	Closed reduction of a forearm fracture [10]

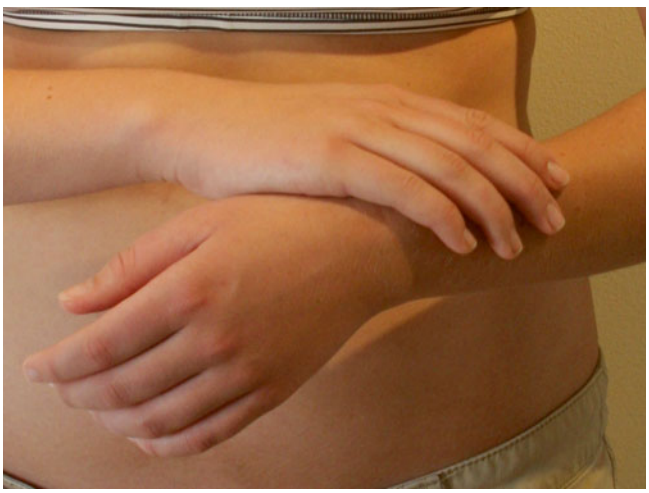

Fig. 36.1 Patient description of pain with superficial radial nerve entrapment (Image courtesy of Andrea Trescot, MD)

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hyperpronation, increase the pain. The pain of SRN entrapment is usually not a problem at night [14], though patients frequently have pain at rest [13].

SRN entrapment can cause or mimic Complex Regional Pain Syndrome (CRPS), resulting in swelling, hyperesthesia, allodynia, and pain on movement. These symptoms are seen initially at the thumb or radial aspect of the dorsum of the hand, but can spread to the whole hand and forearm. Patients often try to protect the hand from stimulation with gloves or bandages [2].

Entrapments of the SRN and posterior interosseous nerve (PIN) (see Chap. 35) may coexist [14]. In the setting of a systemic neuropathy such as diabetes, SRN entrapment along with *median nerve* (see Chap. 37) and *ulnar nerve* (see Chap. 38) entrapments would give a "glove" pattern of pain and/or numbness. Other commonly coexisting conditions include *carpal tunnel syndrome* (CTS) in 16 % [8] to 57 % [14], *lateral antebrachial cutaneous nerve* (*LABC*) neuroma [15], and *de Quervain's tenosynovitis* in 17 % [7] to 50 % [8] of patients with SRN entrapment.

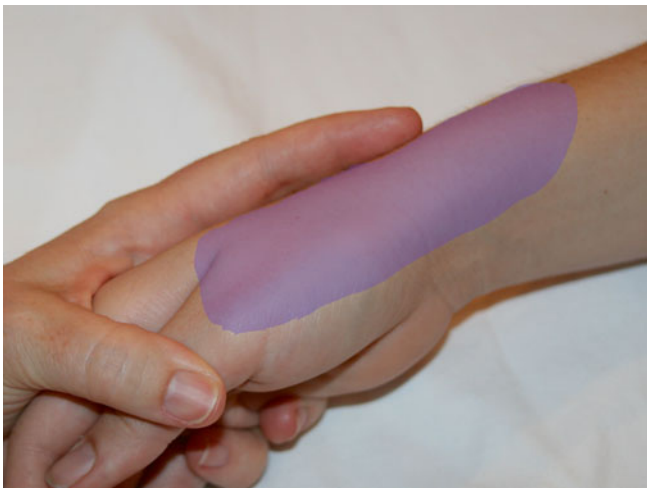

Fig. 36.2 Pain pattern associated with superficial radial nerve entrapment (Image courtesy of Andrea Trescot, MD)

Anatomy (Table 36.2)

The radial nerve arises from the posterior cord of the brachial plexus. It spirals around the humerus and descends down the humerus to the elbow. At the elbow, the radial nerve divides into a superficial branch and deep branch (Fig. 36.3) (see Chap. 35). The *superficial radial nerve* (*SRN*) travels between the *brachioradialis muscle* (BR) and *extensor carpi radialis logus* (ECRL) to emerge superficially at the distal radial forearm (Fig. 36.4), passing across the tendon of the *extensor pollicis longus* (EPL), past the *dorsal tubercle of the radius* (*Lister's tubercle*) and over the *extensor retinaculum* (ER) (Fig. 36.5), traveling to the thumb and dorsum of the hand (Figs. 36.5 and 36.6). The radial, median, and ulnar nerves together provide the sensation to the hand (Fig. 36.7).

Abrams et al. [5] dissected 20 arms. In the most common pattern, seen in ten cases, the SRN arose from the radial nerve at or near the level of the lateral epicondyle. It then ran deep to the BR and usually emerged (in 18 arms) between the tendons of the BR and the ECRL piercing the forearm fascia an average of 9 cm proximal to Lister's tubercle (Fig. 36.5). In two cases, the SRN pierced the tendon of the BR. Once the nerve becomes superficial, it usually divides into two branches, an average of 5 cm proximal to the radial styloid: the *major palmar* and *major dorsal* branches. In 35 % of the cases in the above investigation (7/20), there was a third branch lying directly over the center of the first dorsal wrist compartment. The major palmar branch always continued distally to become the *dorsoradial digital nerve of the thumb*. The major dorsal branch had many branching configurations, ending in the *dorso-ulnar digital nerve to the thumb* and the *dorsoradial digital nerve*

Table 36.2 Superficial radial nerve anatomy

Origin	A direct continuation of the posterior cord of the brachial plexus, C5–T1
General route	Spirals around the posterior humerus, then through the lateral intermuscular septum about 10 cm proximal to the elbow. Divides in/near the radial tunnel at the level of the lateral epicondyle into deep and superficial branches. Superficial branch and radial artery travel deep to the brachioradialis muscle (BR), then wind around the lateral border of the radius, becoming subcutaneous between BR and the tendon of the extensor carpi radialis longus (ECRL), approximately 9 cm proximal to the styloid process of the radius [5]. Divides into the dorsal digital nerves to the radial thenar eminence and to a variable area on the dorsum of the first digit, both sides of the second digit, and the radial half of the third digit
Sensory distribution	Exclusive territory: dorsal thumb web space. Much overlap with lateral antebrachial cutaneous nerve (LABC). Dorsolateral hand and proximal first 3 fingers
Motor innervation	None
Anatomic variability	90 % (18/20) run between BR and ECRL, the remainder through the BR tendon [5]
Other relevant structures	Sensory overlap with LABC nerve, the terminal branch of the musculocutaneous nerve [15]

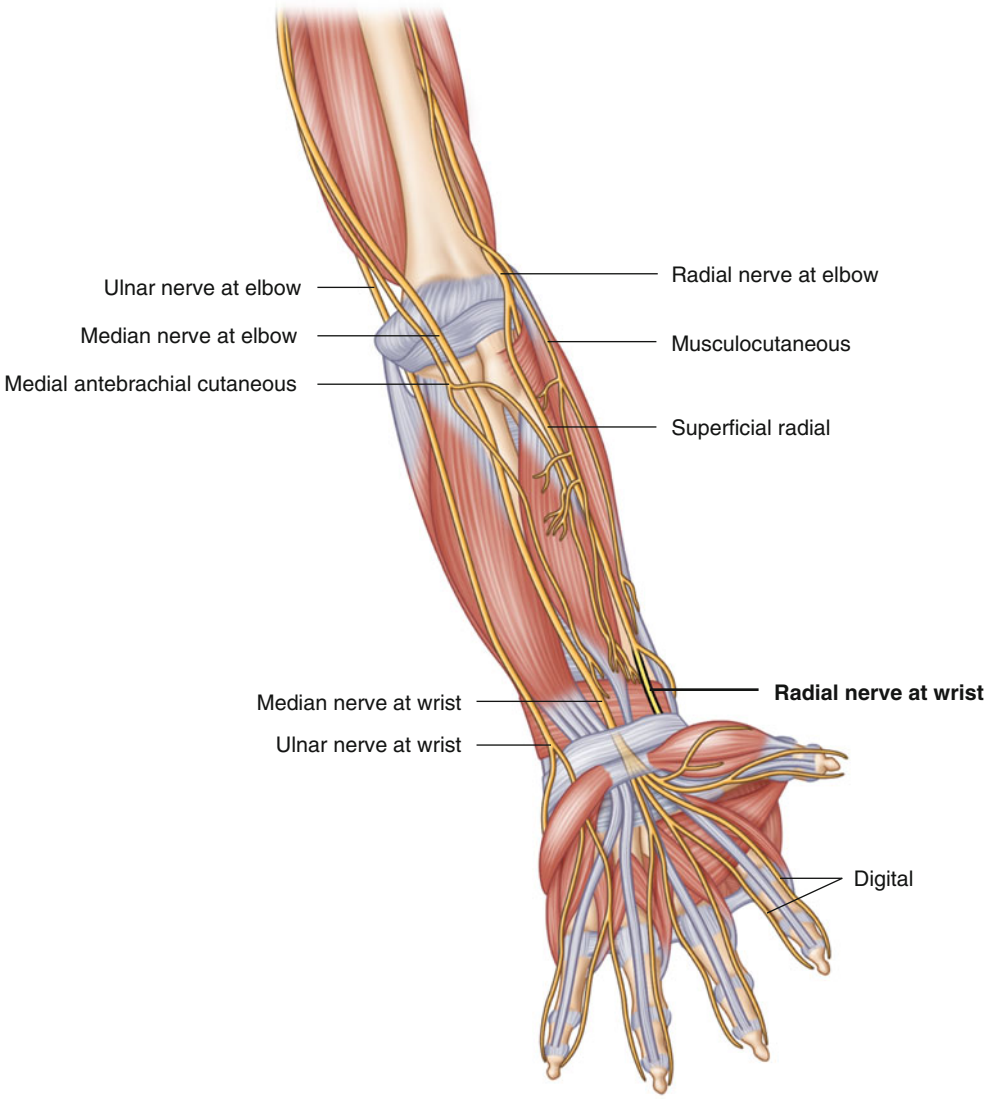

Fig. 36.3 Forearm anatomy (Image by Springer)

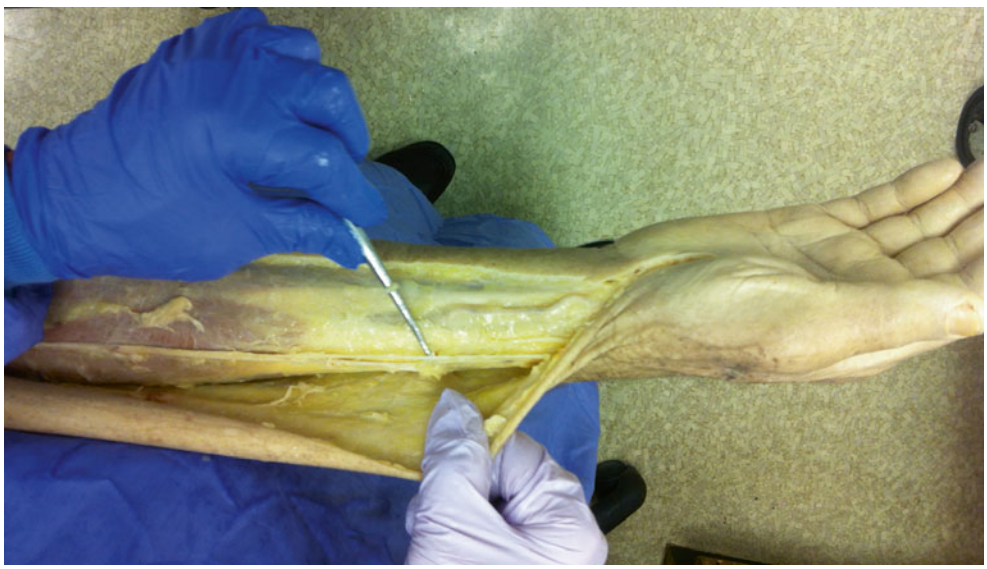

Fig. 36.4 Dissection of the forearm, with probe under the superficial radial nerve (Dissection by Umeshraya Pai, MD; Image courtesy of Andrea Trescot, MD)

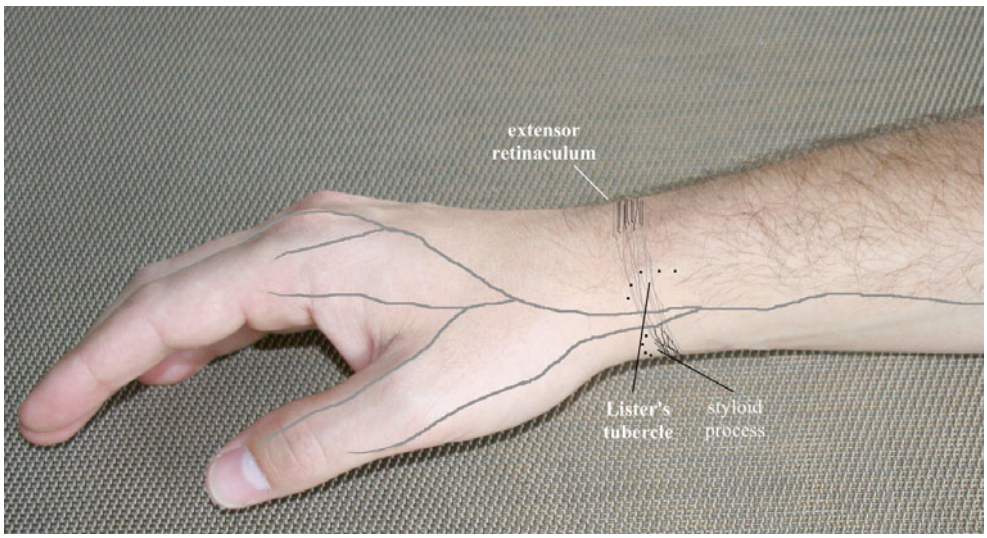

Fig. 36.5 Anatomy of the superficial radial nerve (Image courtesy of Andrea Trescot, MD)

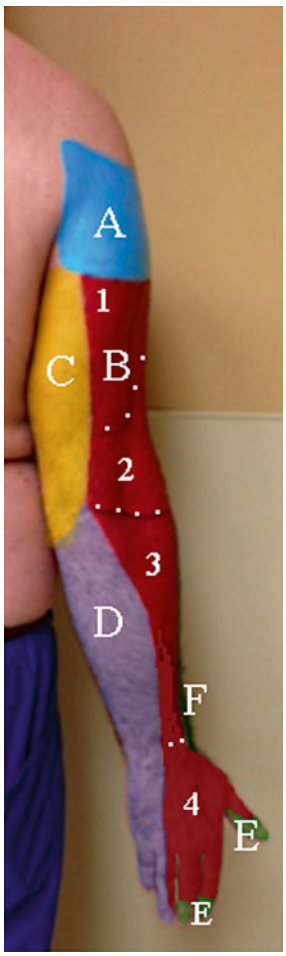

Fig. 36.6 Distribution of arm nerves: *A* axillary nerve, *B* radial nerve (*1* posterior cutaneous nerve of the arm, *2* inferior lateral cutaneous nerve, *3* posterior cutaneous nerve of the forearm, *4* superficial radial nerve), *C* intercostal brachial nerve, *D* medial cutaneous nerve of the forearm, *E* median nerve, *F* lateral cutaneous nerve of the forearm (Image courtesy of Terry Dallas-Prunskis, MD)

to the index finger. Branches of the major dorsal or third branch became the *dorso-ulnar and dorsoradial digital nerves of the index and long fingers*, respectively. In seven cases, there were connections between the SRN and branches of the LABC.

Ali et al. [16] dissected 16 forearms; they found that all of the SRNs crossed the EPL tendon within 2.69 cm of the distal edge of the ER. In 13 specimens (81.25 %), the cephalic vein crossed the SRN once, while in three specimens it crossed the nerve twice. The *dorsal vein of the thumb* always crossed over one of the two main branches of the SRN; 37.5 % of the cases showed the SRN passing over the first dorsal compartment of the wrist. All of the branches of the SRN were noted to be found in the radial half of an isosceles triangle formed by the radial styloid, Lister's tubercle, and the exit of the SRN from underneath the BR (Fig. 36.8).

Entrapment

Entrapment of this nerve can occur in one of several ways. The first is the result of activity that causes spasm or hypertrophy of the BR, such as hammering or using a computer mouse. SRN entrapment can occur where it becomes superficial between the BR and ECRL, about 9 cm proximal to the styloid process of the radius. When the forearm is supinated, the BR and ECRL tendons are parallel (Fig. 36.9); pronation twists them such that the lateral ECRL crosses deep to the BR tendon, thereby decreasing the area through which the nerve passes [7]. This compression is aggravated by ulnar flexion of the wrist, hyperpronation, and other movements that stretch the nerve. These patients may also have pain at the lateral epicondyle (Fig. 36.10).

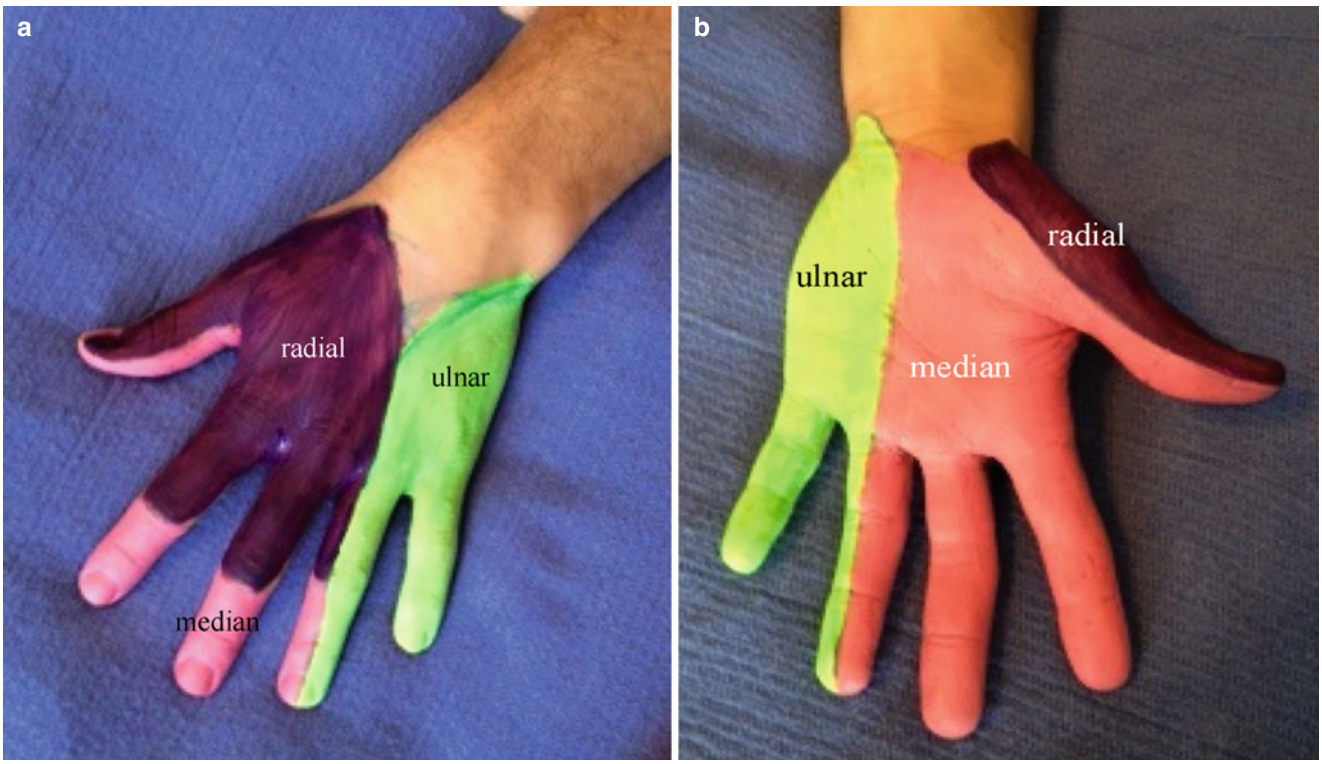

Fig. 36.7 Dermatomal distribution of the nerves of the hand. (**a**) dorsal. (**b**) palmar (Image courtesy of Terri Dallas-Prunskis)

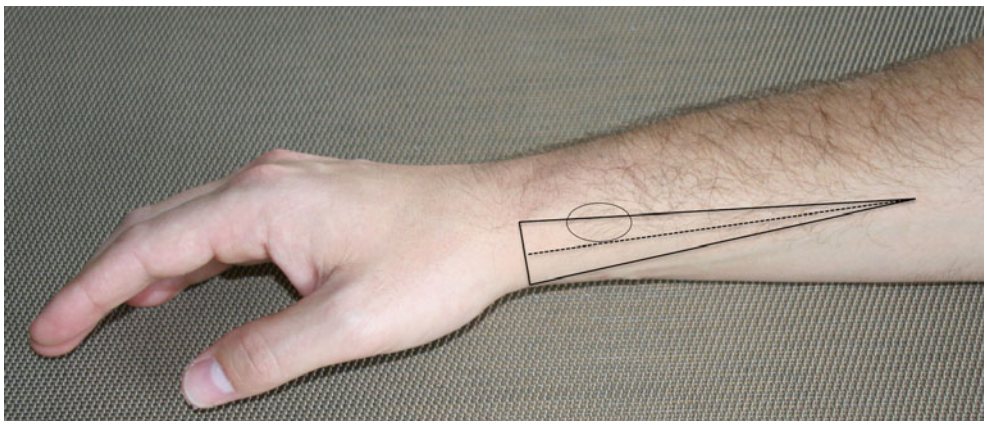

Fig. 36.8 The isosceles triangle formed by the radial styloid, Lister's tubercle, and the exit of the SRN. All of the branches of the superficial radial nerve can be found in the radial half of the triangle. The oval represents the site suggested by Ali et al. [16] as a "safe zone" for placement of Kirschner wires (Image courtesy of Andrea Trescot, MD)

A second group has had trauma to the radial aspect of the forearm (such as infiltration of an IV in the "intern's vein," banging the forearm on the edge of a table, steroid injections for de Quervain's tenosynovitis [3], or a distal radius (e.g., Colles' fracture).

Abrams et al. [5] noted that the branches of the SRN near the *extensor retinaculum* are at risk from a dorsal compartment release of a *de Quervain's tenosynovitis*. They also noted that the usual position for pins for application of an external fixator (i.e., for a Colles' fracture) is very near where the SRN travels in the subcutaneous tissues on the radial aspect of the forearm. During repair of the collateral ligaments of the thumb metacarpophalangeal (MCP) joint, the nearby dorsal digital branches are easily injured.

Physical Exam

The ulnar aspect of the forearm rests in the palm of the clinician's non-examining hand, while the examining thumb is used to roll across the radial aspect of the distal forearm, just distal to the edge of the BR or between the BR and the ECRL (Video 36.1) (Fig. 36.11).

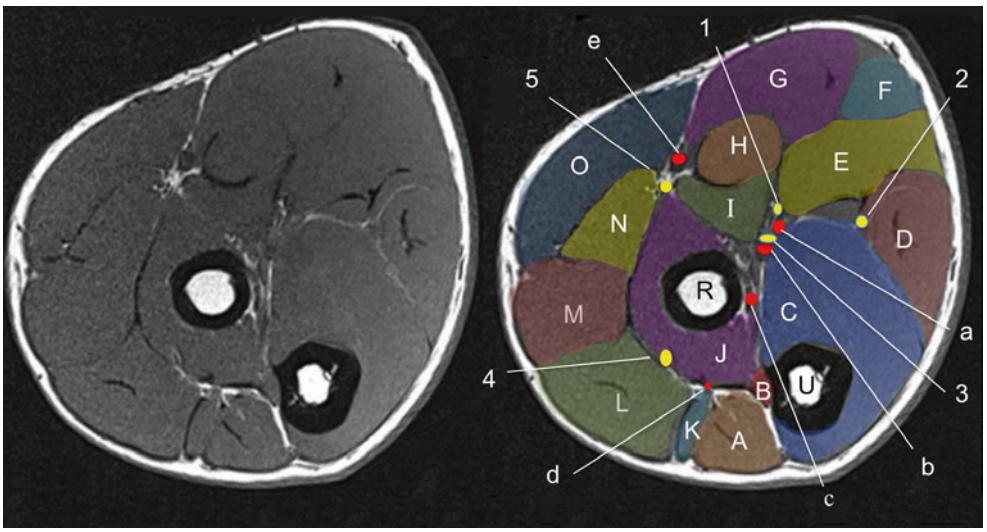

Fig. 36.9 MRI axial image of the distal forearm. *A* anconeus muscle, *B* biceps tendon, *C* flexor digitorum profundus muscle, *D* flexor carpi ulnaris muscle, *E* flexor digitorum superficialis muscle, *F* palmaris longus muscle, *G* flexor carpi radialis muscle, *H* pronator teres muscle (humeral head), *I* pronator teres muscle (ulnar head), *J* supinator muscle, *K* extensor digiti minimi muscle, *L* extensor digitorum muscle, *M* extensor carpi radialis brevis muscle, *N* extensor carpi radialis longus muscle, *O* brachioradialis muscle, *a* ulnar artery, *b* common interosseus artery, *c* posterior interosseus artery, *d* interosseus artery, *e* radial artery, *1* median nerve, *2* ulnar nerve, *3* anterior osseous nerve, *4* deep branch radial nerve, *5* superficial branch radial nerve (Image courtesy of Andrea Trescot, MD)

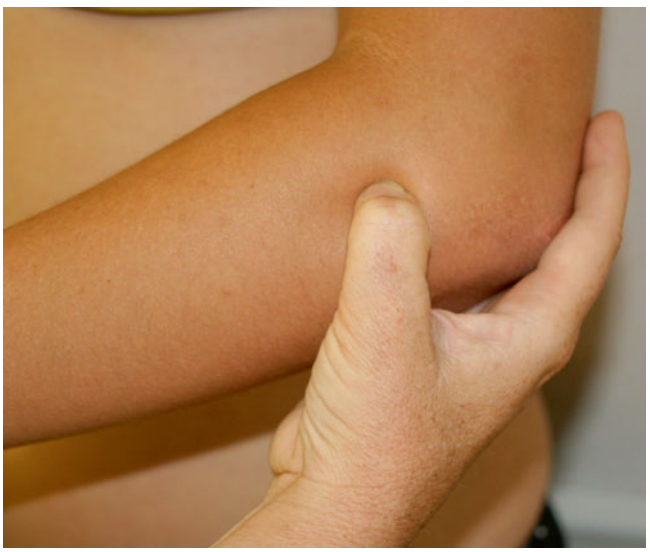

Fig. 36.10 Lateral epicondyle examination (Image courtesy of Andrea Trescot, MD)

Percussion of the mid-forearm where the SRN emerges about 9 cm proximal to the radial styloid may reproduce the patient's symptoms (*Tinel's sign*). Many patients have exacerbation of their symptoms when the patient encloses their own thumb in their fist and the examiner provides forced ulnar deviation (positive *Finkelstein test*) [14]. Patients may also have decreased pinch and grip strength due to pain.

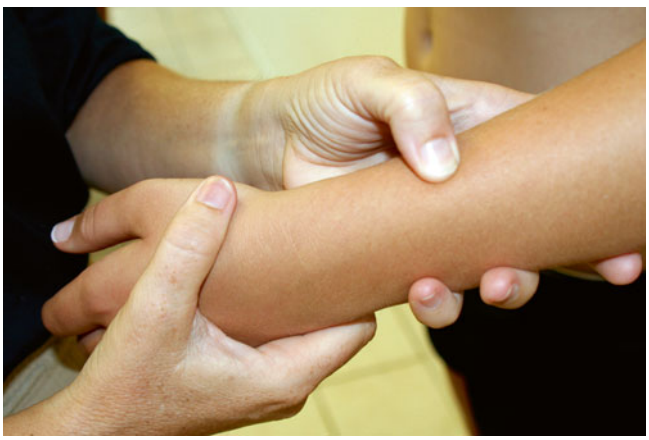

Fig. 36.11 Physical examination of the superficial radial nerve (Image courtesy of Andrea Trescot, MD)

Differential Diagnosis (Table 36.3)

A more proximal radial neuralgia, cervical radiculopathy, thumb MCP pathology, and de Quervain's tenosynovitis are part of the differential diagnosis of dorsoradial wrist pain. A patient with de Quervain's tenosynovitis will be tender over the radial styloid; that tenderness is easiest to elicit with the thumb abducted, as though one were hitchhiking. Patients who have cervical radiculopathy may have proximal muscle weakness and associated neck and/or shoulder pain. Careful examination of the MCP joint of the thumb should identify specific joint tenderness.

Table 36.3 Differential diagnosis of dorsoradial wrist pain

	Potential distinguishing features
de Quervain's tenosynovitis of the first extensor compartment (abductor pollicis longus and brevis tendons)	No Tinel over SRN, have pain with resisted thumb motion [7]. Pain and tenderness over the radial styloid. No paresthesias or increased pain with hyperpronation [15]. Associated with collagen vascular diseases, e.g., rheumatoid arthritis [15]
Lateral antebrachial cutaneous nerve (LABC) neuroma or neuritis	Block this nerve first [15]
More proximal radial nerve, brachial plexus, or cervical spine pathology	Weakness in the PIN-innervated or RN-innervated muscles
Thumb joint pathology	X-ray changes of MCP joint, crepitus, and tenderness over the MCP/PIP joints

Table 36.4 Diagnostic tests for superficial radial nerve entrapment

	Potential distinguishing features
Physical exam	Tenderness to palpation over the SRN
Provocative test	Forced forearm pronation reproduces symptoms within 1 min [7]
Diagnostic injection	Should improve pinch and grip limited by pain
Ultrasound	SRN is small at this level; easiest to locate if tracked proximal to distal
MRI	Not useful
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies	Helpful if positive [13]

The diagnostic studies for the superficial radial nerve are listed in Table 36.4

Identification and Treatment of Contributing Factors

Removing tight watches or jewelry, splinting, changing work ergonomics, and administration of anti-inflammatory medications are all first-line approaches to treatment. In one series, 71 % of 29 patients had excellent or good pain relief (three of these also had a steroid injection) after nonoperative interventions [8].

Ali et al. [16] noted that all of the branches of the SRN were found in the radial half of an isosceles triangle formed by the radial styloid, Lister's tubercle, and the exit of the SRN from underneath the BR (Fig. 36.7). However, they also identified an elliptical area just proximal to Lister's tubercle that was not crossed by any nerves or tendons and suggested this site as an appropriate area for the placement of *Kirschner wires* (K-wires) for pinning the distal forearm.

Fig. 36.12 Landmark-directed superficial radial injection (Image courtesy of Andrea Trescot, MD)

Injection Technique

Landmark-Guided Technique

The SRN is very superficial in the forearm, hence, the name. It is readily palpated and amenable to blind techniques. The index and middle fingers of the non-injecting hand straddle the nerve, similar to starting a wrist IV. A 27-gauge needle is then advanced (like an IV) toward the nerve at an acute angle (Video 36.2) (Fig. 36.12). A peripheral nerve stimulator (PNS) may help aid localization. Deposteroid and a small volume of local anesthetic (less than 2 cc) should be injected deeply enough in the tissues to avoid steroid atrophy of the skin, at the same time taking care not to injure the nerve.

Fluoroscopy-Guided Technique

There are no described fluoroscopic techniques.

Ultrasound-Guided Technique

The SRN is small at this level and difficult to see under ultrasound unless the exam is started proximally at the elbow and the radial nerve followed distally (Fig. 36.13). However, if the nerve itself is not visualized, the radial artery, adductor pollicis longus, and extensor pollicis brevis are easily seen, and a peripheral nerve stimulator can aid in its identification. The nerve can be injected using in-plane or out-of-plane techniques.

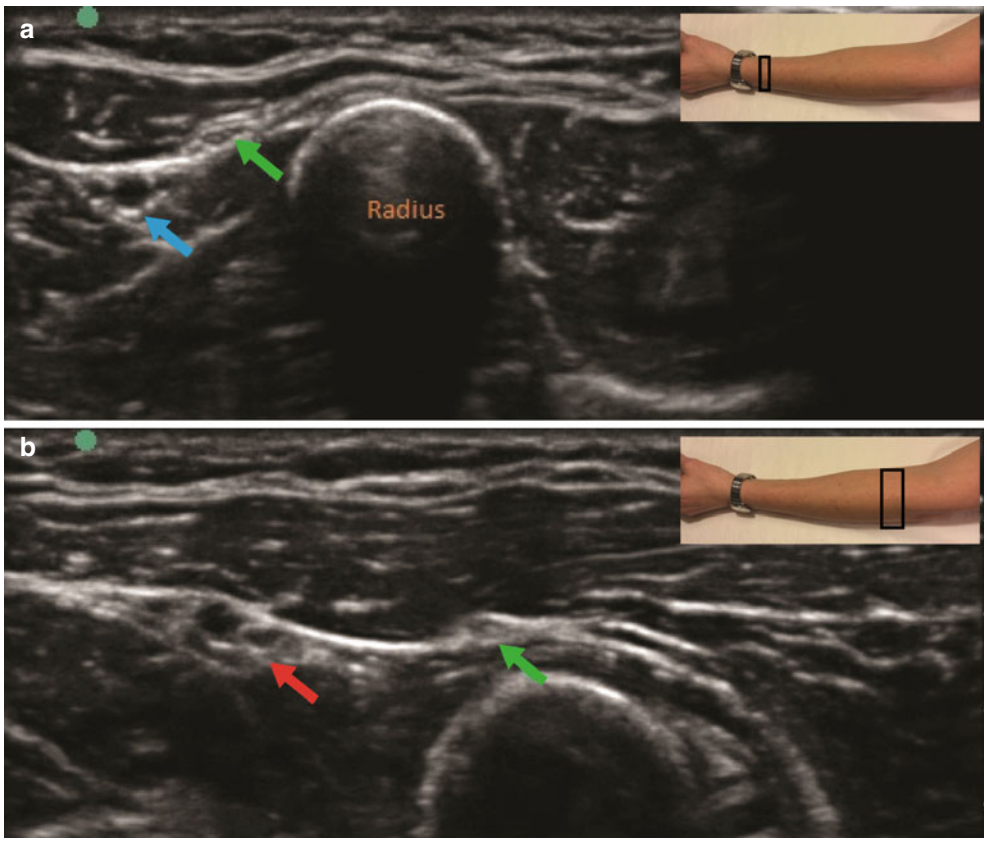

Fig. 36.13 Ultrasound anatomy of the distal radial nerve. (**a**) At the distal forearm superficial radial; (**b**) just below the elbow. Superficial radial nerve (*green arrow*), cephalic vein (*blue arrow*), deep radial nerve (*red arrow*) (Image courtesy of Agnes Stogicza, MD)

Neurolytic/Surgical Technique

Cryoneuroablation

When it is available, cryoneuroablation is the treatment of choice for patients who have not responded to the nonoperative interventions above. This is most easily done with a closed technique. The area of maximal tenderness is identified, usually between the brachioradialis and extensor carpi radialis muscles. If possible, a 12-gauge intravenous catheter is used, advanced proximally parallel to the nerve (Fig. 36.14). The 2.0 mm probe is then placed through the catheter. Sometimes the tissues are too thin to accept the larger probe, at which time a 14-gauge catheter and 1.4 mm probe must be used. Smaller gauge, handheld cryo devices such as iovera™ (see Chap. 8) may be useful in these situations.

Davies et al. described six patients treated with cryoneuroablation via open visualization of the nerve or neuroma [2]. Patients were followed for a mean of 11 months; all returned to work and reported good to excellent relief.

Radio-frequency Lesioning

Although RF lesioning (or pulsed RF lesioning) of the SRN has not been described in the literature, similar nerves have been successfully treated with pulsed radio frequency.

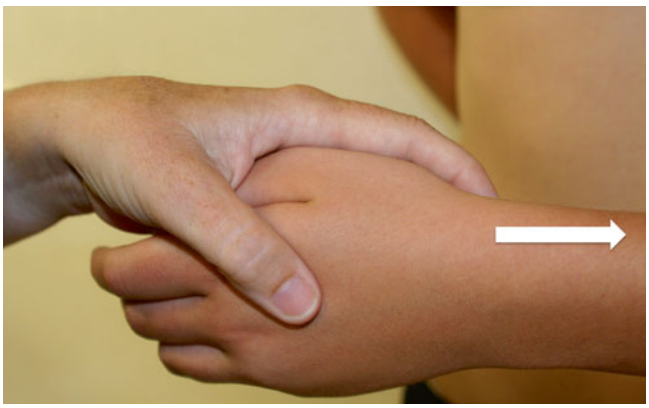

Fig. 36.14 Direction of the cryoprobe for cryoneuroablation of the superficial radial nerve (Image courtesy of Andrea Trescot, MD)

Surgery

Surgical exploration and release should be reserved for intractable cases. If this is required, the diagnosis of SRN entrapment should be clear, and the procedures should include neurolysis from all compressive structures and microsurgical internal neurolysis if necessary [7].

Braidwood [10] described 12 cases of SRN entrapment; 7 cases had a history of trauma (ORIF of the forearm, IV cutdown, radial fracture, surgical de Quervain's release), but the remaining 5 cases did not have a clear-cut cause of

entrapment. Two patients improved with steroid injections; the rest underwent excision of the nerve at the brachioradialis muscle. All of the patients were left with an area of hypoesthesia but "felt this was a small price to pay."

Complications

Because the nerve is superficial, steroid injections in this area have the potential to cause skin atrophy. Chodoroff described skin atrophy and SRN injury after an injection for de Quervain's tenosynovitis [3].

Summary

Superficial radial neuralgia can mimic a variety of conditions, including CRPS, carpal tunnel syndrome, and de Quervain's tenosynovitis. Appropriate diagnosis will lead to appropriate treatment.

References

1. Keefe DT, Lintner DM. Nerve injuries in the throwing elbow. Clin Sports Med. 2004;23(4):723–42, xi.
2. Davies E, Pounder D, Mansour S, Jeffery IT. Cryosurgery for chronic injuries of the cutaneous nerve in the upper limb. Analysis of a new open technique. J Bone Joint Surg Br. 2000;82(3):413–5.
3. Chodoroff G, Honet JC. Cheiralgia paresthetica and linear atrophy as a complication of local steroid injection. Arch Phys Med Rehabil. 1985;66(9):637–9.
4. Massey EW, Pleet AB. Handcuffs and cheiralgia paresthetica. Neurology. 1978;28(12):1312–3.
5. Abrams RA, Brown RA, Botte MJ. The superficial branch of the radial nerve: an anatomic study with surgical implications. J Hand Surg Am. 1992;17(6):1037–41.
6. Glanvill R, Boon JM, Birkholtz F, Meiring JH, van Schoor AN, Greyling L. Superficial radial nerve injury during standard K-wire fixation of uncomplicated distal radial fractures. Orthopedics. 2006;29(7):639–41.
7. Dellon AL, Mackinnon SE. Radial sensory nerve entrapment in the forearm. J Hand Surg Am. 1986;11(2):199–205.
8. Lanzetta M, Foucher G. Entrapment of the superficial branch of the radial nerve (Wartenberg's syndrome). A report of 52 cases. Int Orthop. 1993;17(6):342–5.
9. Lanzetta M, Foucher G. Association of Wartenberg's syndrome and De Quervain's disease: a series of 26 cases. Plast Reconstr Surg. 1995;96(2):408–12.
10. Braidwood AS. Superficial radial neuropathy. J Bone Joint Surg Br. 1975;57(3):380–3.
11. Stopford JSB. Neuritis produced by wristlet watch. Lancet. 1922;199:993–4.
12. Ehrlich W, Dellon AL, Mackinnon SE. Classical article: cheiralgia paresthetica (entrapment of the radial nerve). A translation in condensed form of Robert Wartenberg's original article published in 1932. J Hand Surg Am. 1986;11(2):196–9.
13. Dang AC, Rodner CM. Unusual compression neuropathies of the forearm, part I: radial nerve. J Hand Surg Am. 2009;34(10):1906–14.
14. Dellon AL, Mackinnon SE. Radial sensory nerve entrapment. Arch Neurol. 1986;43(8):833–5.
15. Saplys R, MacKinnon SE, Dellon AL. The relationship between nerve entrapment versus neuroma complications and the misdiagnosis of de Quervain's disease. Contemp Orthop. 1987;15(5):51–7.
16. Ali AM, El-Alfy B, Attia H. Is there a safe zone to avoid superficial radial nerve injury with Kirschner wire fixation in the treatment of distal radius? A cadaveric study. J Clin Orthop Trauma. 2014;5(4):240–4.

Median Nerve Entrapments

37

Virtaj Singh and William B. Ericson Jr.

Introduction

Median nerve (MN) entrapments are among the most commonly recognized nerve entrapments involving the upper extremity. The most well known is entrapment of the MN at the wrist, also known as *carpal tunnel syndrome* (CTS). In fact, CTS is the most commonly recognized entrapment neuropathy. Given the prevalence of CTS, the majority of this chapter will be devoted to this syndrome. Outside of the carpal tunnel, however, the median nerve is susceptible to entrapment at other, more proximal locations in the forearm. These conditions are described in general as *proximal median nerve entrapments* (PMNEs) and include *anterior interosseous nerve syndrome*, *Kiloh–Nevin syndrome*, *Parsonage–Turner syndrome*, *pronator teres syndrome*, *ligament of Struthers syndrome*, and *lacertus tunnel syndrome.*

Clinical Presentation (Tables 37.1 and 37.2)

These different median nerve entrapments present similarly in terms of symptoms, but the treatment varies depending on the location of entrapment, thus requiring a thorough knowledge of the anatomy of the median nerve throughout the upper extremity. All patients will present with pain in the volar forearm, wrist, or hand (Fig. 37.1). The pattern of pain at the palm seen with median nerve entrapments is shown in Fig. 37.2.

Table 37.1 Occupation/exercise/trauma history relevant to proximal median nerve entrapments

Trauma	Forearm trauma; external pressure on the forearm; forearm compartment syndrome [1]
	Surgery around the elbow [1]
Compression	Fibrous bands, anomalous muscles between the heads of the pronator teres
	Tourniquets
Brachial plexopathy	Neuralgic amyotrophy
	Parsonage–Turner syndrome
Positioning	Typing, writing, using a cell phone or mouse, driving
	Elbow extended/forearm supinated or elbow flexed with forearm pronated

Table 37.2 Occupation/exercise/trauma history relevant to distal median nerve entrapments

Decreased carpal tunnel space	Old trauma, deformity of distal radius or ulna, ectopic calcification in the canal, fibrosis of tenosynovium
	Persistent median artery present in 1–16 % of the hands [2] – thrombosis may cause sudden entrapment [3]
	AV malformation [4]
	Edema – pregnancy, hypothyroidism
Trauma	Colles' fracture, hook of the hamate fracture [5]
	Repetitive, forceful movements [6]
	Chronic vibration, steady pressure on the wrist [7]

Anterior Interosseous Nerve Syndrome (AINS)

Anterior interosseous nerve syndrome (AINS), also known as *Kiloh–Nevin syndrome*, is a MN syndrome that involves the branch of the MN known as the *anterior interosseous nerve* (AIN). The AIN is a mixed motor and sensory nerve (contrary

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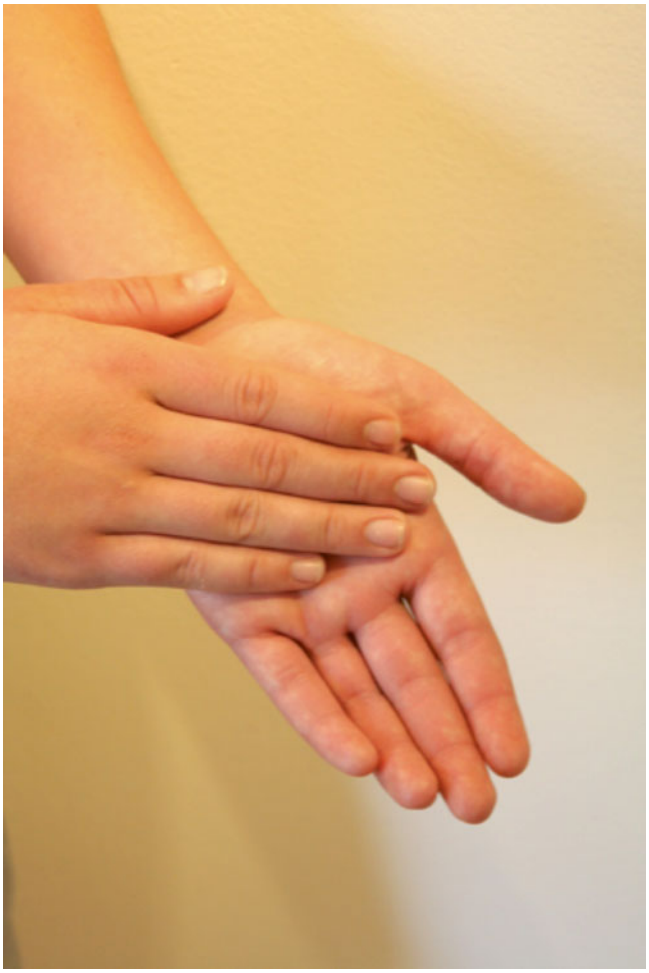

Fig. 37.1 Median nerve pain at the hand (Image courtesy of Andrea Trescot, MD)

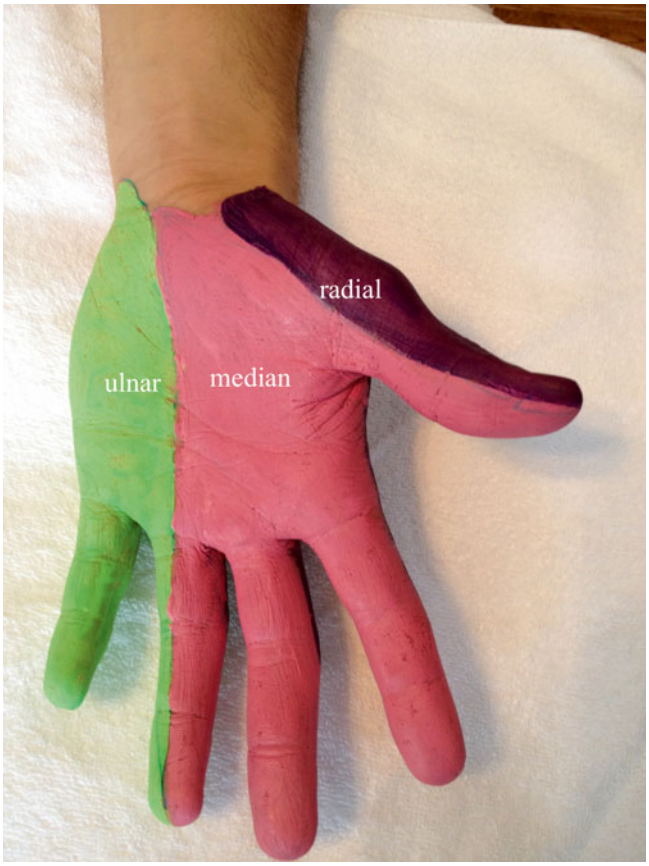

Fig. 37.2 Dermatome pattern of the palm (Image courtesy of Terri Dallas-Prunskis, MD)

to numerous references to the AIN as a "pure motor nerve" in the medical literature). The AIN is a motor nerve to the *flexor pollicis longus* (FPL), *flexor digitorum* profundus of the index finger (FDP IF), and *pronator quadratus* (PQ). It ends in a large sensory branch that innervates the *volar carpus* (i.e., the carpal tunnel bones). The patient presents with an acute palsy of the motor part of the AIN, which may be preceded by pain in the wrist or hand, in the distribution of the sensory branch of the AIN. AINS can be due to forearm trauma, external pressure on the proximal forearm (e.g., while using a sling following shoulder surgery), forearm compartment syndrome, anatomic variations in the forearm related to fibrous bands or anomalous muscles, and idiopathic brachial plexopathy (i.e., *neuralgic amyotrophy* or *Parsonage–Turner syndrome*).

Pronator Teres Syndrome

Pronator teres syndrome refers to entrapment of the median nerve between the two heads of the *pronator teres muscle* (PT). This syndrome mimics CTS, but it does not respond to splinting, NSAIDs, steroid injection, or carpal tunnel surgery. The clinical presentation is typically aching pain in the hand/wrist with activities involving sustained pronation, such as typing, writing, using a mouse or cell phone, or driving a car. The pain is in the distribution of the terminal, sensory branch of the AIN (i.e., the volar wrist). As the syndrome progresses, patients may complain of numbness in the entire hand, particularly at night, especially with any pressure on the proximal forearm or about the medial elbow.

In theory, a way to differentiate CTS from pronator teres syndrome is the presence of sensory symptoms in the thenar eminence. The palmar cutaneous branch of the median nerve travels outside the carpal tunnel and provides sensation in this area. There is weakness of the muscles distal to the site of compression, which typically involves the FPL, FDP IF, and PQ. The pronator teres is ironically spared in pronator teres syndrome. It is important to note that there are no symptoms at the site of nerve compression, but rather where the nerve ends.

Ligament of Struthers Syndrome

As its name implies, *ligament of Struthers syndrom*e is due to entrapment of the median nerve at the *Struthers ligament* (discussed further below). This syndrome presents similarly

to pronator teres syndrome, except that it also includes weakness of the PT, FCR, and PL muscles and greater forearm pain relative to hand/wrist pain.

Lacertus Tunnel Syndrome

The *lacertus fibrosus* is a tendinous attachment to the biceps tendon that wraps around the proximal medial forearm musculature. The lacertus fibrosus is tight when the elbow is in extension combined with the forearm in supination and when the elbow is flexed in combination with forearm pronation. The complaints are vague pain in the distribution of the median nerve distal to the site of compression and forearm/hand fatigue and achiness.

Proximal Median Nerve Entrapment (PMNE)

This term is used to describe any and all entrapments proximal to the carpal tunnel. It is the opinion of these authors that there is a distinct syndrome that encompasses low-grade compression of the median nerve at multiple locations about the elbow and that the specific entrapment needs to be evaluated. The surgical approach to these syndromes is discussed further below.

Carpal Tunnel Syndrome (CTS)

Paget described CTS in 1854 [6]. CTS is the most common entrapment neuropathy of the upper limb, with a reported population prevalence of 3–6 % [7]. Carpal tunnel syndrome classically presents with (A) intermittent aching pain in the wrist and/or (B) numbness at the tips of the thumb, index finger, middle finger, and radial half of the ring finger (i.e., the sensory distribution of the median nerve in the hand) and (C) weakness, specifically of the *opponens muscles* of the thumb. Although these symptoms characterize the classic presentation, CTS can present with numbness in any of the digits. The numbness is most often worse first thing in the morning but can also cause nighttime awakening. In more advanced cases, patients can develop severe thumb weakness with atrophy of the opponens muscles. Although traumatic cases of CTS exist, the usual onset is insidious, with gradual worsening over time. Patients tend to "flick" or shake the affected hand and wrist to try to relieve discomfort. Pryse-Phillips [8] found that 93 % of patients with CTS identify this activity, but, according to Roquer and Herraiz [9], it has no correlation with compression severity.

Anatomy (Table 37.3)

The MN is formed from the medial and lateral cords of the brachial plexus as they combine within the axilla. The median nerve has contributions from the fifth, sixth, seventh, and eighth cervical nerve roots and the first thoracic spinal nerve (thus, essentially all of the spinal nerves within the brachial plexus contribute to the median nerve). The lateral cord provides fibers from C5 to 7, and the medial cord supplies fibers from C8 to T1.

As the MN travels in the arm adjacent to the *brachial artery*, it provides no innervation to any muscle in the upper arm (Fig. 37.3). Proximal to the elbow, a motor branch to the PT muscle exits the main nerve. The MN enters the *antecubital fossa* where it remains accompanied by the brachial artery. The MN is usually medial and posterior to the brachial artery in the antecubital fossa. Distal to the antecubital fossa, the median nerve continues between the two heads of the PT to enter the forearm (Fig. 37.3). As the MN crosses the elbow, it gives off anterior branches to the PL and *flexor carpi radialis* (FCR).

When the MN pierces the pronator teres muscle, it divides to give off its largest branch, the AIN (Fig. 37.4). As described above, the AIN is a mixed motor and sensory nerve. The AIN innervates the FDP of the index finger, the FPL, and PQ. The nerve continues on with a large sensory branch to the *volar carpus*, also known as the *palmar carpal ligament*, which forms the ceiling of the carpal tunnel. The

Table 37.3 Median nerve anatomy

Origin	Contributions from the fifth, sixth, seventh, eighth cervical and first thoracic nerve roots; medial cord (C5–7) and lateral cord (C8–T1) of the brachial plexus
General route	From brachial plexus, medial and posterior to the brachial artery in the antecubital fossa, to between the two heads of the pronator teres. AIN splits off; the rest of nerve passes under the transverse carpal ligament (carpal tunnel)
Sensory distribution	No upper arm innervation; AIN sensory branch to the carpal bones; skin innervation – volar thumb/index/middle fingers, radial half of ring finger; palmar cutaneous branch to thenar area
Motor innervation	No upper arm motor innervation; pronator teres (PT), palmaris longus (PL), flexor carpi radialis (FCR) branches proximal or at the elbow; lumbricales; the thenar muscles from branch distal to the carpal tunnel
Anatomic variability	Numerous anatomic variations of the median nerve have been described in areas of the brachial plexus [1], arm [10], forearm [11], and carpal tunnel [12]
Other relevant structures	In the region of the elbow, relevant structures are the arcade of Struthers, fascia of Struthers, ligament of Struthers, the most superficial proximal fascia of the flexor/pronator muscles, the fascia of the ulnar origin of the pronator teres, and the arch of the superficial flexors; the bicipital aponeurosis may also be tight

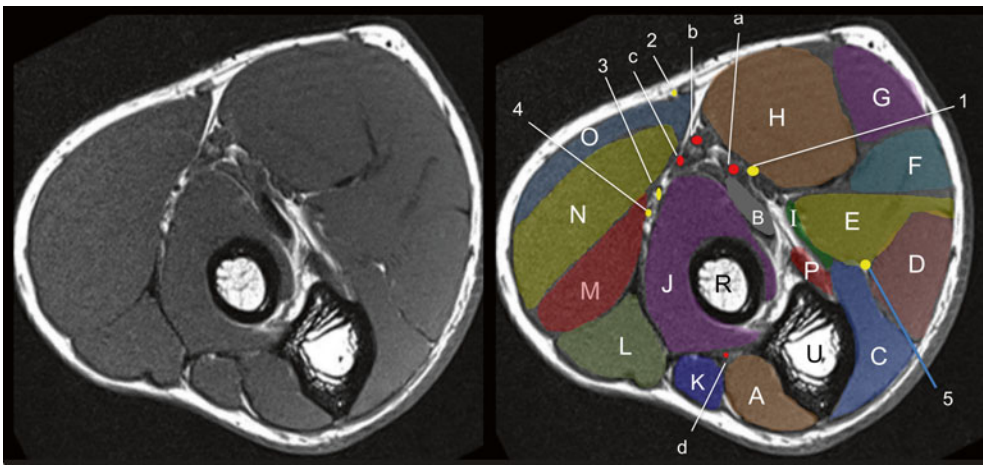

Fig. 37.3 MRI axial image of the proximal forearm. *A* anconeus muscle, *B* biceps tendon, *C* flexor digitorum profundus muscle, *D* flexor carpi ulnaris muscle, *E* flexor digitorum superficialis muscle, *F* palmaris longus muscle, *G* flexor carpi radialis muscle, *H* pronator teres muscle (humeral head), *I* pronator teres muscle (ulnar head), *J* supinator muscle, *K* extensor digiti minimi muscle, *L* extensor digitorum muscle, *M* extensor carpi radialis brevis muscle, *N* extensor carpi radialis longus muscle, *O* brachioradialis muscle, *P* brachialis muscle, *a* ulnar artery, *b* radial artery, *c* radial recurrent artery, *d* recurrent interosseus artery, *1* median nerve, *2* lateral cutaneous nerve of the forearm, *3* superficial branch radial nerve, *4* deep branch radial nerve, *5* ulnar nerve (Image courtesy of Andrea Trescot, MD)

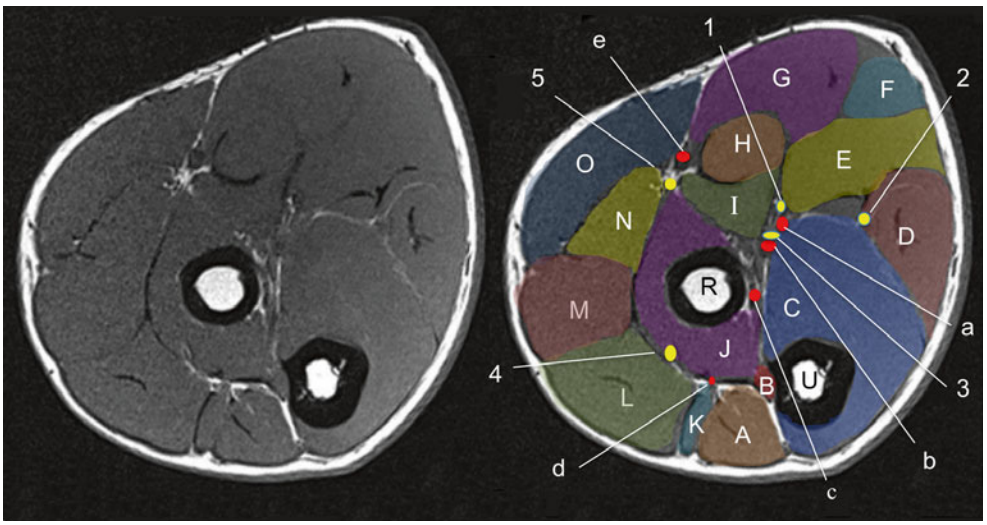

Fig. 37.4 MRI axial image of the distal forearm. *A* anconeus muscle, *B* extensor pollicis longus muscle, *C* flexor digitorum profundus muscle, *D* flexor carpi ulnaris muscle, *E* flexor digitorum superficialis muscle, *F* palmaris longus muscle, *G* flexor carpi radialis muscle, *H* pronator teres muscle (humeral head), *I* pronator teres muscle (ulnar head), *J* supinator muscle, *K* extensor digiti minimi muscle, *L* extensor digitorum muscle, *M* extensor carpi radialis brevis muscle, *N* extensor carpi radialis longus muscle, *O* brachioradialis muscle, *a* ulnar artery, *b* common interosseus artery, *c* posterior interosseus artery, *d* interosseus artery, *e* radial artery, *1* median nerve, *2* ulnar nerve, *3* anterior osseous nerve, *4* deep branch radial nerve, *5* superficial branch radial nerve (Image courtesy of Andrea Trescot, MD)

AIN does not provide sensory innervation to the skin, but rather to the bones of the carpal tunnel. Compression of the AIN at the elbow causes referred pain in the wrist.

The portion of the median nerve that does not separate off to become the AIN continues on through the forearm (Fig. 37.4). Approximately 5 cm proximal to the carpal tunnel, the *palmar cutaneous branch* separates from the main nerve and continues on to the skin over the thenar eminence. The remaining median nerve passes through the carpal tunnel to enter the palm (Figs. 37.5, 37.6, and 37.7). After passing under the *transverse carpal ligament*, the median nerve gives off innervation to the first and second lumbricales and then gives off the recurrent thenar branch to the *abductor pollicis brevis* (APB), superficial head of the *flexor pollicis brevis* (FPB), and the *opponens pollicis* (OP).

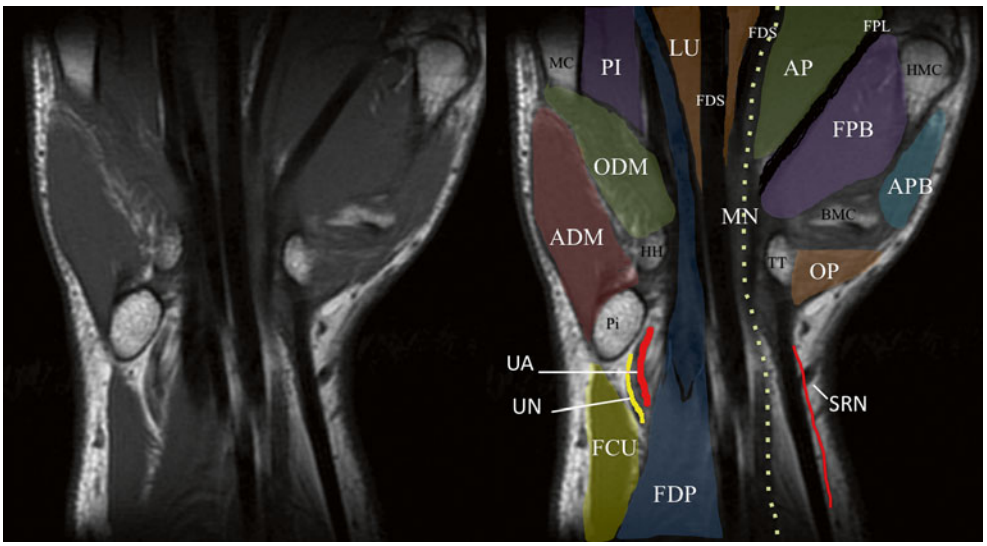

Fig. 37.5 Wrist coronal MRI. *ADM* abductor digiti minimi muscle, *AP* adductor pollicis muscle, *APB* abductor pollicis brevis muscle, *BMC* base of metacarpal bone, *FCU* flexor carpi ulnaris muscle, *FDP* flexor digitorum profundus muscle, *FDS* flexor digitorum superficialis tendon, *FPB* flexor pollicis brevis muscle, *FPL* flexor pollicis longus tendon, *HH* hook of the hamate bone, *HMC* head of the metacarpal bone, *LU* lumbrical muscle, *MC* metacarpal bone, *MN* median nerve, *ODM* opponens digiti minimi, *muscle OP* opponens pollicis, *Pi* pisiform bone, *PI* palmar interossei muscle, *RA* radial artery, *SRN* superficial radial nerve, *TT* trapezium tubercle bone, *UA* ulnar artery, *UN* ulnar nerve (Image courtesy of Andrea Trescot, MD)

The distal MN then provides sensation to the radial aspect of the palm; the volar surfaces of the thumb, index, and middle fingers; and the radial half of the ring finger, particularly at the tips of these digits.

Entrapment

Entrapment of the median nerve can occur at or above the elbow ("high lesions"), in the forearm, or within the carpal tunnel (carpal tunnel syndrome).

Proximal Median Nerve Entrapment (PMNE)

The median nerve can be affected in the upper arm, primarily due to compression such as tourniquets. However, there can be entrapment by the *ligament of Struthers*, which is an anatomic variation consisting of a fascial band extending from the humerus to the medial epicondyle, often with a bony protuberance from the humerus. These "high lesions" can result in attenuation of all motor and sensory function of the median nerve below the elbow. There can be *thenar wasting*, described as a "*simian*" or "*benediction*" *hand* [13]. There can also be numbness of the palmar aspect of the first three and a half fingers, including the thenar eminence.

Rather than discuss each area of proximal entrapment separately (i.e., ligament of Struthers, pronator teres, AINS), we are discussing them in combination, as this format lends itself to the discussion below of a novel surgical treatment for PMNE. Given that the same nerve serves as the culprit in both CTS and PMNE and that symptoms are experienced where the nerve ends rather than where the nerve is compressed, it is not surprising that the differences between these conditions are very subtle.

Carpal Tunnel Syndrome

The *carpal tunnel* refers to the passageway on the palmar side of the wrist through which the median nerve passes en route from the forearm to the hand. The tunnel is covered on the volar side by the *transverse carpal ligament* and on the dorsal side by the carpal bones. In addition to the median nerve, nine separate flexor tendons pass through the carpal tunnel, including two to each finger (FDP and FDS) and one to the thumb (FPL). Given the number of structures that pass through this limited space, it is clear why this area is at risk for entrapment. The tenosynovial linings of the tendons, which nourish and lubricate the tendons, are subject to swelling and fibrosis. The carpal tunnel cannot expand, and the median nerve is subject to compression within the tunnel from changes in the size of the lining of the tendons.

Not to be forgotten, the median nerve can be entrapped by scar tissue from previous trauma or even carpal tunnel surgery. These patients often have good relief initially after

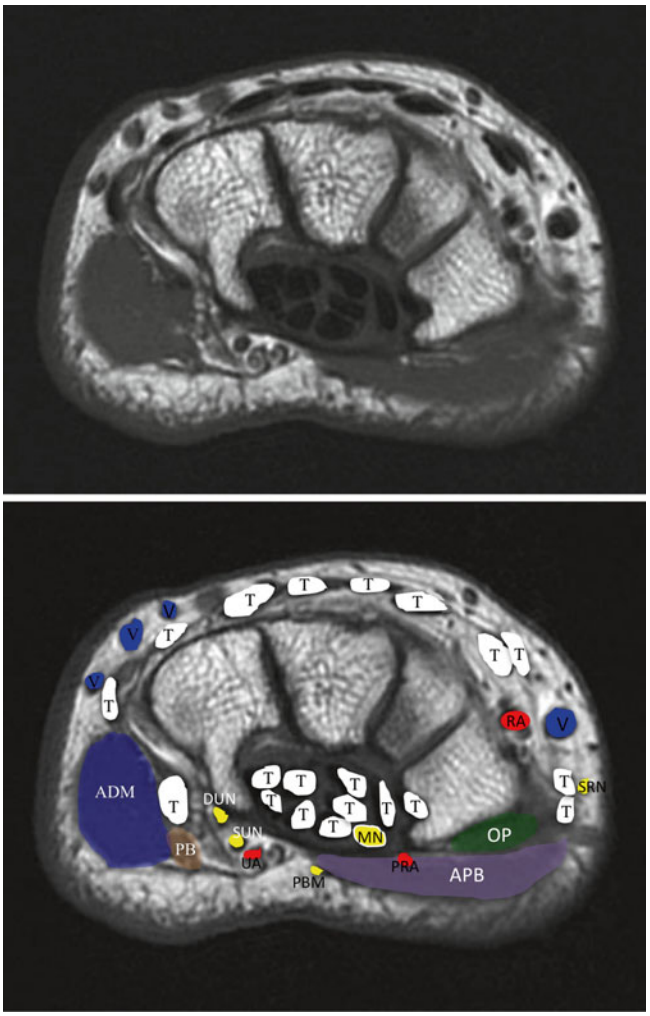

Fig. 37.6 Wrist axial MRI. *A* artery, *APB* abductor pollicis brevis, *DUN* deep branch of the ulnar nerve, *MN* median nerve, *OP* opponens pollicis, *PB* palmaris brevis, *PMN* palmar branch of the median nerve, *PRA* palmar radial artery, *RA* radial artery, *SRN* superficial branch of the radial nerve, *SUN* superficial branch of the ulnar nerve, *T* tendon, *UA* ulnar artery, *V* vein (Image courtesy of Andrea Trescot, MD)

surgery, unfortunately followed by a gradual return of symptoms. Scar neuromas (another type of entrapment) can also form within the scar itself.

Physical Exam

The physical examination for possible median nerve entrapment requires a thorough understanding of upper extremity anatomy and pathophysiology, as well as a thorough musculoskeletal examination, keeping in mind the various diagnoses discussed below that can mimic or exist concomitantly with median nerve entrapments. The examination should include a cervical spine examination to evaluate for cervical radiculopathy, as well as a shoulder area examination for brachial plexopathy. Providing compression of the cervical spine nerve roots (*Spurling's sign*) or traction across the brachial plexus (*upper limb tension test*) (Fig. 37.8) can help to rule these possibilities in or out. Examination of the median nerve at the elbow (Video 37.1) can help to differentiate median nerve entrapment at the elbow from other elbow pathologies such as lateral epicondylitis. Evaluation of reflexes can also help assess whether there is neural impingement at the spinal cord, nerve root, or plexus level. With regard to the neuromuscular examination, there should be a considerable focus on evaluating subtle weakness involving distal median nerve-innervated muscles. In profound cases, thenar atrophy may become apparent, but this may not be the case with a more subtle neuropathy.

One must carefully evaluate the strength of the *abductor pollicis brevis* (APB) and other muscles discussed above. A common, but incorrect, way to evaluate for AIN weakness is the "*OK sign*" (Fig. 37.9). The classic "OK sign" implies intact strength of the AIN-innervated muscles (FPL and FDP IF) but ignores the Blix length–tension curve; strength testing of these flexors must be done with the wrist in neutral position, so one is testing only tension from contraction and not tension from stretch. Isolated APB weakness probably represents CTS, but one must assess whether this weakness is true motor weakness or related to some other condition, such as pain from arthrosis of the thumb carpometacarpal (CMC) joint, which is a common malady. Similar attention must be placed on evaluating the distribution of any sensory deficit (e.g., *two-point discrimination*, *sensory perception threshold*, and pain). Hypoesthesia of the index finger, as opposed to the small finger, is likely a distal median nerve entrapment pattern, but it could also represent a C6 radiculopathy. The distribution of weakness and sensory loss is key in differentiating the various syndromes discussed above.

Specific to carpal tunnel syndrome, common tests include the *carpal compression test*, *Phalen's test*, and *Tinel's sign* at the wrist. Among these tests, the carpal compression test has demonstrated the best sensitivity and specificity (87 % and 90 %, respectively) [14]. As its name implies, the carpal compression test has the examiner use his/her thumb to compress the carpal tunnel over the median nerve and evaluate for recreation of symptoms (Video 37.2) (Fig. 37.10). Phalen's test [15] is less specific and simply uses passive wrist flexion to increase the pressure within the carpal tunnel. Interestingly, the *reverse Phalen's test* (Fig. 37.11) significantly increases intracanal pressure (to a greater extent than the standard Phalen's test), resulting in a test with greater sensitivity [16]. The *Hoffmann–Tinel sign* simply requires tapping over any nerve segment (including the carpal tunnel) and evaluating for reproduction of symptoms.

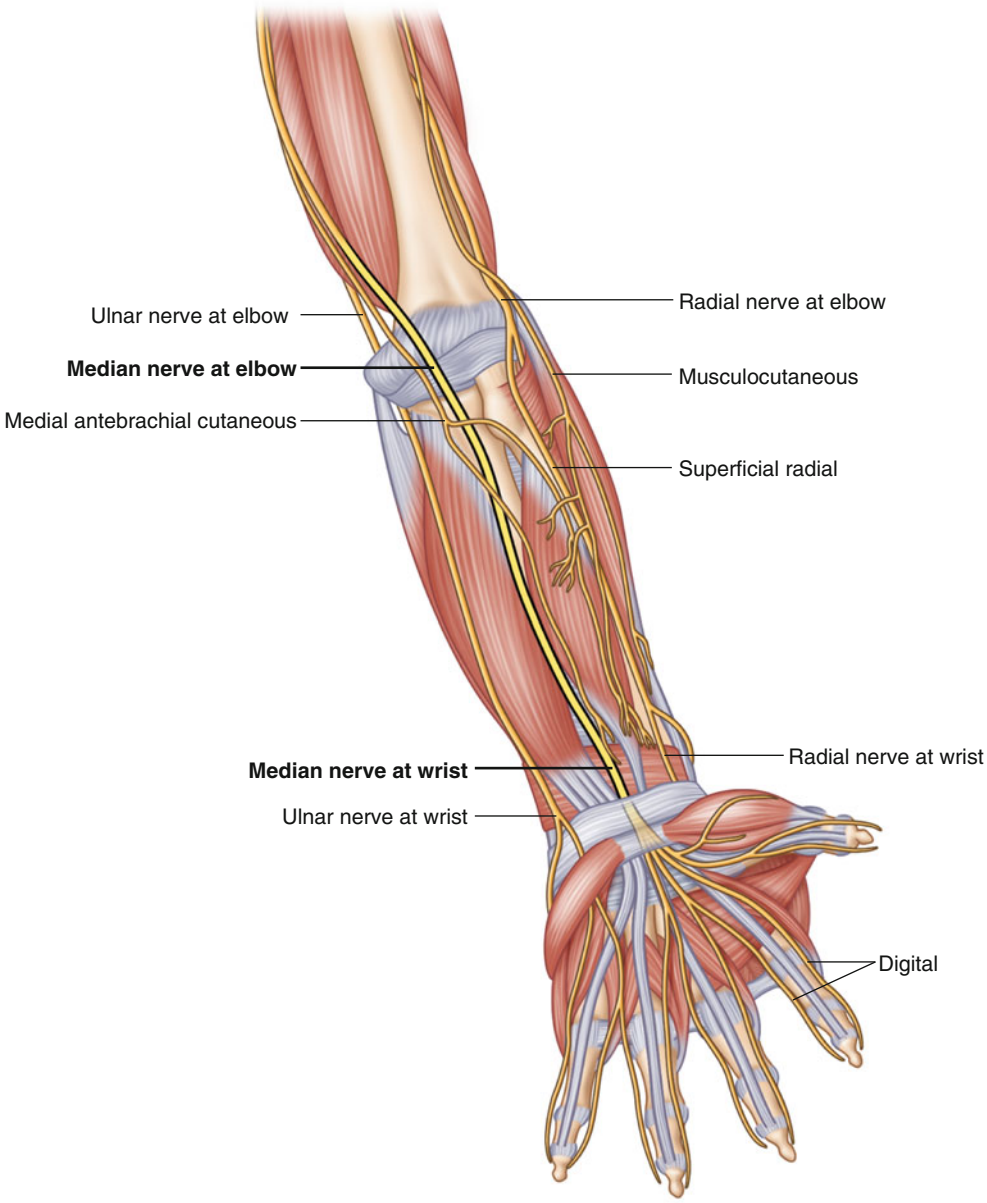

Fig. 37.7 Anatomy of the upper arm, with emphasis on the median nerve (Image by Springer)

Fig. 37.8 Upper limb tension test (ULTT) (Image courtesy of Virtaj Singh, MD)

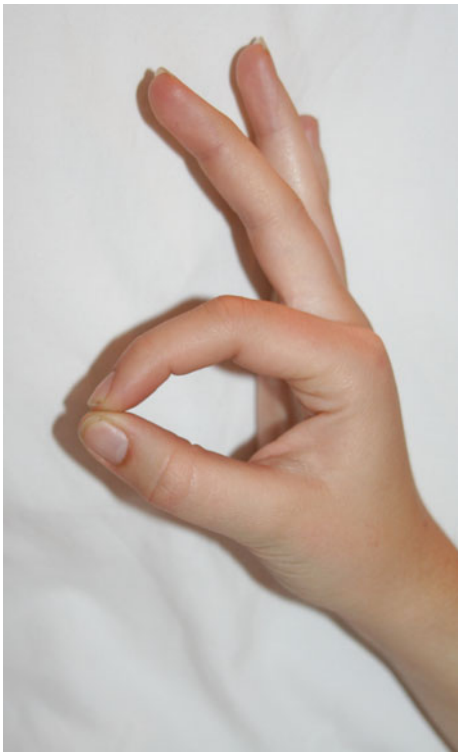

Fig. 37.9 "OK" sign; weakness (i.e., inability to keep fingers together) may be a sign of adductor pollicis brevis (APB) weakness (Image courtesy of Virtaj Singh, MD)

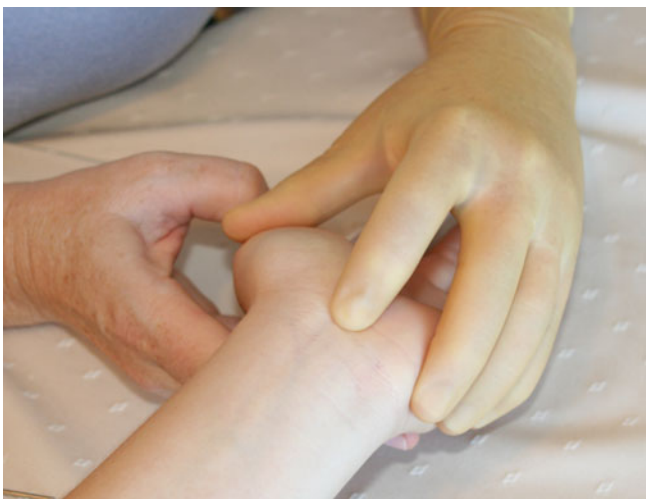

Fig. 37.10 Carpal compression test (Image courtesy of Andrea Trescot, MD)

This test can be performed at the wrist for CTS, but it lacks sensitivity [17]. The *square-shaped wrist evaluation* is the ratio of the anterior–posterior wrist dimension divided by the mediolateral dimension when measured by MRI or ultrasound; if the ratio is greater than 0.70, there may be an increased risk of CTS [6].

Fig. 37.11 Reverse Phalen's test (Image courtesy of Andrea Trescot, MD)

Differential Diagnosis (Tables 37.4 and 37.5)

A variety of different nerve entrapments can present in a manner similar to a median nerve entrapment. In addition, other more proximal nerve entrapments can present concomitantly with median nerve entrapment. Cervical radiculopathy, especially in C6, can present with dysesthesias or pain in the region of the carpal tunnel, similar to CTS. *Neurogenic thoracic outlet syndrome* (see Chap. 33) can also present similarly, although it more commonly mimics an ulnar neuropathy in terms of sensory distribution (see Chap. 38). *Wartenberg's syndrome* causes dysesthesias in the distribution of the superficial sensory branch of the radial nerve (see Chap. 36). *FCR tendonitis* causes pain at the volar radial wrist. Instability as well as arthritis of the thumb carpometacarpal (CMC) joint can cause vague intermittent pain at the volar radial wrist, adjacent to the carpal tunnel. Instability and arthritis of the wrist can cause vague intermittent aching pain in the carpal tunnel area. *De Quervain's tenosynovitis* causes aching pain in the wrist area. A volar wrist *ganglion cyst* may cause aching pain in the wrist in the region of the carpal tunnel. Weakness in the hand may be related to any of these painful conditions.

Diagnostic Tests (Table 37.6)

Kuhlman and Hennessey [20] looked at six carpal tunnel physical exams (Phalen's test, Hoffmann–Tinel sign, hypoesthesia of the index finger, abductor pollicis brevis weakness, median nerve compression, square-shaped wrist) and compared these to the results of nerve conduction studies; the signs were not sensitive, but they were "fairly specific." These authors concluded that the square-shaped wrist was the most sensitive sign.

Table 37.4 Differential diagnosis of upper arm, elbow, or forearm pain

	Potential distinguishing features
Cervical radiculopathy (especially C6)	Dermatomal pain and weakness, decreased brachioradialis reflex, positive Spurling's sign
Brachial plexopathy	Positive upper limb tension test
Neurogenic thoracic outlet syndrome	Usually mimics ulnar, not median entrapment

Table 37.5 Differential diagnosis of wrist pain

	Potential distinguishing features
Wartenberg's syndrome	Superficial radial nerve entrapment in the mid-forearm; causes pain at the radial wrist and hand with ulnar deviation of hand
FCR tendonitis	Pain and swelling at the volar radial wrist
Thumb CMC joint instability	Volar radial wrist pain, tenderness around thumb CMC joint, pain with subluxation
Thumb CMC joint arthritis	Volar radial wrist pain, tenderness around thumb CMC joint, positive "grind" test
Arthritis of the wrist	Tenderness and swelling over the carpal bones, abnormal radiographs
De Quervain's tenosynovitis	Tenderness and swelling at the radial aspect of the distal radius, positive Finkelstein test
Volar ganglion cyst	Palpable cyst or occult (seen on MRI)
Carpal instability	Abnormal wrist motion underload; scaphoid shift test causes subluxation and reproduces wrist pain

Table 37.6 Diagnostic tests for median nerve entrapment

	Potential distinguishing features
Physical exam	Hoffmann–Tinel sign, hypoesthesia of the index finger, abductor pollicis brevis weakness, square-shaped wrist
Diagnostic injection	Potentially therapeutic as well as diagnostic
Ultrasound	Can show compression/swelling of the nerve[a]
MRI	Can show compression/swelling of the nerve[a] [18]
Arteriography	May show anomalous artery [2] or AVM [4]
X-ray	May show bony deformities
Electrodiagnostic studies	Can show slowing across the entrapment site(s)
Provocative tests	Phalen's and reverse Phalen's tests, median nerve compression

[a]Classic triad: (1) nerve flattening in the distal carpal tunnel, (2) nerve swelling within the proximal tunnel itself, and (3) palmar bowing of the flexor retinaculum [19]

Identification and Treatment of Contributing Factors

CTS prevalence tends to increase with age and is much more common in women than men. CTS is also associated with a number of medical conditions including rheumatoid arthritis (3.6 times increased risk), diabetes mellitus (2.3 times increased risk), and pregnancy (2.5 times increased risk) [21]. Carpal tunnel syndrome is also associated with certain occupations. Interestingly, there has been the recognition that patients with CTS are more than twice as likely to have migraine headaches [22], based on a survey of nearly 26,000 Americans, suggesting that "migraines" may also be compressive neuropathies (See Sect. 2).

Contrary to popular belief, CTS is not directly associated with computer keyboard use but is instead associated with occupations that require prolonged use of handheld vibratory tools and those with prolonged and repetitive flexion and extension of the wrist [23].

Patients should be advised to avoid and/or modify activities that trigger CTS. Carlson et al. [24] described the wide variety of nonsurgical options available to treat and avoid CTS, including physical therapy, bracing, steroid injections (see above), yoga, acupuncture, lasers, and magnets. The current literature suggests that bracing and corticosteroid injections may be useful, although the benefits may be short-term; there is limited evidence regarding the efficacy of other treatments.

Injection Technique

Landmark-Guided Technique

Injections for the median nerve can be done using surface anatomy (i.e., without imaging guidance). The technique is generally believed to be safe, as long as one is careful not to inject a nerve, artery, or tendon directly. Needle advancement that causes sharp pain or paresthesias is suggestive of possible incorrect placement, in which case the needle must be removed and redirected.

If the location of the nerve entrapment is easily palpable, such as in the antecubital fossa, the needle can be directed parallel to the nerve, taking care to avoid nerve injury (Video 37.3) (Fig. 37.12). Be aware that large volumes (greater than 2–3 cc) of injectate can increase the pressure in an area that is possibly already tight and therefore should be avoided.

Injections into the carpal tunnel (Video 37.4) (Fig. 37.13) are useful for diagnosis and treatment of carpal tunnel syndrome, but can also be used to perform a lysis of adhesions after surgery. To begin, the patient can either be seated or supine, with the forearm supinated with the palm facing up and the wrist neutral. The flexor carpi radialis (FCR) and the palmaris longus (PL) (if present) are identified by palpation. The injection is done at the wrist, just ulnar to the PL tendon, under sterile conditions. The injection must be radial to the ulnar neurovascular bundle and ulnar to the median nerve, which is typically deep to the PL tendon. As the needle is advanced, there should be minimal pain,

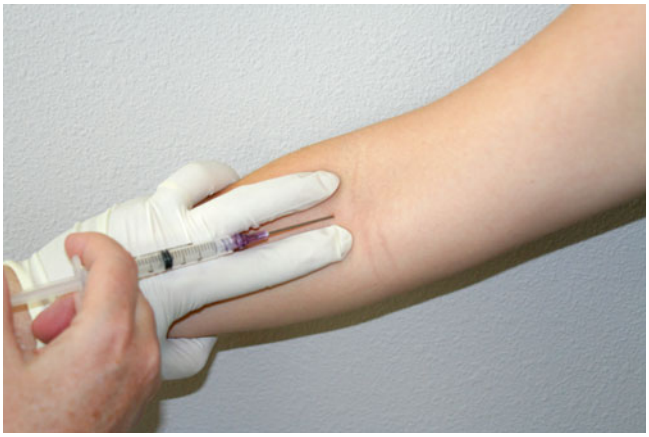

Fig. 37.12 Landmark-guided injection of the median nerve at the elbow (Image courtesy of Andrea Trescot, MD)

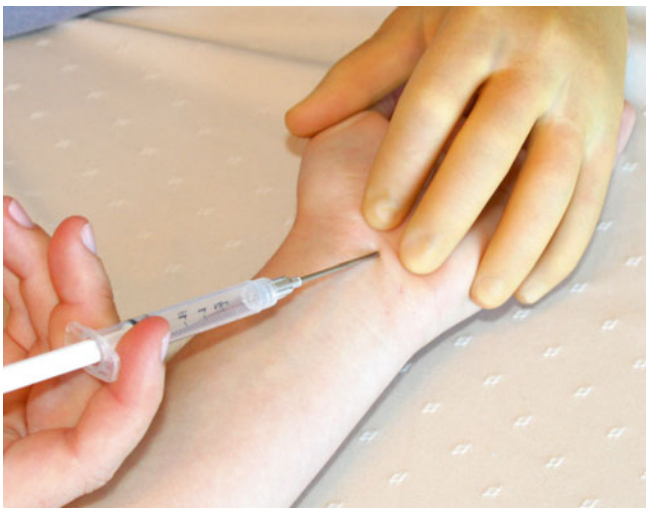

Fig. 37.13 Landmark-guided injection into the carpal tunnel (Image courtesy of Andrea Trescot, MD)

except as the skin is punctured, and there should be no resistance. If arterial blood is encountered, one must remove the needle and redirect. One should move the patient's fingers from flexion to extension to ensure that the injection will not be into a tendon. The local anesthetic and steroid solution is then slowly injected into the tenosynovium lining the tendons at the wrist. Direct intraneural, intra-arterial, or intratendinous injections can cause severe, irreversible complications, but these can be avoided using careful technique.

Fluoroscopy-Guided Technique

For fluoroscopic injections of the median nerve at the elbow, the patient should be supine with the arm extended

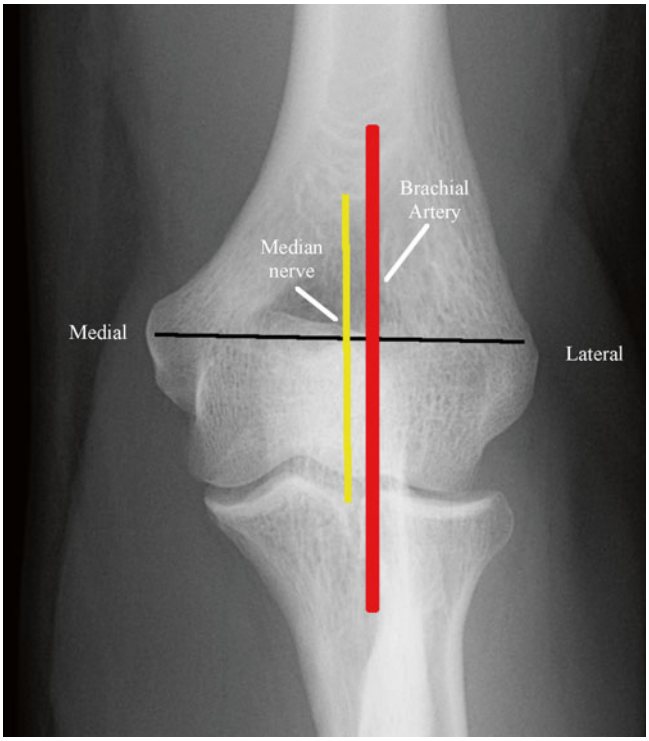

Fig. 37.14 Fluoroscopic image of the elbow (Image courtesy of Andrea Trescot, MD)

and the elbow also extended. The median nerve will be found medially to the brachial artery at the distal humerus. Position the C-arm straight A–P, prep and drape the flexor surface of the elbow, and identify the midpoint between the medial and lateral epicondyle (Fig. 37.14). Inject subcutaneous local anesthetic, and then insert the blunt, beveled, or stimulating needle vertically, just at the medial or lateral border of the humerus. Slowly insert the needle until the peripheral nerve stimulator produces the appropriate paresthetic response. If the humerus is touched, the needle is too deep and must be withdrawn. It is advisable to use a wrist restraint so that an inadvertent movement by the patient does not cause damage from movement of the needle. When aspiration is negative, the therapeutic solution (no more than 1 mL) may be injected.

For the carpal tunnel fluoroscopic injection, the hand is positioned supine with the wrist slightly extended, using a similar needle approach as the landmark-guided technique, with the needle passing from proximal to distal underneath the carpal ligament. A blunt-tipped needle should decrease the risk of nerve injury. Contrast is injected (Fig. 37.15), and a fluid hydrodissection can be performed, especially if prior surgery has resulted in scarring of the median nerve. However, there must be adequate runoff of the fluid (proven with the contrast) to avoid compression of the nerve.

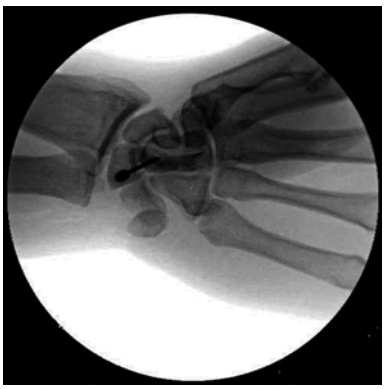

Fig. 37.15 Fluoroscopic image of contrast in the carpal tunnel (Image courtesy of Andrea Trescot, MD)

Ultrasound-Guided Technique

Ultrasound guidance allows for precise placement of the needle, and it can allow one to inject safely if one is not familiar with the surface anatomy. There are two main approaches: the short axis (out of plane) and the long axis (in plane). The patient is positioned either seated or supine, with the forearm supinated, the palm facing up, and the wrist in slight extension using a rolled-up towel. A high-frequency linear transducer is placed transversely in the antecubital fossa, and the nerve is traced distally to the proximal flexion crease at the wrist, at the entrance to the carpal tunnel (Fig. 37.16). Careful evaluation will show the nerve transitioning from a deep position to a superficial position just before it enters the carpal tunnel.

For a carpal tunnel injection, the median nerve is identified in the carpal tunnel (Fig. 37.17). Using the short-axis (out-of-plane) approach, one injects near the median nerve within the carpal tunnel by approaching proximal to distal. With the long-axis (in-plane) approach, the needle is advanced in a radial to ulnar direction (Fig. 37.18). In another described technique, the transducer is placed in a more ulnar orientation, and, after identifying the ulnar nerve and artery, the needle passes superficial to the ulnar nerve and artery with a shallow trajectory until the needle is close to the ulnar side of the median nerve [25].

In 2013, Ustün et al. [26] compared landmark-guided CTS injections to those under US guidance in a randomized prospective study; both groups improved, but the US-guided injections resulted in earlier and more effective improvement.

US-Guided Hydrodissection Technique

Some of the pathology of CTS, especially postoperatively, is felt due to adhesions within the canal. In 2008, Smith et al. [25] used a real-time ultrasound-guided hydrodissection carpal tunnel injection for nonsurgical treatment of carpal tunnel syndrome. During the injection, the hydrodissection could be seen disrupting adhesions between the median nerve in the carpal tunnel and the adjacent connective tissues, allowing the injection material to encircle the target nerve.

Neurolysis/Surgical Technique

Carpal Tunnel Release

Under local, regional, or general anesthesia, the patient's transverse carpal ligament is exposed and then divided. This procedure can be performed using a variety of techniques. Patients generally resume activities as tolerated, most often without casting, splinting, or therapy. Office workers can return to work promptly, as soon as surgery-related swelling and bruising resolve. Heavy laborers may need to be off work for several months.

Proximal Median Nerve Release

The general approach advocated by one author (WBE) is to use two small incisions and to release the nerve from about 10 cm proximal to the elbow to about 10 cm distal to the elbow. The relevant structures for release include the fascia of Struthers, the bicipital aponeurosis, the deep fascia of the ulnar origin of the pronator teres, and the fascial arch of the superficial flexors. Most patients can resume use of the arm promptly, with no cast, splint, or therapy needed postoperatively.

Complications

Kopell first described steroid injections for the treatment of CTS in 1958 [27], and the technique is widely used and is usually safe. However, there are several potential complications, such as nerve injury, muscle atrophy, and skin changes. Frederick et al. [28] described three cases of nerve injury after wrist injections – one of the ulnar nerve and two of the median nerve. Kim and Park [29] performed a literature review of median nerve injuries after carpal tunnel injections; based on their findings, they recommended the use of blunt needles while avoiding intrafascicular/intraneural injections, deep sedation, and large volumes of injectate. They also suggested that pain after a median nerve injection lasting greater than 48 h be considered a nerve injury; US will show a swollen and then atrophied nerve, and nerve testing will show a prolonged latency of the compound muscle action potentials.

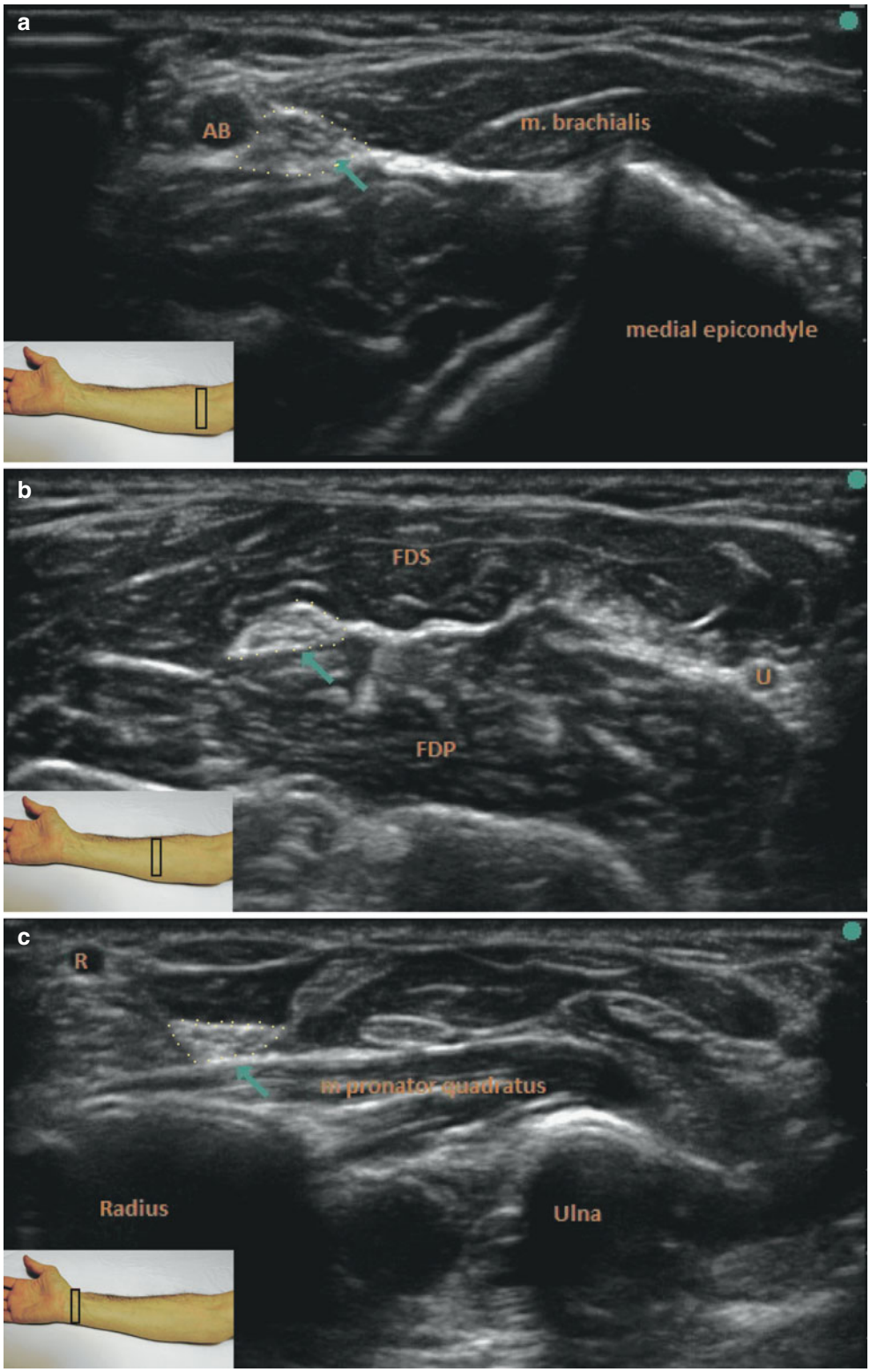

Fig. 37.16 Ultrasound evaluation of the median nerve from the elbow to the wrist. (**a**) Median nerve at the cubital region, marked by *green arrow*, immediately next to the brachial artery (AB). (**b**) Median nerve at the mid-forearm, *U* ulnar artery, *FDS* flexor digitorum superficialis muscle, *FDP* flexor digitorum profundus muscle. (**c**) Medial nerve at the wrist as it enters the carpal tunnel, *R* radial artery (Image courtesy of Agnes Stogicza, MD)

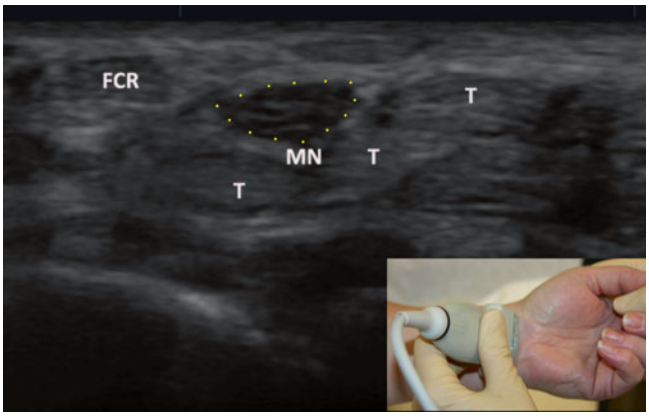

Fig. 37.17 Ultrasound image of the wrist at the carpal tunnel. *T* tendon, *FCR* flexor carpi radialis, *MN* median nerve (Image courtesy of Virtaj Singh, MD)

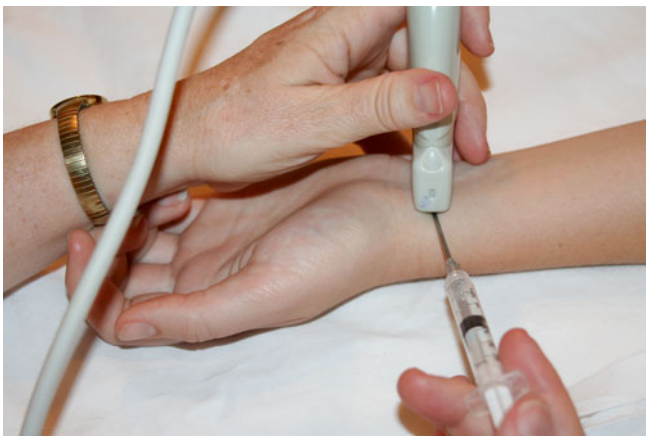

Fig. 37.18 Ultrasound-directed injection of the median nerve; in-plane radial approach (Image courtesy of Andrea Trescot, MD)

Although rare, complications from surgery do exist and include but are not limited to bleeding, infection, nerve injury, incomplete relief, worsening pain or symptoms, hypertrophic scarring, and anesthesia-related concerns, including death.

Summary

Median nerve entrapment can occur at several sites in the arm and wrist, and the presentation (as well as the treatment) is different for each entrapment site. Though CTS is the most common entrapment, several median nerve entrapments will present similarly to CTS, sometimes leading to surgical failure. Proper treatment requires proper diagnosis.

References

1. Figus A, Iwuagwu FC, Elliot D. Subacute nerve compressions after trauma and surgery of the hand. Plast Reconstr Surg. 2007;120(3):705–12.
2. Coleman SS, Anson BJ. Arterial patterns in the hand based upon a study of 650 specimens. Surg Gynecol Obstet. 1961;113:409–24.
3. Levy M, Pauker M. Carpal tunnel syndrome due to thrombosed persisting median artery. A case report. HAND. 1978;10(1):65–8.
4. Chopra JS, Khanna SK, Murthy JM. Congenital arteriovenous fistula producing carpal tunnel syndrome. J Neurol Neurosurg Psychiatry. 1979;42(9):815–7.
5. Bishop AT, Beckenbaugh RD. Fracture of the hamate hook. J Hand Surg Am. 1988;13(1):135–9.
6. Golovchinsky V. Peripheral entrapment syndromes. Double-Crush Syndrome. Norwell: Klower Academic Publishers; 2000. p. 1–53.
7. Robinson LR. Electrodiagnosis of carpal tunnel syndrome. Phys Med Rehabil Clin N Am. 2007;18(4):733–46, vi.
8. Pryse-Phillips WE. Validation of a diagnostic sign in carpal tunnel syndrome. J Neurol Neurosurg Psychiatry. 1984;47(8):870–2.
9. Roquer J, Herraiz J. Validity of flick sign in CTS diagnosis. Acta Neurol Scand. 1988;78(4):351.
10. Jacobson JA, Fessell DP, Lobo Lda G, Yang LJ. Entrapment neuropathies I: upper limb (carpal tunnel excluded). Semin Musculoskelet Radiol. 2010;14(5):473–86.
11. Dang AC, Rodner CM. Unusual compression neuropathies of the forearm, part II: median nerve. J Hand Surg Am. 2009;34(10):1915–20.
12. Robbins H. Anatomical study of the median nerve in the carpal tunnel and etiologies of the carpal-tunnel syndrome. J Bone Joint Surg Am. 1963;45:953–66.
13. deSouza R-M, Choi D. Peripheral nerve lesions. Neurosurgery. 2012;3(30):149–54.
14. Durkan JA. A new diagnostic test for carpal tunnel syndrome. J Bone Joint Surg Am. 1991;73(4):535–8.
15. Phalen GS. The carpal-tunnel syndrome. Clinical evaluation of 598 hands. Clin Orthop Relat Res. 1972;83:29–40.
16. Werner RA, Bir C, Armstrong TJ. Reverse Phalen's maneuver as an aid in diagnosing carpal tunnel syndrome. Arch Phys Med Rehabil. 1994;75(7):783–6.
17. Gellman H, Gelberman RH, Tan AM, Botte MJ. Carpal tunnel syndrome. An evaluation of the provocative diagnostic tests. J Bone Joint Surg Am. 1986;68(5):735–7.
18. Linda DD, Harish S, Stewart BG, Finlay K, Parasu N, Rebello RP. Multimodality imaging of peripheral neuropathies of the upper limb and brachial plexus. Radiographics. 2010;30(5):1373–400.
19. Buchberger W. Radiologic imaging of the carpal tunnel. Eur J Radiol. 1997;25(2):112–7.
20. Kuhlman KA, Hennessey WJ. Sensitivity and specificity of carpal tunnel syndrome signs. Am J Phys Med Rehabil. 1997;76(6):451–7.
21. Stevens JC, Beard CM, O'Fallon WM, Kurland LT. Conditions associated with carpal tunnel syndrome. Mayo Clin Proc. 1992;67(6):541–8.
22. Law HZ, Amirlak B, Cheng J, Sammer DM. An association between carpal tunnel syndrome and migraine headaches-National Health Interview Survey, 2010. Plast Reconstr Surg Glob Open. 2015;3(3):e333.
23. Palmer KT, Harris EC, Coggon D. Carpal tunnel syndrome and its relation to occupation: a systematic literature review. Occup Med (Oxford, Engl). 2007;57(1):57–66.

24. Carlson H, Colbert A, Frydl J, Arnall E, Elliot M, Carlson N. Current options for nonsurgical management of carpal tunnel syndrome. Int J Clin Rheumatol. 2010;5(1):129–42.
25. Smith J, Wisniewski SJ, Finnoff JT, Payne JM. Sonographically guided carpal tunnel injections: the ulnar approach. J Ultrasound Med. 2008;27(10):1485–90.
26. Ustün N, Tok F, Yagz AE, Kizil N, Korkmaz I, Karazincir S, et al. Ultrasound-guided vs. blind steroid injections in carpal tunnel syndrome: a single-blind randomized prospective study. Am J Phys Med Rehabil. 2013;92:999–1004.
27. Kopell HP. Carpal tunnel compression median neuropathy treated nonsurgically. N Y State J Med. 1958;58:744–5.
28. Frederick HA, Carter PR, Littler JW. Injection injuries to the median and ulnar nerves at the wrist. J Hand Surg Am. 1992;17(4):645–7.
29. Kim HJ, Park SH. Median nerve injuries caused by carpal tunnel injections. Korean J Pain. 2014;27(2):112–7.

Ulnar Nerve Entrapment

38

Virtaj Singh and Andrea M. Trescot

Introduction

Entrapment of the ulnar nerve (UN) is the second most common upper extremity peripheral nerve entrapment (with carpal tunnel entrapment being the most common) [1, 2]. The UN innervates the muscles of grasping, including the hypothenar muscles and the interosseous adductor muscles. There are primarily two peripheral locations at which the ulnar nerve is commonly entrapped. The most common is at the elbow and is often referred to as *cuboid tunnel* [3] or *cubital tunnel syndrome;* other names include *sulcus ulnaris syndrome* or *retrocondyle groove syndrome*. Distally, the ulnar nerve can be entrapped at the wrist in a location known as *Guyon's canal*. This is called *Guyon's canal syndrome or palsy* or *handlebar palsy*. Each of these will be discussed separately.

Clinical Presentation (Tables 38.1 and 38.2)

Patients with ulnar entrapment can have a sudden or a gradual onset of pain in the elbow (Fig. 38.1) or paresthesias, numbness, weakness, or clumsiness of the hand. There may be a history of trauma, including habitual elbow leaning, repeated elbow flexion and extension, habitual elbow flexion in sleep, or recent general anesthesia. There can be associated diabetes, chronic renal failure, arthritis (such as rheumatoid), hypothyroidism, or cervical spondylosis. Sports injuries are a common cause of ulnar nerve entrapment (Tables 38.1 and 38.2). According to Dugas [4], there has been a nearly tenfold increase in the number of surgical reconstructions of the ulnar structures in "throwing" athletes with *cubital tunnel syndrome* and *Guyon's tunnel syndrome.*

Table 38.1 Occupation/exercise/trauma history relevant to ulnar nerve (elbow) entrapment

Sports injury	Throwing sports [4] – baseball is most common
	Overhead sports [4] – volleyball, racquet sports
	Swimming [4]
	Gymnastics [4]
	Golf [4]
Occupation	"Holding a tool in position" [5]
	Carpenter
	Typist
Conditions	Supracondylar fracture
	Smoking [2]
	Obesity [2]

Table 38.2 Occupation/exercise/trauma history relevant to ulnar nerve (forearm and wrist) entrapment

Compression	Cycling [6], especially with wrist cocked up
	Compartment syndrome, such as seen in hemophilia [7]
	Lipomas, synovial cysts [7], ganglion cysts [8]
Occupation	Tools pressed into the base of the hand [7]
	Heavy gripping or twisting
	Prolonged flexion with ulnar bending
Trauma	Styloid fracture [8]
	Distal radius fracture [9]
	Hamate fracture (golfers who hit the ground and baseball batting)
	Wrist steroid injections [7]
	Endoscopic carpal tunnel release [7]
	Blood clot to the upper extremity

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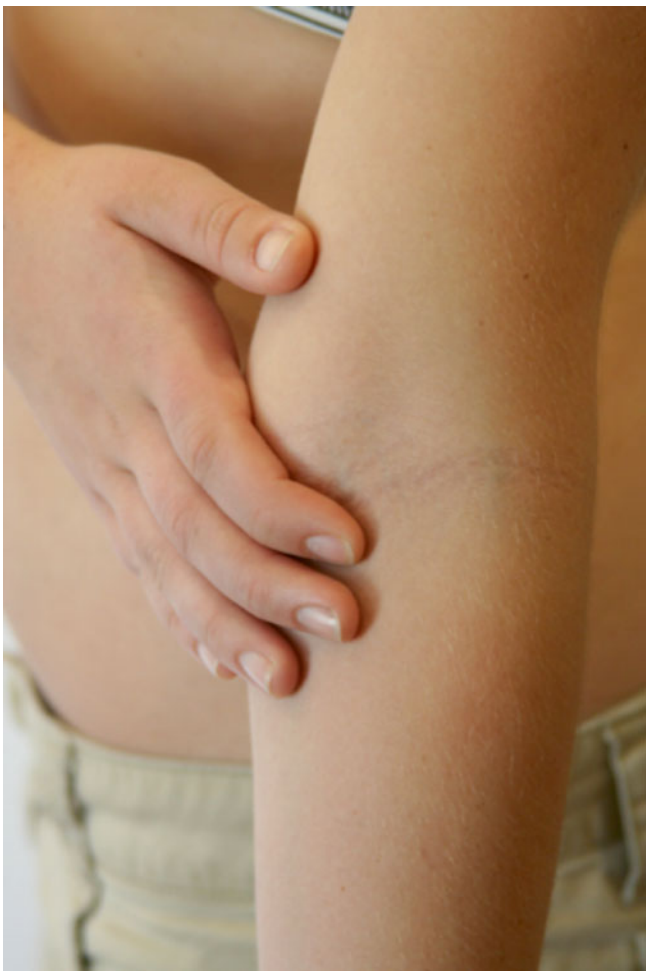

Fig. 38.1 Patient description of ulnar nerve pain at the elbow (Image courtesy of Andrea Trescot, MD)

Fig. 38.2 Distribution of arm nerves: *A* axillary nerve, *B* radial nerve (*1* posterior cutaneous nerve of the arm, *2* inferior lateral cutaneous nerve, *3* posterior cutaneous nerve of the forearm, *4* superficial radial nerve), *C* intercostal brachial nerve, *D* ulnar nerve, *E* median nerve, *F* lateral cutaneous nerve of the forearm (Image courtesy of Terri Dallas-Prunskis, MD)

Cubital tunnel syndrome: An ulnar nerve entrapment at the elbow is known as *cubital tunnel syndrome*, the second most common entrapment after the carpal tunnel syndrome (see Chap. 37). The typical presentation is a patient with pain at the elbow with dysesthesias and numbness radiating into the hand (Fig. 38.2) to both the palmar and dorsal sides of the fourth and fifth fingers (Figs. 38.3 and 38.4). Ulnar entrapment at the elbow tends to cause weakness of the small muscles of the hand while sparing the forearm muscles [7]. Severe cases can lead to weakness in a pattern known as an *ulnar negative hand* (the ring and little fingers remain flexed) (Fig. 38.5). Atrophy may be initially noticed in the first dorsal interosseous space or hypothenar muscles [7, 10], with a claw deformity developing in severe cases. Occasionally, there may be progressive muscle wasting with no sensory loss [7]. Weakness of intrinsic muscles of the hand leads to clumsiness and dropping objects [11]. Symptoms tend to increase with increased use, especially if the activity includes flexion of the elbow.

Guyon's canal entrapment: As discussed above, the ulnar nerve can also be entrapped within *Guyon's canal*, a fibro-osseous structure of the ulnar wrist (see Anatomy section (Sect. 3)). There are multiple clinical patterns that can develop from ulnar nerve compression in Guyon's canal. There can be weakness of the interossei, lumbricals, and hypothenar muscles without sensory deficits; weakness of the interossei, lumbricals, and hypothenar muscles with sensory loss in the distal palm, ring, and little fingers; or a pattern of purely sensory loss in the last two fingers (Figs. 38.3 and 38.4). When combined with *carpal tunnel syndrome* (CTS) (see Chap. 37) and *superficial radial neuralgia* (see Chap. 36), this creates a "glove" pattern of symptoms that may mimic peripheral neuropathy.

The area of hypoesthesia is usually along the ulnar aspect of the fourth and fifth fingers. The exact distribution of numbness can help to differentiate between cubital tunnel,

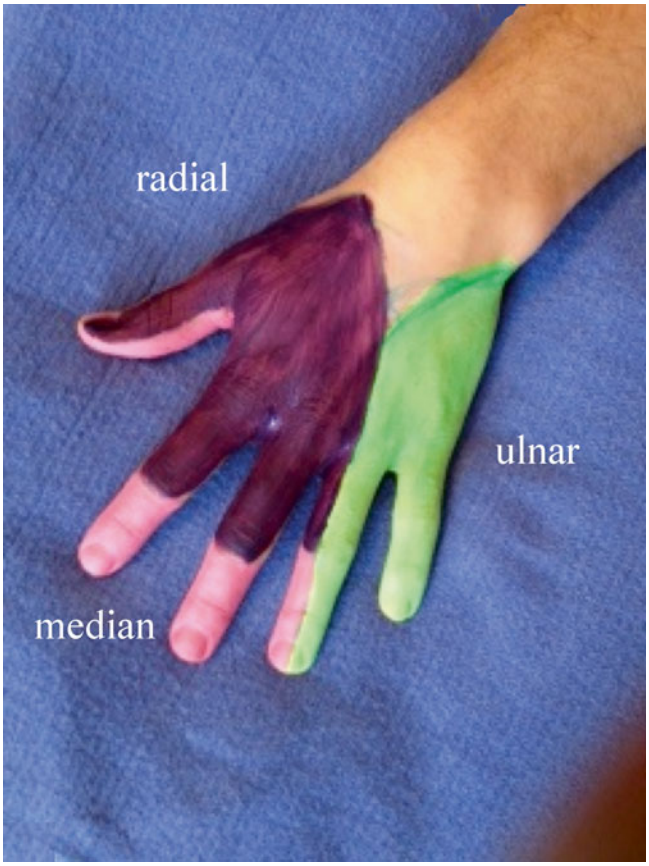

Fig. 38.3 Dorsal dermatomal distribution of hand innervation (Image courtesy of Terri Dallas-Prunskis, MD)

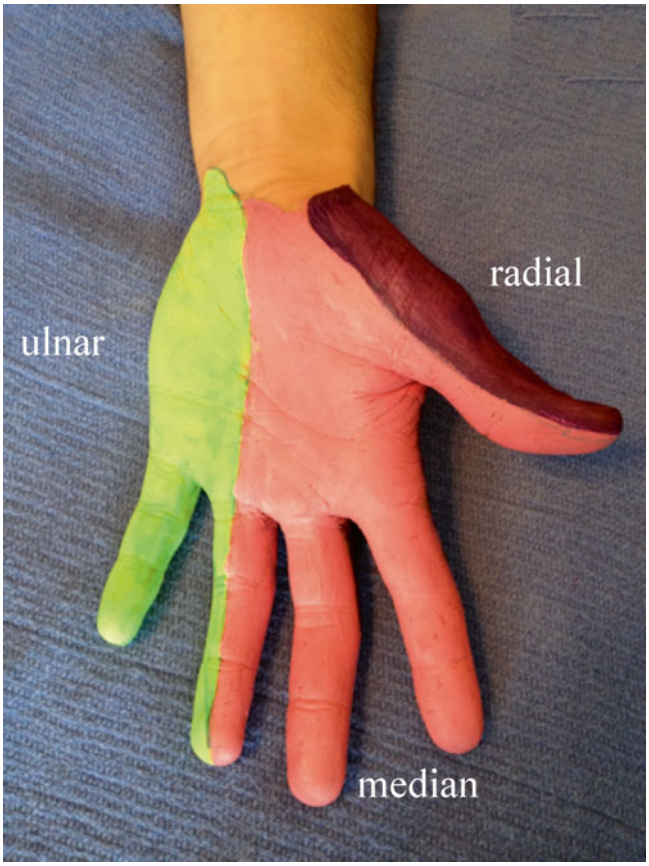

Fig. 38.4 Palmar dermatomal distribution of hand innervation (Image courtesy of Terri Dallas-Prunskis, MD)

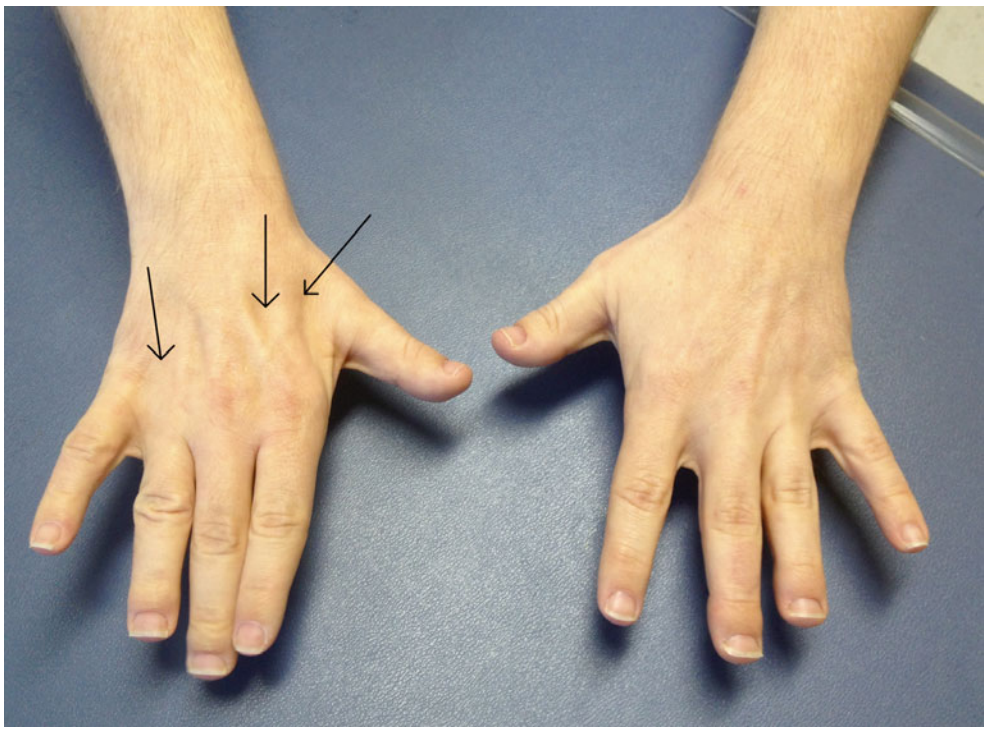

Fig. 38.5 Interosseous atrophy (see *arrows*) (Image courtesy of Andrea Trescot, MD)

Guyon's canal, and a brachial plexus/T1 pathology [7]. The clinician also needs to check for *neurogenic thoracic outlet syndrome (NTOS)* (see Chap. 33), because NTOS may coexist with ulnar entrapment [3].

Anatomy (Table 38.3)

The ulnar nerve is derived primarily from the C8 and T1 nerve roots with some minor contributions from C7. The ulnar nerve comes directly off the medial cord of the brachial plexus. Within the axilla, the nerve runs with the axillary artery and vein. As it enters the arm, it travels with the brachial artery and vein. By the middle of the upper arm (Fig. 38.6), the ulnar nerve passes posteriorly, through the *medial intermuscular septum*, descending medial to the triceps, potentially underneath the *arcade of Struthers* (a usually thin but potentially thick tissue attached to the septum). It then passes into the medial epicondyle of the humerus at the retrocondylar groove, under the *humeroulnar arcade* (*Osborne's ligament*), entering the forearm through the *cubital tunnel* (Fig. 38.7). The roof of the cubital tunnel consists of the aponeurotic arch and muscle fibers of the flexor carpi ulnaris muscle (FCU), while the medial ligaments of the elbow as well as other FCU muscle fibers form the floor. The ulnar nerve then innervates the FCU and *flexor digitorum profundus* (FDP) to the fourth and fifth digits. As it innervates these muscles, it also travels through them (Figs. 38.8 and 38.9).

Table 38.3 Ulnar nerve anatomy

Origin	Elements from the medial cord of the brachial plexus (C8 and T1, with occasional C7) converge to form the ulnar nerve (UN)
General route	The UN leaves the axilla and travels in the median bicipital groove on the intermuscular septum, medial to the brachial artery. Then passes from the anterior (flexor) to the posterior (extensor) compartment, potentially underneath the arcade of Struthers. Does not branch until it reaches the elbow
Sensory distribution	Depends on the level
	Elbow joint [12]
	Palmar and dorsal cutaneous branches
Motor innervation	Flexor carpi ulnaris (FCU)
	Flexor digitorum profundus (FDP) 4th and 5th digits
	Intrinsic hand muscles
Anatomic variability	Depends on the level
Other important structures	Arcade of Struthers [13]
	Cubital tunnel

The ulnar nerve then passes into the middle of the forearm where it gives off the *palmar cutaneous branch*, providing sensation to the medial, proximal palm. As the ulnar nerve travels towards the wrist, it gives off another sensory branch, the *dorsal cutaneous branch*, which gives sensation to the medial aspect of the dorsum of the hand and the fourth and fifth digits.

The UN then enters the wrist at *Guyon's canal* (Figs. 38.10, 38.11, and 38.12). Since it is really a tunnel, it is sometimes called the *ulnar tunnel* or the *carpal ulnar neurovascular space*. This canal or tunnel is classically described as formed by the *hook of the hamate* laterally and the *pisiform* medially; the floor of the tunnel is formed by the *transverse carpal ligament*, and the roof is formed by the *pisohamate ligament*. However, more recently, the description has changed; the border is considered to be formed by the junction of the *palmar carpal ligament* (including the *palmaris brevis muscle*) to the *flexor retinaculum* and tendons of origin of the thenar muscles [14]. In the canal, the UN divides into a mainly *sensory superficial branch* and a *deep motor branch*. The motor branch gives off innervation to the *hypothenar muscles*, the *third and fourth lumbricals*, the *first dorsal interossei*, the deep head of the *flexor pollicis brevis*, and the *adductor pollicis*.

Bozkurt et al. [15] dissected 37 upper extremities; a fibrous, compressive arch between the hook of the hamate and the pisiform was detected in 21 (56.7 %) hands, while in 6 hands (16.2 %) there was an anomalous muscle in Guyon's canal that was positioned over both the superficial and deep branches (or just the deep branch) of the UN.

Entrapment

Elbow

The UN can be compressed when the upper arm hangs over a sharp edge, such as when a patient is in a coma or inebriated; misuse of crutches or tourniquets may also compress the UN in the upper arm. About 9–10 cm above the medial epicondyle, the UN travels with the ulnar collateral artery through the *medial intermuscular septum*, makes a slight change of direction, and may be compressed there by the *arcade of Struthers* (AS) (present in 70–80 % of the population) (Fig. 38.7) [12, 13]. The AS is a fibrous canal with its narrowest portion positioned proximally [13].

When the elbow is extended, the cubital tunnel has a somewhat circular shape and is at its roomiest. With elbow flexion, the distance between the medial epicondyle and the olecranon increases by about 1 cm, causing the flexor carpi ulnaris aponeurosis that joins them to tighten over the nerve [7].

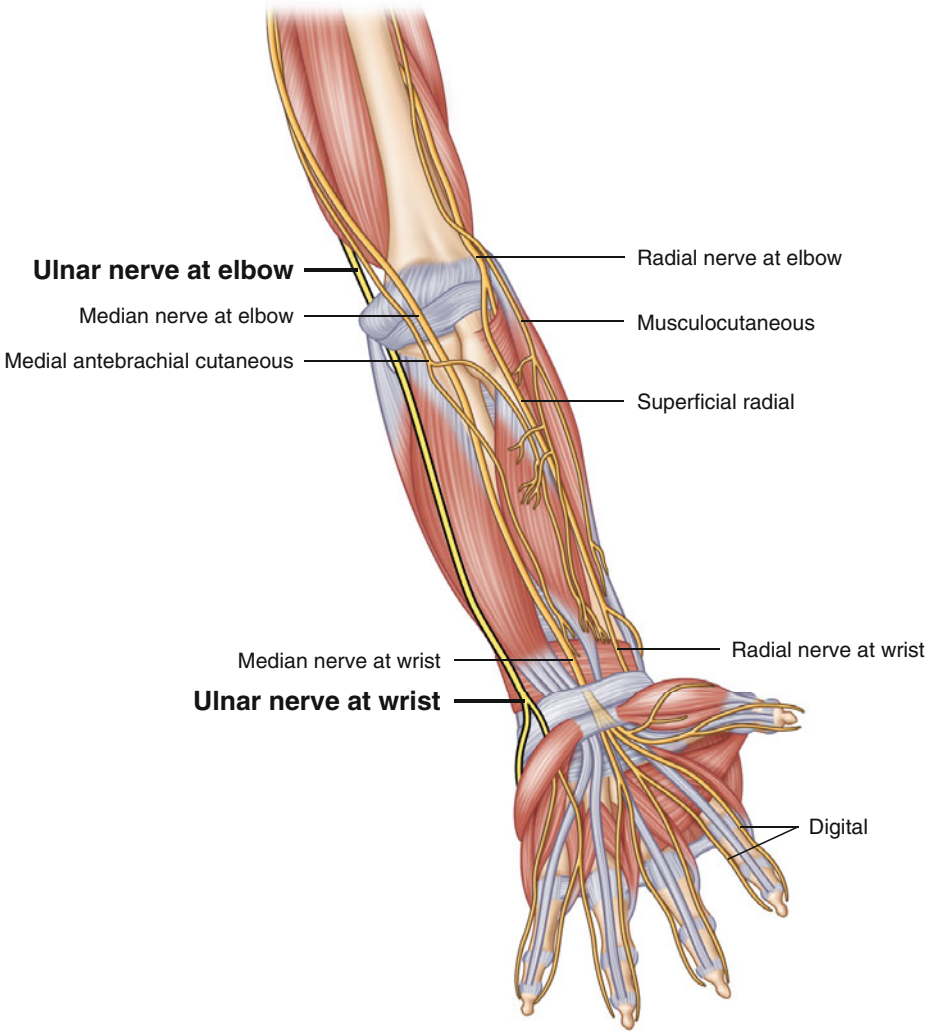

Fig. 38.6 Anatomy of the forearm (Image by Springer)

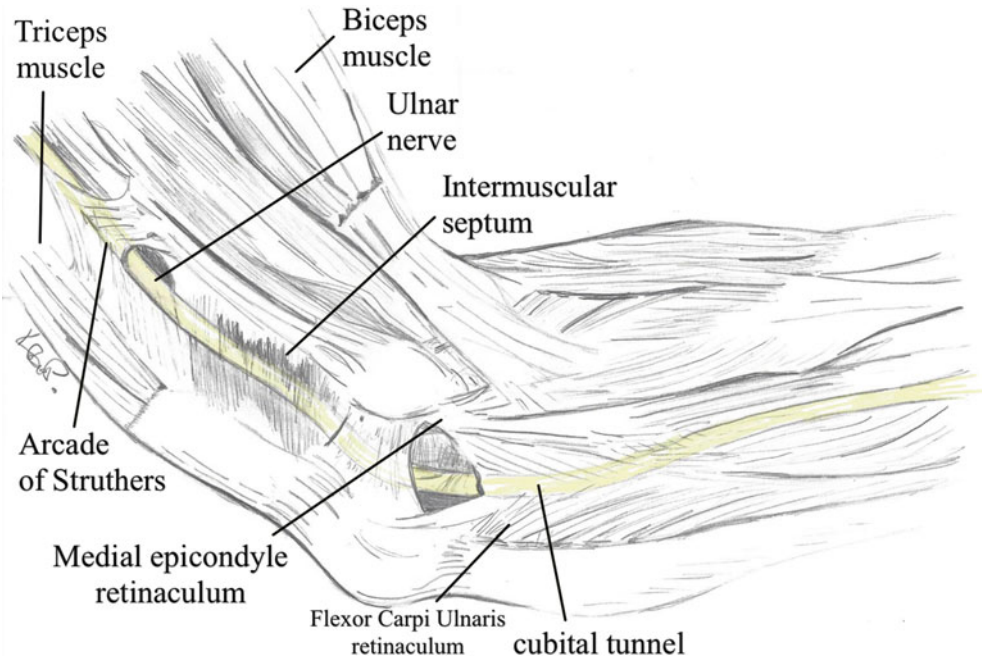

Fig. 38.7 Cubital tunnel anatomy (Image courtesy of Kristen Prunskis)

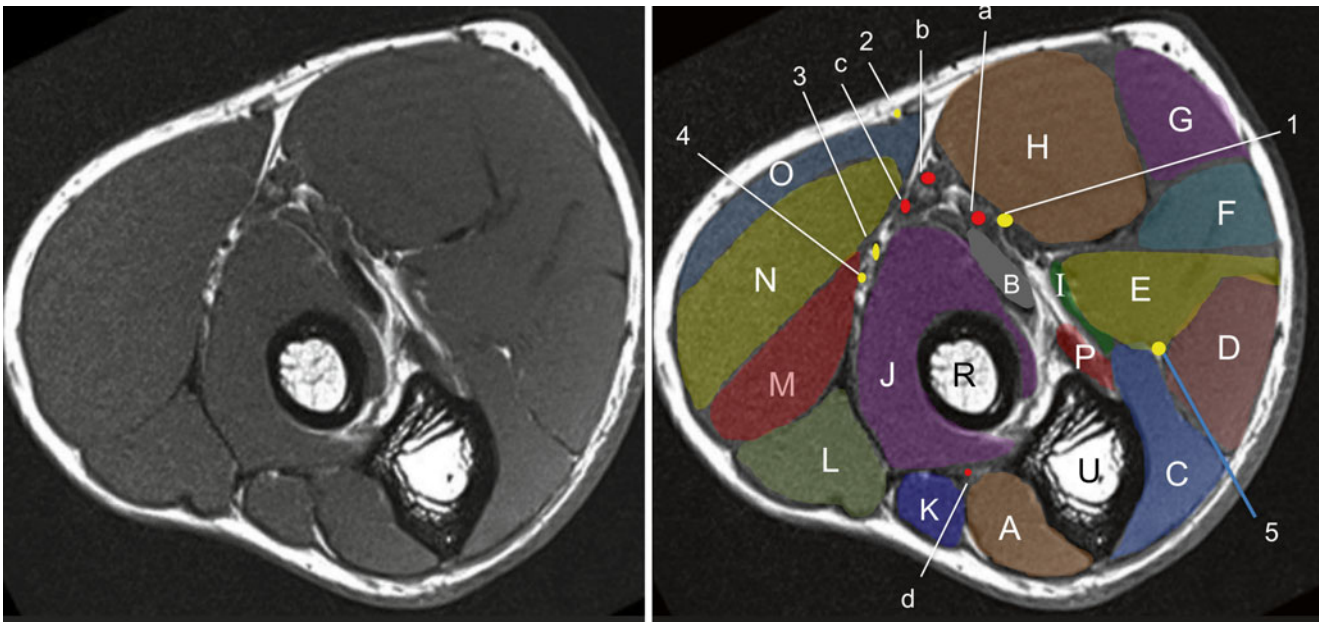

Fig. 38.8 MRI axial image of the proximal forearm. *A* anconeus muscle, *B* biceps tendon, *C* flexor digitorum profundus muscle, *D* flexor carpi ulnaris muscle, *E* flexor digitorum superficialis muscle, *F* palmaris longus muscle, *G* flexor carpi radialis muscle, *H* pronator teres muscle (humeral head), *I* pronator teres muscle (ulnar head), *J* supinator muscle, *K* extensor digiti minimi muscle, *L* extensor digitorum muscle, *M* extensor carpi radialis brevis muscle, *N* extensor carpi radialis longus muscle, *O* brachioradialis muscle, *P* brachialis muscle, *a* ulnar artery, *b* radial artery, *c* radial recurrent artery, *d* recurrent interosseous artery, *1* median nerve, *2* lateral cutaneous nerve of the forearm, *3* superficial branch radial nerve, *4* deep branch radial nerve, *5* ulnar nerve (Image courtesy of Andrea Trescot, MD)

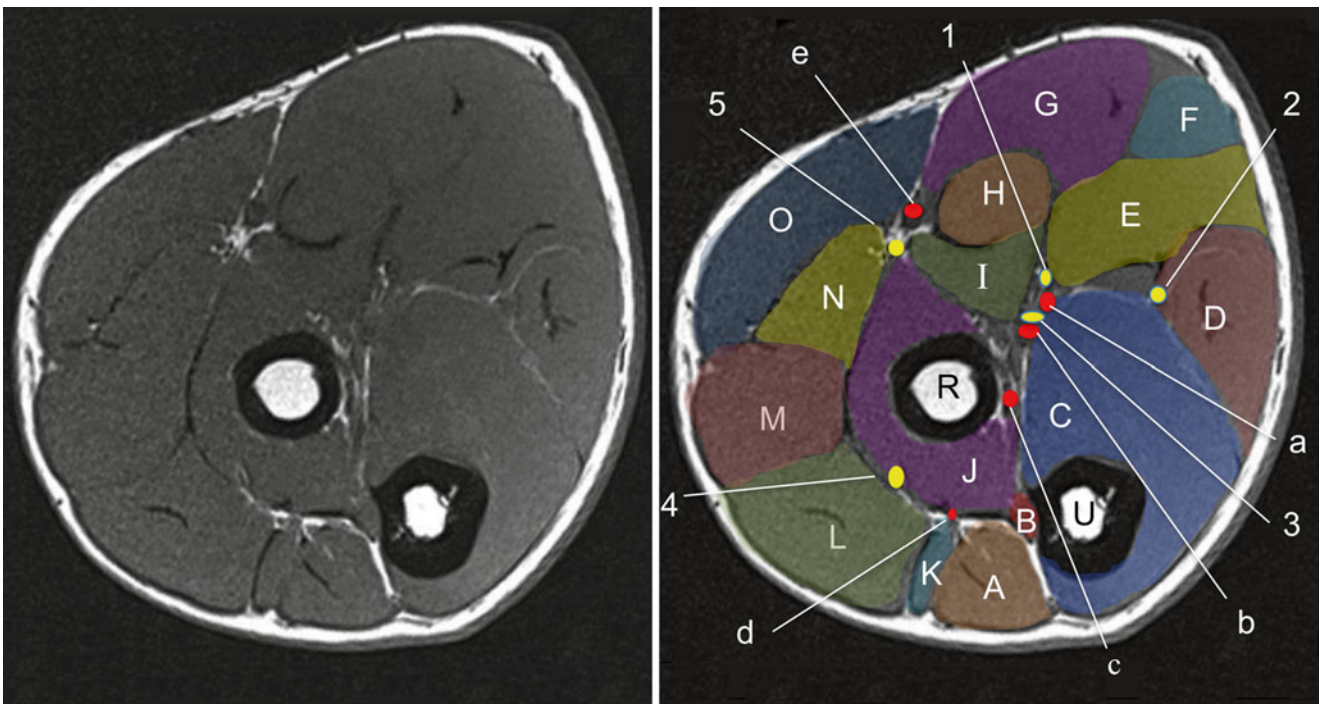

Fig. 38.9 MRI axial image of the distal forearm. *A* anconeus muscle, *B* extensor pollicis longus muscle, *C* flexor digitorum profundus muscle, *D* flexor carpi ulnaris muscle, *E* flexor digitorum superficialis muscle, *F* palmaris longus muscle, *G* flexor carpi radialis muscle, *H* pronator teres muscle (humeral head), *I* pronator teres muscle (ulnar head), *J* supinator muscle, *K* extensor digiti minimi muscle, *L* extensor digitorum muscle, *M* extensor carpi radialis brevis muscle, *N* extensor carpi radialis longus muscle, *O* brachioradialis muscle, *a* ulnar artery, *b* common interosseous artery, *c* posterior interosseous artery, *d* interosseous artery, *e* radial artery, *1* median nerve, *2* ulnar nerve, *3* anterior osseous nerve, *4* deep branch radial nerve, *5* superficial branch radial nerve (Image courtesy of Andrea Trescot, MD)

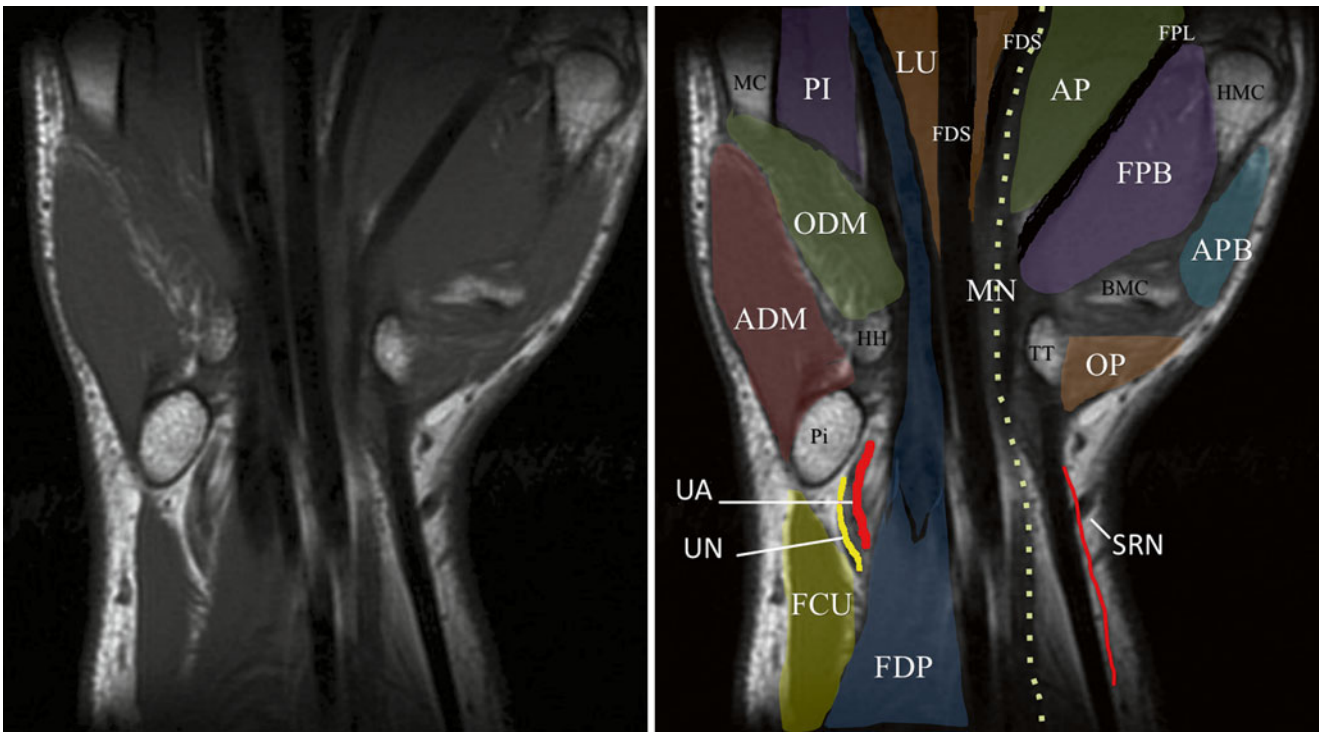

Fig. 38.10 Wrist coronal MRI. *ADM* abductor digiti minimi muscle, *AP* adductor pollicis muscle, *APB* abductor pollicis brevis muscle, *BMC* base of metacarpal bone, *FCU* flexor carpi ulnaris muscle, *FDP* flexor digitorum profundus muscle, *FDS* flexor digitorum superficialis tendon, *FPB* flexor pollicis brevis muscle, *FPL* flexor pollicis longus tendon, *HH* hook of the hamate bone, *HMC* head of metacarpal bone, *LU* lumbrical muscle, *MC* metacarpal bone, *MN* median nerve, *ODM* opponens digiti minimi, *muscle OP* opponens pollicis, *Pi* pisiform bone, *PI* palmar interossei muscle, *RA* radial artery, *SRN* superficial radial nerve, *TT* trapezium tubercle bone, *UA* ulnar artery, *UN* ulnar nerve (Image courtesy of Andrea Trescot, MD)

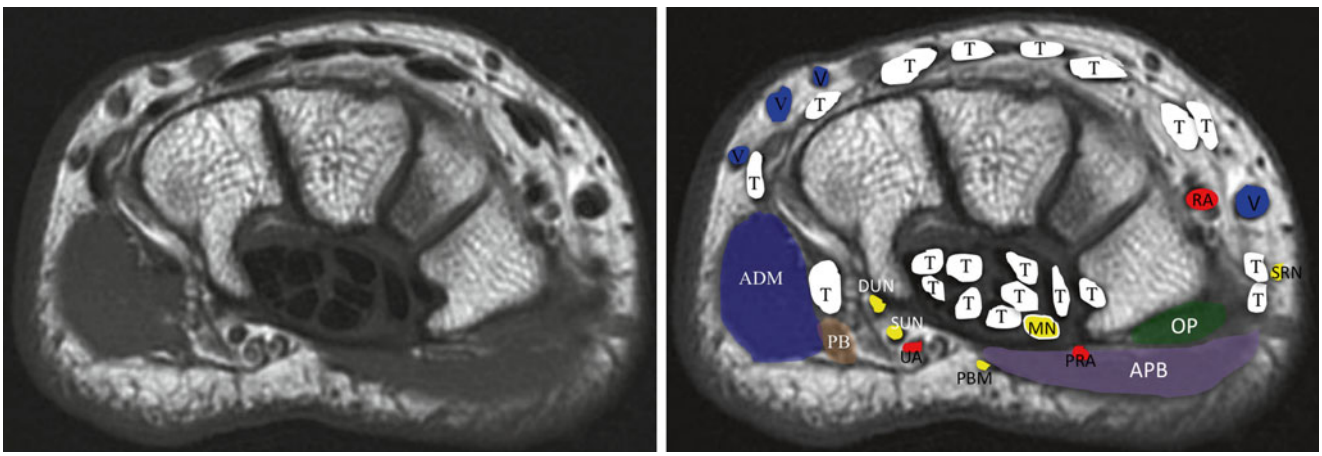

Fig. 38.11 Wrist axial MRI. *A* artery, *APB* abductor pollicis brevis, *DUN* deep branch of the ulnar nerve, *MN* median nerve, *OP* opponens pollicis, *PB* palmaris brevis, *PMN* palmar branch of the median nerve, *PRA* palmar radial artery, *RA* radial artery, *SRN* superficial branch of the radial nerve, *SUN* superficial branch of the ulnar nerve, *T* tendon, *UA* ulnar artery, *V* vein (Image courtesy of Andrea Trescot, MD)

Forearm and Wrist

After leaving the elbow, the UN goes into the cubital tunnel where it can be entrapped between the heads of the FCU by a fascial band, the *arcuate ligament,* or *Osborne's band*, which connects the two heads of the FCU [12]. Compartment syndromes from bleeding, such as might be seen in hemophiliacs or after trauma, can compress the nerve in the forearm [7]. Naran et al. evaluated 74 patients with *cubital ulnar tunnel syndrome* (CUTS); there was noted to be a significant relationship between the age of the patient and signs of ulnar irritation (interosseous weakness and muscle atrophy) [16].

In Guyon's canal, the ulnar artery travels with the UN, but, unlike the carpal tunnel, there are no tendons in the

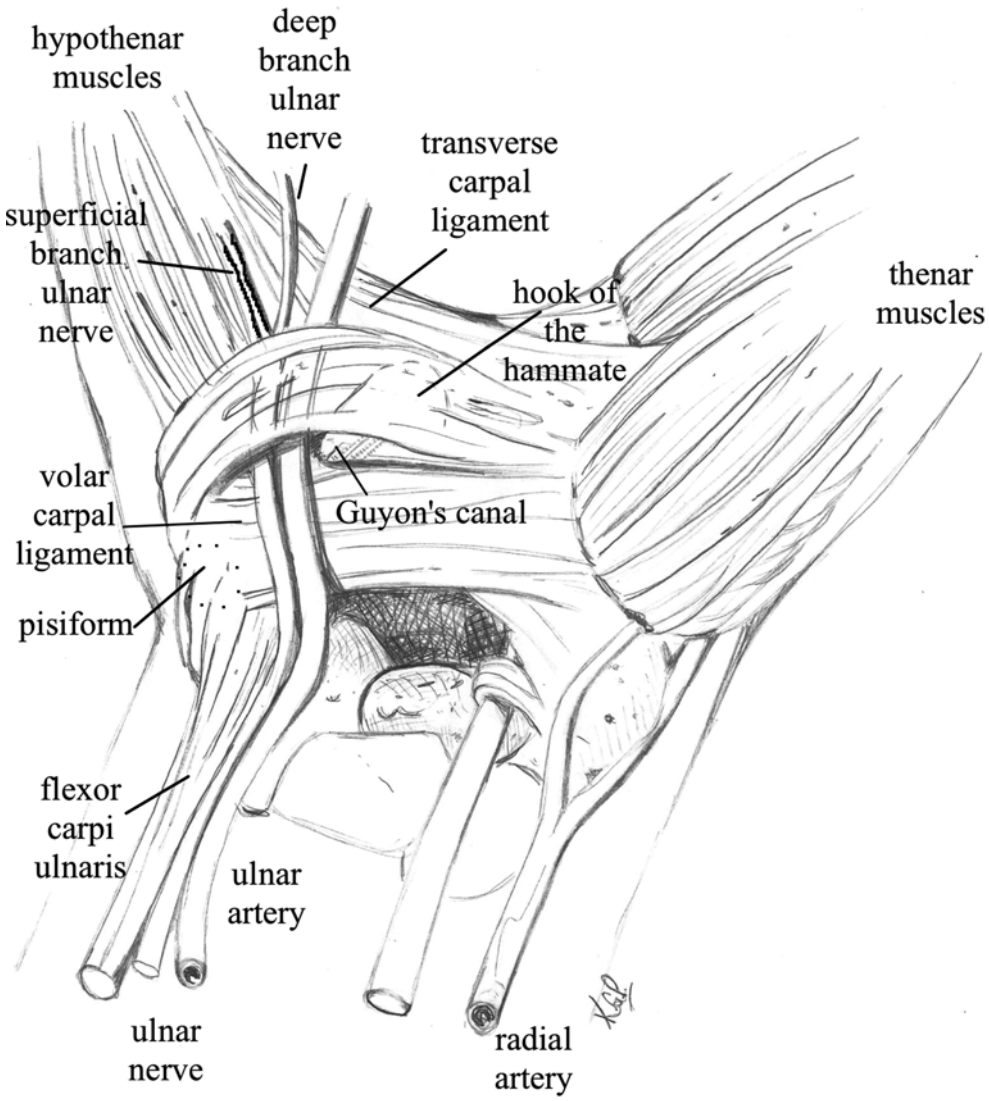

Fig. 38.12 Guyon's canal anatomy (Image courtesy of Kristen Prunskis)

canal. The most frequent causes of neuropathy in Guyon's canal include ganglia, hematoma, or cysts, which may present with ulnar hand numbness or pain, as well as weakness of the abductor digiti minimi (ADM). The pain from entrapment in Guyon's canal tends to be worse at night and with extremes of wrist movements [15]. The deep branch of the ulnar nerve is likely to be compressed at the *pisohamate hiatus*, which lies between the *pisohamate ligament* and the fibrous arch at the origin of the hypothenar muscles [15]. Accessory muscles coursing through Guyon's canal and the fibrous arch overlying the superficial and deep branches of the ulnar nerve may predispose to ulnar entrapment [15].

Physical Exam

Physical exam is necessary to stage the degree and location of entrapment of the UN [17]. *Froment's sign* is a test for the loss of adductor pollicis function; when the patient grasps a piece of paper between the thumb and index finger, the interphalangeal joint flexes. Similarly, *Jeanne's sign* (metacarpophalangeal joint hyperextension with thumb/index pinching) is also a sign of loss of adductor pollicis function. High ulnar injuries lead to less loss of muscle function than a lower lesion; this is known as the "*ulnar paradox*" and is due to the fact that the medial FDP is also paralyzed and the hand is more relaxed [18]. Asking the patient to abduct the fingers against resistance will identify ulnar weakness. Instability of the elbow can typically be demonstrated on physical examination, and in many cases, it can be visualized as patients flex their elbow beyond 90°.

Holding the elbow in the palm of the examining hand, palpation of the cubital tunnel is performed by moving the thumb across the tendon attachment of the FCU (Video 38.1); the nerve will be found to the ulnar side of the tendon and should be tender to the touch (Fig. 38.13). Palpation of the nerve at the elbow may trigger numbness and tingling in the hand and may be a provocative test.

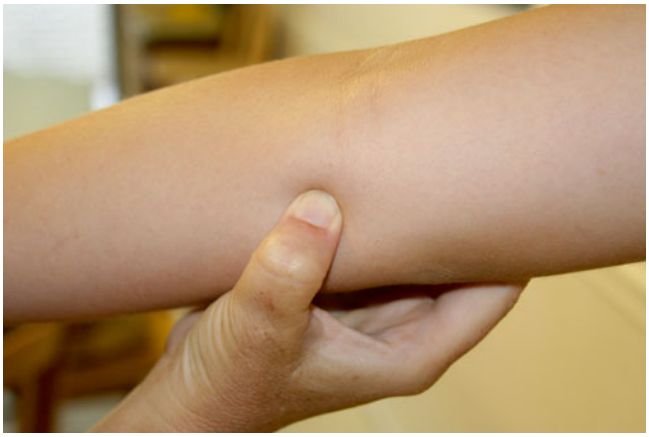

Fig. 38.13 Examination of the elbow (Image courtesy of Andrea Trescot, MD)

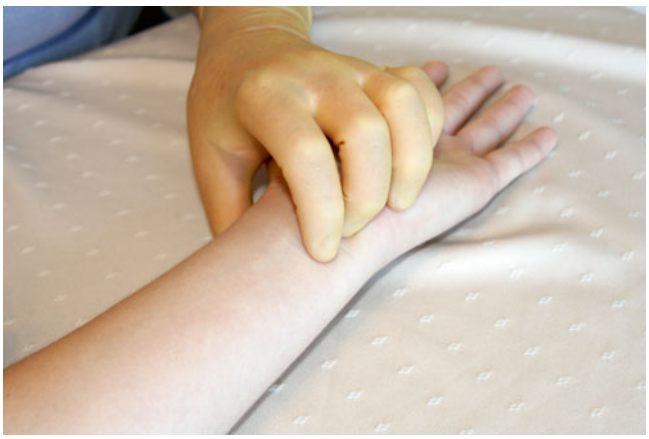

Fig. 38.14 Examination of the wrist (Image courtesy of Andrea Trescot, MD)

A careful examination of the nerve itself, as opposed to the tendon, should allow the examiner to distinguish between UN entrapment and *medial epicondylitis*.

The physical examination of Guyon's canal entrapment is performed by placing the wrist in slight extension (or hyperextension) and palpating along the ulnar aspect of the wrist, just proximal to the volar crease (*Guyon's canal compression test*) (Video 38.2) (Fig. 38.14). *Tinel's sign* (percussion of the nerve) and *Phalen's sign* (flexing the wrist) may also be positive.

Differential Diagnosis (Tables 38.4, 38.5, 38.6, and 38.7)

Other conditions can present similarly to an ulnar neuropathy at the elbow or wrist. A cervical radiculopathy, especially C8 or T1, can have a similar presentation. Neurogenic thoracic outlet syndrome (NTOS) (see Chap. 33) can also present with a similar clinical picture. In addition, one can have a double-crush-type injury (see Chap. 1) in which there is compression of the ulnar nerve distally while there is also a concomitant proximal compression of either a cervical root or within the brachial plexus, such as with NTOS.

Table 38.4 Differential diagnosis of elbow pain

	Potential distinguishing features
Neurogenic thoracic outlet syndrome – NTOS [3]	When elbow symptoms are most prominent and EDX confirms cubital tunnel, decompress the elbow first. If the symptoms are more in the shoulder and there is a good response to a scalene block, decompress the thoracic outlet
Valgus extension overload – VEO [4]	Forcing the elbow into terminal extension while applying a valgus stress to the elbow causes olecranon pain
Proximal olecranon stress fracture [4]	Positive bone scan
Elbow bursitis [19]	Tenderness and swelling over the olecranon
Elbow joint instability [19]	Pain is usually lateral, and the joint may "click" or "snap"
Lateral or medial epicondylitis [19]	Tenderness over the tendon attachment
Radial tunnel syndrome [19]	Pain is usually lateral

Table 38.5 Differential diagnosis of ulnar wrist pain

	Potential distinguishing features
Distal ulnar stress fracture	Positive bone scan
Rheumatoid arthritis	Elevated ESR/CRP, synovitis on MRI
Wartenberg's syndrome	Superficial radial nerve
FCR tendonitis	Pain at the volar radial wrist
Thumb CMC arthritis	Volar radial wrist pain, tenderness over CMC joint
Arthritis of the wrist	Tenderness over the carpal bones
De Quervain's tenosynovitis	Tenderness over the "anatomic snuff box"
Volar ganglion cyst	Palpable cyst

Table 38.6 Diagnostic tests for ulnar nerve (elbow) entrapment

	Potential distinguishing features
Physical exam	Tenderness over cubital tunnel
Diagnostic injection	Injection at cubital tunnel
Ultrasound	May show compression of the UN in the cubital tunnel with flexion and extension
MRI	May show edema of the nerve in the cubital tunnel
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies	NCV across the elbow [7]; controversial [2]; not as sensitive as NCV for CTS [20]
Provocative tests	Elbow flexion test [17] (fully flexing the elbow for 1–5 min and evaluating exacerbation of symptoms)

The diagnostic tests for ulnar entrapment are found on Table 38.7. Ultrasound can show the entrapment of the UN by cysts, hematoma, swelling, or nerve enlargement (Fig. 38.15).

Identification and Treatment of Contributing Factors

For the elbow, overhead sports appear to be the greatest risk, while the prolonged gripping of bicycle handles often triggers the wrist pain. High pressures in the carpal tunnel can transmit to Guyon's canal, causing a UN entrapment [24].

Table 38.7 Diagnostic tests for ulnar nerve (wrist) entrapment

	Potential distinguishing features
Physical exam	Tenderness over Guyon's canal; FCU and FDP function spared; ulnar hypoesthesia and weakness of all ulnar intrinsic muscles
Diagnostic injection	Injection of Guyon's canal
Ultrasound	The nerve is well visualized under US and can show compression [21]. High-definition US can show the branches of the UN in the canal [22]
MRI	Increased T2 signal or enlargement of the nerves in 97 % of cases [7]; 90 % specificity [23]
Arteriography	Not useful
X-ray	Not useful except to show bony deformities
Electrodiagnostic studies	NCV slowing across the wrist; not as sensitive as NCV for carpal tunnel [20]; recording the ulnar-innervated interosseous muscles and comparing the median-innervated 2nd lumbrical muscle may be a more sensitive test [7]
Provocative tests	Guyon's canal compression test (Fig. 38.11)

There is very poor evidence that physical therapy improves outcomes compared to no treatment [25]. There is no evidence that "nerve gliding" improves outcome better than splinting or no treatment [19, 26]. Splinting and avoiding pressure on the nerve, however, are recommended by the European HANDGUIDE study [6]. Because of the risk of "double crush," with lesions at both the wrist and the elbow, it is important to test both proximally and distally with both US and electrodiagnostics [27]. Smith et al. [28] evaluated 70 cyclists with ulnar neuropathy and identified both distal (Guyon's canal) and proximal (thoracic outlet syndrome) pathology in a significant number of these athletes.

Injection Techniques

Landmark-Guided Technique

Elbow

For an injection in the cubital canal, the patient is positioned sitting or supine, and the non-injecting hand straddles the canal, from above or from below (Fig. 38.16). The short-beveled or blunt-tipped needle is then advanced parallel to the nerve into the canal from distal to proximal (Video 38.3). Although some authors recommend obtaining a paresthesia [29], these authors avoid a paresthesia if possible. The use of large volumes (3–5 cc) of local anesthetic and steroids has also been described [29], but these authors limit total volume to no more than 1 cc, injected slowly to avoid high pressures in a closed space. A peripheral nerve stimulator may assist in nerve localizing.

Wrist

With the wrist supported and the patient sitting or supine, the practitioner identifies the flexor carpi ulnaris (lateral) and the ulnar artery (medial), and then a short-beveled or blunt-tipped needle is advanced parallel to the nerve from proximal

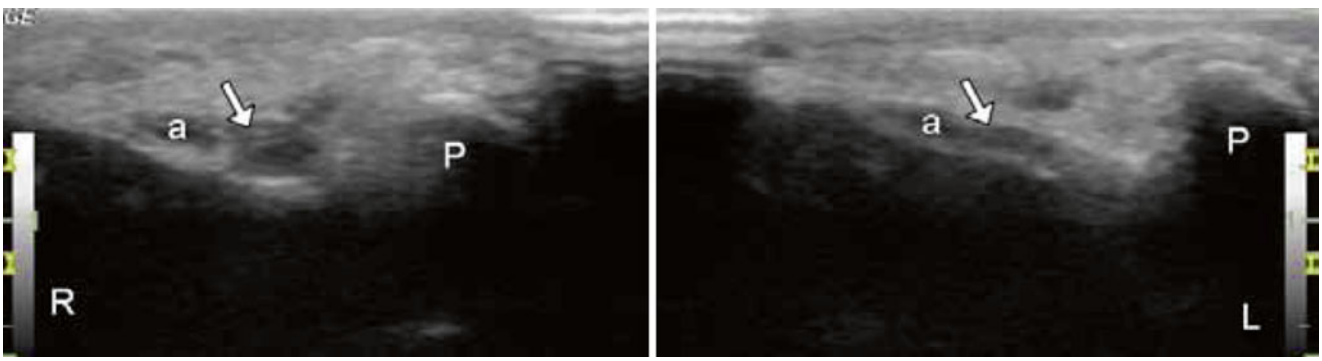

Fig. 38.15 Ultrasound axial view of the both wrists showing the ulnar nerves in the ulnar tunnels (*arrows*), demonstrating enlargement of the ulnar nerve on the right (From Yalcin et al. [21]. Reprinted with permission from American Society of Interventional Pain Physicians)

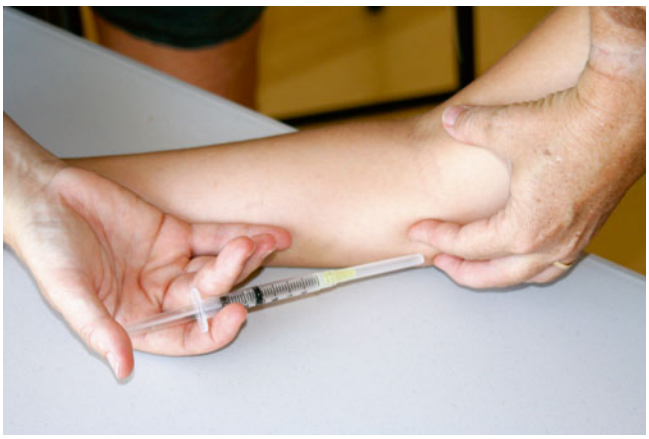

Fig. 38.16 Landmark-guided injection of the elbow (Image courtesy of Andrea Trescot, MD)

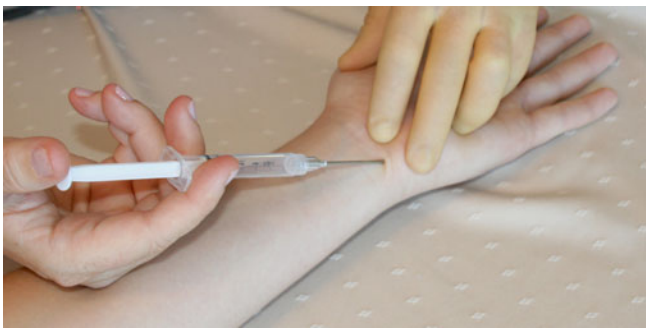

Fig. 38.17 Landmark-guided injection of the wrist (Image courtesy of Andrea Trescot, MD)

to distal (Video 38.4) (Fig. 38.17). Although some authors recommend obtaining a paresthesia [29], these authors avoid a paresthesia, if possible. Use of large volumes (3–5 cc) of local anesthetic and steroids have also been described [29], but these authors limits total volume to no more than 1 cc, injected slowly to avoid high pressures in a closed space. A peripheral nerve stimulator may assist in nerve localizing.

Fluoroscopy-Guided Technique

For fluoroscopy-guided injection of the ulnar nerves at the elbow, the patient should be supine with the arm extended and the elbow also extended. Position the C-arm straight A-P, and identify the medial epicondyle (Fig. 38.18). Insert a blunt-tipped, short-beveled, or stimulating needle vertically, just at the medial border of the humerus. Slowly insert the needle until the stimulator produces the appropriate stimulation response. If the humerus has been touched, the needle is too deep and must be withdrawn. It is advisable to use a wrist restraint so that an inadvertent

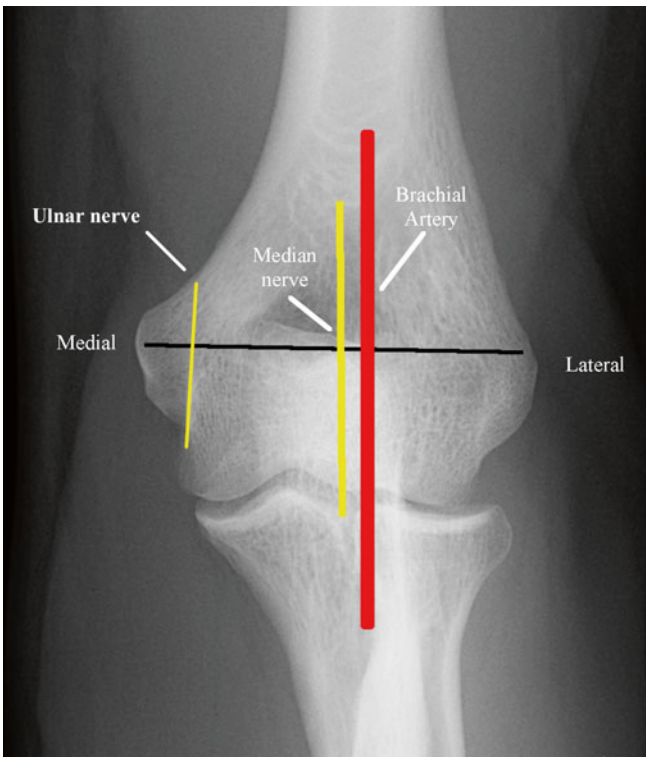

Fig. 38.18 Fluoroscopic landmarks for proximal ulnar injection (Image courtesy of Andrea Trescot, MD)

movement by the patient does not cause damage from movement of the needle.

Ultrasound-Guided Technique

Elbow

The linear probe is placed transversely across the distal ulnar elbow. The ulnar nerve is a bright triangular or oval structure on the ulnar side of the ulnar artery, surrounded by the flexor digitorum superficialis (FDS), flexor digitorum profundus (FDP), and flexor carpi ulnaris (FCU) muscles (Fig. 38.19a, b). The nerve can be injected from this position (out of plane), or the probe can be rotated, keeping the UN centered, and then injected in plane, with the needle parallel to the nerve.

Wrist

The linear probe is placed transversely across the wrist, similar to the US evaluation of the median nerve (see Chap. 37). The ulnar nerve is found on the ulnar side of the ulnar artery, just radial to the pisiform (Fig. 38.19c). The nerve can be injected from this position (transversely, in-plane) (Fig. 38.20), or the probe can be rotated, keeping the UN centered, and then injected in-plane, with the needle parallel to the nerve.

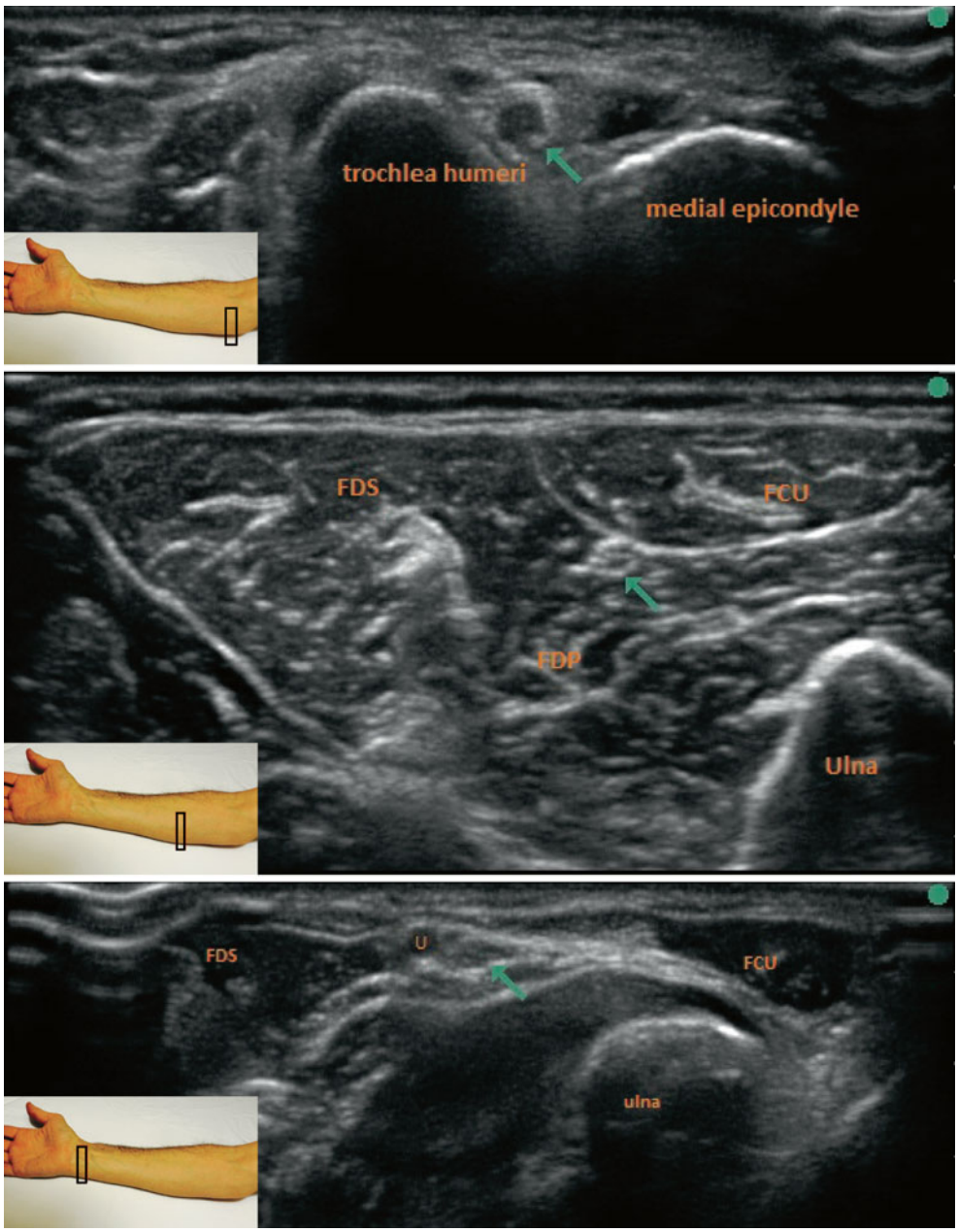

Fig. 38.19 Ultrasound evaluation of the ulnar nerve. (**a**) Ulnar nerve at the ulnar grove at the elbow, marked by a *green arrow*. (**b**) Ulnar nerve (*green arrow*) at the mid-forearm. *FDS* flexor digitorum superficialis muscle, *FDP* flexor digitorum profundus muscle, *FCU* flexor carpi ulnaris muscle. (**c**) Ulnar nerve (*green arrow*) at the wrist, immediately by ulnar artery (U) (Image courtesy of Agnes Stogicza, MD)

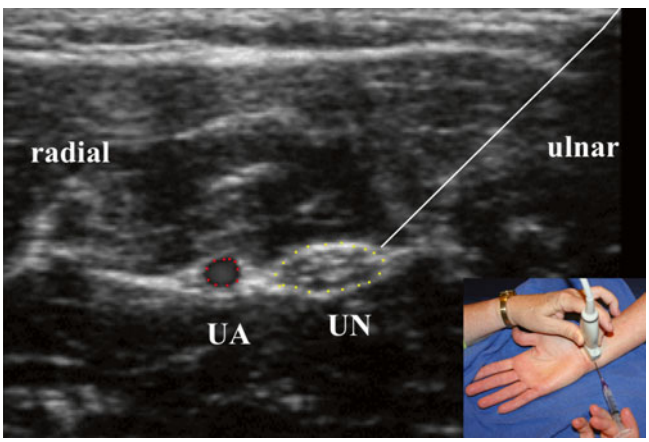

Fig. 38.20 Ultrasound image of the ulnar nerve in Guyon's canal at the wrist, with simulated needle. *UN* ulnar nerve, *UA* ulnar artery (Image courtesy of Andrea Trescot, MD)

Neurolytic Technique

Cryoneuroablation/Radiofrequency Lesioning

Since the ulnar nerve is a significant motor nerve, cryoneuroablation at the cubital tunnel or Guyon's canal would be expected to result in significant (albeit temporary) motor weakness; there would be little use for this technique other than to treat neuromas impinging on the nerve or phantom limb pain [30]. Pulsed radiofrequency lesioning might have similar indications.

Surgical Technique

There are several surgical approaches to ulnar entrapment at the elbow, including simple decompression, medial epicondylectomy, or anterior transposition. Kim DH et al. [31] described 654 surgical outcomes over a 30-year period; 460 entrapments (70 %) were at the elbow, while other pathologies included lacerations, stretch injuries, and gunshot wounds. There were two injection-induced injuries. Almost all of the entrapment cases showed significant intraoperative conduction slowing, even if the preoperative studies were normal.

If the entrapment is in the condylar groove, anterior transposition is the treatment of choice. Since anterior transposition of the nerve requires release of the fascia of the cubital tunnel, it may treat both entrapments [7]. According to a recent systematic review, there is no difference in outcome with and without transposition [19]. Kokkalis et al. [11] recommended wrapping the ulnar nerve with a section of autologous saphenous vein if two or more attempts at decompression have failed, noting that decompression with or without transposition had ~25 % failure rate.

Standard *medial epicondylectomy* may cause elbow instability, ulnar nerve subluxation, pronator weakness, and elbow flexion contractures. According to Kim et al. [32], minimal medial epicondylectomy can improve sensory and motor function for patients with moderate to severe cubital tunnel syndrome.

Most recently, Kamat et al. [33] looked at a 20-year analysis of ulnar nerve transposition vs. neurolysis (480 patients); both were felt to be effective, with only a slight statistical advantage of neurolysis over transposition.

Complications

The UN, especially in the cubital tunnel, is vulnerable to injection injury. Kim DH et al. [31] described injection injuries at the elbow that required surgical repair. Frederick et al. [34] described three cases of nerve injury after wrist injections – one of the ulnar nerve and two of the median nerve. The UN is also vulnerable to injury during endoscopic carpal tunnel release.

Summary

Ulnar nerve entrapment can occur at several sites along the upper extremity, and the presentation as well as the treatment depends largely on the site of entrapment. Accurate diagnosis is necessary for effective treatment and requires differentiation of proximal versus distal pathology.

References

1. Caliandro P, La Torre G, Padua R, Giannini F, Padua L. Treatment for ulnar neuropathy at the elbow. Cochrane Database Syst Rev. 2012;7:CD006839.
2. Frost P, Johnsen B, Fuglsang-Frederiksen A, Svendsen SW. Lifestyle risk factors for ulnar neuropathy and ulnar neuropathy-like symptoms. Muscle Nerve. 2013;48(4):507–15.
3. Machanic BI, Sanders RJ. Medial antebrachial cutaneous nerve measurements to diagnose neurogenic thoracic outlet syndrome. Ann Vasc Surg. 2008;22(2):248–54.
4. Dugas JR. Valgus extension overload: diagnosis and treatment. Clin Sports Med. 2010;29(4):645–54.
5. Svendsen SW, Johnsen B, Fuglsang-Frederiksen A, Frost P. Ulnar neuropathy and ulnar neuropathy-like symptoms in relation to biomechanical exposures assessed by a job exposure matrix: a triple case-referent study. Occup Environ Med. 2012;69(11):773–80.
6. Hoogvliet P, Coert JH, Friden J, Huisstede BM. How to treat Guyon's canal syndrome? Results from the European HANDGUIDE study: a multidisciplinary treatment guideline. Br J Sports Med. 2013;47(17):1063–70.
7. Stewart JD. The variable clinical manifestations of ulnar neuropathies at the elbow. J Neurol Neurosurg Psychiatry. 1987;50(3):252–8.
8. Yang H, Wang B, Wang J, Li R, Zhao S, Bu W. Clinical research of ulnar styloid fracture complicated with wrist dorsal branch of ulnar nerve injury. Zhongguo xiu fu chong jian wai ke za zhi = Zhongguo xiufu chongjian waike zazhi = Chin J Reparative Reconstr Surg. 2013;27(9):1028–31.
9. Cho CH, Kang CH, Jung JH. Ulnar nerve palsy following closed fracture of the distal radius: a report of 2 cases. Clin Orthop Surg. 2010;2(1):55–8.
10. Payan J. Electrophysiological localization of ulnar nerve lesions. J Neurol Neurosurg Psychiatry. 1969;32(3):208–20.
11. Kokkalis ZT, Jain S, Sotereanos DG. Vein wrapping at cubital tunnel for ulnar nerve problems. J Shoulder Elbow Surg. 2010;19(2 Suppl):91–7.
12. Brabston EW, Genuario JW, Bell J-E. Anatomy and physical examination of the elbow. Oper Tech Orthop. 2009;19(4):190–8.
13. von Schroeder HP, Scheker LR. Redefining the "Arcade of Struthers". J Hand Surg Am. 2003;28(6):1018–21.
14. Cobb TK, Carmichael SW, Cooney WP. Guyon's canal revisited: an anatomic study of the carpal ulnar neurovascular space. J Hand Surg Am. 1996;21(5):861–9.
15. Bozkurt MC, Tagil SM, Ozcakar L, Ersoy M, Tekdemir I. Anatomical variations as potential risk factors for ulnar tunnel syndrome: a cadaveric study. Clin Anat. 2005;18(4):274–80.

16. Naran S, Imbriglia JE, Bilonick RA, Taieb A, Wollstein R. A demographic analysis of cubital tunnel syndrome. Ann Plast Surg. 2010;64(2):177–9.
17. Dellon AL. Review of treatment results for ulnar nerve entrapment at the elbow. J Hand Surg Am. 1989;14(4):688–700.
18. deSouza R-M, Choi D. Peripheral nerve lesions. Neurosurgery. 2012;3(30):149–54.
19. Rinkel WD, Schreuders TA, Koes BW, Huisstede BM. Current evidence for effectiveness of interventions for cubital tunnel syndrome, radial tunnel syndrome, instability, or bursitis of the elbow: a systematic review. Clin J Pain. 2013;29(12):1087–96.
20. Suk JI, Walker FO, Cartwright MS. Ultrasonography of peripheral nerves. Curr Neurol Neurosci Rep. 2013;13(2):328.
21. Yalcin E, Akyuz M, Unlu E. Ulnar tunnel syndrome with ultrasonographic nerve imaging. Pain Physician. 2015;18(3):E437–8.
22. Niitsu M, Kokubo N, Nojima S. Variations of the ulnar nerve in Guyon's canal: in vivo demonstration using ultrasound and 3 T MRI. Acta Radiol (Stockholm, Sweden: 1987). 2010;51(8):939–46.
23. Kollmer J, Baumer P, Milford D, Dombert T, Staub F, Bendszus M, et al. T2-signal of ulnar nerve branches at the wrist in Guyon's canal syndrome. PLoS One. 2012;7(10), e47295.
24. Ozdemir O, Calisaneller T, Gulsen S, Caner H. Ulnar nerve entrapment in Guyon's canal due to recurrent carpal tunnel syndrome: case report. Turk Neurosurg. 2011;21(3):435–7.
25. Verhagen AP, Bierma-Zeinstra SM, Burdorf A, Stynes SM, de Vet HC, Koes BW. Conservative interventions for treating work-related complaints of the arm, neck or shoulder in adults. Cochrane Database Syst Rev. 2013;12:CD008742.
26. Svernlov B, Larsson M, Rehn K, Adolfsson L. Conservative treatment of the cubital tunnel syndrome. J Hand Surg Eur Vol. 2009;34(2):201–7.
27. Kim D, Hwang M. Double crush syndrome of the ulnar nerve: wrist and elbow. Clin Neurophysiol. 2008;119, e52.
28. Smith TM, Sawyer SF, Sizer PS, Brismee JM. The double crush syndrome: a common occurrence in cyclists with ulnar nerve neuropathy-a case-control study. Clin J Sport Med. 2008;18(1):55–61.
29. Monkowski D, Collante L. Distal upper extremity blocks. Tech Reg Anesth Pain Manag. 2006;10:132–6.
30. Moesker AA, Karl HW, Trescot AM. Treatment of phantom limb pain by cryoneurolysis of the amputated nerve. Pain Pract. 2014;14(1):52–6.
31. Kim DH, Han K, Tiel RL, Murovic JA, Kline DG. Surgical outcomes of 654 ulnar nerve lesions. J Neurosurg. 2003;98(5):993–1004.
32. Kim KW, Lee HJ, Rhee SH, Baek GH. Minimal epicondylectomy improves neurologic deficits in moderate to severe cubital tunnel syndrome. Clin Orthop Relat Res. 2012;470(5):1405–13.
33. Kamat AS, Jay SM, Benoiton LA, Correia JA, Woon K. Comparative outcomes of ulnar nerve transposition versus neurolysis in patients with entrapment neuropathy at the cubital tunnel: a 20-year analysis. Acta Neurochir (Wien). 2014;156(1):153–7.
34. Frederick HA, Carter PR, Littler JW. Injection injuries to the median and ulnar nerves at the wrist. J Hand Surg Am. 1992;17(4):645–7.

Part VI

Abdominal Wall Pain

Tiffany Zhang

Introduction

Chronic abdominal pain is a common complaint encountered by primary care as well as specialist providers, such as gastroenterologists, emergency medicine specialists, general surgeons, and gynecological surgeons. A 1999 review article reported that up to 5 % of a family practitioner's workload is related to upper gastrointestinal symptoms, which only account for part of chronic abdominal pain complaints [1]. In other studies and reviews, it was also noted that 75 % of otherwise healthy adolescent students, and about half of adults, reported abdominal pain when answering health-related questionnaires [2, 3].

It is estimated that 10–30 % of chronic abdominal pain is related to chronic abdominal wall pain syndrome (CAWP) [4, 5], including abdominal wall nerve entrapments (abdominal cutaneous nerves, genitofemoral nerve, intercostal nerves). Unfortunately, this entity is often overlooked or undiagnosed. These patients often have undergone extensive laboratory testing, imaging, and even invasive procedures and surgery, without significant findings or improvement. This delay in appropriate diagnosis and treatment of CAWP is often related to the lack of knowledge of the providers about its diagnosis and treatment [5, 6].

There are several clues that may point to an abdominal wall source of pain. Unlike visceral pain, abdominal wall pain is usually unaffected by eating or bowel movements. It tends to be chronic, "nagging," and nonprogressive, and it may be related to position (standing, sitting, lying down). While intra-abdominal pain usually is better when the abdominal wall is tensed ("guarding"), the pain from abdominal wall sources is unchanged or increased with tensing of the abdominal wall (a positive Carnett's sign) [5].

In this section, we describe details of several relatively commonly encountered peripheral nerve entrapment syndromes in the abdominal and pelvic regions, including anterior cutaneous nerve entrapment syndrome (ACNES); ilioinguinal, iliohypogastric, and genitofemoral nerve entrapment syndrome; intercostal nerve entrapment; and lumbar plexus entrapment. We review their clinical presentation, findings on physical examination, and diagnostic and interventional options.

References

1. Haycox A, Einarson T, Eggleston A. The health economic impact of upper gastrointestinal symptoms in the general population: results from the Domestic/International Gastroenterology Surveillance Study (DIGEST). Scand J Gastroenterol Suppl. 1999;231:38–47.
2. Hyams JS, Burke G, Davis PM, Rzepski B, Andrulonis PA. Abdominal pain and irritable bowel syndrome in adolescents: a community-based study. J Pediatr. 1996;129(2):220–6.
3. Heading RC. Prevalence of upper gastrointestinal symptoms in the general population: a systematic review. Scand J Gastroenterol Suppl. 1999;231:3–8.
4. Srinivasan R, Greenbaum DS. Chronic abdominal wall pain: a frequently overlooked problem. Practical approach to diagnosis and management. Am J Gastroenterol. 2002;97(4):824–30.
5. Lindsetmo RO, Stulberg J. Chronic abdominal wall pain – a diagnostic challenge for the surgeon. Am J Surg. 2009;198(1):129–34.
6. Suleiman S, Johnston DE. The abdominal wall: an overlooked source of pain. Am Fam Physician. 2001;64(3):431–8.

Intercostal Nerve Entrapment: Abdomen

39

Sola Olamikan, Amitabh Gulati, and Andrea M. Trescot

Introduction

Intercostal neuralgia (IN) is a condition of nerve pain secondary to intercostal nerve pathology. The term "intercostal" refers to the location between two ribs; so technically, the T12 nerve is not an "intercostal" nerve, because there is no rib below it. Like the *anterior cutaneous nerve entrapment* (ACNE) syndrome (see Chap. 42) to which it is related, the pain of intercostal neuralgia is neuropathic in origin. The most common causes of intercostal nerve damage-related abdominal wall pain are postherpetic neuralgia, lower thoracic/upper abdominal surgery, and diabetic thoracic neuropathy [1]. Other causes of intercostal neuralgia include direct nerve injury, stretching, entrapment, and inflammation. Direct nerve injury may be due to physical trauma or may be an aftereffect of surgery. Stretching injuries include traction on the chest wall from an expanding gravid uterus or ascites. Entrapment can be caused by neoplasm, sarcoidosis, and pleural mesothelioma. The pain roughly parallels the rib over the affected nerve. The condition has also been referred to as "*intercostal nerve syndrome*." At times, it can be extremely painful, to the point of being debilitating.

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Clinical Presentation (Table 39.1)

Carnett first described IN as a cause of abdominal pain in 1926 [2] and IN as a cause of pain mimicking gallbladder disease in 1927 [3]. IN is easily misdiagnosed due to the ambiguity of its symptoms. Patients may complain of "rib pain" or "chest pain" (see Chap. 29) but may also describe upper abdominal pain (Fig. 39.1). The history of onset may be clearly related to a specific event such as blunt trauma or rib fracture, a nerve injury such as *shingles*, or *thoracotomy* and *abdominal surgery,* but the onset may also be insidious with no identifiable preceding event. The pain is often perceived as stabbing, sharp, spasm-like, tearing, tender, aching, or gnawing. The pain may wrap around the chest in a dermatomal pattern or radiate from the back toward the front of the chest in a band-like pattern (Fig. 39.2). It may occur in sporadic episodes or may be dull and constant. The pain may intensify with exertion such as with heavy lifting, twisting, or turning the torso. Breathing, coughing, laughing, or sneezing may be painful. It may be associated with numbness or tingling, and allodynia may be present. Pain may start in the back and wrap around

Table 39.1 Occupation/exercise/trauma history relevant to intercostal entrapment

Surgery	Thoracotomy, cholecystectomy
Neuropraxia, stretching injury	Pregnancy, with tension on nerves by gravid uterus; retractor trauma during thoracotomy; frequent, prolonged coughing or ascites
Trauma	Rib fracture or rib contusion; prolonged positioning
Neuropathy	Post-herpetic neuralgia, diabetic peripheral neuropathy
Entrapment	Entrapment at lateral rectus border (ACNE syndrome – see Chap. 42); pleural fibrosis [1]; osteoporotic compression fracture, degenerative disk disease, scoliosis, rib articulation arthritis, exaggerated lumbar lordosis
Postoperative neuroma	Thoracotomy

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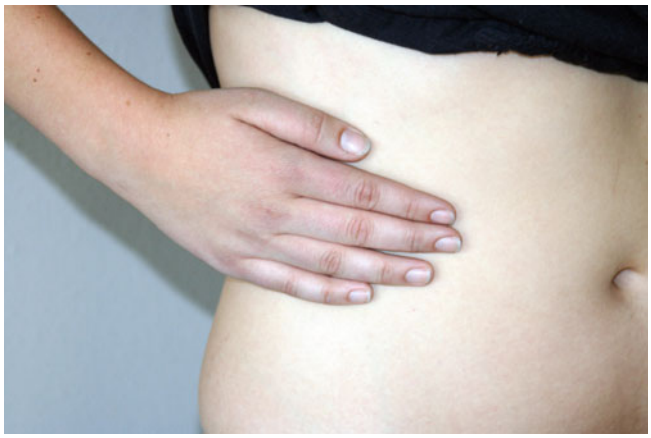

Fig. 39.1 Patient description of intercostal nerve upper abdominal pain (Image courtesy of Andrea Trescot, MD)

the front, or it may be felt only in the front or back. If the subcostal/subgastric nerve is involved, patients may believe they are suffering from gallbladder disease.

Haynsworth and Noe [4] described a patient with 4 months of severe right upper abdominal pain radiating into the spine, associated with nausea and weight loss. When the workup was negative (including laparoscopy, which showed only "incidental scarring" over the liver), the patient underwent intercostal nerve blocks at T10, T11, and T12. Pain was replicated and then relieved with the T11 intercostal injection. Carnett and Bates [5] described intercostal neuralgia from anemia (that resolved with transfusions).

The lower intercostal nerves travel around to the upper abdomen and can get trapped at the edge of the *rectus abdominis* (Fig. 39.3), the muscle that runs vertically from

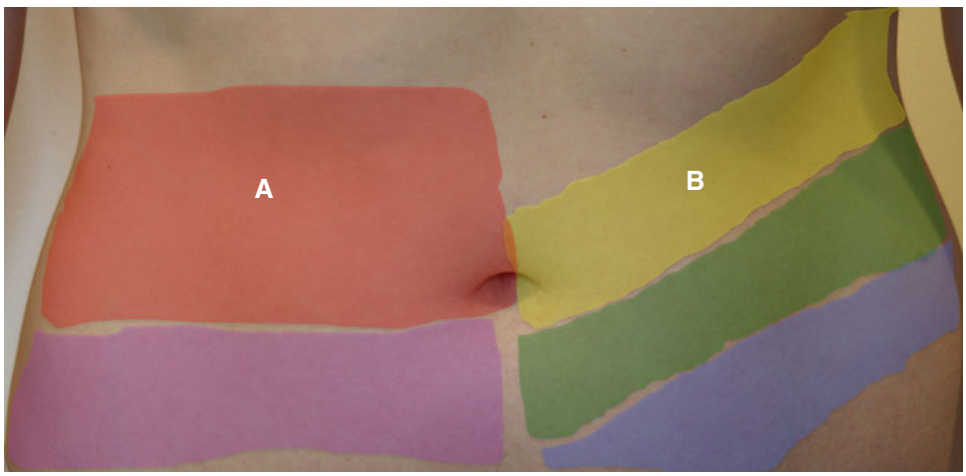

Fig. 39.2 Pain patterns of intercostal abdominal neuralgia; *A* = "band-like" pattern, *B* = dermatomal pattern (Image courtesy of Andrea Trescot, MD)

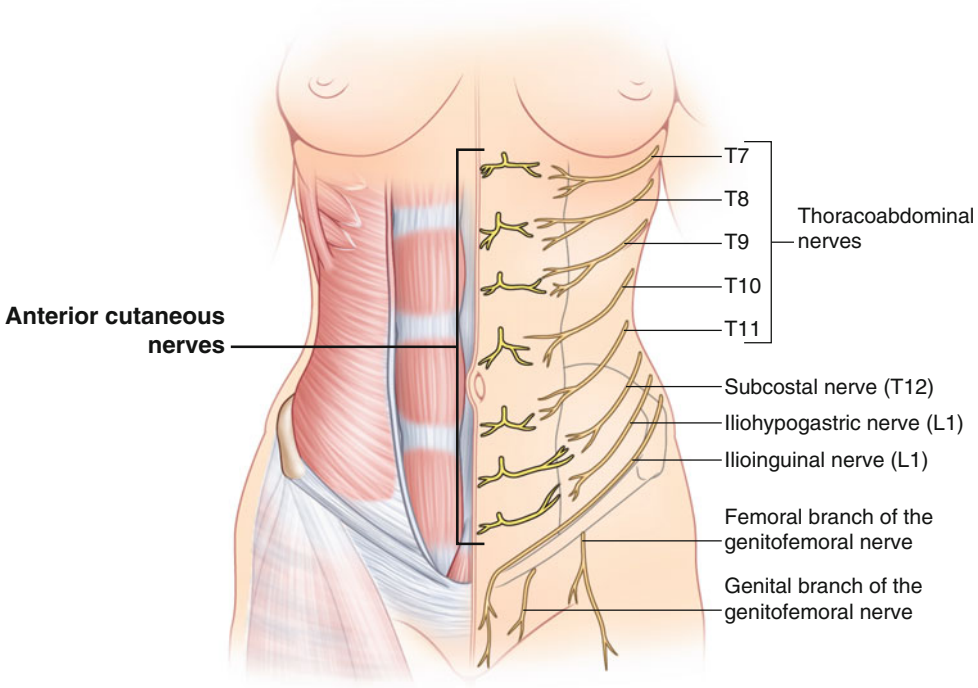

Fig. 39.3 Site of entrapment at the anterior abdominal wall from lower intercostal entrapment (Image by Springer)

the bottom of the breastbone to the top of the pubic bones, causing an *anterior cutaneous nerve entrapment* (ACNE) syndrome, discussed in Chap. 42.

Anatomy (Table 39.2)

Intercostal nerves are the anterior rami of the first 11 thoracic spinal nerves. The anterior ramus of the 12th thoracic nerve travels to the abdomen as the subcostal (also known as subgastric) nerve. The intercostal nerve travels from the *intervertebral foramen*, under the inferior edge of the rib with the *intercostal artery and vein*, to the anterior chest or abdominal wall (Fig. 39.4). Each intercostal nerve enters the corresponding intercostal space between the *posterior intercostal membrane* and the *parietal pleura*. The intercostal nerve then travels forward with the intercostal vessels in the subcostal groove of the corresponding rib, between the innermost intercostal and internal intercostal muscles. The first six intercostal nerves terminate within their respective intercostal spaces in the anterior chest. The lower intercostal nerves leave their intercostal spaces anteriorly (after innervating the structures within) and pass to the anterior abdominal wall (Fig. 39.3).

The *main intercostal nerve* runs in the subcostal groove contained in a fibrous sheath with the intercostal vessels. A typical intercostal nerve has four major branches [6] (Fig. 39.4). The *gray ramus communicans* consists of unmyelinated postganglionic fibers that interface with the sympathetic chain. The *posterior cutaneous intercostal branch* innervates the muscles and skin of the paraspinal area. The *lateral cutaneous nerve* courses with the main intercostal nerve until it penetrates the *intercostal muscles* to arrive at

Table 39.2 Intercostal nerve anatomy

Origin	Anterior rami of the 1st 11 thoracic nerve roots; T12 anterior ramus is called the subcostal nerve (not technically an "intercostal" nerve because there is no 13th rib)
General route	Main intercostal runs in subcostal groove to anterior chest or abdominal wall – 4 branches:
	Gray ramus communicans connects to the sympathetic chain
	Posterior cutaneous innervates the muscles and skin of the paraspinal area
	Lateral cutaneous separates from the main nerve in the anterior axillary line, and provides the skin sensation to the chest and abdominal wall
	T2 (sometimes also T3) lateral cutaneous nerve is called *the intercostobrachial nerve*, crosses the axilla to the medial arm
	Anterior cutaneous provides innervation of the abdominal wall, piercing the fascia of the abdominal wall at the lateral border of the rectus abdominis muscle
Sensory distribution	Skin of the chest wall and abdominal wall
Motor innervation	Intercostal muscles and abdominal wall
Anatomic variability	Collateral nerves connect with a variable number of nerves
Other relevant structures	Subcostal groove, lateral rectus border

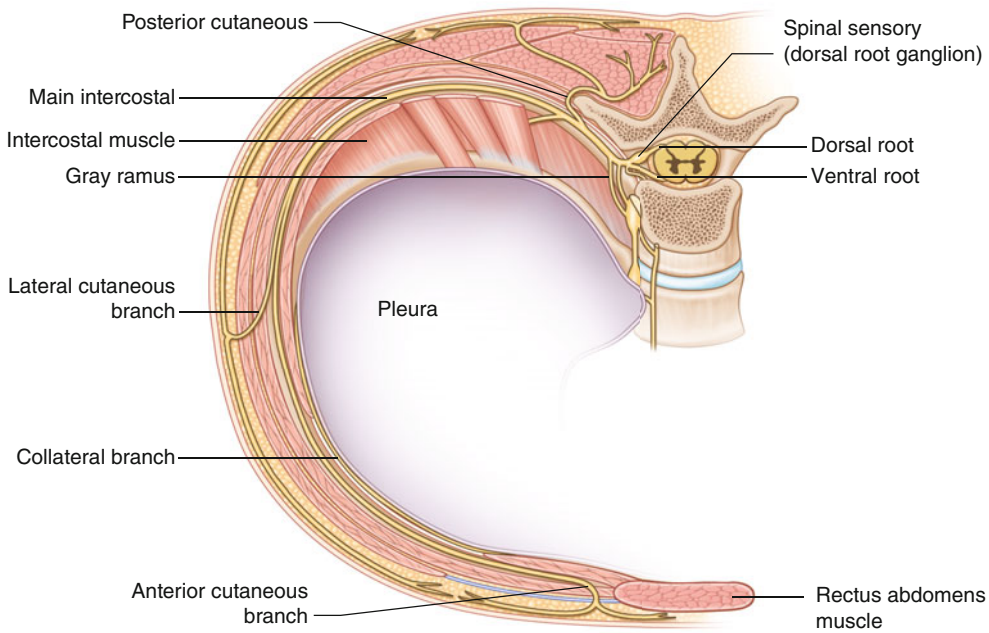

Fig. 39.4 Branches of the intercostal nerve (Image by Springer)

the skin along the midaxillary line; and the *anterior cutaneous nerve*, providing innervation of the abdominal wall, pierces the fascia of the abdominal wall at the lateral border of the rectus abdominis muscle. At this point, the nerve, accompanied by an epigastric artery and vein, makes a sharp turn anteriorly to supply sensation to the anterior abdominal wall; the nerve passes through a firm fibrous ring as it transits the fascia, which results in a site of entrapment. Although traditionally considered unilateral structures, occasionally a given intercostal nerve will cross the midline to provide sensation to the contralateral side.

There is also a *collateral branch* with a less predictable path, though it usually travels near the superior border of the rib below for at least part of its route. Usually, neighboring intercostal nerves communicate with each other via variable additional nerve branches (Table 39.3).

Table 39.3 Branches of intercostal nerves (see Fig. 39.4)

Nerve	Anatomic course and function
Rami communicans	Connects the intercostal nerves to the sympathetic trunk
Collateral branch	Runs parallel to main nerve on upper border of the rib below
Posterior cutaneous	Innervates the muscles and skin of the paraspinal area
Lateral cutaneous branch	Innervates the skin on the side of the thoracic wall by dividing into anterior and posterior branches
Anterior cutaneous branch	Terminal portion of the intercostal nerves, which innervate the skin near the midline of chest
Muscular branches	Supply all the muscles of the intercostal spaces
Pleural branches	Sensory branches to the parietal pleura
Peritoneal sensory branches	Similar to the pleural sensory branches but arise from the lower intercostal nerves and supply the abdominal peritoneum

The 12th intercostal nerve, also called the *subgastric* or *subcostal nerve*, is unique in that it joins with the first lumbar nerve root, thus becoming part of the lumbar plexus.

Causes of Intercostal Neuralgia

A variety of mild to severe diseases, disorders, and conditions may lead to the development of intercostal neuralgia. A chest or rib injury, such as a fractured rib or bruised chest sustained in a motor vehicle accident or participation in sports that involve high speeds or contact with other athletes (e.g., skiing, snowboarding, football, wrestling, and rugby), can be precipitating events. The most common site of *herpes zoster* eruptions is along the path of the intercostal nerve (thoracic *shingles*), which represents 55 % of all shingles cases [7]. Trejjo-Gabriel-Galan et al. [1] described intercostal neuralgia caused by compression of the nerves by a fibrotic mass from a prior tuberculosis infection with chronic pleuritis.

In pregnancy, the increasing size of a growing baby alters the structure of the torso and rib cage such that pressure on the nerves between the ribs can occur. Intercostal neuralgia in pregnancy has been associated with pain and numbness in the ribs, abdomen, and back. Ascites will cause a similar pressure on the nerves.

Oesch et al. [8] documented 14 cases of intercostal neuralgia resulting from an impingement of the intercostal nerve at the level of the anterior rectus sheath, presenting as acute or chronic abdominal pain. This is also known as anterior cutaneous nerve entrapment (ACNE) syndrome, as described in Chap. 42. The authors were able to identify the correct diagnosis upon noting a positive *Carnett test* (pain intensification during palpation while contracting the abdominal muscles by raising the head, while lying supine) (Fig. 39.5) and relief of pain after injecting local anesthesia at the point of maximal tenderness.

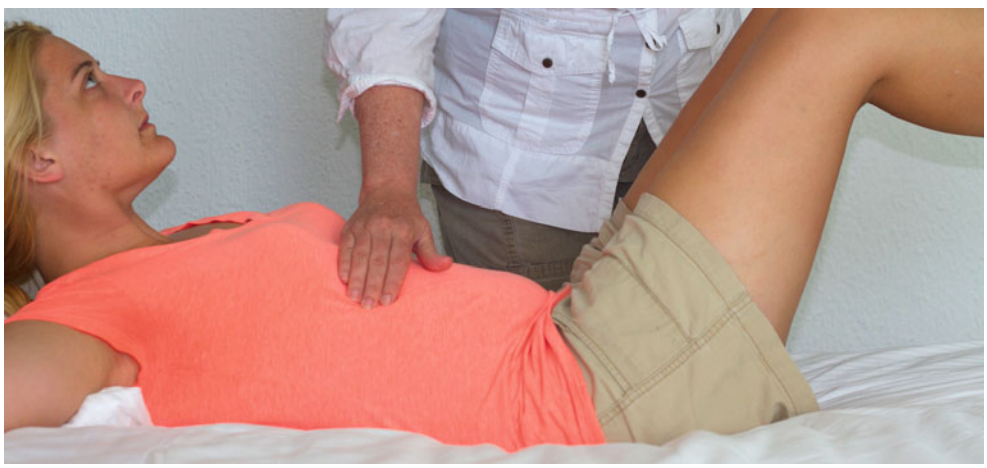

Fig. 39.5 Carnett test (Image courtesy of Andrea Trescot, MD)

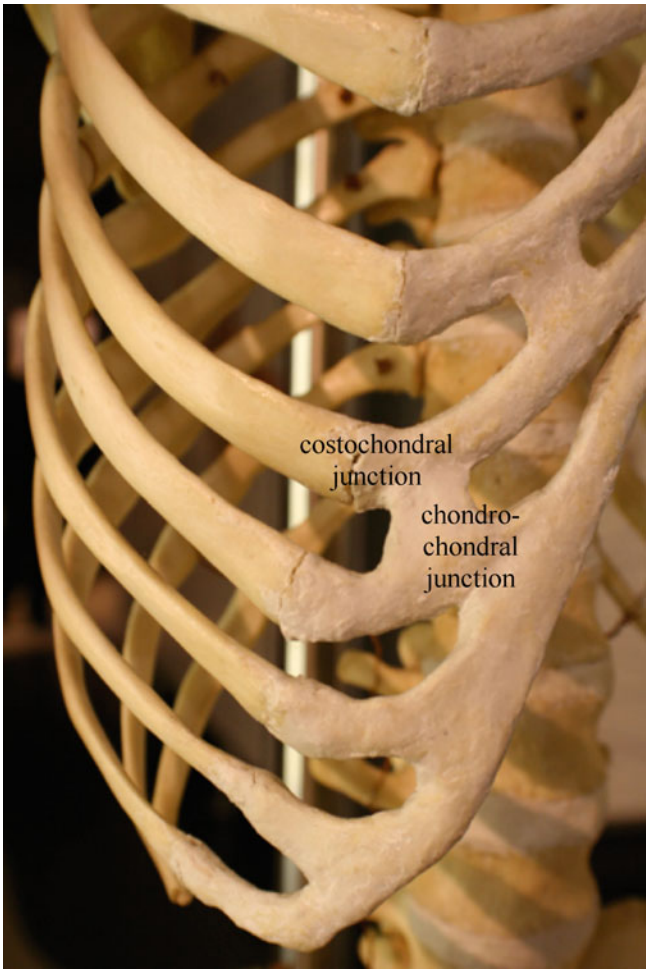

Fig. 39.6 Costochondral and chondrochondral junction of the ribs (Image courtesy of Andrea Trescot, MD)

The intercostal nerve also innervates the *costochondral junction* (where the rib attaches to the cartilage) and the *chondrochondral junction* (where the cartilage attaches to the cartilage above) (Fig. 39.6). These junctions are traumatized by forces less than necessary to fracture the rib itself (visualize how easy it is to pop the cartilage off the end of a barbequed spare rib), but will not show up on X-ray. The patient may describe a sharp, painful, slipping, or popping sensation in the anterior chest with movement; smokers, perhaps because they have poor blood flow to the tissues or because of chronic cough, seem to be most susceptible.

Intercostal nerve injury has long been suspected as a complication of *thoracotomy* procedures. Rogers et al. [9] recorded motor-evoked potentials from intercostal muscles in 13 patients undergoing thoracotomy. Intercostal nerves functioned normally before and after entering the pleural space. After the rib retractor was removed, there was a total conduction block in the nerve immediately above the incision in every patient. In the nerves above this, six had a total block, one had a partial block, and three had normal conduction. There was a total conduction block in the nerve immediately below the incision in all but one patient. Of the nerves below this, four had a total block and two a partial block, and three had normal conduction. In a single patient where rib retraction was not employed, there was no impairment of the intercostal nerves throughout the operation. This study demonstrates that intercostal nerve injury occurs routinely due to rib retraction during thoracotomy.

Wildgaard et al. [10] reviewed literature published from 2000 to 2008 on post-thoracotomy pain syndrome. Although the authors noted the limit of their ability to compare data from the various studies, they concluded that intercostal nerve injury does appear to be the most important pathogenic factor in chronic post-thoracotomy pain. *Intercostal neuromas* have also been identified as a source of pain after aesthetic and reconstructive breast implant surgery [11].

Shingles (also termed *herpes zoster* or *zoster*) is a disease caused by reactivation of a previous infection with the herpes zoster virus (the same virus that causes chickenpox, termed *varicella zoster virus*). After a chickenpox outbreak, the virus remains dormant for many years, usually in the roots of sensory nerves. Upon reactivation, it typically causes unilateral pain, burning, or tingling and blistering rash in the sensory distribution of the nerve. Shingles can involve an intercostal nerve and results in a painful rash along the chest or abdominal wall. The risk of the disease increases with age, with about half of all cases occurring among men and women 60 years of age or older. Conditions that may trigger reactivation include stress, fatigue, weakened immune system, cancer, and HIV. As many as 25 % of people over 50 years old with shingles develop postherpetic neuralgia [12], where significant pain remains even after the rash has resolved. It can be very difficult to treat.

Physical Exam

The physical examination will generally reveal minimal physical findings, unless there is a history of previous thoracic or subcostal surgery or cutaneous findings of herpes zoster involving the thoracic dermatomes. In contrast to musculoskeletal chest wall pain syndromes, the patient does not attempt to splint or protect the affected area. Careful sensory examination of the affected dermatomes may reveal decreased sensation or allodynia. With significant motor involvement of the subcostal nerve, the patient may complain that his or her abdomen bulges out (Fig. 39.7). Pressure or stretching over the site of the nerve injury will typically reproduce symptoms (Video 39.1) (Fig. 39.8). Finally, the single most effective diagnostic test for intercostal neuralgia is an intercostal nerve block.

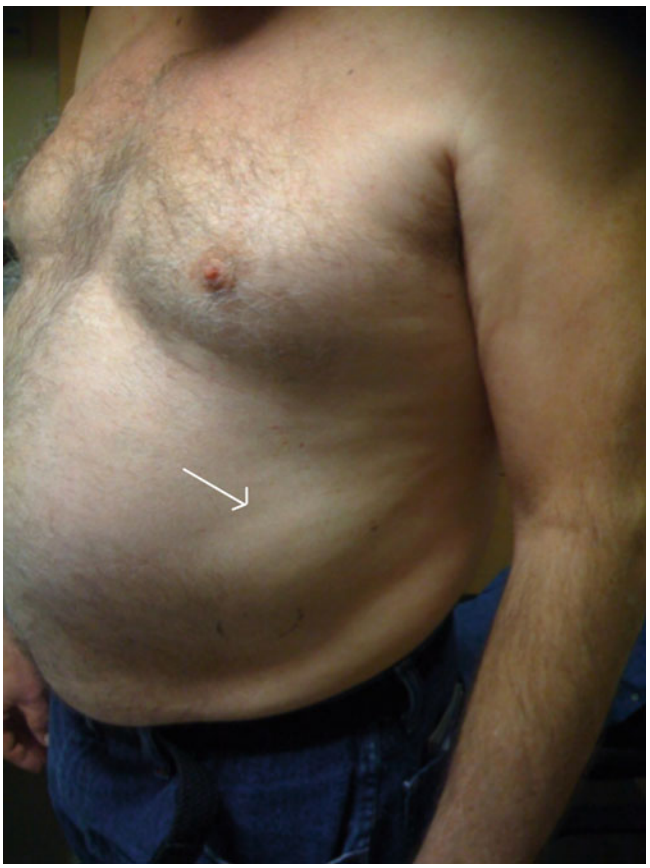

Fig. 39.7 Bulging abdominal wall caused by intercostal nerve injury – *arrow* shows the abdominal wall bulge (Image courtesy of Andrea Trescot, MD)

Fig. 39.8 Palpation of the intercostal nerve (Image courtesy of Andrea Trescot, MD)

Differential Diagnosis (Table 39.4)

The diagnosis of intercostal neuralgia is typically one of exclusion. Abdominal pathology should be ruled out. Although uncommon, thoracic disk herniation should be excluded. Intercostal nerve conduction study has proved to be an accurate technique in the diagnosis of thoracic radiculopathy. Johnson et al. [13] performed intercostal nerve conduction studies in 161 patients, 80 of whom had subsequent posterior rhizotomy (81 % of those noted relief of pain with surgery). The only significant complication of the intercostal nerve conduction study is an 8.8 % incidence of pneumothorax. Pradhan and Taly [14] then developed a surface nerve conduction technique to study the intercostal nerves.

Chen et al. [15] described two patients with pain after shingles, presumed because of post-herpetic neuralgia; both patients complained of burning, shooting, aching pain, and allodynia (consistent with neuropathic pain), localized to the site of the previous lesions. However, they were able to identify trigger points in the intercostal muscles just below the hyperesthetic region; injections of local anesthetic in this region replicated and then abolished the pain. Although the patients required several sessions of injections, both were virtually pain-free 6 months later. The authors felt that the relief was unlikely to be due to an intercostal nerve block, since they injected only 1 cc of local anesthetic at the superior (not inferior) edge of the rib. However, it is conceivable that, by relaxing the intercostal muscle, they released an entrapment of the intercostal nerve (Table 39.5).

Table 39.4 Differential diagnosis of abdominal wall pain

Disorders	Potential distinguishing features
Cholecystitis, appendicitis, diverticulitis, spleen pathology	History, abdominal CT, sonography, endoscopic tests or MRI, lab work
Endometriosis	History, abdominal CT, sonography or MRI, lab work, manual examination
Abdominal wall hernia, Spigelian hernia	History, abdominal CT, sonography, lab work, palpation of the hernia or abdominal wall defect
Herpes zoster	History, skin rash, examination
Interstitial cystitis	History, cystography, sonography or MRI, lab work, manual examination
Irritable bowel disease	History, abdominal CT, sonography, endoscopic tests, lab work
Rectus sheath hematoma	History, abdominal CT, sonography and exam with evidence of hematoma
Slipped rib syndrome	History, chest X-ray, examination with pain on pulling on lower rib, palpable popping with subluxation

Table 39.5 Diagnostic tests for intercostal neuralgia

	Potential distinguishing features
Physical exam	Tenderness over the rib or rib cartilage junction; positive Carnett's sign
Diagnostic injection	Intercostal nerve block is diagnostic
Ultrasound	Useful for nerve localization
MRI/CT	May show rib fracture
Arteriography	Nondiagnostic
X-ray	May show rib fracture or calcification of cartilage
Electrodiagnostic studies	May show conduction slowing

Identification and Treatment of Contributing Factors

Carnett and Bates described exaggerated *lumbar lordosis* as the most frequent direct cause of chronic intercostal neuralgia, especially in patients younger than 35 years old [5]. They also described intercostal neuralgia from *scoliosis*, usually from the side of the concavity, noting that it is not uncommon to find neuralgic pain in the abdomen on one side and chest wall pain on the other side. Chronic cough or increased abdominal girth (obesity, pregnancy, and ascites) will put tension on the intercostal nerve. As noted above, smokers appear to be more likely to have rib nonunion or nonhealing cartilage issues.

For thoracic disk herniations, percutaneous diskectomy can offer relief. For chronic cough leading to costochondral pathology, the use of cough suppressants may help. Topical medications can give relief from post-herpetic neuralgia.

Carnett and Bates [5] noted that those patients with their most severe pain at night or when they first awaken usually improve when shifting to a firmer bed. It is interesting to note that they also recommended X-ray treatment ("About 15–20 % of an erythema skin dose is employed four to six times at intervals of five to seven days"), a large radiation dose that would not be recommended today.

Intercostal Nerve Block Techniques

The technique may be performed with landmark guidance, fluoroscopic guidance, or with the use of ultrasound guidance. Radiological guidance is advised for neurolytic blocks.

An intercostal nerve block with local anesthetic and corticosteroid serves as a diagnostic test for intercostal neuralgia and offers short-term (and potentially long-term) therapeutic relief. The immediate effect is usually from the local anesthetic injected. This wears off in a few hours. The deposteroid starts working in about 24–48 h, and its effect can last for several days to a few months. If the first injection does not relieve symptoms in about a week or 2, a second injection may be recommended. Repeat injections can be done about 1 week apart, if needed.

Landmark-Guided Technique

The patient is placed in the prone position with the patient's arm hanging loosely off the side of the table. Alternatively, this block can be done with the patient in the sitting or lateral position. Because of the overlapping innervation of the chest and upper abdominal wall, the intercostal nerves above and below the nerve suspected of subserving the painful condition will likely need to be injected as well.

There are several sites for injecting an intercostal nerve, based on the location of pathology. Perhaps the most common site is at the angle of the rib about 7 cm lateral to the midline in adults. The landmark-guided ("blind") injection technique should be reserved for those patients in whom the ribs and rib interspace can be reliably palpated.

The skin is first drawn cephalad about one cm with the palpating hand, and a 1.5–2 in. (5 cm) 22- to 27-gauge (for single shot injection), short-bevel needle is introduced through the chosen site of entry at a 20° cephalad angle, with the bevel facing cephalad. The needle is advanced until it contacts the rib at a depth of less than 1 cm (for most nonobese patients). At this moment, a small amount of local anesthetic may be injected to anesthetize the periosteum. With the palpating hand holding the needle firmly and resting securely on the patient's back, the injecting hand gently walks the needle caudally while the skin is allowed to move back over the rib (Video 39.2). The needle is now advanced about 3 mm, still maintaining the 20° tilt angle cephalad. A subtle "give" or "pop" of the fascia of the internal intercostal muscle may be felt, especially if a short-bevel needle is used. As the distance from the posterior aspect of the rib to the pleura averages 8 mm, advancement of the needle much beyond 3 mm increases the risk of pneumothorax. Paresthesia, while not actively sought, confirms needle placement. A peripheral nerve stimulator may aid in confirmation (Fig. 39.9). After negative aspiration to ensure the needle is not in a blood vessel or intrapleural, local anesthetic and steroid are injected. It is desirable to block at least one intercostal nerve cephalad and one caudad because of overlapping innervation.

Because the perpendicular approach has a significant risk of pneumothorax, an alternative approach places the needle more parallel to the bottom of the rib, sliding the needle up underneath the inferior edge of the rib (Fig. 39.10).

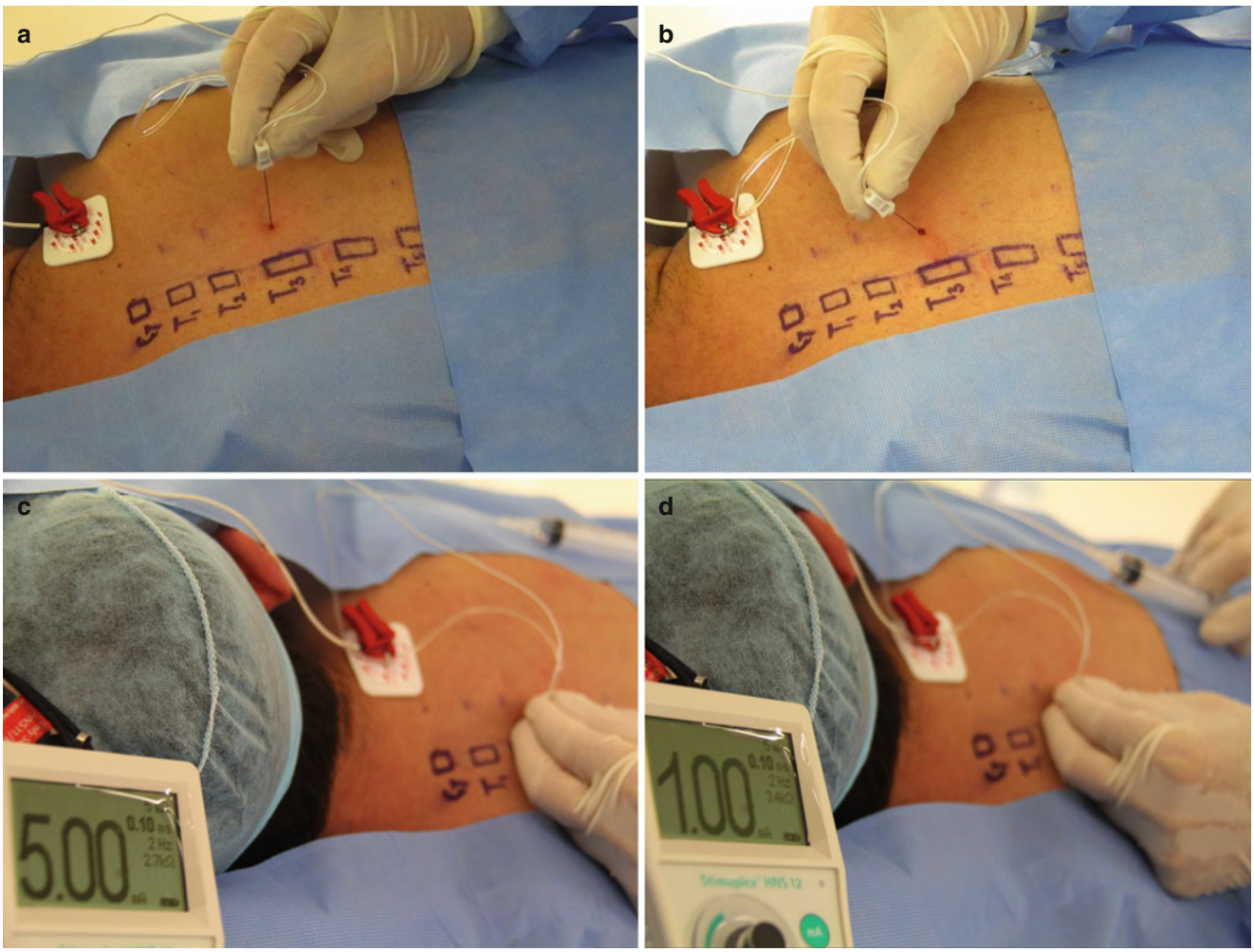

Fig. 39.9 Landmark-guided intercostal nerve block. (**a**) The needle is placed perpendicular to the skin; (**b**) the needle is walked inferiorly; (**c**) stimulator is used to identify the nerve location at a higher voltage; (**d**) stimulator is used at a lower voltage to identify the nerve (Image courtesy of Thialgo Nouer Frederico, MD)

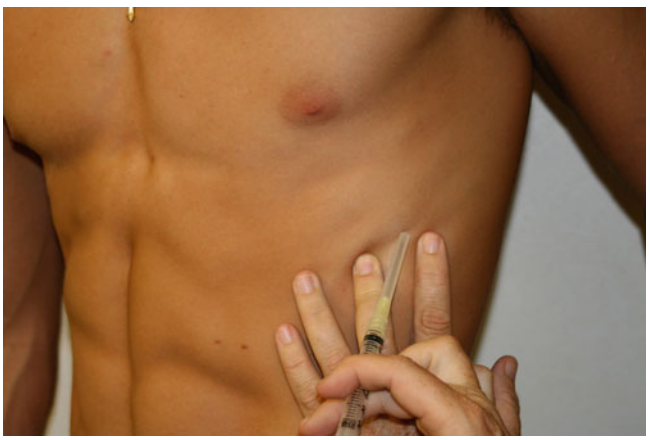

Fig. 39.10 Landmark-guided intercostal nerve block (alternative technique) – needle is more parallel to the inferior rib (Image courtesy of Andrea Trescot, MD)

For the lower intercostal nerves, the entrapment may be at the rectus border. The patient is positioned supine and the lateral border of the rectus palpated (Fig. 39.11a). The injection technique is then the same as used for the ACNE syndrome (see Chap. 42), with the needle advanced obliquely to the lateral border of the rectus (Fig. 39.11b). A peripheral nerve stimulator may aid in localization.

Fluoroscopic-Guided Technique

Use of fluoroscopy allows precise localization of the rib, potentially decreasing the risk of pneumothorax. The patient is positioned prone, supine, or lateral, depending on the site of pain. Instead of placing the needle perpendicular to the rib (Fig. 39.12), the needle can be placed obliquely at the inferior

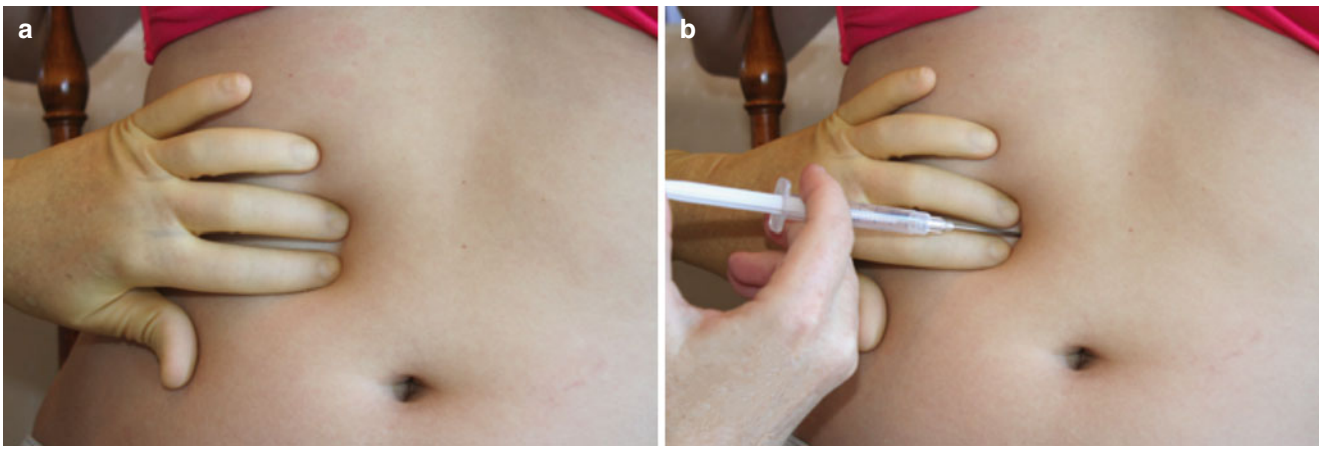

Fig. 39.11 Injection of the intercostal at the rectus border. (**a**) Palpation of the lateral rectus border; (**b**) injection at the lateral rectus border (Image courtesy of Andrea Trescot, MD)

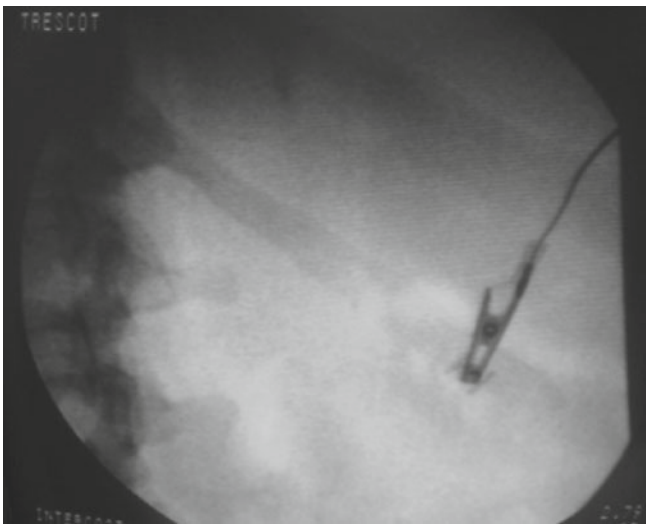

Fig. 39.12 Fluoroscopic standard intercostal nerve injection; the needle is perpendicular to the nerve (Image courtesy of Andrea Trescot, MD)

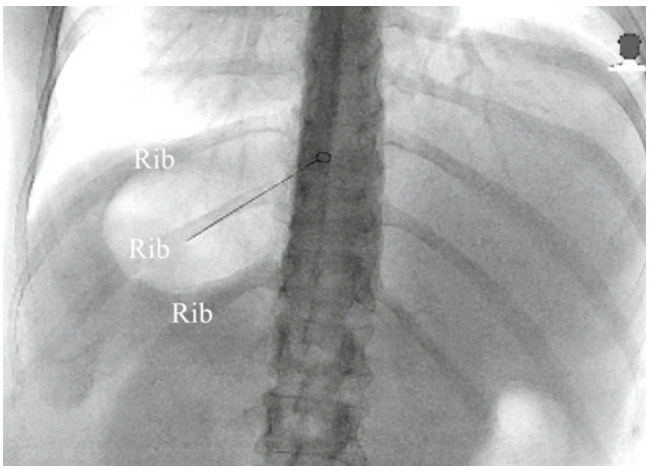

Fig. 39.13 Fluoroscopic intercostal nerve injection; the needle is parallel to the inferior edge of the rib and then is slid up under the rib (Image courtesy of Andrea Trescot, MD)

edge of the rib, sliding up underneath the edge of the rib (Fig. 39.13). A peripheral nerve stimulator will help to identify the nerve (Fig. 39.14), and contrast will confirm the perivascular spread of medication (Fig. 39.15).

Ultrasound-Guided Technique

Use of ultrasound has dramatically decreased the risk of pneumothorax, by allowing clear identification of the rib at the bedside. The patient can be positioned prone, lateral, or sitting. With the US probe positioned vertically (Fig. 39.16a), the US images demonstrate three layers of the intercostal muscles (external, internal, and innermost) covering the pleural line (Fig. 39.16b) [16]. The neurovascular bundle lies between the internal and innermost intercostal muscles. The US probe is then placed horizontally (Fig. 39.17), and the needle introduced in-plane.

Neurolytic Technique

Intercostal nerve blocks can predict how a patient will respond to neurolytic techniques. A good nerve block response usually means the patient will benefit from neurolytic procedures as well. Fluoroscopic or ultrasound guidance improves the accuracy of these blocks and minimizes complications.

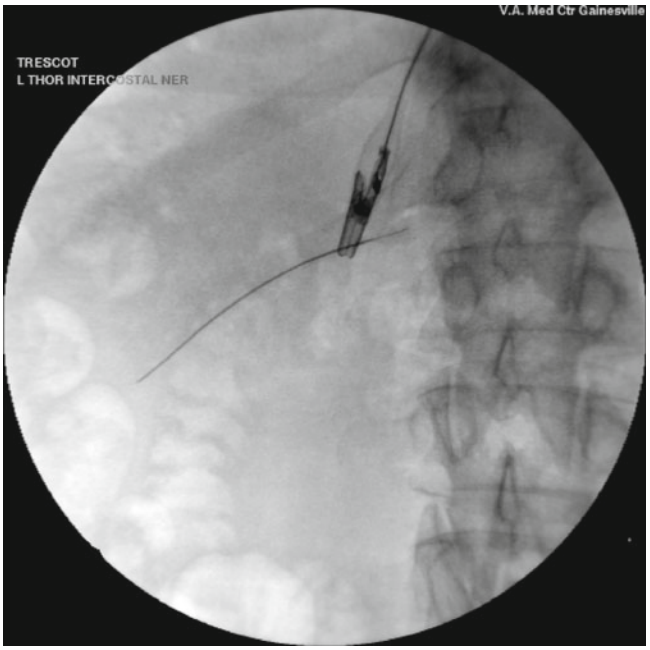

Fig. 39.14 Fluoroscopic intercostal nerve injection; the needle is parallel to the inferior edge of the rib and then is slid up under the rib using a peripheral nerve stimulator (Image courtesy of Andrea Trescot, MD)

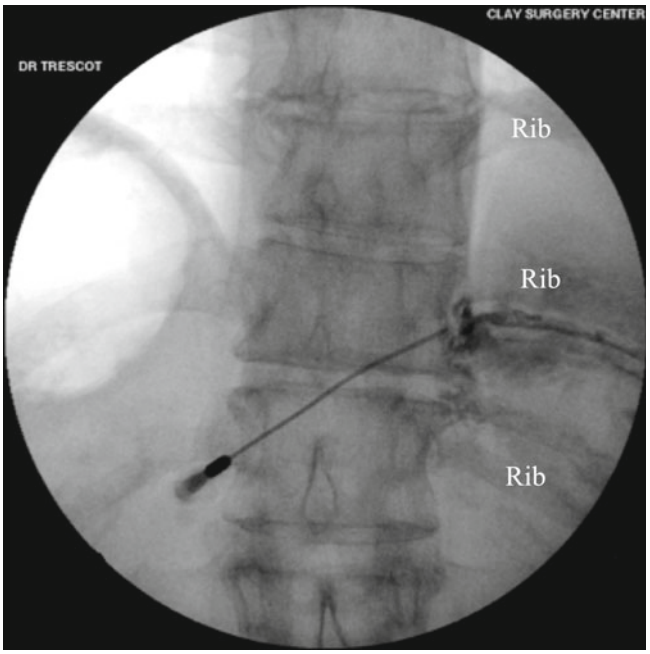

Fig. 39.15 Fluoroscopic intercostal nerve injection; the needle is parallel to the inferior edge of the rib, using a peripheral nerve stimulator, with position confirmed by contrast (Image courtesy of Andrea Trescot, MD)

Cryoneuroablation

One of the most commonly described uses of cryoneuroablation has been for intercostal neuralgia. The 12- or 14-gauge introducer is introduced at the inferior border of the rib and advanced laterally; the stylet is then removed and the probe

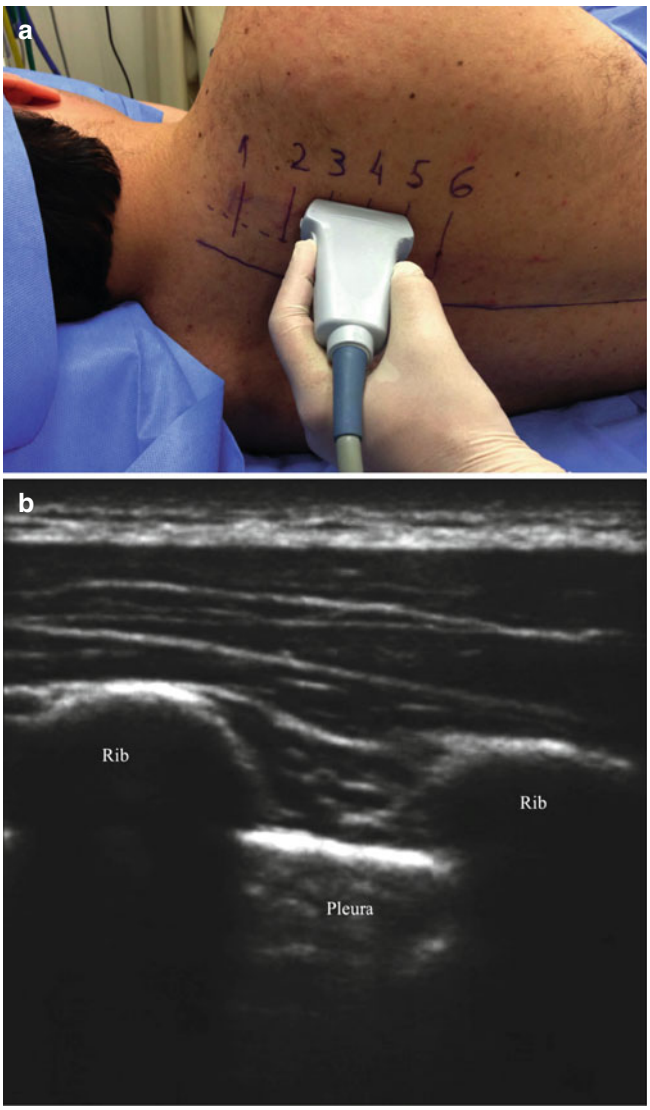

Fig. 39.16 Ultrasound identification of the intercostal space, with the US probe placed vertically. (**a**) Location of probe; (**b**) US image showing rib, intercostal space, and pleura (Images courtesy of Thialgo Rouer Frederico, MD)

introduced underneath the inferior edge of the rib (Figs. 39.18 and 39.19).

Green and colleagues [17] retrospectively looked at 43 patients with chronic chest pain treated with cryoneuroablation. The mean duration of pain prior to cryoneurolysis was 31 months (range: 0.5 months to 24 years). Sixty percent of the patients reported a decrease in their pain immediately after the procedure. Three months after cryoneurolysis, 50 % of the patients reported significant pain relief.

Radiofrequency Lesioning

A case series by Engel [18] reports efficacy of conventional radiofrequency neurolysis in refractory intercostal neuralgia due to blunt chest wall trauma. Garcia Cosamalòn et al. [19]

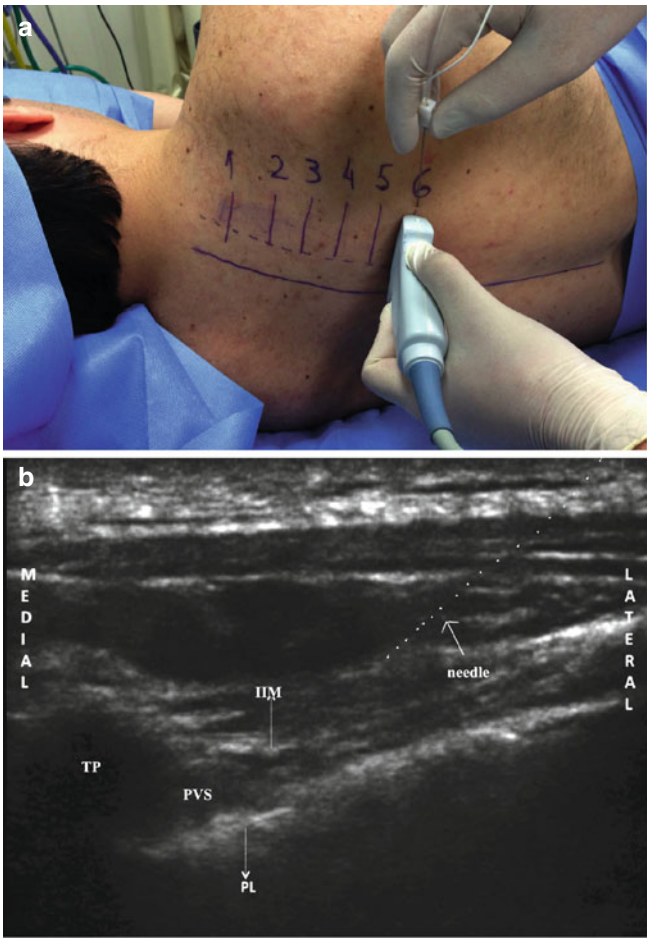

Fig. 39.17 Ultrasound injection of the intercostal space. (**a**) Location of probe; (**b**) US image. *PL* pleural space, *TP* transverse process, *PVS* paravertebral space, *IIM* internal intercostal membrane (Images courtesy of Thialgo Rouer Frederico, MD)

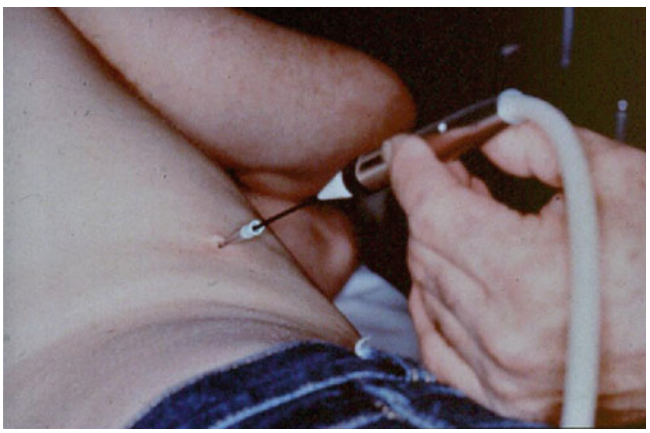

Fig. 39.18 Intercostal cryoneuroablation (Image courtesy of Epimed®, with permission)

describe the use of CT-guided dorsal percutaneous radiofrequency rhizotomy for intercostal neuralgia. Akkaya and Ozkan [20] described good relief for three intercostal neuralgia patients using pulsed radiofrequency under US guidance.

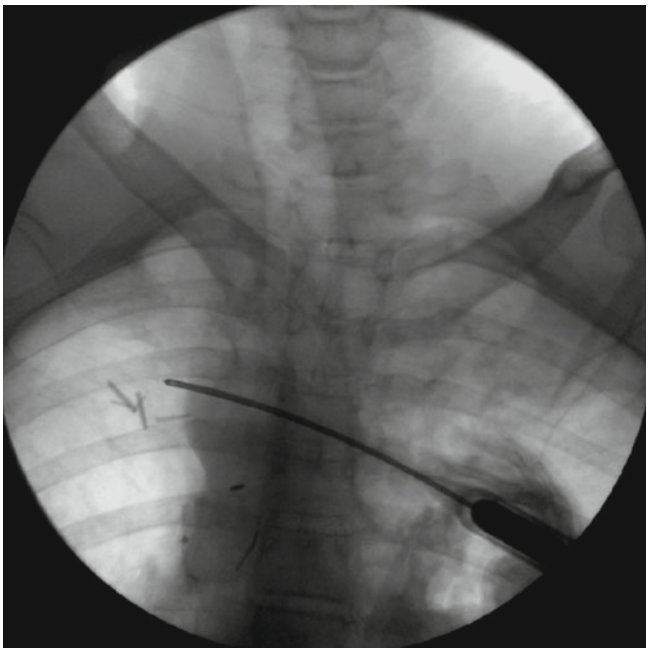

Fig. 39.19 Location of the cryoprobe for intercostal neuralgia (Image courtesy of Andrea Trescot, MD)

Chemical Neurolysis

Alcohol and phenol are the preferred agents for neurolytic procedures because they cause axonal degeneration within minutes and effectively interrupt the central transmission of pain impulses. Chemical neurolysis can result in immediate and total pain relief in selected patients, but it should only be considered for refractory neuralgic pain due to intercostal nerve injury from rib fracture or post-thoracotomy and for refractory postherpetic neuralgia. Complications include unintended spread of alcohol or phenol to the root cuff, epidural space, or cerebrospinal fluid.

Neuromodulation

Although peripheral nerve stimulation has been used for intercostal chest wall pain (see Chap. 29) and lower abdominal wall pain (see Chaps. 40 and 42), there are no reports of the use of peripheral nerve stimulation for the lower thoracic/upper abdomen region (perhaps because of the increased movement in the area).

Surgical Decompression/Neurectomy

Oesch et al. [8] reported results of a retrospective study of 14 patients with intercostal nerve impingement at the level of the anterior rectus sheath, called anterior cutaneous nerve entrapment (ACNE) (see Chap. 42). All patients were treated with neurectomy (surgical resection) at the point of

exit of the nerve from the anterior rectus sheath. Minimal side effects were noted, but follow-up was limited. In a double-blind, randomized, controlled trial, Boelens et al. [21] studied 44 patients with anterior cutaneous nerve entrapment from 2008 to 2010. Half of the patients underwent neurectomy at the level of the abdominal wall. The other half underwent a sham surgical procedure. Of the 22 neurectomy patients, 16 reported a successful pain response at 6 weeks postoperatively. The authors of the study concluded that neurectomy is an effective surgical procedure for pain reduction in anterior cutaneous nerve entrapment syndrome.

Williams et al. [22] studied five consecutive patients who underwent neurectomy of one or more intercostal nerves and implantation of the cut nerve(s) into the latissimus dorsi or into the rib. The average follow-up after surgery was 8.8 months. Preoperatively, mean average pain level was 8 (range: 7–9). Mean average pain level postoperatively was 2.2 (range: 0–7). The authors concluded that neurectomy and implantation of the cut intercostal nerve into the latissimus dorsi or into the rib was an efficacious treatment for this small group of patients.

Concerns still exist regarding the overall efficacy of neurectomy as a treatment for intercostal nerve impingement. Neurectomy complications include neuroma formation, which may result in worsened pain. According to Shapiro, Woodall, and Muller [23], nerve transections of rat sciatic nerves performed by use of a scalpel or a CO2 laser resulted in neuroma formation in all cases.

Complications

Pneumothorax (often signaled by, but *not* caused by, a cough) and penetration of the peritoneum and abdominal viscera are potential complications. Absorption of local anesthetic from the intercostal space is rapid; toxicity is always a concern with multiple or continuous intercostal injections. Because the dural sheath can extend up to 8 cm laterally, there is a rare chance of spinal anesthesia.

Summary

Intercostal neuralgia can cause severe, debilitating pain that may be difficult to treat. Causes include chest wall injury, pregnancy, abdominal impingement, and shingles. The syndrome may also occur iatrogenically, following thoracotomy procedures and abdominal surgery. The condition is also implicated in abdominal wall pain. When conservative treatments fail, the first line of more aggressive management consists of a series of anesthetic and corticosteroid nerve blocks. In patients who do not respond to repeat nerve blocks, cryoneuroablation or radiofrequency neurolysis may provide pain relief. Additional measures described in the literature include neurectomy.

References

1. Trejo-Gabriel-Galan JM, Macarron-Vicente JL, Lazaro L, Rodriguez-Pascual L, Calvo I. Intercostal neuropathy and pain due to pleuritis. Pain Med. 2013;14(5):769–70.
2. Carnett JB. Intercostal neuralgia as a cause of abdominal pain and tenderness. Surg Gynecol Obstet. 1926;42:625.
3. Carnett JB. The simulation of gallbladder disease by intercostal neuralgia of the abdominal wall. Ann Surg. 1927;86(5):747–57.
4. Haynsworth Jr RF, Noe CE. An unusual presentation of intercostal neuralgia. Anesthesiology. 1990;73(4):779–80.
5. Carnett JB, Bates W. The treatment of intercostal neuralgia of the abdominal wall. Ann Surg. 1933;98(5):820–9.
6. Davies F, Gladstone RJ, Stibbe EP. The anatomy of the intercostal nerves. J Anat. 1932;66(Pt 3):323–33.
7. Trescot AM. Shingles and interventional pain treatment. ASIPP News. 2006(Fall):22–3.
8. Oesch A, Kupfer K, Bodoky A. Intercostal nerve syndrome. Swiss Surg = Schweizer Chirurgie = Chirurgie suisse =Chirurgia svizzera. 2001;7(2):82–5.
9. Rogers ML, Henderson L, Mahajan RP, Duffy JP. Preliminary findings in the neurophysiological assessment of intercostal nerve injury during thoracotomy. Eur J Cardiothorac Surg: Off J Eur Assoc Cardiothorac Surg. 2002;21(2):298–301.
10. Wildgaard K, Ravn J, Kehlet H. Chronic post-thoracotomy pain: a critical review of pathogenic mechanisms and strategies for prevention. Eur J Cardiothorac Surg: Off J Eur Assoc Cardiothorac Surg. 2009;36(1):170–80.
11. Nguyen JT, Buchanan IA, Patel PP, Aljinovic N, Lee BT. Intercostal neuroma as a source of pain after aesthetic and reconstructive breast implant surgery. J Plast Reconstr Aesthet Surg. 2012;65(9):1199–203.
12. Gimenez-Mila M, Busquets C, Ojeda A, Fauli A, Moreno LA, Videla S. Neuropathic pain with features of complex regional syndrome in the upper extremity after herpes zoster. Pain Pract. 2014;14(2):158–61.
13. Johnson ER, Powell J, Caldwell J, Crane C. Intercostal nerve conduction and posterior rhizotomy in the diagnosis and treatment of thoracic radiculopathy. J Neurol Neurosurg Psychiatry. 1974;37(3):330–2.
14. Pradhan S, Taly A. Intercostal nerve conduction study in man. J Neurol Neurosurg Psychiatry. 1989;52(6):763–6.
15. Chen SM, Chen JT, Kuan TS, Hong CZ. Myofascial trigger points in intercostal muscles secondary to herpes zoster infection of the intercostal nerve. Arch Phys Med Rehabil. 1998;79(3):336–8.
16. Sakai F, Sone S, Kiyono K, Imai S, Izuno I, Oguchi M, et al. High resolution ultrasound of the chest wall. RoFo: Fortschr Gebiete Rontgenstrahlen Nuklearmedizin. 1990;153(4):390–4.
17. Green CR, de Rosayro AM, Tait AR. The role of cryoanalgesia for chronic thoracic pain: results of a long-term follow up. J Natl Med Assoc. 2002;94(8):716–20.
18. Engel AJ. Utility of intercostal nerve conventional thermal radiofrequency ablations in the injured worker after blunt trauma. Pain Physician. 2012;15(5):E711–8.
19. Garcia Cosamalon PJ, Mostaza A, Fernandez J, Vinuela J, Mazabel M, Costilla S, et al. Dorsal percutaneous radiofrequency rhizotomy guided with CT scan in intercostal neuralgias. Technical note. Acta Neurochir (Wien). 1991;109(3–4):140–1.

20. Akkaya T, Ozkan D. Ultrasound-guided pulsed radiofrequency treatment of the intercostal nerve: three cases. J Anesth. 2013;27(6): 968–9.
21. Boelens OB, van Assen T, Houterman S, Scheltinga MR, Roumen RM. A double-blind, randomized, controlled trial on surgery for chronic abdominal pain due to anterior cutaneous nerve entrapment syndrome. Ann Surg. 2013;257(5):845–9.
22. Williams EH, Williams CG, Rosson GD, Heitmiller RF, Dellon AL. Neurectomy for treatment of intercostal neuralgia. Ann Thorac Surg. 2008;85(5):1766–70.
23. Samlaska S, Dews TE. Long-term epidural analgesia for pregnancy-induced intercostal neuralgia. Pain. 1995;62(2): 245–8.

Ilioinguinal and Iliohypogastric Nerve Entrapment: Abdominal

40

Neel Amin, Daniel Krashin, and Andrea M. Trescot

Introduction

Analogous to the intercostal nerves (Chap. 39) in both origin and function, the *ilioinguinal nerve* (IIN) and the *iliohypogastric nerve* (IHN) are sensory and motor nerves, arising from the thoracolumbar junction and traveling anteriorly to the rectus border and inguinal canal. The IHN supplies the skin over the abdomen and inguinal region, whereas the IIN supplies the skin of the inguinal canal and the superomedial aspect of the thigh, as well as the base of the penis and anterior scrotum (or mons pubis and labium majus). *Ilioinguinal nerve entrapment* (IINE) is a common cause of chronic abdominal, suprapubic, and pelvic pain, especially after lower abdominal surgery or hernia repair. Spontaneous IINE is considered very rare, and most cases are due to blunt trauma or previous abdominal or pelvic surgeries. Both nerves can be injected for the diagnosis and treatment of chronic abdominal, suprapubic, and inguinal pain. IHN and IIN injections can be utilized for the management and treatment of nerve entrapment syndromes as well as neuralgias; they are often treated together and thus are considered together in this chapter. Their role in pelvic pain is discussed in Chap. 44.

Clinical Presentation (Table 40.1)

Ilioinguinal and iliohypogastric nerve entrapments are easily mistaken for other pain syndromes involving the lower GI tract and genitourinary tract, as well as orthopedic conditions of the pelvis and hip (Fig. 40.1). The sensory distribution of the ilioinguinal and iliohypogastric nerves is wide, from the lower abdominal wall to the cutaneous innervation of the inguinal crease, the scrotum or labia majora, and superomedial thigh (Fig. 40.2). Therefore, depending on the location of the entrapment, the clinical picture may vary [7].

Entrapment of the IIN as it passes through the *external oblique* and *internal oblique* muscles and their fascia, at the level of, or medial to, the *anterior superior iliac spine* (ASIS), results in pain originating in the iliac fossa and radiating into the ipsilateral groin and anteromedial thigh. This is sometimes accompanied by sensory changes such as dysesthesia or hypesthesia in the nerve distribution, with a trigger point site where the nerve exits through the abdominal muscles.

Table 40.1 Occupation/exercise/trauma history relevant to ilioinguinal and iliohypogastric nerve entrapment

Surgery	Inguinal hernia repair [1]
	Pelvic open and laparoscopic surgeries [2]
	Trocar trauma from laparoscopic surgery [3]
	Appendectomy, hysterectomy [4]
Trauma	Blunt abdominal trauma [5]
Entrapment	At the rectus border (ACNE)
	At the iliac crest
	At the inguinal surgical site
Spontaneous	Variations in the musculoaponeurotic connective tissue [6]

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More commonly, the entrapment occurs postsurgically, particularly after *herniorrhaphy* (as much as 54 % of hernia patients) [8], but also after appendectomy, laparoscopic procedures in the abdomen, or surgeries requiring a Pfannenstiel incision (Table 40.1) [7]. This pain is sometimes noticed acutely during recovery from the procedure, but more often develops over weeks or months, due to scarring or fibrosis at the surgical area. In many cases of persistent pain after hernia surgery, the ilioinguinal nerve will be discovered during reoperation to be caught up in sutures, surgical staples, or mesh [7]. The pain in this form of entrapment is usually replicable by pressure at the entrapment site and is worse with activity, especially activity that involves the lower abdominal wall or underlying muscles, such as the psoas. This entrapment is also associated with altered sensation in the nerve's distribution. Patients may note pain with hip extension and therefore will try to keep the hip flexed [6]. They may also walk flexed at the hips to avoid tension on the abdominal muscles [9]. Patients will often experience increase in pain intensity and frequency with menstruation, salt intake, and sexual intercourse [10]. The main clue to the ilioinguinal nerve entrapment is stabbing pain while exercising, with improvement at rest. The diagnostic triad for ilioinguinal neuralgia is:

1. Symptoms elicited by movements and pressing at the anterior iliac spine or lateral rectus border
2. Hyperesthesia and/or a dysesthesia in the nerve distribution areas
3. Improvement of pain after a nerve injection [2]

The relief from diagnostic injections may only be temporary; however, for some patients, long-term or even permanent relief can result from a single injection [11].

Fig. 40.1 Patient description of ilioinguinal and iliohypogastric nerve entrapment (Image courtesy of Andrea Trescot, MD)

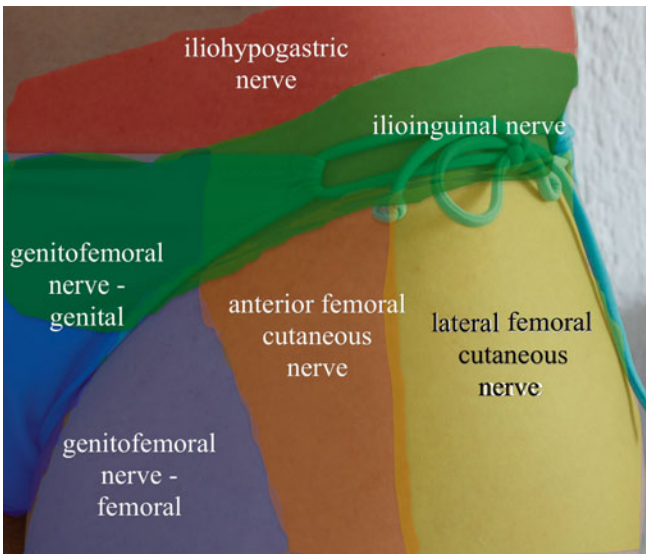

Fig. 40.2 Pain patterns from iliohypogastric, ilioinguinal, genitofemoral, lateral femoral cutaneous, and anterior femoral cutaneous nerves (Image courtesy of Andrea Trescot, MD)

Anatomy (Table 40.2)

Both the iliohypogastric and ilioinguinal nerves originate from the T12 to L2 spinal nerve roots (Table 40.2). The nerves emerge from the lateral border of the psoas major muscle, under the peritoneum, and travel around the abdominal wall, between the layers of the transversus abdominis muscle and the internal oblique muscle, emerging superficially at a point about 2–3 cm medial to the *anterior superior iliac spine* (ASIS) (Fig. 40.3). The motor portion of these nerves innervates the lowest portions of the *transversus abdominis* and *internal oblique muscles* and fascia and travels somewhat parallel to the *inguinal ligament* (Fig. 40.4). At the area of the inguinal ligament, the nerves travel between the internal and external oblique muscles. This is the area where they are most likely to be entrapped due to variations of musculoaponeurotic connections [1].

The size of the ilioinguinal nerve is inversely proportional to the iliohypogastric nerve. In some patients, the ilioinguinal nerve merges into the iliohypogastric nerve, or one of the nerves may be entirely absent. "The variations in the emergence and distribution of the ilioinguinal nerve are the cause of the failures of the ilioinguinal block and the difficulties in interpreting the ilioinguinal nerve syndrome" [1].

Klaassen et al. [12] dissected 100 cadavers (200 specimens) and found that the IIN entered the abdominal wall 2.8 ± 1.1 cm medial and 4 ± 1.2 cm inferior to the ASIS and terminated 3 ± 0.5 cm lateral to the midline. The IHN entered the abdominal wall 2.8 ± 1.3 cm medial and 1.4 ± 1.2 cm inferior to the ASIS and terminated 4 ± 1.3 cm lateral to the midline. For both nerves, the distance between the ASIS and the midline was 12.2 ± 1.1 cm. See Table 40.3 for the origin of the nerves. Note that the IHN percentages do not equal 100, because there were a significant number of specimens where no separate IHN could be identified.

In a study published by Ndiaye et al. [1], the ilioinguinal nerve was absent in seven cadavers and duplicated in one cadaver. The IIN emerged from the internal oblique muscle, passing nearby the inguinal ligament (1 ± 0.8 cm) and close to the ASIS (3.33 ± 2 cm).

Whiteside et al. [3] studied the course of the ilioinguinal and iliohypogastric nerve in 11 cadavers and then correlated the path of the nerves to the usual sites for laparoscopic trocar ports. On average, the IIN entered the abdominal wall 3.1 cm medial and 3.7 cm inferior to the ASIS and then followed a linear course to terminate 2.7 cm lateral to the midline and 1.7 cm superior to pubic symphysis. The IHN entered the abdominal wall on average 2.1 cm medial and 0.9 cm inferior to the ASIS and then followed a linear course to terminate 3.7 cm lateral to the midline and 5.2 cm superior to pubic symphysis.

Table 40.2 Ilioinguinal/iliohypogastric nerve anatomy

Origin	Anterior ramus T12, L1, and L2 nerve roots
General route	IHN – exits L1 (sometimes T12), travels with the IIN for a short distance, passes through the psoas, extends diagonally across the ventral surface of the quadratus lumborum to the iliac crest, and then continues between the transversus abdominis and internal oblique to ASIS and then almost horizontal to superficial inguinal ring. Lateral cutaneous branch (LCB) to trochanter
	IIN – exits L1 (sometimes L2) with IHN and then separates, passes through psoas, leaves internal oblique, past ASIS, and travels below aponeurosis of external oblique along spermatic cord/round ligament through superficial abdominal ring
Sensory distribution	IHN – groin and symphysis; LBN – skin of lateral pelvis, hip, and trochanter region
	IIN – ribbon-shaped area over inguinal region up to iliac crest, over symphysis, root of penis, proximal scrotum/labia, and small area of the anterior and medial thigh
Motor innervation	Transversus abdominis and external oblique muscles
Anatomic variability	There is an inverse size of the IIN and IHN; they may be joined or one may be absent
Other relevant structures	ASIS, inguinal canal, spermatic cord/round ligament

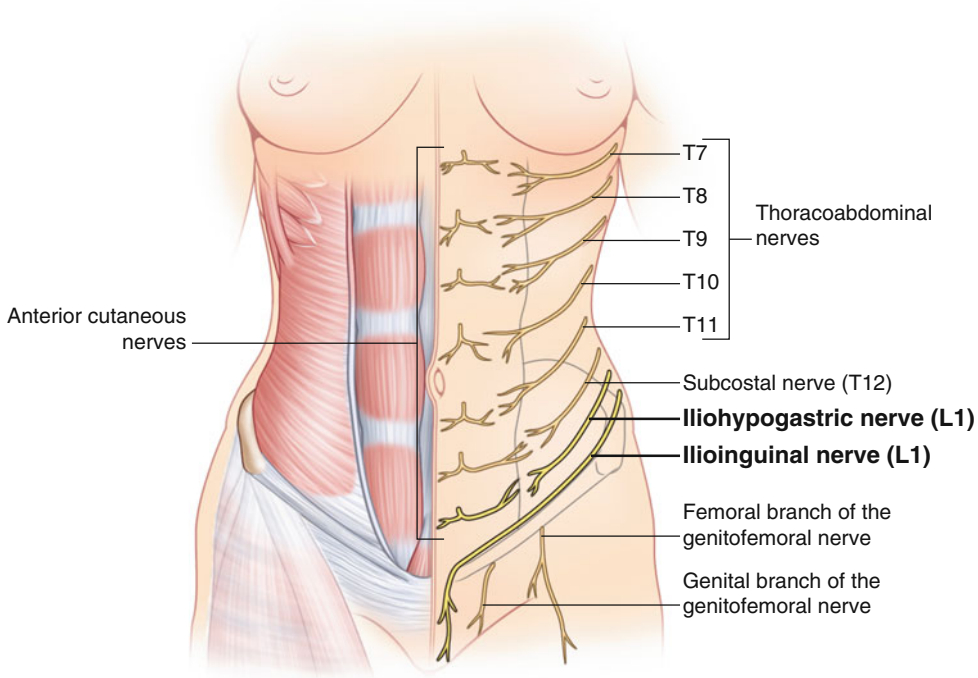

Fig. 40.3 Anatomy of the abdominal wall (Image by Springer)

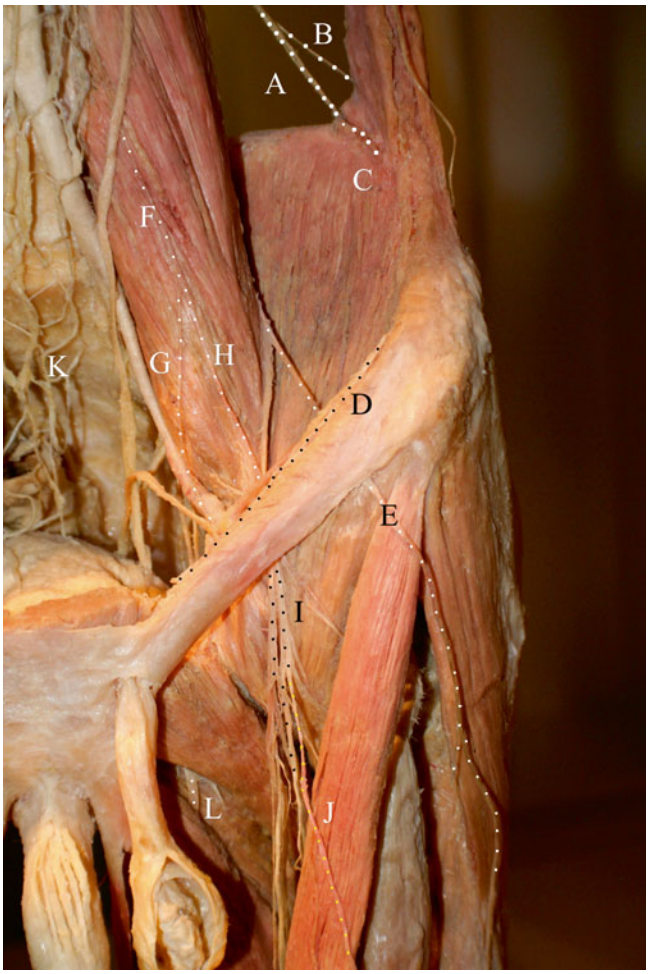

Fig. 40.4 Lumbar plexus nerves, modified from an image from *Bodies, The Exhibition*, with permission. *A* ilioinguinal nerve, *B* iliohypogastric nerve, *C* site of ilioinguinal nerve entrapment at the external oblique, *D* ilioinguinal nerve over the inguinal ligament, *E* lateral femoral cutaneous nerve, *F* genitofemoral nerve; *G* genital branch of the genitofemoral nerve, *H* femoral branch of the genitofemoral nerve, *I* femoral nerve; *J* saphenous nerve, *K* inferior hypogastric plexus, *L* obturator nerve (Image courtesy of Andrea Trescot, MD)

Table 40.3 Origin of the ilioinguinal and iliohypogastric nerve

Origin	Number of specimens (%)
Ilioinguinal nerve	
L1	130 (65 %)
T12 and L1	28 (14 %)
L1 and L2	22 (11 %)
L2 and L3	20 (10 %)
Iliohypogastric	
T12	14 (7 %)
T12 and L1	28 (14 %)
L1	20 (10 %)
T11 and T12	12 (6 %)

Chart created from data in Klaassen et al. [12]

Entrapment

The exact prevalence of IIN entrapment is unknown. According to Knockaert et al. [13], IIN entrapment is a little-known cause of abdominal pain. The IIN is commonly trapped at the rectus border, the iliac crest, and the paravertebral area. Entrapment at the rectus border is part of the *abdominal cutaneous nerve entrapment (ACNE) syndrome* discussed in Chap. 42. Most cases are likely undiagnosed or misdiagnosed, but some level of persistent pain occurs after up to 10 % of open and laparoscopic surgery, primarily hernia repairs and Pfannenstiel incisions [6].

There is less knowledge and published scientific literature about "spontaneous entrapment" of the IIN. The nerve is exposed to microtrauma as it passes through the transverse and internal oblique muscles [9]. The nerve can be entrapped as it traverses "in a step-like or zigzag" fashion, exposed to mechanical rubbing by the fibrous fibers under strain, when "the [muscle] steps are tightened against the nerve" [9]. Patients with Pfannenstiel incisions from previous surgery are at an increased risk for ilioinguinal nerve entrapment due to tissue scarring or fibrosis.

Physical Exam

The physical exam is an extension of the history, and the examination should concentrate on the relevant historical information. Most patients presenting with IINE have history of surgery, and the physical exam should concentrate on visualization to identify any likely etiology of mass or other anatomic structural cause of pressure in the area. For the physical exam of the rectus border entrapment, position the patient supine or standing, and check for point tenderness at the rectus border (Fig. 40.5). This occurs inferior to the

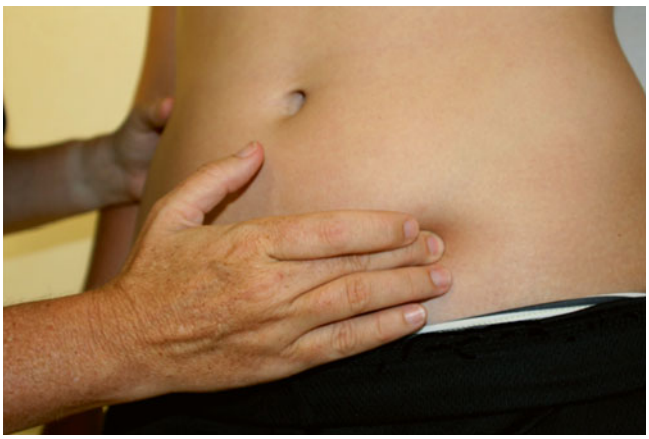

Fig. 40.5 Ilioinguinal nerve exam at the rectus border (Image courtesy of Andrea Trescot, MD)

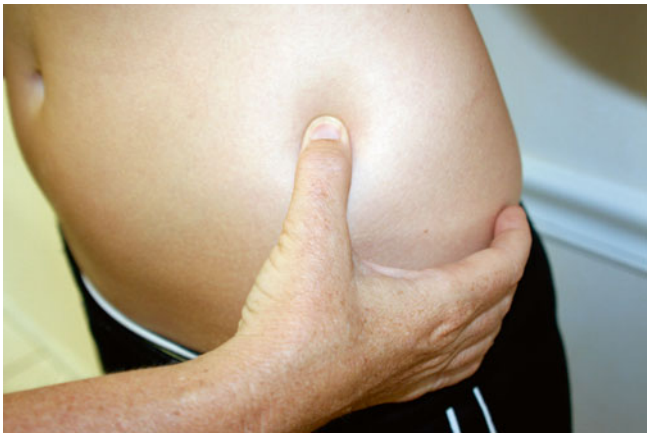

Fig. 40.6 Ilioinguinal nerve exam at the iliac crest (Image courtesy of Andrea Trescot, MD)

umbilicus, in a similar position as the abdominal cutaneous nerves (see Chap. 42). To check for iliac crest entrapment, stand in front of the patient and place hands on the iliac crest laterally (Fig. 40.6). Feel the edge of the external oblique with your thumbs (Video 40.1). Tenderness at the external oblique tendon attachment can indicate ilioinguinal nerve entrapment. For paravertebral entrapment, palpate the paravertebral L1 vertebrae. Tenderness here can indicate proximal ilioinguinal entrapment. This exam is helpful if the treatment needs to be more proximal.

Table 40.4 Differential diagnosis of abdominal wall pain

	Potential distinguishing features
Appendicitis, diverticulitis	History, abdominal CT, sonography, endoscopic tests or MRI, lab work
Endometriosis	History, abdominal CT, sonography or MRI, lab work, manual examination
Abdominal wall hernia, spigelian hernia	History, abdominal CT, sonography, lab work, palpation of the hernia or abdominal wall defect
Herpes zoster	History, skin rash, examination
Interstitial cystitis	History, cystography, sonography or MRI, lab work, manual examination
Irritable bowel disease	History, abdominal CT, sonography, endoscopic tests, lab work
Rectus sheath hematoma	History, abdominal CT, sonography and exam with evidence of hematoma
Tendonitis of abdominal muscle	History, abdominal CT, sonography, tenderness at muscle attachment on the pubic tubercle examination
Upper lumbar facet pathology	History, spine CT or MRI, examination with tenderness over upper lumbar facets, pain relief with facet injection
Lumbar radiculopathy	History, MRI or CT findings, relief with transforaminal or intercostal injection
Myofascial pain	History, examination with local tenderness

Differential Diagnosis (Table 40.4)

Consider the diagnosis of IINE in patients presenting for evaluation of chronic abdominal pain, especially after hernia surgery (Table 40.4). Although there are many causes of postsurgical pain, 81 % of patients presenting with chronic pain after inguinal hernia surgeries are found on reoperation to have entrapment of this nerve [14, 15]. When patients present with chronic intractable pain after hernia surgery, the IIN can be caught in sutures, surgical staples, or mesh [7]. Patients with IIN entrapment present with burning pain over the lower abdomen radiating to the inner portion of the upper thigh and scrotum or labia majora [16]. The IIN entrapment pain needs to be differentiated from the clinical findings of *genitofemoral nerve* (GFN) entrapment (see Chap. 41), with burning sensations in the inguinal region and radiation of pain to the skin of the genitalia and upper medial thigh. Standing, walking, and other movements necessitating hip extension will worsen the pain as well. Any increase in intra-abdominal pressure with increasing Valsalva, coughing, or sneezing will also worsen the clinical condition.

Despite motor innervation of transversus abdominis and internal oblique, there is no motor exam that is confirmatory of the diagnosis. However, there may be a subtle bulge in the abdominal wall with nerve paresis (Fig. 40.7). Knockaert et al. [17] detected electromyographic abnormalities in only 15 of 25 (60 %) patients with a probable diagnosis of ilioinguinal-iliohypogastric nerve entrapment syndrome, with definite diagnosis in only 6 of 16 (37 %) of those. The low sensitivity makes this not a useful test.

Both IIN and GFN entrapments (see Chap. 41) can be clinically worsened by walking and improved by rest [16]. Distinguishing between IIN and GFN neuropathy is difficult because of the overlap of the sensory innervation territories of these nerves. Therefore, local and specific diagnostic blocks need to be performed to determine the nerves involved as accurately as possible. Think about the diagnostic triad mentioned above under the *Clinical Presentation*: the worsening of symptoms by movement, exacerbation of pain with pressure at the ASIS (site of the IIN crossing the iliac crest), and improvement of pain after the specific nerve block (Table 40.5) [7].

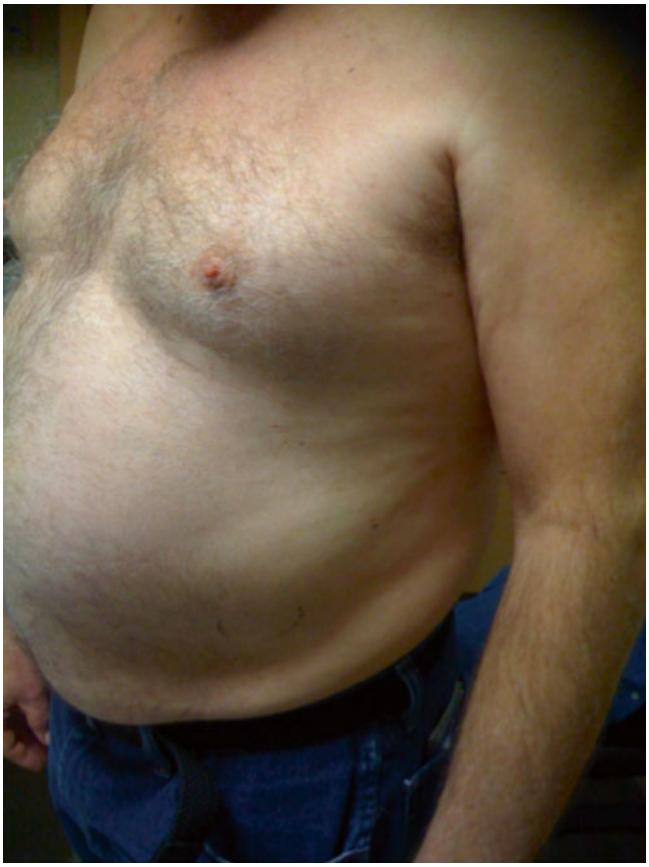

Fig. 40.7 Abdominal bulge due to proximal ilioinguinal entrapment (Image courtesy of Andrea Trescot, MD)

Table 40.5 Diagnostic tests for ilioinguinal and iliohypogastric nerve entrapment

	Potential distinguishing features
Physical exam	Tenderness at the rectus border or the ASIS
Diagnostic injection	At rectus border or the ASIS
Ultrasound	Used for injection but not diagnosis
MRI	Not useful, except to address differential diagnosis
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies	Not useful

Identification and Treatment of Contributing Factors

IINE is often seen as a postsurgical complication after hernia surgery. Although there are many causes of postsurgical pain, 81 % of patients presenting with chronic pain after inguinal hernia surgeries are found on reoperation to have entrapment of ilioinguinal nerve [14, 15]. Perimenstrual fluid retention can lead to monthly abdominal pain. Pfannenstiel incisions can lead to IINE at the lateral corner of the incision. Inguinal hernia repairs, especially with mesh, can lead to early (immediately postoperatively) and late (sometimes years after the surgery) entrapment.

Desensitization therapy is reported to be helpful in some cases. Unfortunately, this nerve entrapment is often triggered by abdominal wall exercises, which would be expected to help abdominal wall pain. Giving diuretics (hydrochlorothiazide or furosemide) 5 days before scheduled menses can sometimes prevent the perimenstrual edema that may trigger entrapment (personal communication, Andrea Trescot, MD).

Injection Technique

Temporary relief should be achieved in patients with IINE outside the pelvis. Complete long-term relief is reported in approximately 60 % of patients after a series of one to three injections [18, 19].

Landmark-Guided Technique

For entrapment that occurs at the rectus border, palpate the lateral border of the rectus with the non-injecting hand, straddling the nerve (Fig. 40.8). After a sterile skin prep, advance a 25-gauge 2-in. needle to the lateral rectus border (Video 40.2). Inject 2 cc maximum volume of local anesthetic and deposteroid. Using a peripheral nerve stimulator to identify the nerve may help with specificity.

For a more proximal injection, locate the ASIS by palpation. Next, draw a line 2 cm medial and 2 cm *superior* to the ASIS (Fig. 40.9 – site A). After a sterile skin prep, infiltrate the skin with local anesthetic. Insert a blunt-tip needle perpendicular to the skin at the skin puncture site. As the needle moves through the external oblique muscle, there will be resistance appreciated. When the needle transverses the external oblique to pass between it and the internal oblique muscle, there will be a "pop" and a loss of resistance. Continue inserting the needle further through the internal oblique to appreciate another "pop" and loss of resistance, indicating that the needle is in between the internal oblique and the transversus abdominis muscles. After negative aspiration for blood, inject 2 cc of a local anesthetic and steroid to block the nerve.

In another technique, the anterior superior iliac spine (ASIS) is palpated in the supine position; the injection point is 2 in. medial and 2 in. *inferior* to that point (Fig. 40.9 – site B).

For entrapment at the iliac crest, position the patient in either the supine or lateral decubitus position. Advance a 25-gauge 2-in. needle to the tendon attachment of the external oblique onto the iliac crest (Fig. 40.10). Inject a 2 cc maximum volume of local anesthetic with or without

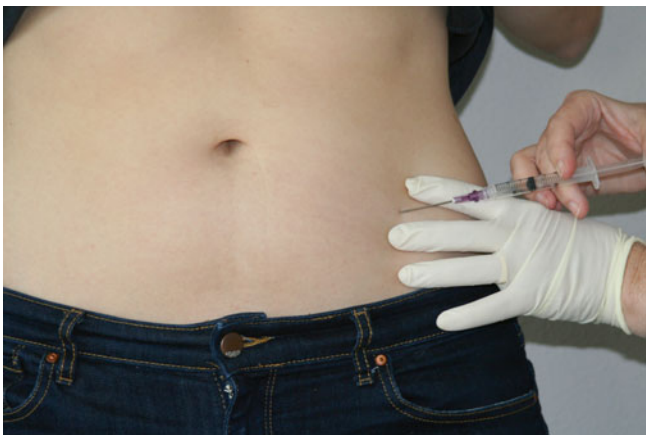

Fig. 40.8 Landmark-guided ilioinguinal nerve injection at the rectus border (Image courtesy of Andrea Trescot, MD)

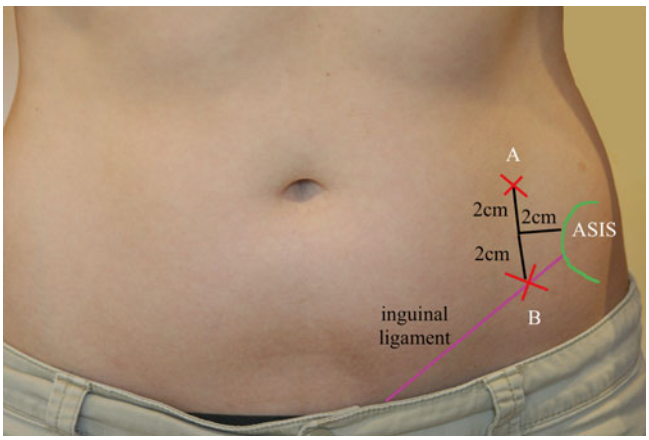

Fig. 40.9 Site for landmark-guided ilioinguinal/iliohypogastric injection at ASIS. *A* site 2 cm superior, *B* site 2 cm inferior (Image courtesy of Andrea Trescot, MD)

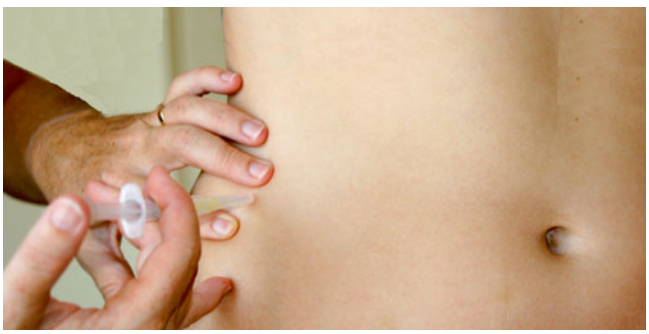

Fig. 40.10 Landmark-guided injection of the ilioinguinal/iliohypogastric nerve at the iliac crest (Image courtesy of Andrea Trescot, MD)

deposteroid. This injection is useful in patients with multiple prior groin surgeries (because the injection site is proximal to the surgical pathology); a peripheral nerve stimulator is often used for this type of injection. If the patient's physique makes body landmarks difficult to identify, consider using fluoroscopy for the procedure to identify the iliac crest.

Fluoroscopic-Guided Technique

There are no specific bony landmarks for the ilioinguinal or iliohypogastric nerve (other than the fluoroscopic location of the iliac crest in obese patients), but the proximal approach at T12, L1, or L2 is done under fluoroscopic control. The procedure is done in a manner very similar to a transforaminal epidural or selective nerve root block. A peripheral nerve stimulator is used to confirm the level of pathology, since the origin of this nerve is sometimes variable. Stimulation should allow the patient to identify the "that's it" site. For paravertebral injections, position the patient prone under fluoroscopy, and identify the foramen (Fig. 40.11). Use a selective nerve root technique (extra-foraminal), rather than the transforaminal technique, with a peripheral nerve stimulator to identify the ilioinguinal nerve. If you notice twitching in the patient's lower back, this usually represents stimulation of the *superior cluneal nerve* (see Chap. 50); reposition the needle slightly more anterior to obtain ilioinguinal and iliohypogastric stimulation. Inject a 1 cc maximum volume of local anesthetic and non-particulate steroid.

Ultrasound-Guided Technique

Ultrasound techniques with visualization of the IIN have been described [18], and the nerve is most easily visualized at the ASIS. The linear probe is initially placed perpendicular to the ASIS (Fig. 40.12a) and then slightly rotated from a transverse to an oblique plane to be perpendicular to the anatomical course of both the IIN and the IHN, with the lateral/caudal part of the transducers brought into contact with the iliac crest (Fig. 40.12b). At this time, the fascial layers between the external oblique, the internal oblique, and the transversus abdominis should be visible as bright hypoechoic white lines; the IHN and IIN will be hypoechoic oval structures between the internal oblique and the transversus abdominis muscles (Fig. 40.13).

Bischoff et al. [20] described a randomized, double-blind, placebo-controlled crossover trial with 12 patients with severe persistent inguinal pain after herniorrhaphy and 12 normal controls. Each subject underwent a lidocaine injection under ultrasound guidance followed by mapping of the subsequent hypoesthesia. This study found only 1 responder to lidocaine injection out of 12 patients; however, the cutoff for being a responder was an 80 % reduction in pain ratings, rather than the more usual 50 % reduction. The authors concluded that ultrasound-guided lidocaine blocks of the ilioinguinal and iliohypogastric

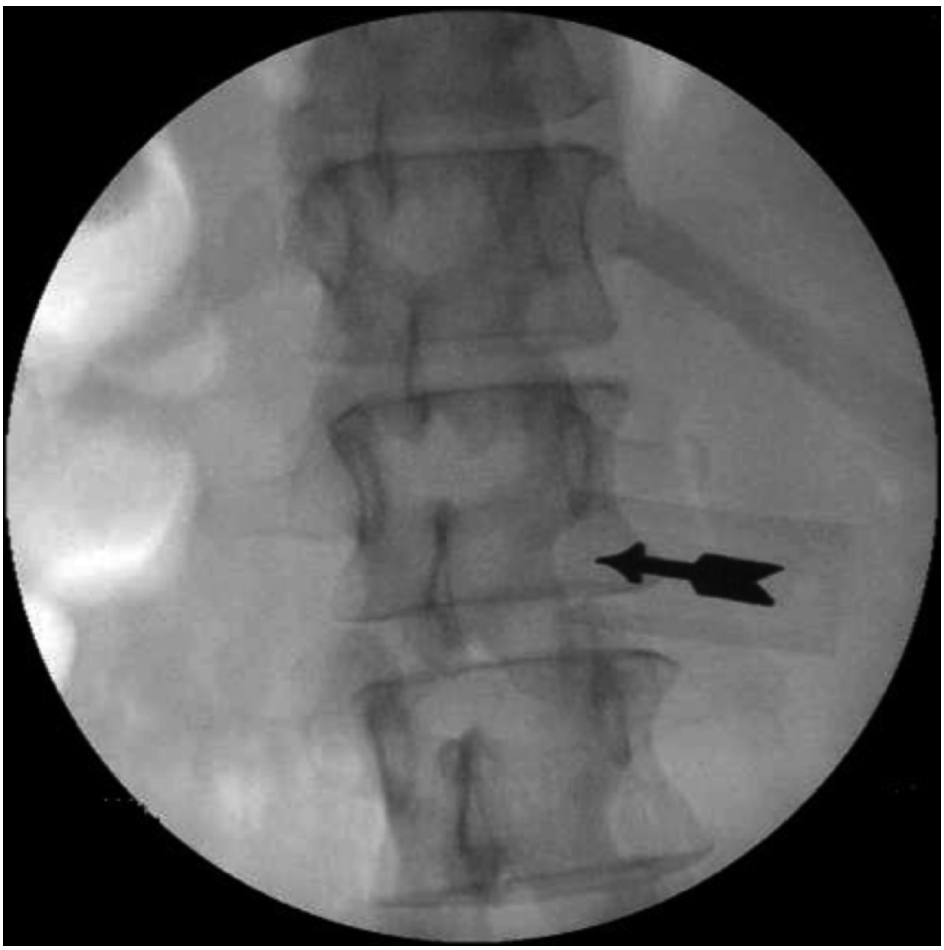

Fig. 40.11 Location of the L1 vertebral foramen (Image courtesy of Andrea Trescot, MD)

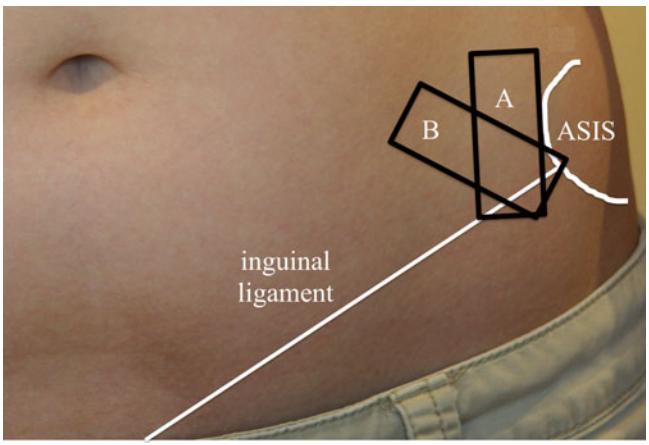

Fig. 40.12 Location of the ultrasound probe. Probe is initially placed perpendicular to the anterior superior iliac spine (site *A*) and then rotated parallel to the ilioinguinal and iliohypogastric nerve (site *B*) (Image courtesy of Andrea Trescot, MD)

nerves, at the level of the anterior superior iliac spine, were not useful in diagnosis and management of persistent inguinal post-herniorrhaphy pain. The authors offered several possible explanations for these findings: first, the complex sensory innervation of the groin, consisting of branches of the ilioinguinal, iliohypogastric, and genitofemoral nerves, which share origins from the T12-L2 spinal nerves. Communicating branches between these nerves and anatomic variations are common, meaning that interruption of one nerve may not abolish sensory transmission from a specific area [21]. Second, in some patients, sensory branches to the painful area may leave the main nerves proximal to the ASIS, so that even a properly performed nerve injection in that area will have no effect on the pain. Third, a more prominent role of the GFN in persistent post-herniorrhaphy pain has recently been suggested [16]. Ultrasound-guided techniques for a selective block of the GFN proximal to the groin area are not currently available, though there are techniques described in Chaps. 41 and 45 for more distal genitofemoral injections. Fourth, a peripheral nerve block lasting only a short duration may not be adequate to affect persistent postsurgical pain, which is thought to be maintained by central sensitization [22].

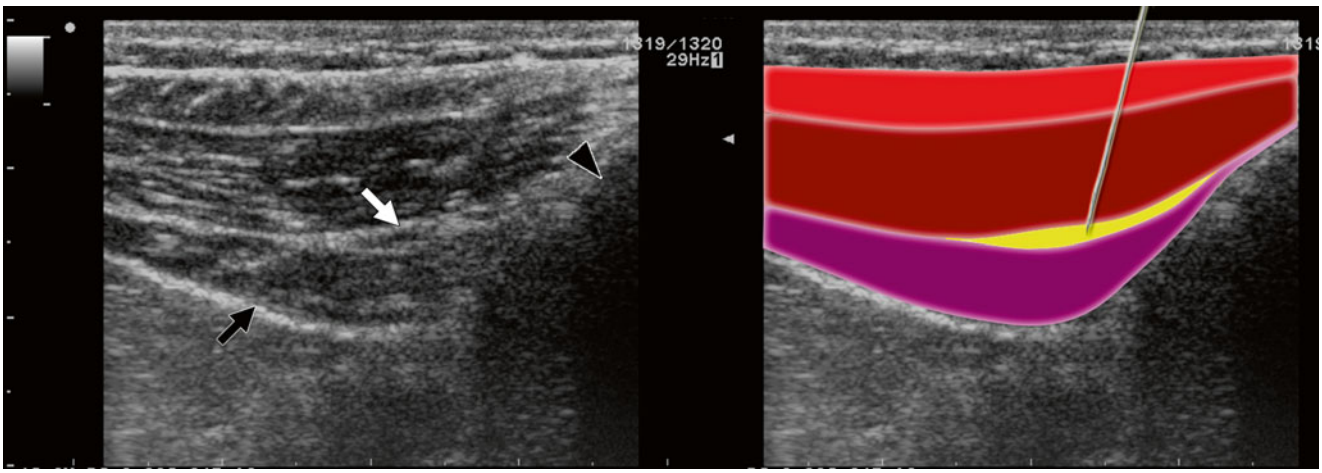

Fig. 40.13 Ultrasound image of the ilioinguinal and iliohypogastric nerves (*white arrow*) and simulation of the ultrasound placement of a needle onto the ilioinguinal nerve. *Orange* external oblique muscle, *red* internal oblique muscle, *yellow* location of ilioinguinal and iliohypogastric nerves with local anesthetic, and *pink* transversus abdominis muscle. *Black arrow* represents peritoneum; *black arrowhead* represents anterior superior iliac spine (From Kastler et al. [26]. Reprinted with permission from American Society of Interventional Pain Physicians)

Ultrasound-Directed Hydrodissection

Adler et al. [23] described the use of a 14-gauge angiocatheter placed from a lateral approach and an 18-gauge Tuohy needle from a medial approach at the ASIS, advanced to within 0.25 cm of the IHN under US guidance (Fig. 40.14). They used normal saline to distend the perineural space and then followed with cryoneuroablation through the angiocatheter (see section "Cryoneuroablation") or surgical resection with the catheter as the intraoperative guide (see section "Surgical Technique").

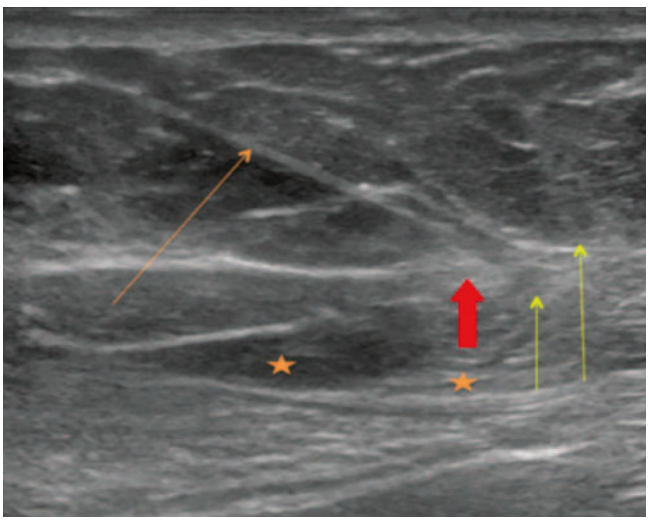

Fig. 40.14 Ultrasound-guided 14-gauge angiocatheter insertion (*orange arrow*) for hydrodissection (*orange star*) of the iliohypogastric nerve (*red arrow*) with catheter insertion (*yellow arrow*) (Image courtesy of Adam Adler, MD, with permission)

Neurolytic Technique

Cryoneuroablation

For persistent ilioinguinal and iliohypogastric pain, cryoneuroablation is an appropriate further therapy. After superficial infiltration with local anesthetic and deep infiltration with saline containing epinephrine 1:200,000, a 12-gauge intravenous catheter is used as the introducer for the 2.0 mm cryoprobe, usually attempting to place the probe parallel to the nerve from lateral to medial (Fig. 40.15). Location may be optimized with the use of US guidance, as well as the peripheral nerve stimulator.

Fanelli et al. [24] described ten patients with ilioinguinal, genitofemoral, or combined neuralgia after inguinal hernia repair treated with cryoneuroablation. After cryotherapy, patients reported overall pain reduction of 0–100 % (mean, 77.5 %; median, 100 %); 80 % reported decreased analgesic use, and 90 % reported increased physical capacity. Two patients underwent additional cryotherapy, one for incomplete relief and one for recurrent pain, both with 100 % efficacy. Wound infection ($n = 1$) was the only complication.

Neurolytic Injections

Phenol and alcohol have been used to treat IIN or IHN entrapment [25], but there is the possibility of tissue necrosis around the nerve as well as a significant risk of neuritis.

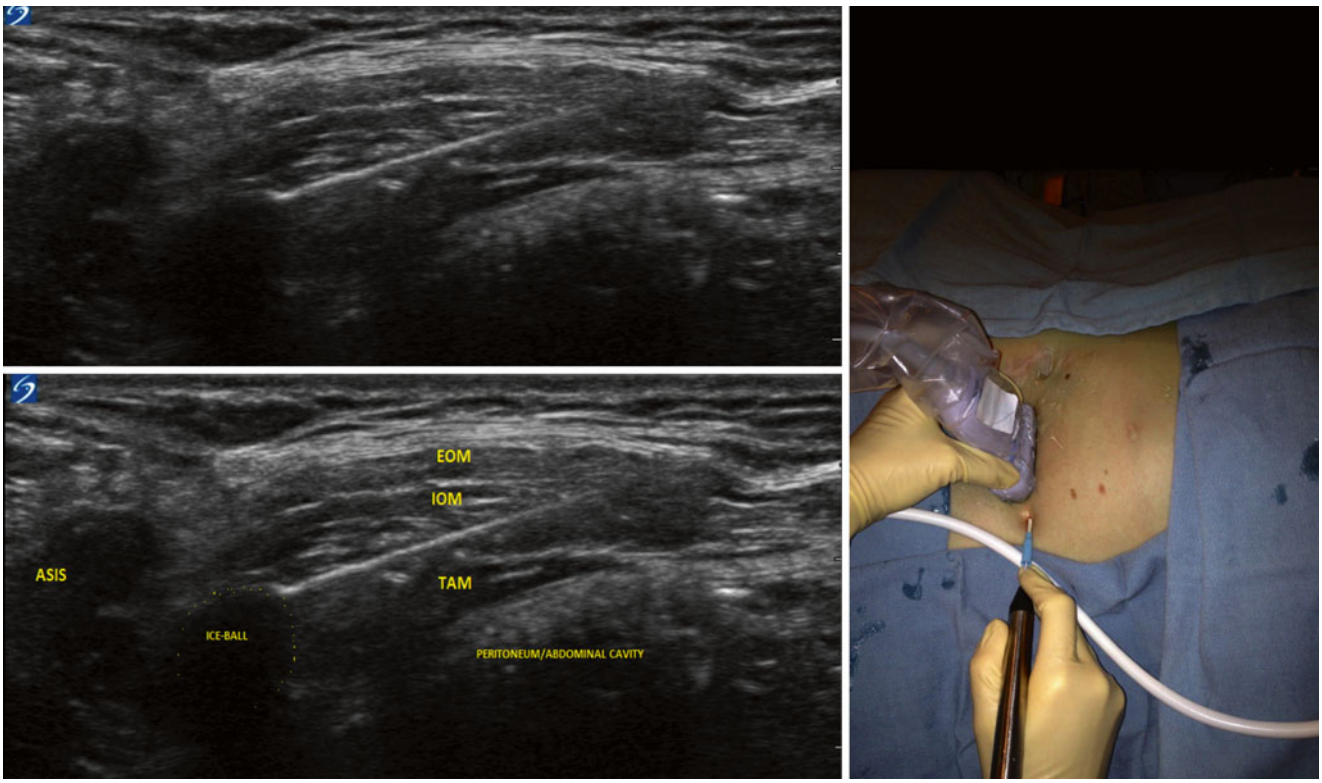

Fig. 40.15 Cryoneuroablation of the ilioinguinal nerve under ultrasound guidance. *ASIS* anterior superior iliac spine, *EOM* external oblique muscle, *IOM* internal oblique muscle, *TAM* transversus abdominis muscle (Image courtesy of Agnes Stogicza, MD)

Radiofrequency Lesioning

Standard, neuro-destructive radiofrequency ablation has been evaluated. Kastler et al. [26] compared local anesthetic blockade to radiofrequency ablation (RFA). A 22-gauge 5 mm active-tip RFA needle was placed at the ASIS, with computerized tomography (CT) guidance (Fig. 40.16). There was no mention of threshold, but stimulation was used to finalize needle position. Three ablations were performed, each for 90 s, at 70 °C, 80 °C, and 90 °C. Results showed an average of 12.5 months relief in the RFA arm, compared to 1.6 months in the local anesthetic/steroid arm.

Several groups have used pulsed radiofrequency to treat persistent IIN pain [19]. Long-term relief using pulsed radiofrequency of the ilioinguinal nerve is a promising alternative to conventional injections.

Neurostimulation

Peripheral nerve stimulation has been used for chronic groin pain [27, 28]. The trial electrodes are placed percutaneously through introducers, and, if there is significant temporary relief, the leads can be placed permanently (Fig. 40.17). Rauchwerger et al. [29] described three cases of severe ilioinguinal neuralgia (after vaginal hysterectomy or inguinal hernia repair) treated using peripheral nerve stimulation, with the leads placed over the surgical hernia scar (in the inguinal hernia repairs), or field stimulation with the lead placed subcutaneously near the IIN at the site of greatest pain (in the vaginal hysterectomy patient).

Surgical Technique

Entrapment neuralgia arising from the ilioinguinal nerve can be treated by the surgical release of the entrapped nerve. Kim et al. [4] were able to retrospectively review charts on 33 patients; 23 had ilioinguinal neuralgia, while 10 had combined ilioinguinal and iliohypogastric neuralgia. All had a positive preoperative local anesthetic injection; 30 of the 33 had "considerable" pain relief after surgical release. Zacest and colleagues [30] noted complete or partial pain relief in 13 of 19 (66.7 %) patients undergoing ilioinguinal nerve release, with a mean follow-up of 35 months. Hahn [7], in a crossover prospective study, randomized 19 women to medication management or surgical release of the ilioinguinal nerve. The surgery patients noted good relief. Nine of the ten women initially randomized to the medication arm discontinued the medications due to side effects; they were then offered surgery and also noted good relief.

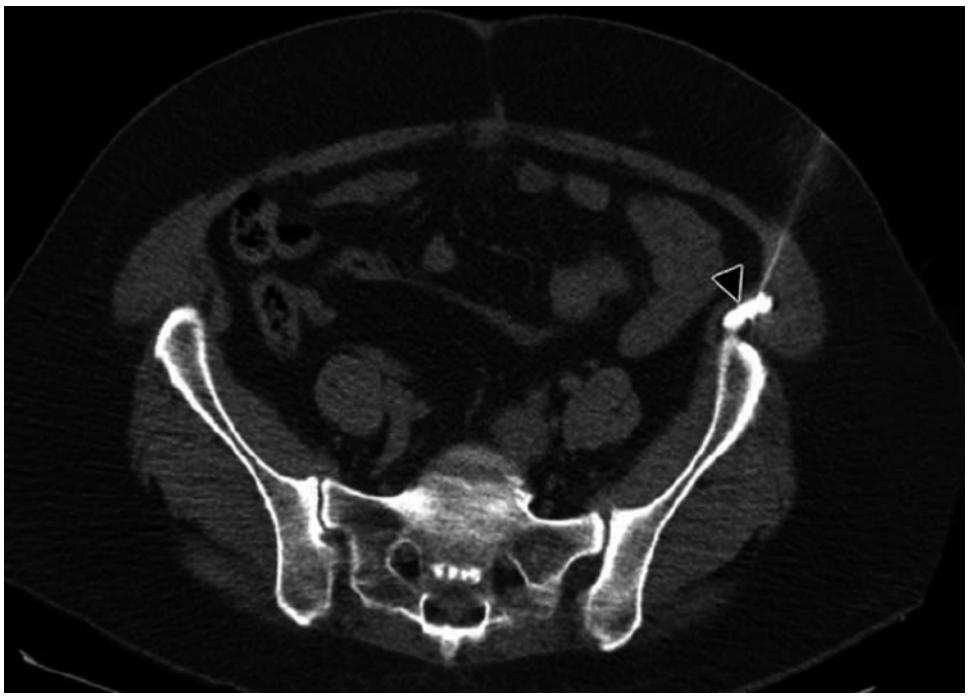

Fig. 40.16 CT-guided radiofrequency lesioning at the anterior superior iliac spine, between the transversus abdominis and internal oblique muscles (From Kastler et al. [26]. Reprinted with permission from Pain Physician)

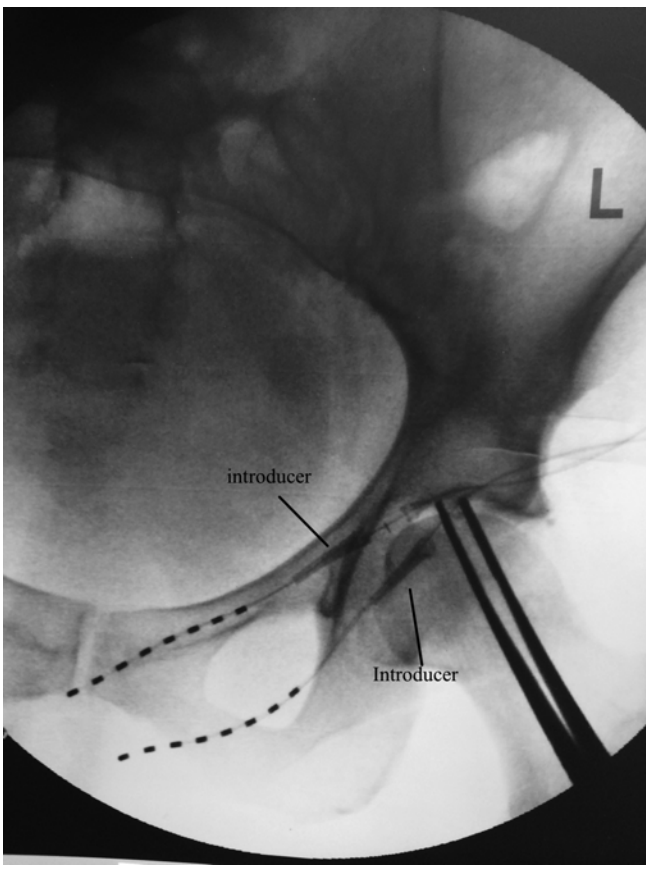

Fig. 40.17 Peripheral nerve stimulation of the abdominal wall (Image courtesy of Gladstone MacDowell, MD)

Excision of the scar neuroma can give relief, but the neuroma is often difficult to localize. One technique that has been successful (Trescot, personal correspondence) is mixing local anesthetic with methylene blue. If the local anesthetic abolishes the pain, then the neuroma is within the dyed tissue, and the surgeon is instructed to "take out everything blue."

Complications

As with all types of injections, aseptic preparation and good technique will limit potential risks from the injection. Because proper performance of ilioinguinal/iliohypogastric blocks requires small volume injections, the possibility of local anesthetic toxicity is remote. As the injection is limited to the lower abdominal wall and inguinal region, hemodynamic changes would be unusual. As with other injections, the patient is advised to protect the anesthetized area from trauma.

Proper performance of ilioinguinal/iliohypogastric blocks places the injection medial and superior to the anterior superior iliac spine. Some texts advocate performing the block from a point medial and inferior to the anterior superior iliac spine, which essentially places the injection within or inferior to the inguinal ligament. This may result in a lateral femoral cutaneous or femoral block with little or no ilioinguinal/iliohypogastric anesthesia. However, even properly performed ilioinguinal/iliohypogastric blocks can result in

transient femoral anesthesia, with a reported incidence of 3.7–5 % [31], due to tracking of local anesthetic proximally along the iliac fascia. Perforations of the bowels [32] and pelvic hematomas [33] have been reported after ilioinguinal/iliohypogastric blocks; this illustrates the importance of using blunt needles to appreciate the loss of resistance as the needle traverses the layers of the abdominal wall. Damage to the L1 nerve root can leave an abdominal wall weakness (Fig. 40.5).

Summary

IIN and IHN entrapments are significant but often unrecognized causes of groin, abdominal, and pelvic pain. The marked variability of the anatomy and the clinical presentation makes it important to keep IIN and IHN in mind as a pain etiology.

References

1. Ndiaye A, Diop M, Ndoye JM, Konate I, Ndiaye AI, Mane L, et al. Anatomical basis of neuropathies and damage to the ilioinguinal nerve during repairs of groin hernias. (about 100 dissections). Surg Radiol Anat. 2007;29(8):675–81.
2. Stulz P, Pfeiffer KM. Peripheral nerve injuries resulting from common surgical procedures in the lower portion of the abdomen. Arch Surg. 1982;117(3):324–7.
3. Whiteside JL, Barber MD, Walters MD, Falcone T. Anatomy of ilioinguinal and iliohypogastric nerves in relation to trocar placement and low transverse incisions. Am J Obstet Gynecol. 2003;189(6):1574–8.
4. Kim DH, Murovic JA, Tiel RL, Kline DG. Surgical management of 33 ilioinguinal and iliohypogastric neuralgias at Louisiana State University Health Sciences Center. Neurosurgery. 2005;56(5):1013–20; discussion – 20.
5. Starling JR, Harms BA, Schroeder ME, Eichman PL. Diagnosis and treatment of genitofemoral and ilioinguinal entrapment neuralgia. Surgery. 1987;102(4):581–6.
6. Toussaint CP, Perry 3rd EC, Pisansky MT, Anderson DE. What's new in the diagnosis and treatment of peripheral nerve entrapment neuropathies. Neurol Clin. 2010;28(4):979–1004.
7. Hahn L. Treatment of ilioinguinal nerve entrapment – a randomized controlled trial. Acta Obstet Gynecol Scand. 2011;90(9):955–60.
8. Eichenberger U, Greher M, Kapral S, Marhofer P, Wiest R, Remonda L, et al. Sonographic visualization and ultrasound-guided block of the third occipital nerve: prospective for a new method to diagnose C2-C3 zygapophysial joint pain. Anesthesiology. 2006;104(2):303–8.
9. Kopell HP, Thompson WA, Postel AH. Entrapment neuropathy of the ilioinguinal nerve. N Engl J Med. 1962;266(1):16–9.
10. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
11. Racz GB, Hagstrom D. Iliohypogastric and ilioinguinal nerve entrapment: diagnosis and treatment. Pain Dig. 1992;2:43–8.
12. Klaassen Z, Marshall E, Tubbs RS, Louis RGJ, Wartmann CT, Loukas M. Anatomy of the ilioinguinal and iliohypogastric nerves with observations of their spinal nerve contributions. Clin Anat. 2011;24:454–61.
13. Knockaert DC, D'Heygere FG, Bobbaers HJ. Ilioinguinal nerve entrapment: a little-known cause of iliac fossa pain. Postgrad Med J. 1989;65(767):632–5.
14. Vuilleumier H, Hübner M, Demartines N. Neuropathy after herniorrhaphy: indication for surgical treatment and outcome. World J Surg. 2009;33(4):841–5.
15. Starling JR, Harms BA. Diagnosis and treatment of genitofemoral and ilioinguinal neuralgia. World J Surg. 1989;13(5):586–91.
16. Giger U, Wente M, Büchler M, Krähenbühl S, Lerut J, Krähenbühl L. Endoscopic retroperitoneal neurectomy for chronic pain after groin surgery. Br J Surg. 2009;96(9):1076–81.
17. Knockaert DC, Boonen AL, Bruyninckx FL, Bobbaers HJ. Electromyographic findings in ilioinguinal-iliohypogastric nerve entrapment syndrome. Acta Clin Belg. 1996;51(3):156–60.
18. Suresh S, Patel A, Porfyris S, RYEE MY. Ultrasound–guided serial ilioinguinal nerve blocks for management of chronic groin pain secondary to ilioinguinal neuralgia in adolescents. Pediatr Anesth. 2008;18(8):775–8.
19. Mitra R, Zeighami A, Mackey S. Pulsed radiofrequency for the treatment of chronic ilioinguinal neuropathy. Hernia. 2007;11(4):369–71.
20. Bischoff JM, Koscielniak-Nielsen ZJ, Kehlet H, Werner MU. Ultrasound-guided ilioinguinal/iliohypogastric nerve blocks for persistent inguinal postherniorrhaphy pain: a randomized, double-blind, placebo-controlled, crossover trial. Anesth Analg. 2012;114(6):1323–9.
21. Rab M, Ebmer J, Dellon LA. Anatomic variability of the ilioinguinal and genitofemoral nerve: implications for the treatment of groin pain. Plast Reconstr Surg. 2001;108(6):1618–23.
22. Kehlet H, Jensen TS, Woolf CJ. Persistent postsurgical pain: risk factors and prevention. Lancet. 2006;367(9522):1618–25.
23. Adler AC, Smith DI, Parikh PM. Chronic pain localized to the iliohypogastric nerve: treatment using an ultrasound-guided technique of hydrodissection for catheter placement as a guide for surgical iliohypogastric nerve resection. Plast Reconstr Surg. 2014;134(1):182e–3.
24. Fanelli RD, DiSiena MR, Lui FY, Gersin KS. Cryoanalgesic ablation for the treatment of chronic postherniorrhaphy neuropathic pain. Surg Endosc. 2003;17(2):196–200.
25. Hakeem A, Shanmugam V. Current trends in the diagnosis and management of post-herniorrhaphy chronic groin pain. World J Gastrointest Surg. 2011;3(6):73–81.
26. Kastler A, Aubry S, Piccand V, Hadjidekov G, Tiberghien F, Kastler B. Radiofrequency neurolysis versus local nerve infiltration in 42 patients with refractory chronic inguinal neuralgia. Pain Physician. 2012;15(3):237–44.
27. Rosendal F, Moir L, de Pennington N, Green AL, Aziz TZ. Successful treatment of testicular pain with peripheral nerve stimulation of the cutaneous branch of the ilioinguinal and genital branch of the genitofemoral nerves. Neuromodulation. 2013;16(2):121–4.
28. Banh DP, Moujan PM, Haque Q, Han TH. Permanent implantation of peripheral nerve stimulator for combat injury-related ilioinguinal neuralgia. Pain Physician. 2013;16(6):E789–91.
29. Rauchwerger JJ, Giordano J, Rozen D, Kent JL, Greenspan J, Closson CW. On the therapeutic viability of peripheral nerve stimulation for ilioinguinal neuralgia: putative mechanisms and possible utility. Pain Pract. 2008;8(2):138–43.
30. Zacest AC, Magill ST, Anderson VC, Burchiel KJ. Long-term outcome following ilioinguinal neurectomy for chronic pain. J Neurosurg. 2010;112(4):784–9.
31. Rosario DJ, Jacob S, Luntley J, Skinner PP, Raftery AT. Mechanism of femoral nerve palsy complicating percutaneous ilioinguinal field block. Br J Anaesth. 1997;78(3):314–6.
32. Johr M, Sossai R. Colonic puncture during ilioinguinal nerve block in a child. Anesth Analg. 1999;88(5):1051–2.
33. Vaisman J. Pelvic hematoma after an ilioinguinal nerve block for orchialgia. Anesth Analg. 2001;92:1048–9.

Genitofemoral Nerve Entrapment: Abdominal

41

Thais Khouri Vanetti, Alexandra Tavares Raffaini Luba, Fabrício Dias Assis, and Charles Amaral de Oliveira

Introduction

The *genitofemoral nerve* (GFN) is a branch of lumbar plexus and may become entrapped in the abdominal region, causing pain in the distribution of this nerve, such as the lower abdomen/pelvic region, groin, scrotum or labia majora, and anterior proximal thigh area. The pelvic presentation is discussed in Chap. 45, and the proximal thigh presentation is discussed in Chap. 60. In this chapter, we will focus on the abdominal aspect of GFN entrapment.

It is very important to remember that the GFN and *ilioinguinal* and *iliohypogastric* nerves originate from similar levels of spinal nerve roots; therefore, it is often difficult to distinguish which of the three nerves is causing the pain. Diagnostic injections are a critical tool for differentiation.

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Clinical Presentation (Table 41.1)

Magee first reported the syndrome of genitofemoral neuralgia in 1942, but it remains a rarely encountered (or diagnosed) clinical entity [6]. The most common clinical presentation of GFN entrapment consists of intermittent or constant pain, burning dysesthesia, and sensory changes in the inguinal region. Paresthesias and persistent pain in the lower abdomen (Fig. 41.1), groin (Fig. 41.2), scrotum or labia majora, and anterior proximal thigh area may indicate GFN neuropathy. Pain may be misdiagnosed as interstitial cystitis, endometriosis, and inguinal or femoral hernias. Pain is aggravated with thigh extension because of psoas compression of the nerve; activities such as walking, running, and hyperextension of the thigh will exacerbate the pain, while lying down and thigh flexion will help to relieve the pain [7].

Although the precise incidence of GFN entrapment is unknown, the incidence of groin pain after inguinal hernia

Table 41.1 Occupation/exercise/trauma history relevant to genitofemoral nerve entrapment

Compression	Psoas spasm
	Psoas abscess
	Pelvic hematoma
	Lymphoma
Trauma	Pubic ramus fracture
	Pubic symphysis irritation (pubic synovitis)
	Needle trauma during lumbar sympathetic injections [1]
Surgery	Inguinal hernia repair (especially with mesh) [2]
	Laparoscopic inguinal hernia repair [3]
	C-section/appendectomy [2]
	Lumbar fusion
	Retroperitoneal surgery
Neuritis	Alcohol/phenol/RF to the lumbar sympathetic chain [4, 5] or celiac plexus
	Thermal damage from renal radio-frequency lesioning (renal cell CA) [5]

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repair has been reported as high as 20 % [8]. In contrast to ilioinguinal neuralgia, Tinel's signal cannot be induced (obtained) [9].

There is significant overlap between the pain patterns of the ilioinguinal nerve (Chap. 40), iliohypogastric nerve (Chap. 40), lateral femoral cutaneous nerve (Chap. 61), and GFN (Fig. 41.3).

Anatomy (Table 41.2)

The GFN arises from the upper *lumbar plexus* (see Chap. 43), emerges at L1 and L2 (primarily L2), and consists mainly of sensory fibers, with a motor component for the *cremaster muscle* (*cremasteric reflex*). The nerve penetrates obliquely the two bodies of the *psoas muscle* anteriorly at the L3 and L4 level (as part of the lumbar plexus – see Chap. 49), travels behind the ureter, and descends in the retroperitoneal space with the iliac vessels to the medial border of the *psoas muscle* (Fig. 41.4), where it divides into a genital and femoral branch, just above the *inguinal ligament* (Fig. 41.5). The *genital branch* (also known as the *external spermatic nerve*) [11] travels down the psoas

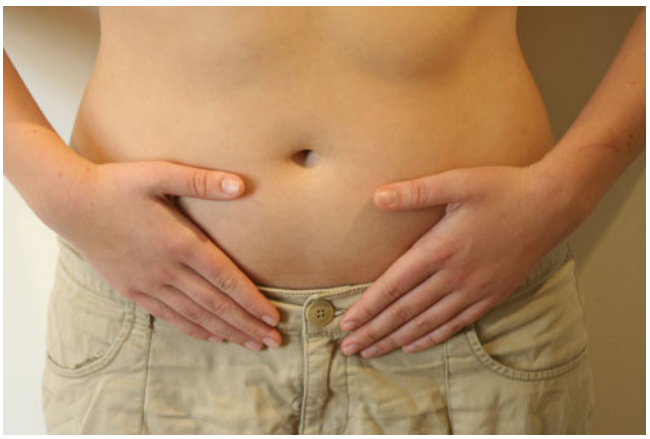

Fig. 41.1 Lower abdominal pattern of genitofemoral pain (Image courtesy of Andrea Trescot, MD)

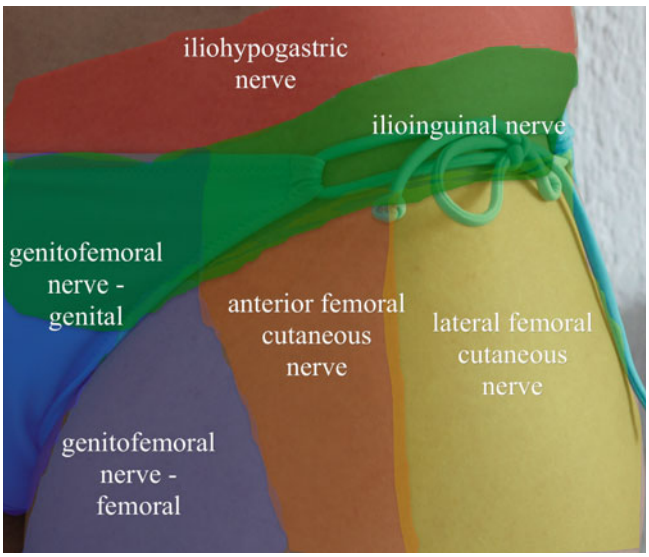

Fig. 41.3 Pattern of pain from the iliohypogastric, ilioinguinal, lateral femoral cutaneous, and genitofemoral nerves (Image courtesy of Andrea Trescot, MD)

Fig. 41.2 Groin pattern of genitofemoral pain (Image courtesy of Andrea Trescot, MD)

muscle to the inguinal ligament, crossing the transversus abdominus and internal oblique muscles. In males, the genital branch enters the *internal inguinal ring* (*deep inguinal ring*) together with the *spermatic cord* and the *ilioinguinal nerve*, supplying motor fibers to the cremaster muscle and sensation to the *scrotum*. In females, it accompanies the round ligament, innervating the *mons pubis* and the *labia majora*. The *femoral branch* is located caudally and laterally to the genital branch and travels alongside the *external iliac artery*, under the inguinal ligament, passing through the *fascia lata* to the *femoral sheath* (lateral to the femoral artery). There, it innervates the skin of the anterior superior part of the thigh (the *femoral triangle*, which is bounded superiorly by the inguinal ligament, laterally by the *sartorius muscle*, and medially by the *adductor longus muscle*) (Fig. 41.6) [12, 13].

Table 41.2 Genitofemoral nerve anatomy

Origin	L1 and L2
General route	Perforates the psoas at L3 and L4, descends along the medial psoas border, and divides into genital and femoral branches just above the inguinal ligament
	Genital branch
	Males: inside internal inguinal ring with spermatic cord to scrotum
	Females: accompanies the round ligament to the mons pubis and labia majora
	Femoral branch
	Located caudally and laterally to the genital branch, traveling caudally with external iliac artery, behind inguinal ligament through fascia lata to femoral sheath
Sensory distribution	Anterior medial thigh, scrotum, mons pubis, and labia majora
Motor innervation	Cremasteric muscle
Anatomic variability	Location where genital and femoral branches split (occasionally within the psoas). Location of spermatic cord relative to genital branch. Communication between ilioinguinal and GFN
	200 cadavers [10]: single trunk in 80 % and 2 trunks in 20 %
	Single trunk came from L1 and L2 or L2 and L3
	Two trunks came from L1 and L2 or from L1, L2, and L3
Other relevant structures	Pubic tubercle, psoas muscle, inguinal canal, femoral canal

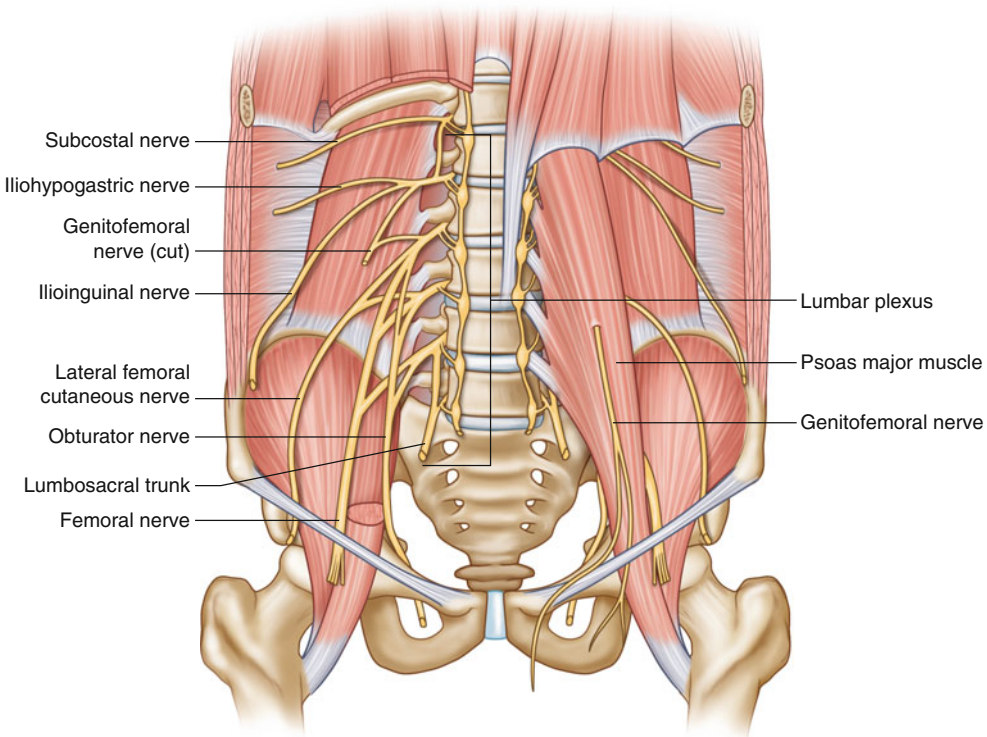

Fig. 41.4 Lumbar plexus (Image by Springer)

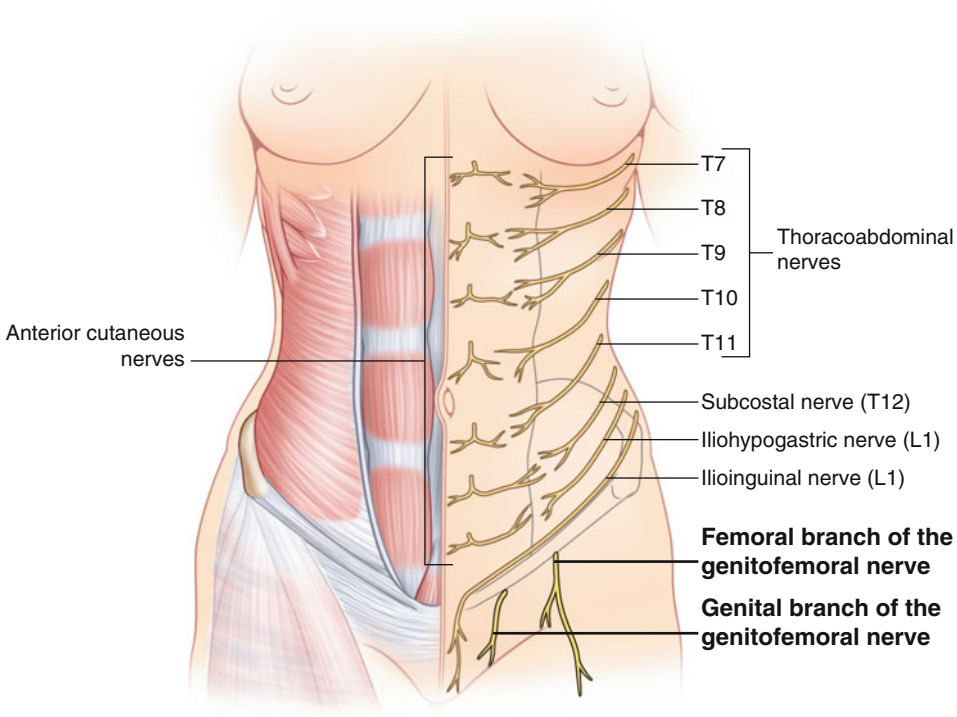

Fig. 41.5 Genital and femoral branches of the genitofemoral nerve (Image by Springer)

Though the course of this nerve and its branches is similar in men and women, anatomical studies suggest great variability among individuals, with the minority of nerves following the course traditionally described [13]. Moreover, the location where genital and femoral branches split before reaching the inguinal ligament is quite variable. Also, the relation between the genital branch and the spermatic cord varies considerably; it can travel outside the spermatic cord dorsally, ventrally, or inferiorly [9].

Both the GFN and the *lateral femoral cutaneous nerve* (LFCN) (see Chap. 61) are at risk for injury during lumbar sympathetic injections and neurolysis. Feigl and colleagues [4] dissected the sympathetic trunk of 118 cadavers and evaluated the distance from the *lumbar sympathetic trunk* (LST) to the GFN (in 20 cases, they could only measure from the femoral branch). In the dissection of 186 sides, the GFN passed the LST at a distance of 0–28 mm (mean distance 8.5) at the level of L3/L4, 0–13 mm at L4/L5 in 55 cases, and 9–19 mm at L2/L3 in 19 cases. The authors suggested that, due to the close proximity of the two structures (especially at L3/L4 and L4/L5), needle trauma or neurolytic damage could easily affect the GFN during lumbar sympathetic treatment. Interestingly, the authors found that the GFN was fused with the LFCN in three cases. Similarly, the GFN can also be injured during radio-frequency lesioning of renal cell carcinomas at the level of the psoas muscle [5].

Special attention to the great variation of the nerves in the groin region (ilioinguinal, iliohypogastric, and genitofemoral) is warranted, including to the free communication between these branches. There is a great deal of variability and overlap between these branches. According to a study of 64 groins (32 cadavers) [14], there were four different types of patterns of the ilioinguinal and GFN cutaneous innervation:

- In type A (43.7 %), the GFN provided the innervation of the scrotal/labial and the ventromedial thigh region, with no contribution from the ilioinguinal nerve.
- In type B (28.1 %), there was a dominance of the ilioinguinal nerve; the GFN shared a branch with the ilioinguinal nerve and gave motor fibers to cremaster muscle in the inguinal canal, but had no sensory branch to the groin.
- In type C (20.3 %), with a dominance of the genitofemoral nerve, the ilioinguinal nerve had sensory branches to the mons pubis and inguinal crease as well as the anterioproximal part of the root of the penis or labia majora. The GFN was found to share a branch with the iliohypogastric nerve.
- In type D (7.9 %), cutaneous branches emerged from both the ilioinguinal nerve and the GFN.

Entrapment

The GFN passes anteriorly through the psoas major muscle and may therefore become entrapped on its path or by reflex spasm of the psoas muscle [15]. Although entrapment symptoms of this primarily sensory lumbosacral nerve have not

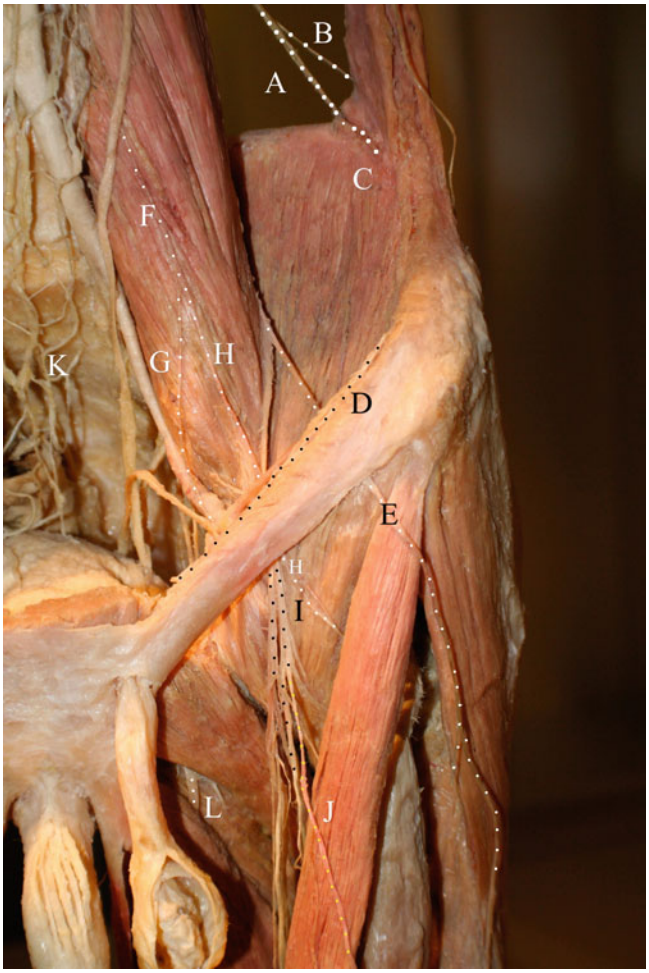

Fig. 41.6 Lumbar plexus nerves, modified from an image from *Bodies, The Exhibition*, with permission. *A* ilioinguinal nerve, *B* iliohypogastric nerve, *C* site of ilioinguinal nerve entrapment at the external oblique, *D* ilioinguinal nerve over the inguinal ligament, *E* lateral femoral cutaneous nerve, *F* genitofemoral nerve, *G* genital branch of the genitofemoral nerve, *H* femoral branch of the genitofemoral nerve, *I* femoral nerve, *J* saphenous nerve, *K* inferior hypogastric plexus, *L* obturator nerve (Image courtesy of Andrea Trescot, MD)

been specifically related to psoas major trigger points, this possibility should be considered when the patient suffers from pain and sensory alterations along the distribution of this nerve [15]. The GFN can be entrapped in relation to the psoas muscle, in the presence of hematoma, abscess, or trigger points. Retroperitoneal hematoma, lymphoma, aortic aneurism repair, and failed lumbar surgery can also cause entrapment of this nerve [15]. Operations such as appendectomy, nephrectomy, vasectomy, urethral sling [16], and laparoscopic procedures may contribute to injury of the GFN and therefore pain arising along the distribution of the GFN [2, 17]. Other causes include blunt trauma and appendectomy [11].

Inguinal hernia repair, especially laparoscopic repairs with mesh, is particularly associated with GFN entrapment at the *pubic tubercle*. This is because the mesh has to be

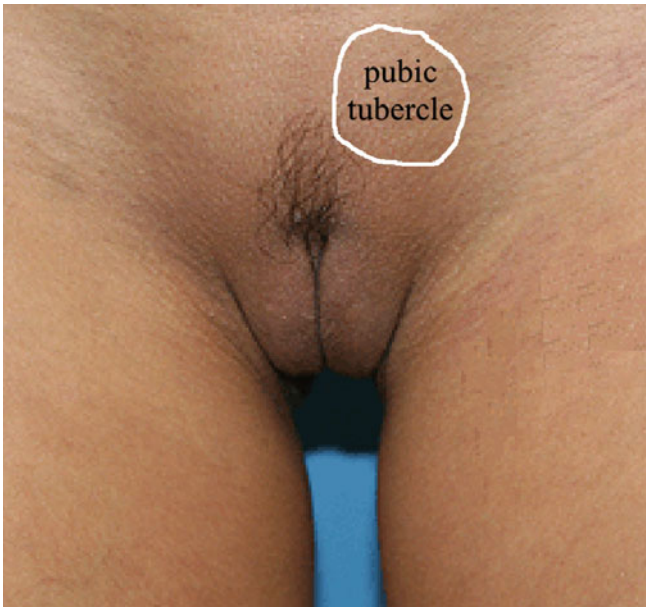

Fig. 41.7 Location of tenderness at the pubic tubercle (Image courtesy of Andrea Trescot, MD)

secured on *Poupart's ligament*, at the pubic attachment of the inguinal ligament onto the *symphysis pubis*, which is the site of the GFN transit across the pubic tubercle [11]. The GFN is also traumatized by *Pfannenstiel incisions* (at the pubic level), as well as lumbar fusions and retroperitoneal surgery (at the psoas level).

Murovic et al. [18] reviewed ten cases of GFN neuralgia treated at their institution; two cases were after vasectomies, three were after gynecologic surgery, four cases suffered blunt trauma and one case was after herniorrhaphy.

Physical Examination

A thorough neurologic exam may find sensory changes, which can be subtle, in the lower abdomen/pelvic area, groin area, anterior proximal thigh, and scrotum or labia majora. Sensory changes range from hypoesthesia to total loss of sensation, paresthesias, or even allodynia. Pain may also worsen on performing a Valsalva maneuver, on coughing, on rising, on side-bending to the contralateral side [3], on abducting the ipsilateral thigh [3], and on peristaltic movements. Female patients may observe worsening of pain during menstruation and sexual intercourse [16, 19]. In advanced neuropathy, loss of *cremasteric reflex* can occur. Maneuvers to activate or tighten the psoas muscle may also aggravate the pain.

It is difficult to palpate the anterior aspect of the psoas muscle where the GFN runs. However, the most common site of tenderness to palpation is on the pubic tubercle, which can be confirmed by fluoroscopy (Fig. 41.7).

Differential Diagnosis (Table 41.3)

Lumbar plexopathies and lumbar spinal diseases may manifest in similar presentations. There is considerable overlapping of neuropathies related to GFN, ilioinguinal nerve, or iliohypogastric nerve, because of the overlapping in nerve distribution and/or communication among those nerves. As such, it may be quite difficult to differentiate between the three nerves [9]. In this situation, it is important to perform specific diagnostic injections to exclude pain originating from these other two nerves (see *Injections* below) [23]. Murovic et al. [18] pointed out the importance of a Tinel's sign; since the GFN is not associated with a positive Tinel's sign, eliciting a paresthesia when tapping over a herniorrhaphy or Pfannenstiel scar should suggest an ilioinguinal or iliohypogastric nerve entrapment, rather than GFN. Table 41.4 lists the diagnostic tests for the GFN.

Table 41.3 Differential diagnosis of groin pain

	Potential distinguishing features
Adductor or rectus abdominis tendonitis/myofascial spasm	Tenderness at pubis; palpable spasm of the muscle
Avascular necrosis femoral head	X-ray and MRI show femoral head collapse
Pubic symphysitis/osteitis [20]	Bone scan positive
L1–L3 radiculopathy [21]	Sensory loss, weakness, EMG positive
Distal psoas tendonitis [21]	Tenderness on the lesser trochanter
Abdominal wall hernia [20]	Abdominal wall defect
Hip joint pathology [22]	Stiffness, limited range of motion, crepitus, clicking
Ilioinguinal nerve injury/ entrapment [20]	Increased abdominal wall tension can result in groin pain; may be tender near ASIS; increased pain with hip hyperextension
Adductor strain [22]	Tenderness over adductors. Usual site is at the muscle-tendon intersection; sometimes at tendon-bone

Table 41.4 Diagnostic tests for genitofemoral nerve entrapment

	Potential distinguishing features
Physical exam	Tenderness at the pubic tubercle, negative Tinel's sign
Diagnostic injection	At the pubic tubercle, the psoas muscle, or the L1 or L2 foramen
Ultrasound	Not useful for diagnosis
MRI	Not useful for diagnosis
Arteriography	Not useful for diagnosis
X-ray	Not useful for diagnosis, unless surgical clips are seen at the area of tenderness
Electrodiagnostic studies	Not useful for diagnosis, but may help to rule out other causes of groin pain

Identification and Treatment of Contributing Factors

Strenuous exercise and overstretching of the psoas muscle will trigger spasm of the psoas, entrapping the GFN. Past abdominal or pelvic surgeries, scars, mesh placement, neuroma, and hematoma formation are associated GFN entrapment. Pregnancies, with pressure on the GFN or the psoas or at the pelvic rim from the gravid uterus, can result in paresthesias or hypoesthesias that usually resolve with delivery.

Laparoscopic hernia repairs with mesh use sutures or clips to secure the mesh to the abdominal wall from the *anterior superior iliac spine* (ASIS) to the symphysis pubis, placed preperitoneally without dissection or visualization of the surrounding nerves, potentially jeopardizing all the nerves of the lumbar plexus [3]. Stretching of the genital branch of the GFN occurs because of traction on the spermatic cord during open hernia repairs, and ligation of the cremasteric artery can also damage the genital branch [18]. Other causes of GFN damage during hernia repair include inadvertent electrocoagulation, stricture by ligature, entrapment of the nerve in scar tissue, or inflammation [18]. The femoral branch of the GFN can be entrapped by fibrotic tissue in the femoral canal; laparoscopic hernia repair has lead to an increased incidence of both femoral branch and lateral femoral branch cutaneous nerve injury [18].

Because the GFN can be traumatized by surgery, Bischoff et al. [24] studied whether meticulous identification of the ilioinguinal, iliohypogastric, and genitofemoral nerves during surgery could decrease the incidence of postoperative nerve pain. They were able to identify the GFN in only 21 % of 244 open surgical hernia repair patients (compared to the ilioinguinal nerve in 94 % of the patients and the iliohypogastric nerve in 97 % of the patients); unfortunately, this meticulous identification did not decrease the incidence of chronic postoperative pain. However, Krause [25] was able to identify the femoral branch of the GFN in 19 of 20 patients during laparoscopic hernia repair, which will hopefully lead to a decrease in injuries to this branch.

Injection Technique

Landmark-Guided Technique

Because the pubic tubercle is superficial in all but the most obese patients, the landmark-guided injection of the GFN at the pubic tubercle can be useful, especially as a screening tool. Tenderness to palpation at the tubercle identifies the injection site (Fig. 41.7). Use of a peripheral nerve stimulator (PNS) can aid in locating the nerve.

Trescot described a landmark-guided ("blind") technique to inject the genital branch of the GFN [26]. The patient is placed in the supine position with a pillow under the knees if extending the lower limbs evokes pain. After a sterile prep, the pubic tubercle is palpated, and 1 cc of local anesthetic and deposteroid is injected via a 25-gauge needle, just superior and lateral to the tubercle. Broadman described a similar technique using 10 cc of solution just lateral to the pubic tubercle [27]. Use of a PNS can facilitate the accuracy of the injection. When performing the landmark-guided technique, utmost care must be taken in relation to important spermatic cord structures (testicular artery) and peritoneal cavity transgression.

Fluoroscopy-Guided Technique

With the patient in the supine position, the area of maximal tenderness (just lateral to the pubic tubercle) is identified by fluoroscopy (Fig. 41.8). After a skin prep and local anesthetic infiltration subcutaneously, a 22-gauge Quincke needle is advanced to the periosteum. Use of a PNS will facilitate identification of the nerve (Fig. 41.9). Once the nerve is identified, 1 cc of local anesthetic and deposteroid is injected. Rho et al. [3] described a similar injection technique under fluoroscopy to inject the "tack" used at the pubic tubercle to secure mesh during a laparoscopic hernia repair.

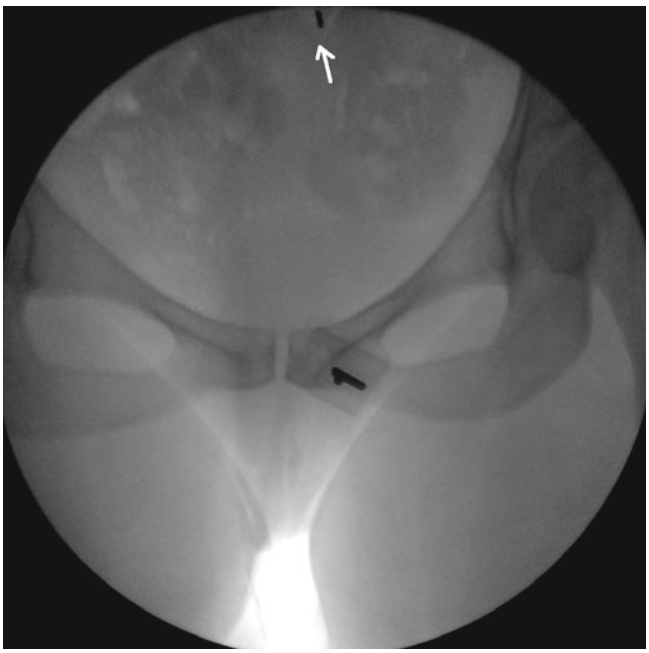

Fig. 41.8 Marker showing tenderness over the pubic tubercle, which is consistent with genitofemoral nerve pain. Note the *white arrow* showing Interstim® placed for "interstitial cystitis" pain that offered no relief (Image courtesy of Andrea Trescot, MD)

Another technique for GFN diagnosis and treatment is the dorsal root ganglion (DRG) local anesthetic block at T12, L1, and L2, ipsilateral to the pain (Fig. 41.10). T12 DRG should be included, as it is common for the ilioinguinal and genitofemoral nerves to communicate. If the block results in

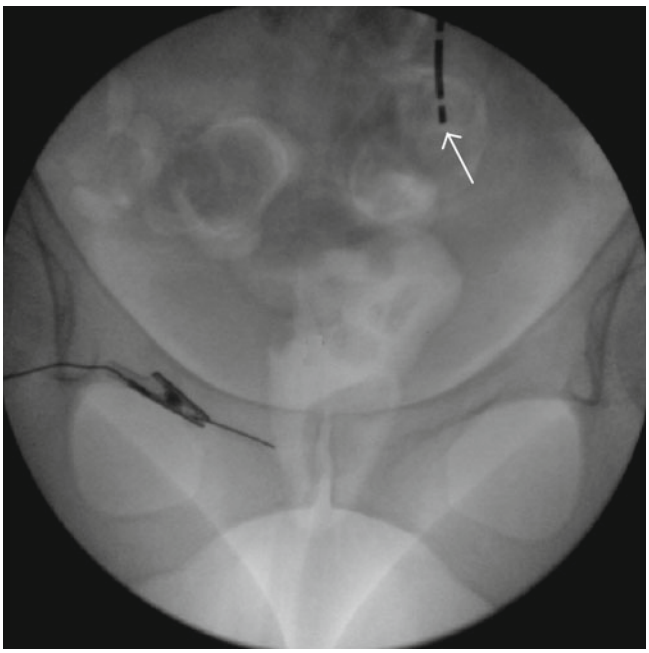

Fig. 41.9 PNS identification of the genitofemoral nerve. Note the *white arrow* showing Interstim® placed for interstitial cystitis pain that offered no relief (Image courtesy of Andrea Trescot, MD)

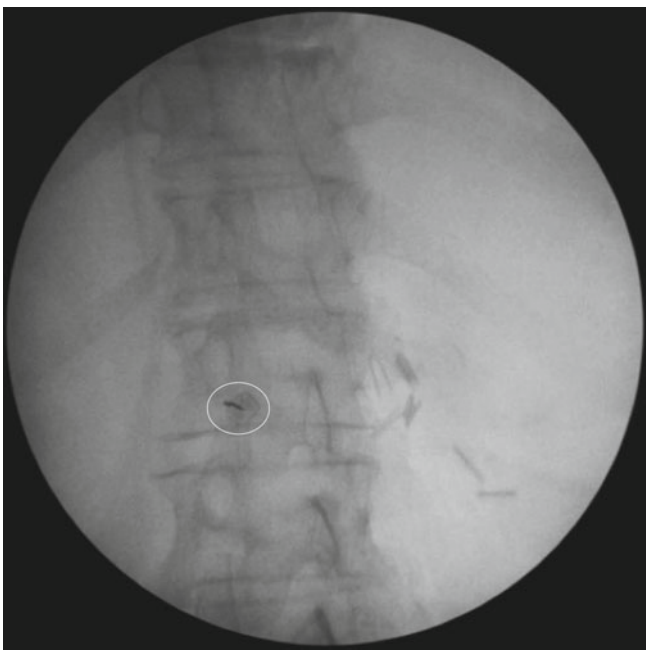

Fig. 41.10 Injection of the dorsal root ganglia at L1 (Image courtesy of Fabrício Assis, MD)

significant pain relief (at least 50 % improvement) but is short-lived, cryoneuroablation or pulsed radio frequency may be applied posteriorly to these ganglia (see *Neurolytics* below) [9].

Ultrasound-Guided Injection

Another approach is to perform a selective block of the genital branch of the GFN under ultrasound guidance. The genital branch is slender and cannot be directly visualized, but the external iliac artery is easily found at the inguinal canal, and the genital branch is immediately medial to it, within the internal inguinal ring [28, 29]. A high-frequency linear probe is oriented perpendicular to the inguinal ligament, just above the femoral artery and vein, about one to three fingerbreadths lateral to the pubic tubercle. It is suggested that the practitioner start in the internal inguinal ring, in which it is possible to visualize the longitudinal (lengthwise) section of the artery. When the probe is advanced cephalad, the *iliac artery* can be identified as it splits into the *femoral artery* and *external iliac artery*; the femoral artery is seen to penetrate deep inside the inguinal ligament. At this point, an oval or round structure (the *inguinal canal*, which contains the medial *cremaster/spermatic cord* and lateral *testicular vessels* in males and the *round ligament* in females), medial and superficial to the femoral artery, can easily be visualized (Fig. 41.11). The probe is then advanced in the medial direction, slowly, and moving away from the femoral artery. The needle may be inserted out-of-plane or in-plane, aiming the needle toward the spermatic cord [11] (Fig. 41.12); care must be taken to avoid vasoconstrictors in the local anesthetic, to avoid the adverse effects of testicular artery vasoconstriction. Because of anatomical variability, it is recommendable to use 5 cc inside and 5 cc outside the spermatic cord [2], though use of a peripheral nerve stimulator should decrease the need for large volumes of injectate.

CT-Guided Injection

The genitofemoral nerve travels in the retroperitoneal space before entering the inguinal canal. This position increases the risk of transgressing the peritoneum, if a posteriorly placed needle is advanced anteriorly. Because of this, a computer tomography (CT)-guided trans-psoas technique was developed, capable of selectively blocking the genitofemoral nerve [13], while avoiding injury to the ureters and intestines (Fig. 41.13). This technique can be used for diagnostic and therapeutic purposes. Since the genitofemoral nerve is

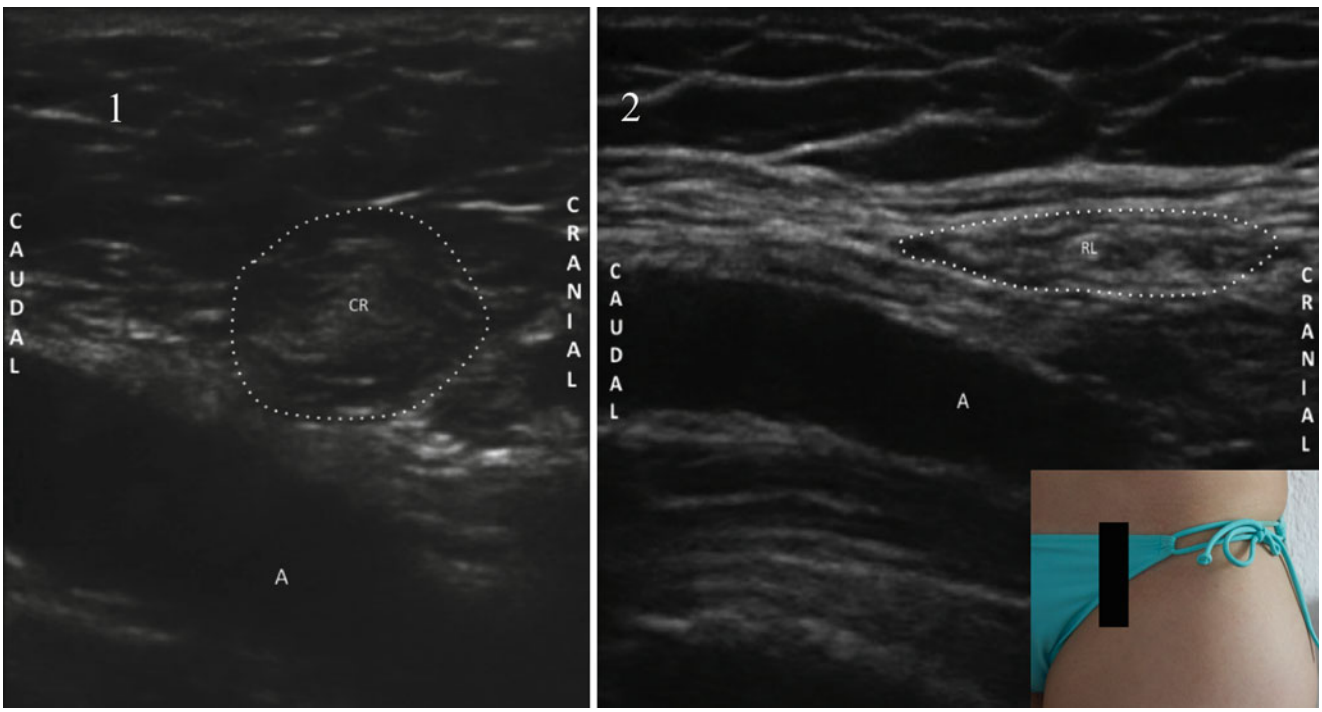

Fig. 41.11 Genitofemoral ultrasound imaging. *1* Cross-sectional ultrasound image of the male internal inguinal ring. *Dotted line* represents the internal inguinal ring; *CR* cremaster, *A* external iliac artery. *2* Cross-sectional ultrasound image of the female internal inguinal ring. *Dotted line* represents the internal inguinal ring; *RL* round ligament, *A* external iliac artery (Image courtesy of Thiago Nouer Frederico, MD)

difficult to visualize, when this technique is done with the aid of a PNS, small volumes of local anesthetic can be used, preventing the spread of medication into the lumbar sympathetic chain. The needle entry point is just above the L4 transverse process, and utilizing the PNS, stimulation radiating to the groin and the upper ipsilateral thigh should be achieved [13].

Neurolytic/Surgical Technique

Cryoneuroablation

Cryoneuroablation at the pubic tubercle can be performed by fluoroscopy-guided or ultrasound-guided techniques. Because the tissue at the pubis is usually relatively thin, it is conceivable that one could identify the GFN by just landmarks and the built-in nerve stimulator on the cryoprobe, but this is not a recommended technique. Trescot [19] described placement of the cryoprobe onto the pubic tubercle, using fluoroscopy and the nerve stimulator to find the nerve (Fig. 41.14). In that same publication, Trescot also described cryoneuroablation at the L1 foramen to treat the GFN proximally (Fig. 41.15). Campos et al. [30] described cryoneuroablation of the femoral branch of the GFN under US guidance (Fig. 41.16). The details of that approach are described more fully in the GFN lower extremity section (Chap. 60).

Radio-Frequency Lesioning

Although conventional RF should be strongly discouraged in this area because of the risk of neuritis and neuroma, pulsed radio-frequency treatment of the GFN has been described. Terkawi and Romdhane [31] treated a young man suffering

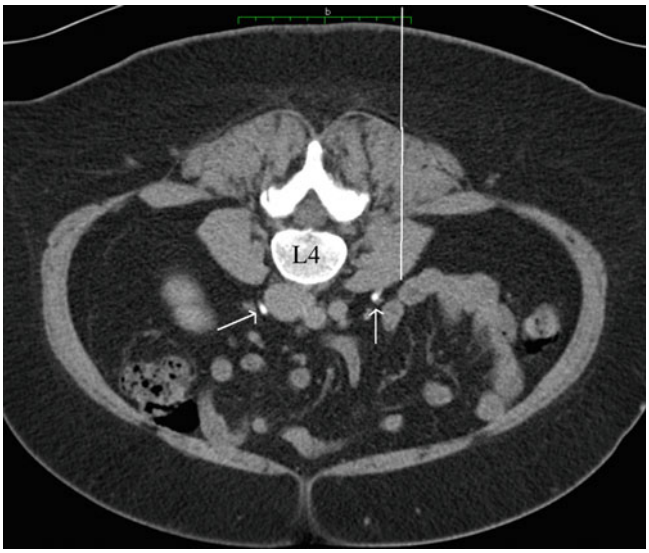

Fig. 41.13 Trans-psoas contrast CT-guided technique (simulated). Per Parris et al. [10], the needle is placed through the psoas muscle; *arrows* = ureters (with contrast) (Image courtesy of Andrea Trescot, MD)

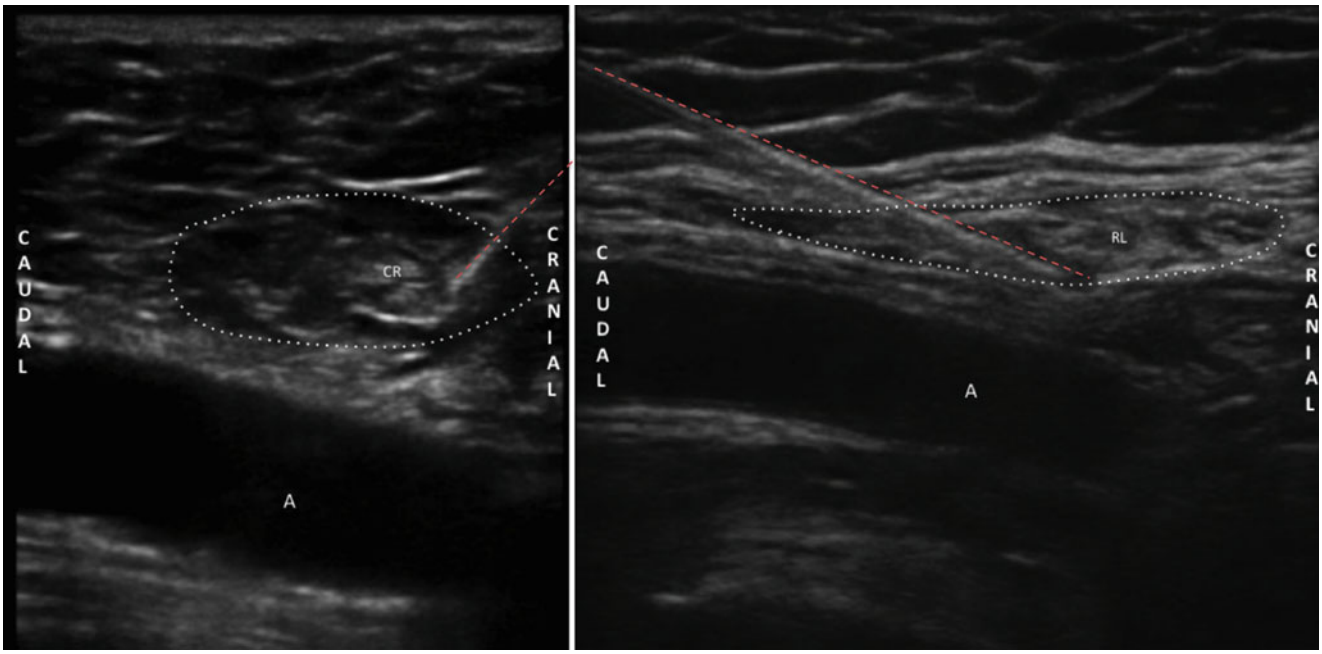

Fig. 41.12 Genitofemoral ultrasound-guided injection. *1* Injection of the male internal inguinal ring filled with local anesthetic and needle tip near the cremaster. *White dotted line* represents the internal inguinal ring; *dashed red line* represents the needle path; *CR* cremaster, *A* external iliac artery. *2* Injection of the female internal inguinal ring filled with local anesthetic and needle tip near the round ligament. *White dotted line* represents the internal inguinal ring; *dashed red line* represents the needle path; *RL* round ligament, *A* external iliac artery (Image courtesy of Thiago Nouer Frederico, MD)

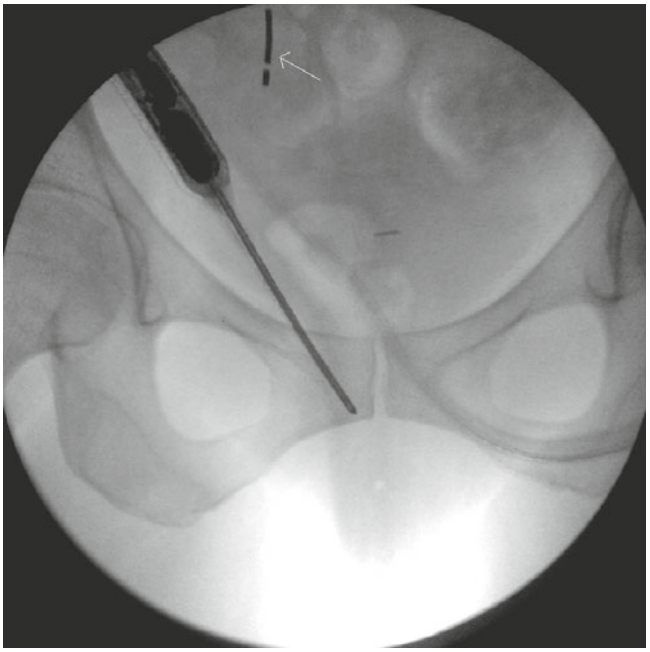

Fig. 41.14 Cryoneuroablation of the genitofemoral nerve at the pubic tubercle. Note the *white arrow* showing Interstim® placed for interstitial cystitis pain that offered no relief (Image courtesy of Andrea Trescot, MD)

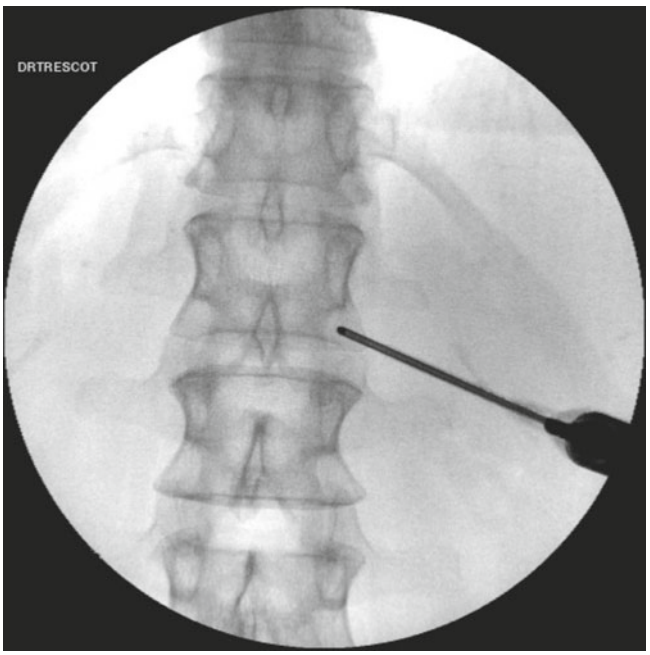

Fig. 41.15 Cryoprobe positioned on the proximal genitofemoral nerve at L1 (Image courtesy of Andrea Trescot, MD)

from chronic orchialgia; a diagnostic injection of the genital branch under US guidance (using a technique similar to that described above) gave excellent but only temporary relief. The patient underwent a pulsed RF lesion, again using the same ultrasound technique. At 7 months, the patient was still noting excellent relief.

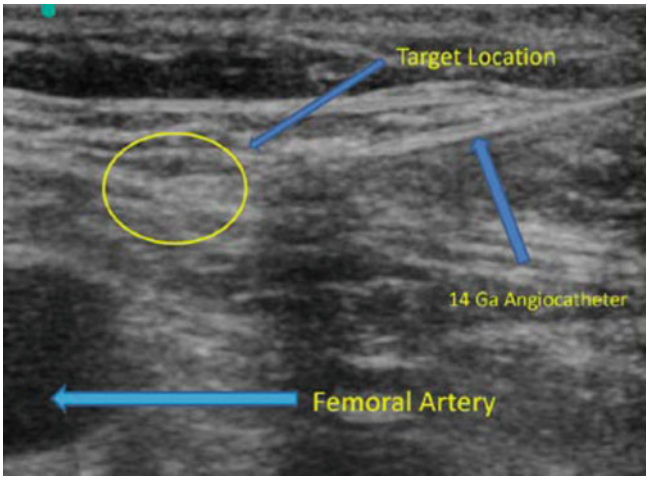

Fig. 41.16 Cryoneuroablation under ultrasound of the genitofemoral nerve (Image courtesy of John Chiles, MD)

Rozen and Ahn [32] described five patients with chronic groin pain treated with pulsed RF at T12, L1, and L2. Although they described the treatment of ilioinguinal neuralgia after inguinal hernia repair, due to the great variability distally and the shared origin at L1and L2, the nerves treated could well have been the GFN.

Phenol

Weksler and colleagues [33] described injecting 4 % phenol onto a variety of painful structures (including the GFN) in 35 patients; they noted good relief and no complications.

Neurostimulation

Peripheral nerve stimulation has been used to treat chronic groin pain (Fig. 41.17) [34, 35]. In a technique similar to that used for ilioinguinal nerve stimulation (see Chap. 40), the trial electrodes are placed percutaneously through introducers, and if there is significant temporary relief, the leads can be placed permanently.

Surgery

Starling and colleagues reviewed 30 patients with ilioinguinal or GFN abdominal pain over a 7-year period. The patients were diagnosed with local anesthetic injections; 10 of the 13 GFN patients were treated with neurectomy proximal to the entrapment noted relief. Triple neurectomy (ilioinguinal, iliohypogastric, and GFN nerves) has also been advocated, with a reported 80 % success rate in relieving postsurgical groin pain [36]. According to Muto et al., retroperitoneal endoscopic lumbar neurectomy is a simple, minimally

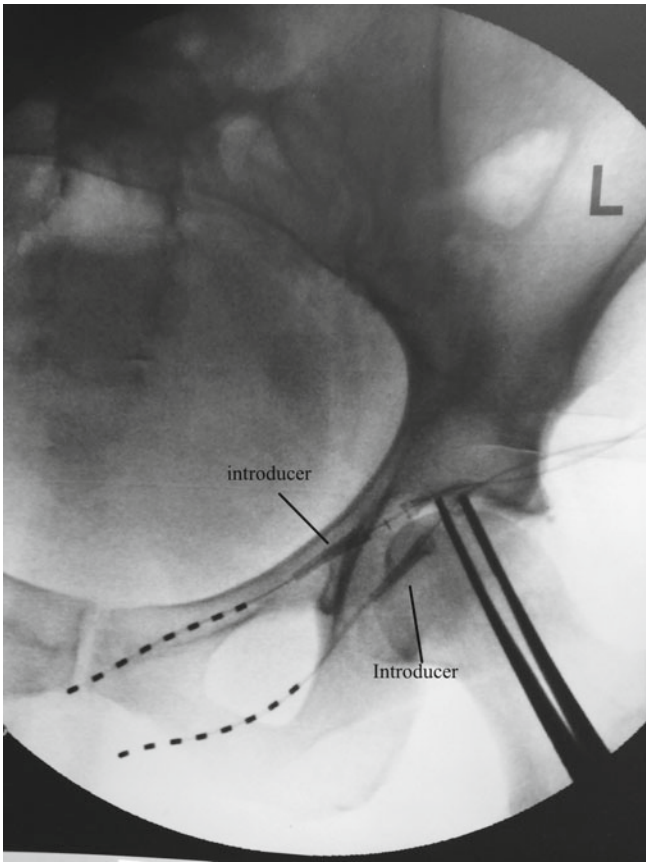

Fig. 41.17 Peripheral nerve stimulation of the abdominal wall (Image courtesy of Gladstone MacDowell, MD)

invasive technique with low morbidity and mortality. In this technique, the genitofemoral nerve is easily visualized where it penetrates the psoas muscle and can be dissected and resected at this site [12].

For the ten cases described by Murovic et al. [18], all were diagnosed as having GFN neuralgia after they noted temporary relief from diagnostic injections at L1 and L2. After a neurectomy of the GFN, all ten patients noted "complete or considerable" relief of their groin pain; there was "usually" a persistent numbness below the resected nerve, "which gradually resolved."

Muto et al. [12] described an endoscopic retroperitoneal approach to the ilioinguinal and genitofemoral nerves and reported good relief on six patients treated with endoscopic neurectomy.

Complications

Any injection may cause the usual complications of bleeding, infection, and nerve damage. Psoas hematoma is a potentially serious complication of a nerve block, but it is usually encountered in the setting of anticoagulation. In the CT-guided trans-psoas technique, the main complications are ureter or intestine perforation, but retroperitoneal or psoas hematoma can also occur. At any rate, in the CT-guided technique, there is theoretically a lower risk of this happening than in the blind or fluoroscopy-guided techniques [13].

After the endoscopic surgical procedure, though uncommon, in men there may be loss of cremasteric reflex and in women a loss of sensation in the mons pubis and labia majora [12].

Summary

The GFN is an under-recognized cause of abdominal pain, as well as pelvic pain (see Chap. 45) and lower extremity pain (see Chap. 60). A careful history and physical, as well as a high index of suspicion, will help the clinician to begin to recognize and then treat GFN entrapments.

Acknowledgments We would like to thank Camille Khan for her translation of this manuscript from Brazilian Portuguese to English.

References

1. Sayson SC, Ramamurthy S, Hoffman J. Incidence of genitofemoral nerve block during lumbar sympathetic block: comparison of two lumbar injection sites. Reg Anesth. 1997;22(6):569–74.
2. Peng PW, Tumber PS. Ultrasound-guided interventional procedures for patients with chronic pelvic pain – a description of techniques and review of literature. Pain Physician. 2008;11(2):215–24.
3. Rho RH, Lamer TJ, Fulmer JT. Treatment of genitofemoral neuralgia after laparoscopic inguinal herniorrhaphy with fluoroscopically guided tack injection. Pain Med. 2001;2(3):230–3.
4. Feigl GC, Dreu M, Ulz H, Breschan C, Maier C, Likar R. Susceptibility of the genitofemoral and lateral femoral cutaneous nerves to complications from lumbar sympathetic blocks: is there a morphological reason? Br J Anaesth. 2014;112(6):1098–104.
5. Boss A, Clasen S, Kuczyk M, Anastasiadis A, Schmidt D, Claussen CD, et al. Thermal damage of the genitofemoral nerve due to radiofrequency ablation of renal cell carcinoma: a potentially avoidable complication. AJR Am J Roentgenol. 2005;185(6):1627–31.
6. Magee RK. Genitofemoral causalgia: (a new syndrome). Can Med Assoc J. 1942;46(4):326–9.
7. Starling JR, Harms BA. Diagnosis and treatment of genitofemoral and ilioinguinal neuralgia. World J Surg. 1989;13(5):586–91.
8. Aasvang E, Kehlet H. Surgical management of chronic pain after inguinal hernia repair. Br J Surg. 2005;92(7):795–801.
9. Rozen D, Parvez U. Pulsed radiofrequency of lumbar nerve roots for treatment of chronic inguinal herniorraphy pain. Pain Physician. 2006;9(2):153–6.
10. Bergman RA, Afifi AK, Miyauchi R. Genitofemoral nerve. Iowa City: Virtual Hospital, University of Iowa Health Care; 2002.
11. Shanthanna H. Successful treatment of genitofemoral neuralgia using ultrasound guided injection: a case report and short review of literature. Case Rep Anesthesiol. 2014;2014:371703.
12. Muto CM, Pedana N, Scarpelli S, Galardo R, Guida G, Schiavone V. Inguinal neurectomy for nerve entrapment after open/laparoscopic hernia repair using retroperitoneal endoscopic approach. Surg Endosc. 2005;19(7):974–6.

13. Parris D, Fischbein N, Mackey S, Carroll I. A novel CT-guided trans-psoas approach to diagnostic genitofemoral nerve block and ablation. Pain Med. 2010;11(5):785–9.
14. Rab M, Ebmer, Dellon AL. Anatomic variability of the ilioinguinal and genitofemoral nerve: implications for the treatment of groin pain. Plast Reconstr Surg. 2001;108(6):1618–23.
15. Ingber RS. Iliopsoas myofascial dysfunction: a treatable cause of "failed" low back syndrome. Arch Phys Med Rehabil. 1989;70(5):382–6.
16. Parnell BA, Johnson EA, Zolnoun DA. Genitofemoral and perineal neuralgia after transobturator midurethral sling. Obstet Gynecol. 2012;119(2 Pt 2):428–31.
17. Reinpold WM, Nehls J, Eggert A. Nerve management and chronic pain after open inguinal hernia repair: a prospective two phase study. Ann Surg. 2011;254(1):163–8.
18. Murovic JA, Kim DH, Tiel RL, Kline DG. Surgical management of 10 genitofemoral neuralgias at the Louisiana State University Health Sciences Center. Neurosurgery. 2005;56(2):298–303.
19. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
20. Ekberg O, Persson NH, Abrahamsson PA, Westlin NE, Lilja B. Longstanding groin pain in athletes. A multidisciplinary approach. Sports Med. 1988;6(1):56–61.
21. Malik A, Simopolous T, Elkersh M, Aner M, Bajwa ZH. Percutaneous radiofrequency lesioning of sensory branches of the obturator and femoral nerves for the treatment of non-operable hip pain. Pain Physician. 2003;6(4):499–502.
22. Tammareddi K, Morelli V, Reyes Jr M. The athlete's hip and groin. Prim Care. 2013;40(2):313–33.
23. Rigaud J, Riant T, Delavierre D, Sibert L, Labat JJ. Somatic nerve block in the management of chronic pelvic and perineal pain. Prog Urol. 2010;20(12):1072–83.
24. Bischoff JM, Aasvang EK, Kehlet H, Werner MU. Does nerve identification during open inguin4l herniorrhaphy reduce the risk of nerve damage and persistent pain? Hernia. 2012;16(5):573–7.
25. Kraus MA. Laparoscopic identification of preperitoneal nerve anatomy in the inguinal area. Surg Endosc. 1994;8(5):377–80.
26. Trescot AM. Cryoneurolysis and the healing response. Annual Meeting American Academy of Pain Management, San Diego. 2005.
27. Broadman L. Ilioinguinal, iliohypogastric, and genitofemoral nerves. In: Gay SG, editor. Regional anesthesia, An Atlas of Anatomy and Techniques. St. Louis: Mosby; 1996. p. 247–54.
28. Recker DC, Perry PM. Postsurgical pain syndromes: chronic pain after hysterectomy and cesarean section. Tech Reg Anesth Pain Manag. 2011;15(3):133–9.
29. Peng PW, Narouze S. Ultrasound-guided interventional procedures in pain medicine: a review of anatomy, sonoanatomy, and procedures: part I: nonaxial structures. Reg Anesth Pain Med. 2009;34(5):458–74.
30. Campos NA, Chiles JH, Plunkett AR. Ultrasound-guided cryoablation of genitofemoral nerve for chronic inguinal pain. Pain Physician. 2009;12(6):997–1000.
31. Terkawi AS, Romdhane K. Ultrasound-guided pulsed radiofrequency ablation of the genital branch of the genitofemoral nerve for treatment of intractable orchalgia. Saudi J Anaesth. 2014;8(2):294–8.
32. Rozen D, Ahn J. Pulsed radiofrequency for the treatment of ilioinguinal neuralgia after inguinal herniorrhaphy. Mt Sinai J Med, New York. 2006;73(4):716–8.
33. Weksler N, Klein M, Gurevitch B, Rozentsveig V, Rudich Z, Brill S, et al. Phenol neurolysis for severe chronic nonmalignant pain: is the old also obsolete? Pain Med. 2007;8(4):332–7.
34. Rosendal F, Moir L, de Pennington N, Green AL, Aziz TZ. Successful treatment of testicular pain with peripheral nerve stimulation of the cutaneous branch of the ilioinguinal and genital branch of the genitofemoral nerves. Neuromodulation. 2013;16(2):121–4.
35. Banh DP, Moujan PM, Haque Q, Han TH. Permanent implantation of peripheral nerve stimulator for combat injury-related ilioinguinal neuralgia. Pain Physician. 2013;16(6):E789–91.
36. Amid P. Causes, prevention, and surgical treatment of postherniorrhaphy neuropathic inguinodynia: triple neurectomy with proximal end implantation. Hernia. 2004;8(4):343–9.

Abdominal Cutaneous Nerve Entrapment Syndrome

42

Eugene D. Kaplan and Helen W. Karl

Introduction

Abdominal cutaneous nerve entrapment syndrome (ACNES) (also known as "intercostal neuralgia" [1], "rectus abdominis nerve entrapment syndrome" [2], chronic abdominal wall pain [3–5], and many others [6]) is abdominal pain due to entrapment of the abdominal cutaneous nerves (ACN). These are the most distal branches of intercostal nerves, which terminate in the *rectus abdominis muscle* and the skin of the anterior abdominal wall. One investigator stated that any patient with the triad of chronic abdominal pain, a positive *Carnett's test* (see below), and normal laboratory/radiologic examinations suffers from ACNES until proven otherwise [7].

Despite the fact that ACNES was initially described in the nineteenth century and is relatively easy to identify and treat, it is still widely underdiagnosed and viewed as a diagnosis of exclusion [8–10]. As a result, patients with abdominal wall pain frequently have multiple studies and surgeries in the interval before correct diagnosis (1 month to 30 years, average 25 months) [5], examples of what one author has termed "visceral thinking" [11]. The average cost of unnecessary evaluations before correct diagnosis was $6,727 per patient (using 2001 prices) in one study [12], and evaluation costs decreased more than 50 % after a pain clinic consultation, when compared to the costs of a diagnostic evaluation for the same symptoms in a primary care clinic (mean cost $541 per pain clinic patient versus $1,133 per primary care clinic patient) [5].

The true incidence and prevalence of ACNES is unknown. It has been estimated that for every 150 patients in a family practice, there are 1–2 patients who have abdominal wall pain [13].

Clinical Presentation (Table 42.1)

Patients with ACNES will present with sharp, constant, or intermittent pain over any part of the anterior abdominal wall (Fig. 42.1). If the pain is on the right, they may be misdiagnosed as having *appendicitis* or *cholecystitis*; if on the left, they may be thought to have *diverticulitis*. The patient may have undergone multiple abdominal surgeries, especially in the presence of objective evidence such as gallstones or endometriosis [18]. Women have ACNES much more often than men (as much as 4:1) [3, 5, 11, 19]. Upper abdominal wall pain is more frequently seen in men, while women more often have pain in the lower abdominal wall [18]. Patients with ACNES are most commonly in 30–50 year age range

Table 42.1 Occupation/exercise/trauma history relevant to abdominal cutaneous nerve entrapment

Abdominal wall stretching	Pregnancy (9 %) [11], ascites, obesity
Neuropathy, radiculopathy	Shingles, diabetes [14]
Musculoskeletal/mechanical trauma	Large holster/belts, seat belt injury, blunt trauma to the abdominal wall [15], playing sports (7 %) [11]
Iatrogenic (20 %) [11]	Abdominal surgery, laparoscopy, drains, postoperative scars
Infection	Herpes zoster
Endocrine/metabolic	Obesity, diabetic mononeuropathy, pregnancy, birth control pills [16, 17]
Neoplasms	Abdominal wall tumors (lipoma, desmoid), metastatic disease

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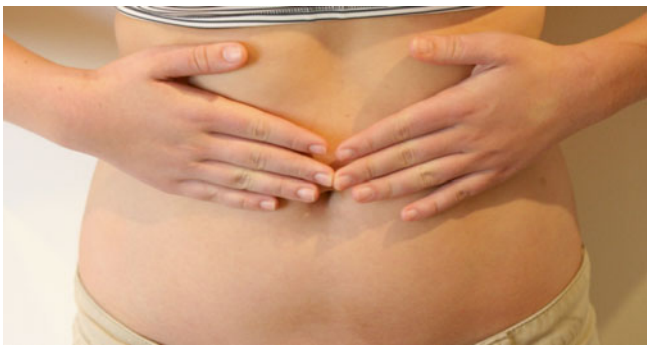

Fig. 42.1 Pattern of pain from ACNES (Image courtesy of Andrea Trescot, MD)

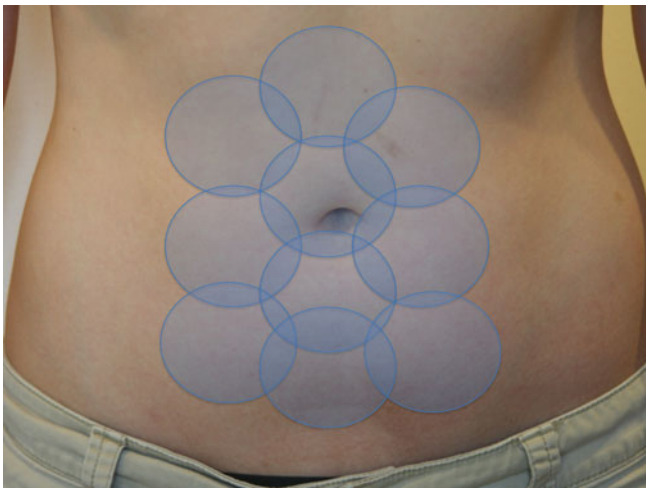

Fig. 42.2 Sites where ACNES patients will identify pain (Image courtesy of Andrea Trescot, MD)

[3, 6, 19], though this condition has been reported in children [20, 21] and the elderly [5]. The pain is more often on the right side [3, 5, 18]. This condition often occurs at 22–23 weeks of pregnancy, especially in muscular young mothers, as the growth of the baby stretches the abdominal musculature. The pain is usually increased by activities that increase abdominal wall tension (e.g., coughing, lifting weights/groceries/children, turning in bed), and patients can usually point with one finger to where it is most intense (Fig. 42.2).

Some patients experience immediate pain after straining the abdominal wall with coughing or other abrupt movement, while others cannot provide definite pain onset details, and a third group develops pain after a surgical intervention. Their medical history is frequently long and filled with multiple unproductive subspecialty consultations, exuberant workup, and trials of medication, physical therapy, massage, acupuncture, psychotherapy, and/or hypnosis. They are often told that they have *fibromyalgia*, *irritable bowel syndrome*, *depression*, or *Munchausen's syndrome*, and they may be confronted as malingerers or drug seekers. Since they are unable to prove the legitimacy of their pain because of their often normal tests, these patients may have a hard time establishing a trusting relationship with a physician.

Anatomy (Table 42.2)

The lowest five pairs of thoracic intercostal nerves plus the nerve from T12 innervate the abdominal wall (Fig. 42.3). At each thoracic spinal level, the junction of the dorsal (posterior sensory) and ventral (anterior motor) roots from the spinal cord forms an approximately 1 cm *spinal nerve*, which exits the intervertebral foramen (Fig. 42.4). A short *posterior cutaneous ramus* loops back to innervate the intrinsic muscles of the back and their overlying skin. The much longer anterior and collateral rami travel forward between the *internal oblique* and *transversus abdominis* muscles in the *transversus abdominis plane* (TAP) [22] as *thoracoabdominal intercostal* (T7–T11) or *subcostal (subgastric)* (T12) nerves and turn 90° anteriorly to pierce the *rectus abdominis muscle* (RAM). Multiple terminal motor branches of these thoracoabdominal nerves supply the RAM [24]. Each of these abdominal wall nerves divides into several branches that, accompanied by an artery and vein, traverse the RAM at each level [24]. Each perforating neurovascular bundle crosses the muscle anywhere from the midline to its lateral border via a fibrous ring to innervate the skin and subcutaneous tissues [24, 25] (Fig. 42.5). The T7 ACN innervates the skin at the level of the xiphoid, the T10 at the umbilicus, and the L1 in the groin as the *iliohypogastric nerve*. The *subcostal (subgastric)* (T12) nerve can communicate with the *iliohypogastric nerve* (see Chap. 40) [23].

RAM anatomy is of particular interest to clinicians interested in rectus sheath or paraumbilical blocks for perioperative [26] and chronic [27] pain management and to those involved in the management of abdominal wall hernias [28] or design of myocutaneous flaps [24, 29]. Thus, investigators from a variety of perspectives have made valuable contributions to understanding the anatomy pertinent to ACNES.

Entrapment

The thoracoabdominal nerves are anchored at six branching points, each a potential site of irritation or entrapment, particularly where the nerve abruptly changes position (Table 42.3) [13, 30]. The most common thoracoabdominal nerve entrapment site is near the lateral border of the RAM, though many other sites over the RAM and at the branching points of the lateral and posterior cutaneous branches are possible. Usually patients have only one site of entrapment (92 %), but multiple sites are possible [5].

Table 42.2 Anatomy of anterior cutaneous nerve entrapment

Origin	Thoracic spinal nerves T7–T12
General route	The anterior rami of these spinal nerves become the intercostal (T7–T11) and subcostal (T12) nerves. Each intercostal nerve gives off a lateral, collateral, and anterior cutaneous branch, with the anterior and collateral branches continuing between the internal oblique and transversus abdominis muscles in the *transversus abdominis plane* (TAP) [22] to the deep surface of the *rectus abdominis muscle* (RAM) [23], where they angle sharply forward to pass through the muscle and anterior *rectus sheath*, accompanied by a branch of the *epigastric artery* and vein [8]
Sensory distribution	To the corresponding dermatomes on the anterior abdominal wall, with overlap from its neighboring nerves
Motor innervation	Anterior abdominal wall and intercostal muscles
Anatomic variability	*Branching and recombination pattern of the anterior and collateral branches of the abdominal wall nerves*: extensive and variable [23] to the point that it has been termed the "intercostal plexus" [22, 24]
	Site of entry of the nerve into the RAM: between the posterior medial part of the muscle (more superior nerves) and the lateral border (more inferior nerves) [22]. At this point, the nerve contains motor and sensory fibers. Often T12 courses anterior to the RAM [24]
	Number of muscular branches to the RAM at each level: 1–20 [24]
	Number of neurovascular perforators at each level: 2–18 [24]
Other relevant structures	*Rectus abdominis muscle* (*RAM*): originates at the symphysis pubis and inserts on the cartilages of the 5–7th ribs. It is surrounded by a posterior (above the arcuate line, see below) and an anterior layer of fascia (*rectus sheath*)
	Linea alba: thick, midline abdominal fascia that extends from the xiphoid process to the pubic symphysis and is formed by fusion of the abdominal muscle aponeuroses from both sides
	Semilunar line: a curved line at the lateral border of RAM formed by fusion of the aponeuroses of the external and internal oblique and transversus abdominis muscles. It stretches from the cartilage of the fifth rib to the pubic tubercle
	Arcuate line: the horizontal line slightly less than halfway between the umbilicus and the pubis formed by the inferior margin of the posterior rectus sheath. Above this line, the anterior rectus sheath is the fused aponeuroses of the external and internal oblique muscles, and the posterior rectus sheath is the aponeuroses of the internal oblique and transversus abdominis muscles. Below the arcuate line, the fused aponeuroses of all three muscles form the anterior rectus sheath; the posterior rectus sheath is only thin transversalis fascia
	Epigastric arteries and veins: branches accompany the anterior cutaneous nerves through the rectus margin

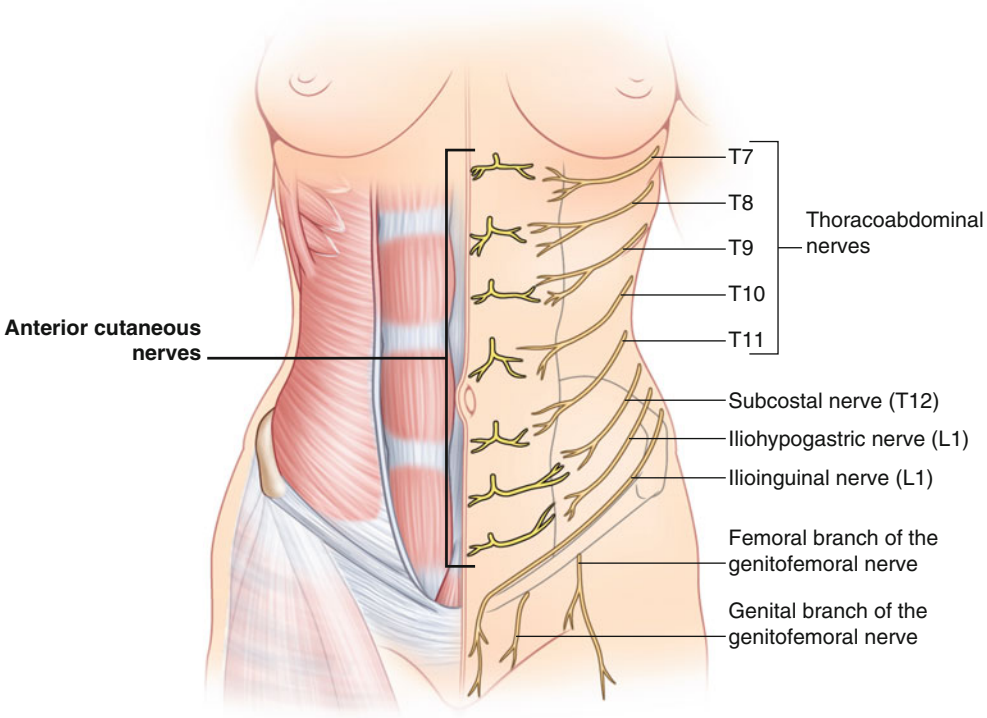

Fig. 42.3 Abdominal wall nerves (Image courtesy of Springer)

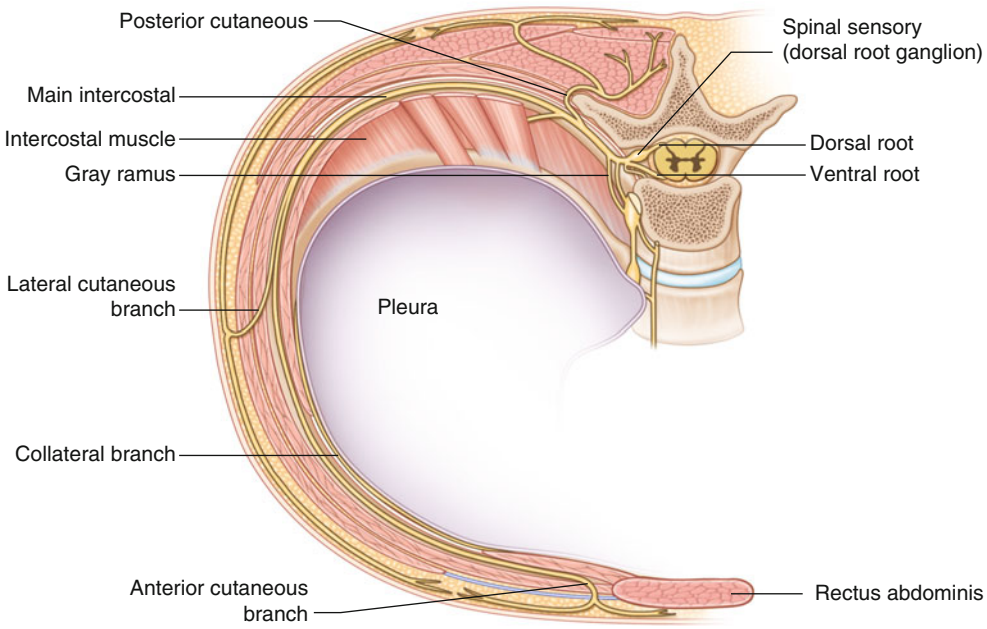

Fig. 42.4 Anatomy of an intercostal nerve and its branches (Image courtesy of Springer)

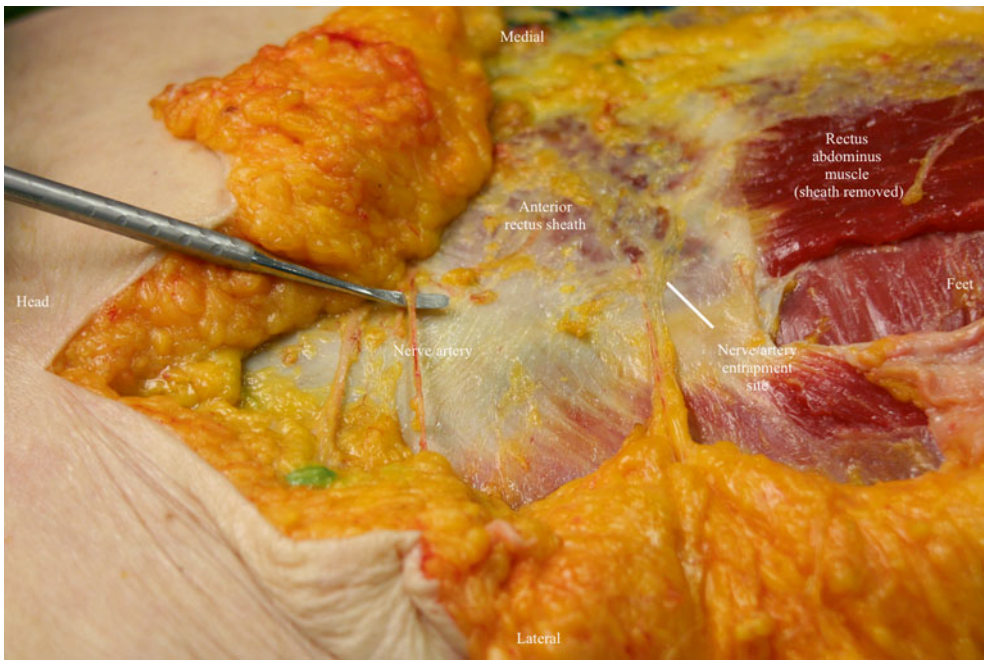

Fig. 42.5 Abdominal wall dissection. Note the neurovascular bundles emerging from the rectus abdominis muscle to innervate and perfuse the subcutaneous tissues and skin (Dissection courtesy of Gabor Balsa, MD, Semmelweis University, Laboratory for Applied and Clinical Anatomy, Budapest, Hungary; Image courtesy of Andrea Trescot, MD)

Table 42.3 Sites of anchoring of anterior cutaneous nerves [13]

Exit from the spinal cord
Origin of the posterior branch
Origin of the lateral branch
90° turn to enter the rectus muscle (most common)
Branching site in the rectus channel
Skin

As the ACN passes through a firm fibrous ring in the rectus muscle, it is accompanied by small branches of the inferior epigastric artery and vein, cushioned by fatty tissue [8, 25] (Fig. 42.5). Local inflammation and scarring impedes ACN gliding through the sheath opening, which causes painful nerve overstretching, frequently associated with a specific body position. Increased intra-abdominal pressure from

any cause can initiate herniation of the neurovascular bundle and its accompanying fat through the fibrous ring [6]. The presence of systemic conditions such as diabetes mellitus makes the ACN more vulnerable to injury [13, 14], and fluid retention due to menstrual cycles or pregnancy may increase the volume of the neurovascular bundle and make entrapment more likely [16, 17].

Scars due to trauma or surgery may create additional points of entrapment of subcutaneous branches [31] and are frequent precursors to ACNES [11].

Physical Exam

Physical examination is directed by the history of symptom development and location, and is initially performed in a standing position. It begins with observation of abdominal muscle tone and the presence of surgical scars or hernias. With the patient supine, ask the patient to put one finger on the most painful spot and then displace it slowly with your finger while palpating for a small indentation on the fascial surface to find the point of the ACN entrapment as it exits from the RAM [32]. Palpating fingers should literally "walk" through the painful area looking for irregularities and the point of maximum tenderness (Fig. 42.6). There is usually a particularly sharp pain localized to a small depression over the lateral RAM (Video 42.1). The entire RAM should be palpated, searching for additional trapped nerves. Then, ask the patient to perform a partial "sit-up" to contract the RAM while you palpate the point of maximum tenderness (*Carnett's test*) (Fig. 42.7) [1, 5, 33].

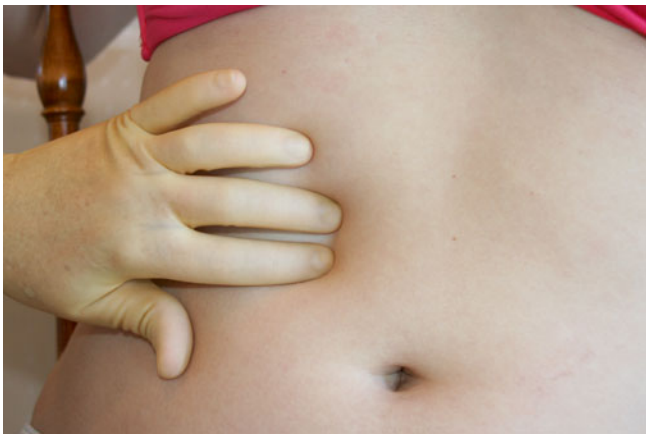

Fig. 42.6 Anterior cutaneous nerve entrapment examination (Image courtesy of Andrea Trescot, MD)

Carnett's test is valuable in differentiating a visceral cause of pain from ACNES. Contraction of the abdominal muscles is thought to decrease visceral pain by protecting the internal organs from the pressure of the examining finger; in ACNES, muscle contraction increases localized pain. Most authors have shown this test to be very useful [4, 5, 18, 34]. In one series of patients with acute abdominal pain seen in the emergency department, it was an unreliable predictor of the absence of a visceral cause for abdominal pain, probably because inflammation of the parietal peritoneum can also lead to a positive Carnett's test [35]. Carnett's sign may be difficult to elicit in patients with an overstretched rectus abdominis (frequently seen in pregnancy and central obesity) or in elderly and/or debilitated patients. The combination of a positive Carnett's test and the presence of highly localized pain is the fundamental criterion for ACNES diagnosis [3, 6].

Complete examination should include palpation of intercostal spaces (see Chap. 39), since ACNES symptoms may be produced by entrapment of the intercostal nerve at any point during its course.

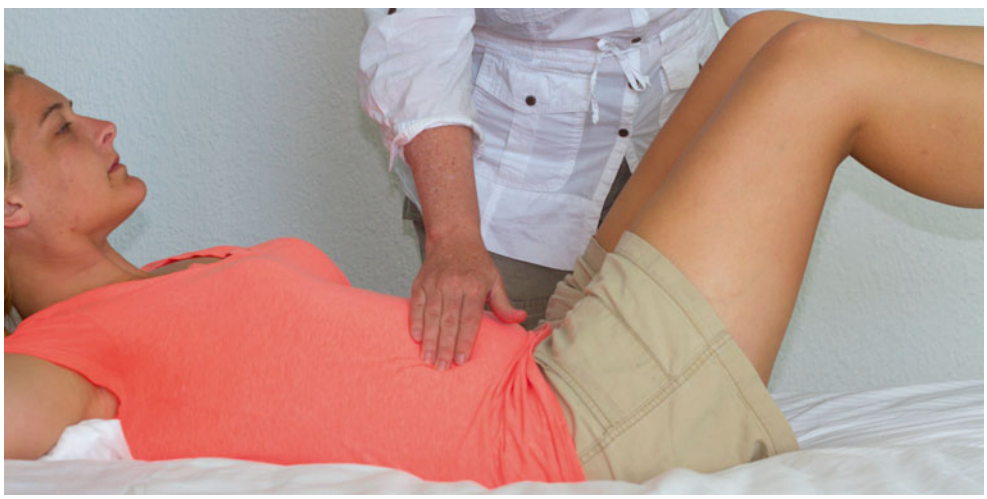

Fig. 42.7 Carnett's test (Image courtesy of Andrea Trescot, MD)

Differential Diagnosis (Table 42.4)

Broadly speaking, the biggest question is whether a patient's chronic abdominal pain is due to a visceral or a parietal (abdominal wall) problem (Table 42.5). Several algorithms have been devised to discriminate between the two [3, 33]. The presence of highly localized pain and tenderness and the increase in pain with abdominal wall tensing make a visceral source of pain much less likely [3, 4]. A good response to at least one injection of local anesthetic is included in the diagnostic criterion by many authors [3, 6, 11, 32, 49]. When algorithms are followed, only a small proportion (<10 %) of patients with chronic abdominal pain are subsequently found to have a visceral cause for their pain [3].

ACNES may resemble different conditions of the abdominal wall and viscera, depending on the location and chronicity of pain [33, 50]. Visceral pathology should be considered and appropriately evaluated with a thorough physical examination. Ultrasound or CT scanning may be useful [51–54]; however, most patients have already had multiple tests for visceral disease because this is usually a provider's first thought.

ACNES may coexist with an intra-abdominal condition [3]. A differential epidural or transversus abdominis plane (TAP) block may relieve somatic abdominal pain (no GI symptoms and positive Carnett's sign) and rarely ameliorates visceral pain (symptoms worsening with food intake and negative Carnett's sign) [55, 56]. A history of an organic abdominal cause, ongoing systemic symptoms, and deep abdominal tenderness is more often associated with a non-musculoskeletal cause of pain. ACNES at points of

Table 42.5 Comparison of intra-abdominal pain to abdominal wall pain

Intra-abdominal	Abdominal wall
Pain tends to be episodic	Pain tends to be constant or fluctuating
Nausea, vomiting, weight loss	May have nausea but rarely emesis
Diarrhea or constipation	Intensity related to position and intra-abdominal pressure
Jaundice	Pain not related to meals or bowel movements
Fever	Positive Carnett's sign
Positive lab or imaging	Very localized tender spot
Significant negative indication for musculoskeletal origin [48]	Significant positive indication of musculoskeletal source of abdominal symptoms
"Has there been any change in bowel habits since the onset of your symptoms?"	"Does taking a deep breath aggravate your symptoms?"
"Does eating aggravate your symptoms?"	"Does twisting your back aggravate your symptoms?"
"Has there been any weight change since the onset of symptoms?"	

Modified from Suleiman [32] and Sparkes [48]. These five questions combined produce specificity of 96 % and sensitivity of 67 % for the musculoskeletal origin of abdominal pain [48]

Table 42.4 Differential diagnosis of abdominal wall pain

	Potential distinguishing features
Cholecystitis, appendicitis, diverticulitis, spleen pathology	Pain not localized to a "fingertip"; abdominal CT, US, endoscopic tests, or MRI; often laboratory abnormalities
Endometriosis	Cyclic pattern of pain; abdominal CT, US, or MRI
Abdominal wall hernia, Spigelian hernia [6, 28, 32, 33]	May occur at laparoscopy port insertion sites or drain sites; visible on abdominal CT, US, and palpable abdominal wall defect
Iliohypogastric nerve entrapment [6]	Localize on physical examination
Herpes zoster	Presence of a skin rash in a dermatomal distribution
Interstitial cystitis	Urinary symptoms; cystoscopy
Irritable bowel disease [3]	May coexist; abdominal CT, US, endoscopy
Rectus sheath hematoma [36–40]	A tender, non-pulsatile mass due to injury of the epigastric blood vessels on physical examination. History of anticoagulation, abdominal CT, US
Slipping rib syndrome [5, 33, 41]	Pain in T7–T10 dermatomes, palpable popping with subluxation, can be differentiated by pulling out on the lower ribs on the involved side, visible on chest X-ray
Abdominal wall tumor [42]	Palpable superficial mass on exam
Intra-abdominal mass	Possible palpable intra-abdominal mass on physical exam, visible on abdominal MRI, CT or MRI
Tendonitis or tear of an abdominal muscle [6]	More likely in athletes, tenderness at muscle attachment (including the xiphoid) on examination; visible on abdominal CT or US
Spinal or vertebral pathology	Back pain [43]; tenderness at the tip of a vertebral transverse process "almost invariably occurs in pain of spinal origin" [44]
Thoracic facet pathology	Tenderness over thoracic facets, pain relief with facet injection; spine CT or MRI
Thoracic radiculopathy [14]	Negative Carnett's sign, position-dependent pain, relief with transforaminal or intercostal injection
Thoracic disk herniation [45, 46]	May have a history of trauma; absence of point tenderness; visible on MRI
Myofascial pain [47]	Local tenderness on examination

laparoscopic equipment insertion and postsurgical scars may produce widespread pain from the involvement of multiple nerves.

Diagnostic Tests (Table 42.6)

Ultrasound

Sonography is handy for quick confirmation of the diagnosis; it is especially valuable when Carnett's test cannot be reliably elicited. The sonographic anatomy of the abdominal wall has been well characterized [29, 56]. The patient should be in the supine position and instructed to breathe slowly and shallowly. Orient yourself by placing the transducer transversely, just off the midline, at the approximate level of the point of maximum tenderness (Fig. 42.8a). The RAM is seen as a hypoechoic area surrounded by its hyperechoic fascia. Above the arcuate line, the posterior rectus sheath consists of the aponeurosis of the transversus abdominis muscle and the transversalis fascia, often seen on ultrasound as a hyperechoic "double layer" (Fig. 42.8b) [56]. Tendinous intersections, the sites of attachment of the rectus muscle to the anterior rectus sheath, are visible on ultrasound [58]. Move the transducer laterally to identify the hyperechoic semilunar line; often the ACN is seen as a hyperechoic dot within the muscle medial to the semilunar line [49]. Its accompanying artery may also be visible and may help to find the nerve; veins at this level are usually too small to produce detectable blood flow.

Apply ultrasound gel on the skin over the painful area and slide a fingertip back and forth with mild pressure and without pulling the skin. This maneuver can help locate the site of origin of the pain, especially when hyperesthesia or allodynia is present; this technique is also useful in identification of a little "pit" (crescent defect of a fascia opening) or a "ball" of a firm fat surrounding the ACN at its exit through the fascia. The ultrasound probe is then placed over the point of maximal pain with minimal pressure, parallel to the lateral rectus border. Sharp pain elicited with a slight increase in the probe pressure or palpation under the probe with the tip of a ballpoint pen is a confirmative sign. The same steps may be utilized for precise needle guidance (see below). Diagnostic ultrasound also may be used to evaluate intra-abdominal pathology [51, 52].

Table 42.6 Diagnostic tests for anterior cutaneous nerve entrapment

	Potential distinguishing features
Physical exam	Point tenderness over the RAM, especially at the lateral edge
	Positive Carnett's test
Local anesthetic injection	≥50 % decrease in pain [3]
X-ray	Not useful
Ultrasound	To evaluate abdominal wall and intra-abdominal pathology [52]
MRI, CT	To evaluate intra-abdominal pathology [53, 57]
Electrodiagnostic studies	Not useful
Arteriography	Not useful

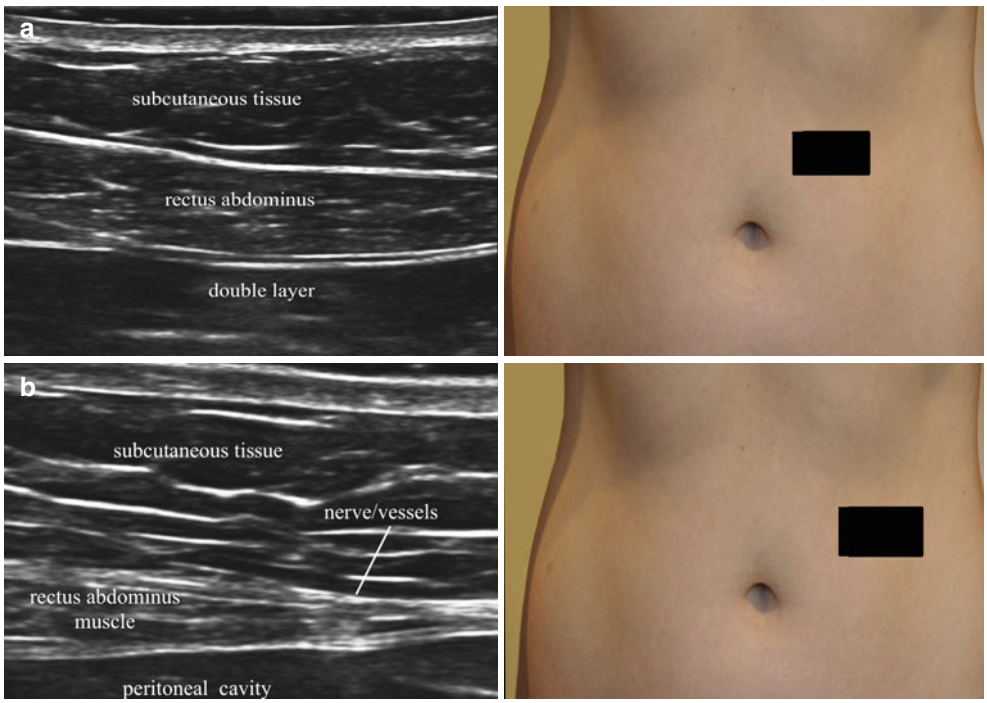

Fig. 42.8 Ultrasound images of the rectus abdominis muscle. *A* ultrasound image at the mid-rectus level, *B* ultrasound image at the lateral rectus border (Image courtesy of Andrea Trescot, MD)

CT or MR Imaging

CT or MR imaging may be useful to evaluate intra-abdominal pathology, although most patients will already have had an extensive workup.

Identification and Treatment of Contributing Factors

Given their often long search for a diagnosis and the seriousness of some of the other possible causes, the reassurance of a positive ACNES examination and that the pain cause is explainable and treatable is frequently life-changing news. The simple reassurance that the cause of the pain is not dangerous often helps relieve the patient.

Many patients have a history of contributing factors, such as those that increase intra-abdominal pressure or lead to abdominal wall weakening, that increase the risk of ACNES [28]. These include obesity (in 84 % of patients with [5]), multiple pregnancies, collagen disorders, prostatic hypertrophy, chronic lung disease (especially with coughing), and trauma (including abdominal surgery). Comorbidities may include gastroesophageal reflux, irritable bowel syndrome, and depression [3, 5]. Systemic signs and symptoms are rare, and diagnostic studies are usually negative or may show gallstones or other misleading causes of abdominal pain.

The ACNES cases that arise due to overstretching the RAM by abdominal obesity or pregnancy may be improved by delivery of the infant or loss of the excess weight. Sports injuries can be prevented with proper exercise technique. Perimenstrual fluid retention can cause swelling and painful "self-suffocation" of the inflamed nerve within the rigid sheath and may result in cyclical abdominal wall pain that mimics endometriosis. Strategies to reduce fluid retention, such as diuretics prior to menstruation, may be helpful.

Injection Techniques

Precise placement of local anesthetic is key for accurate diagnosis [3]. There are several techniques available for diagnostic and therapeutic injections of the anterior abdominal cutaneous nerves.

Landmark-Guided Technique

The patient is placed supine with the involved side close to the injector and instructed to avoid deep breaths and movement. In thin patients, palpation is usually sufficient to identify the target, while a peripheral nerve stimulator (PNS) may be helpful in those who are overweight [10, 59]. Following aseptic precautions, the fingers of the non-injecting hand straddle the tender site, and a 25-gauge 2-in. needle is inserted at a 30° angle through the skin, 2–3 cm from the point of maximal pain (more in an obese patient) (Video 42.2) (Fig. 42.9). The needle should never be directed perpendicular to the abdominal wall; tangential needle position ensures that the needle moves within the abdominal wall, making penetration less likely. The needle is slowly advanced toward the painful spot through two ill-defined layers – the superficial loose fat of "Camper's fascia" and deeper fibrous fat of "Scarpa's fascia." The needle will finally encounter the resistance of the RAM sheath, and the patient usually feels a reproduction of their ACNES pain. Injection of a small amount of local anesthetic and waiting until the pain diminishes makes the procedure less stressful. Needle penetration through the dense anterior rectus sheath can be appreciated as a definite "pop." The inferior and superior epigastric vessels are superficial to the posterior rectus sheath and can be inadvertently damaged with deep needle advancement.

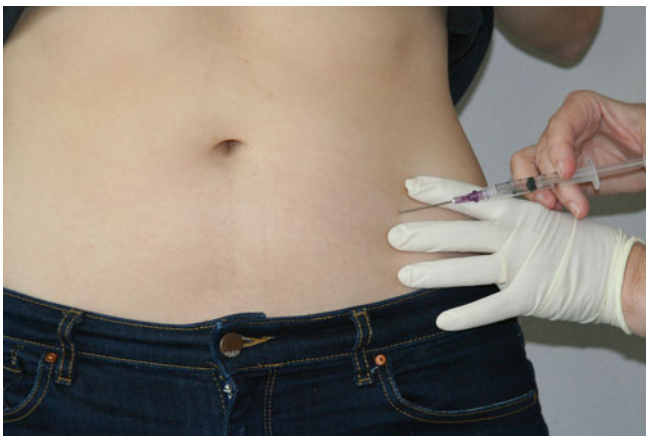

Fig. 42.9 Landmark-guided injection technique for ACNES (Image courtesy of Andrea Trescot, MD)

If the injection is diagnostic or the plan is to use cryoneuroablation (see below), small volumes (<2 cc) of local anesthetic must be used [60]. In order to treat inflammation of the nerve and surrounding tissues, add 10–40 mg of deposteroid. One may also attempt to soften or even expand the nerve canal through the RAM with hydrodissection (see Chap. 7), by using 2–3 cc of local anesthetic followed by 5–10 cc of normal saline solution. Additional forceful palpation of the point of the ACN exit with a fingertip after injection may also help increase the size of the opening and disrupt local scar tissue.

At least one third of patients have long-term pain relief after one or two injections of local anesthetic, with or without added steroid [19].

Ultrasound-Guided Technique

Because the interventionalist can see both the nerve and the needle, injection under US guidance shortens procedure time and improves its safety [49]. Using US allows precise identification of the RAM, its sheath, epigastric vessels, and peritoneum, thereby decreasing the significance of anatomic variations and avoiding injuries [58]. The needle course is similar to the landmark-guided technique (Fig. 42.10). Batistaki et al. [61] described a patient with intractable bilateral abdominal pain radiating to the back after a wide Pfannenstiel who noted excellent relief from a series of anterior cutaneous nerve blocks at T11 and T12 under US guidance (without and then with deposteroid).

Transversus Abdominis Plane (TAP) Block

The TAP block has been used primarily to provide analgesia for abdominal surgical procedures, but it has also been used to differentiate visceral and parietal causes of chronic abdominal pain [56]. It is designed to block the intercostal (T7–T11), subcostal (T12), ilioinguinal, and iliohypogastric nerves, as well as the lateral cutaneous branches of L1–L3 that supply sensory input to the lateral and anterior abdominal walls. Local anesthetic is placed between the internal oblique and transverse abdominal muscles under US guidance as single injections for intra- and postoperative analgesia [62] or via a catheter for a patient with persistent abdominal wall pain after a cholecystectomy [63].

Scar Injection (Neural Therapy)

Abdominal surgical scars frequently entrap distal ACN branches in the dermis and underlying tissue. *Hydrodissection* of the scar bulk, including the dermis, with 2 % lidocaine diluted with normal saline 1:1 or 1:2 and made slightly "hazy" with a small dose of steroid can provide significant immediate and long-term pain relief [E.D. Kaplan personal communication]. An appropriately Quincke spinal sized needle should be used (25-gauge for softer, short scars and 20–22-gauge for dense or long scars). A needle that is too "weak" for a particular scar can break or slow the procedure. Successful infiltration requires applying enough force to overcome the scar tissue density; therefore, syringes with a smaller cross-sectional area (such as a 3-cc syringe) are more effective, since a higher pressure can be generated. The needle length should cover the length of a planned procedure from a single entry point – otherwise the solution will leak through the other needle entry points, dissipating the high injectate pressure necessary for hydrodissection. To preserve skin integrity, only topical local anesthetics are used for preprocedure local anesthesia, so patients should be instructed to apply lidocaine cream 40–60 min before the procedure. Though *ethyl chloride spray* provides up to a minute of immediate skin anesthesia in cosmetic procedures without a compromise in sterility, the data on its use in interventional procedures remains limited [64, 65].

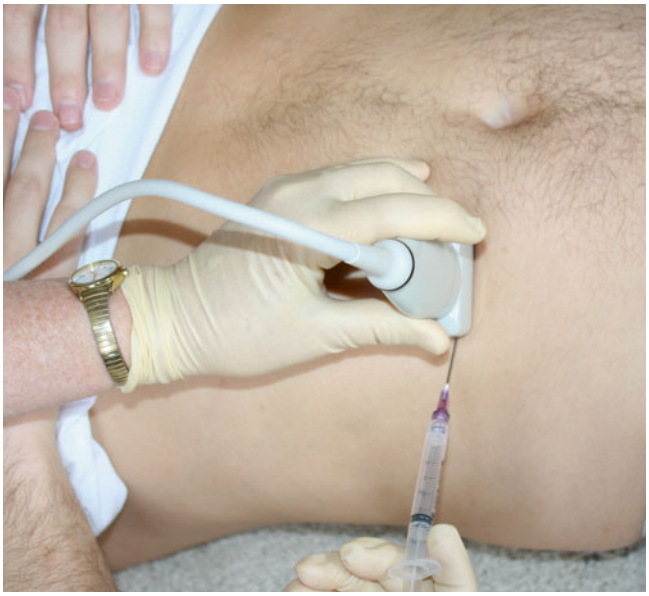

Fig. 42.10 Simulated ultrasound-directed anterior abdominal cutaneous nerve (Image courtesy of Andrea Trescot, MD)

For the procedure, the patient is positioned comfortably, and the site is prepared. The needle is inserted through the skin at about 30° and repositioned almost parallel to the skin in the lower dermis layers. The insertion point should be at least 1 cm from the infiltration area to create a "sleeve" of normal dermis around the needle to prevent solution backflow under high pressure. If the needle movements are very painful, a three-step procedure may provide additional comfort – advance the needle for 0.5–1 cm while injecting 0.5–2 mL of solution, wait for 10–15 s for the onset of local anesthesia, and then hydrodissect the area to create an even, scar-wide "orange peel" (Fig. 42.11). "Stubborn" areas are worked through with several 3–5 mm back and forth needle movements, in order to dissect dense collagen fibers and promote solution spread. When the first pass through the scar is completed, another layer can be placed 3–5 mm deeper or to the side, depending on the scar shape, and the process should continue in the same manner until the scar is well infiltrated. Ultrasound localization significantly simplifies hydrodissection of deeper scar layers.

Infected scars should not be injected. Hydrodissection of recent scars or scars with a history of prolonged and poor healing should be approached with caution.

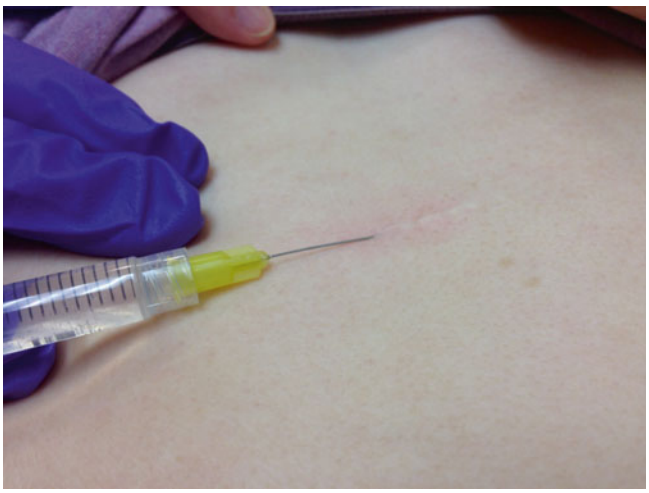

Fig. 42.11 Subcutaneous hydrodissection (neural therapy) (Image courtesy of Andrea Trescot, MD)

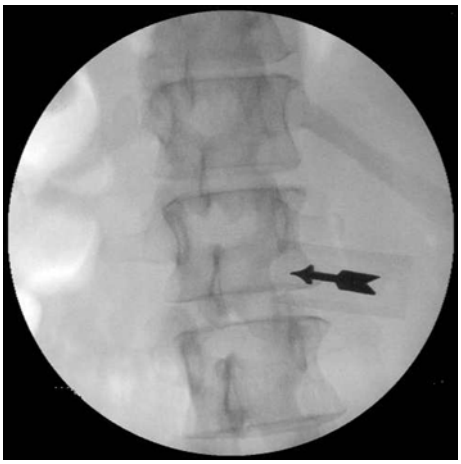

Fig. 42.12 Transforaminal site of proximal abdominal wall nerve injection (Image courtesy of Andrea Trescot. MD)

Fluoroscopic-Guided Technique

Although fluoroscopy is not helpful for injections into the anterior abdominal wall, it may be useful for more proximal injections at the dorsal root ganglion (Fig. 42.12), especially if there is a great deal of scar tissue at the distal entrapment site [66]. There is also a role for proximal injections as a diagnostic tool; temporary relief from a transforaminal injection should effectively eliminate intra-abdominal pathology.

Neurolytic/Surgical Techniques

Cryoneuroablation

For the ACNES patient who has experienced only temporary relief from two to three local anesthetic and steroid injections, consideration should be given to cryoneuroablation for non-surgical pain relief. Cryoneuroablation is usually performed at the entrapment site, but it may also be considered at a more proximal level, especially when finding the ACN neuroma is difficult, as when it is embedded in the scar tissue [60]. The probe is positioned parallel to the anterior abdominal nerve (Fig. 42.13), in order to increase the area of contact with the nerve.

Radiofrequency Neuroablation

In general, conventional RF for ACNES should be discouraged due to the risk of neuritis and neuroma formation. Pulsed RF may be a reasonable choice, though there are no published reports of its use in the abdominal wall, and it is a non-covered procedure in the United States. Successful ACNES treatment in a patient with at least 8 abdominal operations with pulsed RF ablation of the T10 and T11 dorsal root ganglia has been reported [66].

Chemodenervation

Alcohol has been used for pain relief by producing necrosis of the ACN and surrounding tissue. Applegate described successful pain relief with minimal local discomfort using a mixture of 1 cc of absolute alcohol and 0.5 cc of lidocaine 2 % [13]. A small-volume injection of phenol 5–7 % aqueous solution has also been reported to produce long-lasting relief in 56–95 % of patients [10, 59, 67].

Neuromodulation (See Chap. 9)

The use of peripheral nerve stimulation for abdominal wall pain has been rapidly expanding. Several authors have described its use for ilioinguinal/genitofemoral [68], intercostal [69], and abdominal nerves [70]. The leads are placed percutaneously, either parallel (Fig. 42.14) or oblique to the nerve, and then connected to a subcutaneous generator with frequencies and pulse width chosen to provide maximum pain relief for each individual.

Surgical Resection

In patients who fail to improve with nonoperative treatments, including only temporary relief with injections, surgical excision of an entrapped nerve or a scar neuroma is often effective. In a double-blind, randomized, controlled study of anterior neurectomy (neurectomy of one or more ACNs at the level of the anterior RAM sheath), 44 patients with

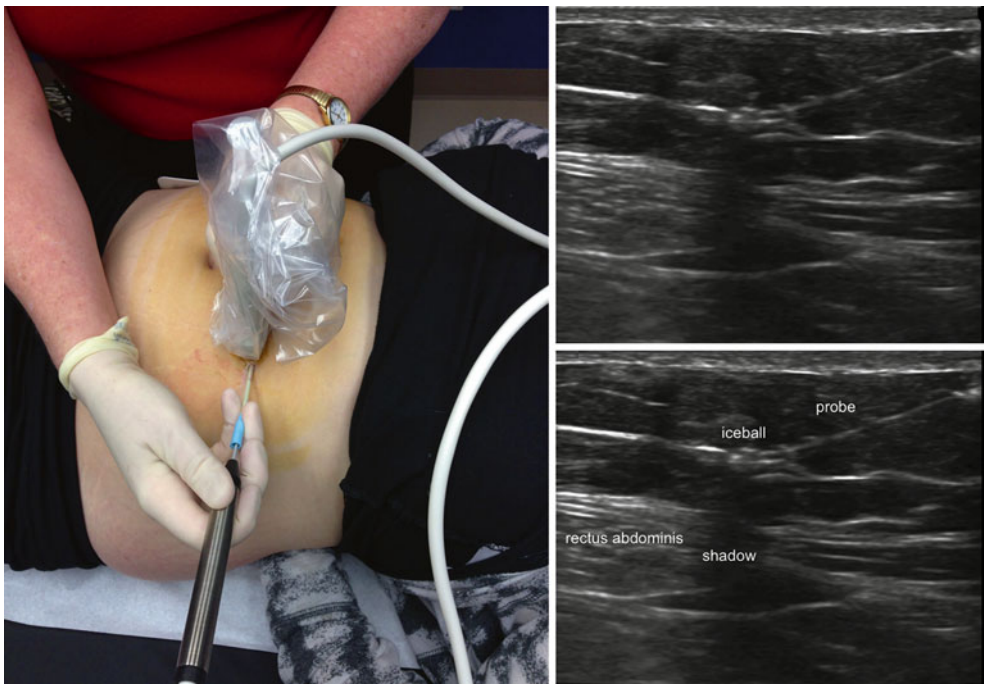

Fig. 42.13 Cryoneuroablation of the abdominal wall nerves (Image courtesy of Andrea Trescot, MD)

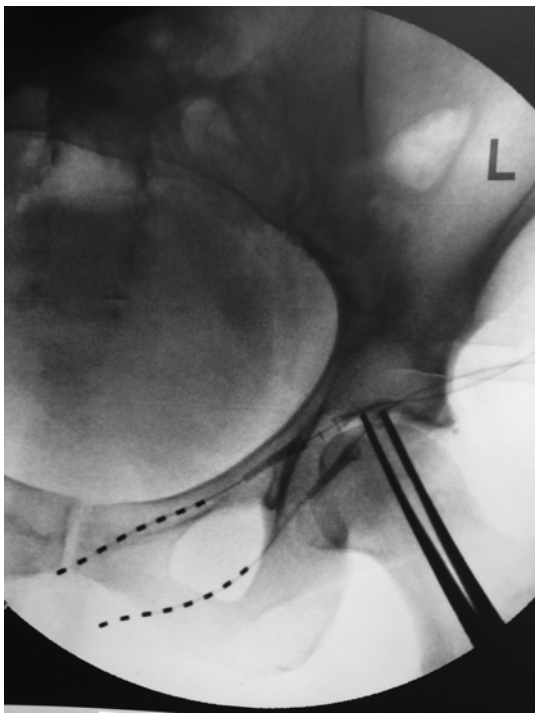

Fig. 42.14 Peripheral nerve stimulation of the abdominal wall (Image courtesy of Gladstone MacDowell, MD)

ACNES who had failed a nonoperative regimen were randomized to anterior neurectomy or a sham operation; success was defined as a 50 % reduction in pain. Anterior neurectomy was successful in 73 % of subjects vs. 18 % in a sham operation. Surgical complications were infrequent and easily treated [7]. These results were confirmed in a larger retrospective study with long-term follow-up. Seventy percent of 181 procedures were successful in the first 3 months, and 61 % maintained the result for an average of almost 3 years, but with a 16 % recurrence rate [19].

Complications

As with other interventional procedures, complications can include poor target localization with ineffective treatment during a landmark-based technique (especially in obese patients), intra-abdominal or intrathoracic needle penetration, intramuscular hematoma, anesthetic toxicity, bleeding, and increased pain. Although there are no reports specifically describing this in ACNES patients, chemodenervation and RF neuroablation can cause scar formation and deafferentation with possible pain increase. Denervation or injury to the lower abdominal nerves may cause an abdominal bulge, due to motor weakness of the abdominal wall (Fig. 42.15).

Summary

ACNES is a commonly overlooked cause of abdominal pain. Patients usually have undergone extensive evaluations and multiple surgeries before being sent to a pain management specialist. Diagnosis is simple and can be done at the bedside, and treatment can be very successful.

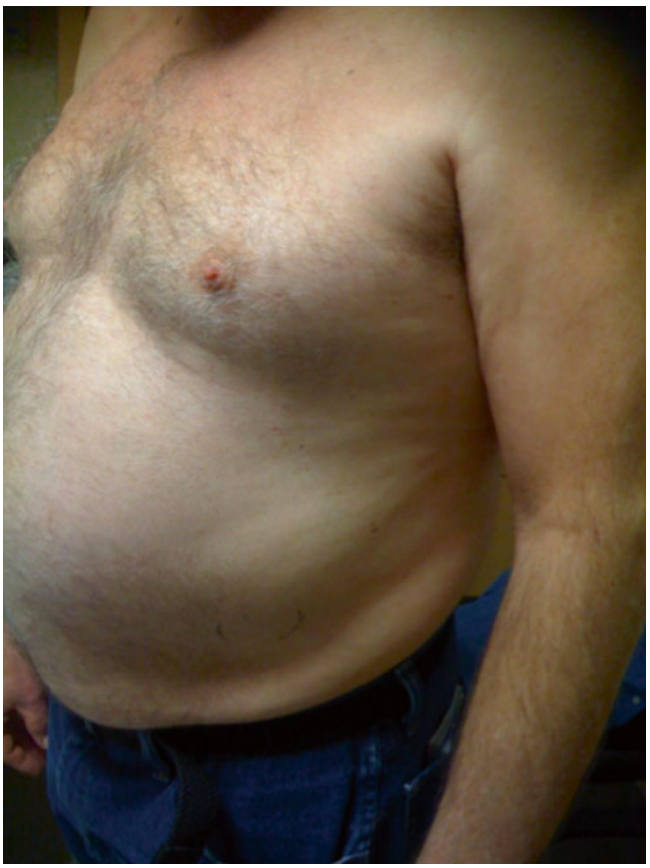

Fig. 42.15 Abdominal bulge after L1 injury (Image courtesy of Andrea Trescot, MD)

References

1. Carnett JB. Intercostal neuralgia as a cause of abdominal pain and tenderness. Surg Gynecol Obstet. 1926;42:625–32.
2. Tung AS, Tenicela R, Giovanitti J. Rectus abdominis nerve entrapment syndrome. JAMA. 1978;240(8):738–9.
3. Greenbaum DS, Greenbaum RB, Joseph JG, Natale JE. Chronic abdominal wall pain. Diagnostic validity and costs. Dig Dis Sci. 1994;39(9):1935–41.
4. Srinivasan R, Greenbaum DS. Chronic abdominal wall pain: a frequently overlooked problem. Practical approach to diagnosis and management. Am J Gastroenterol. 2002;97(4):824–30.
5. Costanza CD, Longstreth GF, Liu AL. Chronic abdominal wall pain: clinical features, health care costs, and long-term outcome. Clin Gastroenterol Hepatol. 2004;2(5):395–9.
6. Lindsetmo RO, Stulberg J. Chronic abdominal wall pain – a diagnostic challenge for the surgeon. Am J Surg. 2009;198(1):129–34.
7. Boelens OB, van Assen T, Houterman S, Scheltinga MR, Roumen RM. A double-blind, randomized, controlled trial on surgery for chronic abdominal pain due to anterior cutaneous nerve entrapment syndrome. Ann Surg. 2013;257(5):845–9.
8. Applegate WV. Abdominal cutaneous nerve entrapment syndrome. Surgery. 1972;71(1):118–24.
9. Hershfield NB. The abdominal wall. A frequently overlooked source of abdominal pain. J Clin Gastroenterol. 1992;14(3):199–202.
10. McGrady EM, Marks RL. Treatment of abdominal nerve entrapment syndrome using a nerve stimulator. Ann R Coll Surg Engl. 1988;70(3):120–2.
11. Boelens OB, Scheltinga MR, Houterman S, Roumen RM. Management of anterior cutaneous nerve entrapment syndrome in a cohort of 139 patients. Ann Surg. 2011;254(6):1054–8.
12. Thompson C, Goodman R, Rowe W. Abdominal wall syndrome: a costly diagnosis of exclusion. Gastroenterology. 2001;120(5):A637.
13. Applegate WV. Abdominal cutaneous nerve entrapment syndrome (ACNES); a commonly overlooked cause of abdominal pain. Perm J. 2002;6(3):1–14.
14. Longstreth GF, Newcomer AD. Abdominal pain caused by diabetic radiculopathy. Ann Intern Med. 1977;86(2):166–8.
15. Ivens D, Wojciechowski M, Vaneerdeweg W, Vercauteren M, Ramet J. Abdominal cutaneous nerve entrapment syndrome after blunt abdominal trauma in an 11-year-old girl. J Pediatr Surg. 2008;43(5):E19–21.
16. Peleg R, Gohar J, Koretz M, Peleg A. Abdominal wall pain in pregnant women caused by thoracic lateral cutaneous nerve entrapment. Eur J Obstet Gynecol Reprod Biol. 1997;74(2):169–71.
17. Peleg R. Abdominal wall pain caused by cutaneous nerve entrapment in an adolescent girl taking oral contraceptive pills. J Adolesc Health. 1999;24(1):45–7.
18. Gallegos NC, Hobsley M. Recognition and treatment of abdominal wall pain. J R Soc Med. 1989;82(6):343–4.
19. van Assen T, Boelens OB, van Eerten PV, Perquin C, Scheltinga MR, Roumen RM. Long-term success rates after an anterior neurectomy in patients with an abdominal cutaneous nerve entrapment syndrome. Surgery. 2015;157(1):137–43.
20. Scheltinga MR, Boelens OB, Tjon A, Ten WE, Roumen RM. Surgery for refractory anterior cutaneous nerve entrapment syndrome (ACNES) in children. J Pediatr Surg. 2011;46(4):699–703.
21. Akhnikh S, de Korte N, de Winter P. Anterior cutaneous nerve entrapment syndrome (ACNES): the forgotten diagnosis. Eur J Pediatr. 2014;173(4):445–9.
22. Rozen WM, Tran TM, Ashton MW, Barrington MJ, Ivanusic JJ, Taylor GI. Refining the course of the thoracolumbar nerves: a new understanding of the innervation of the anterior abdominal wall. Clin Anat. 2008;21(4):325–33.
23. Davies F, Gladstone RJ, Stibbe EP. The anatomy of the intercostal nerves. J Anat. 1932;66(Pt 3):323–33.
24. Yap LH, Whiten SC, Forster A, Stevenson JH. The anatomical and neurophysiological basis of the sensate free TRAM and DIEP flaps. Br J Plast Surg. 2002;55(1):35–45.
25. Applegate WV, Buckwalter NR. Microanatomy of the structures contributing to abdominal cutaneous nerve entrapment syndrome. J Am Board Fam Pract. 1997;10(5):329–32.
26. Courreges P, Poddevin F, Lecoutre D. Para-umbilical block: a new concept for regional anaesthesia in children. Paediatr Anaesth. 1997;7(3):211–4.
27. Skinner AV, Lauder GR. Rectus sheath block: successful use in the chronic pain management of pediatric abdominal wall pain. Paediatr Anaesth. 2007;17(12):1203–11.
28. Skandalakis PN, Zoras O, Skandalakis JE, Mirilas P. Spigelian hernia: surgical anatomy, embryology, and technique of repair. Am Surg. 2006;72(1):42–8.
29. Ali QM. Sonographic anatomy of the rectus sheath: an indication for new terminology and implications for rectus flaps. Surg Radiol Anat. 1993;15(4):349–53.
30. Kopell HP, Thompson WAL. Peripheral Entrapment Neuropathies, 2nd Edition. Robert E. Krieger Pub Co: Huntington, NY; 1976.
31. Ducic I, Larson EE. Outcomes of surgical treatment for chronic postoperative breast and abdominal pain attributed to the intercostal nerve. J Am Coll Surg. 2006;203(3):304–10.
32. Suleiman S, Johnston DE. The abdominal wall: an overlooked source of pain. Am Fam Physician. 2001;64(3):431–8.
33. Gallegos NC, Hobsley M. Abdominal wall pain: an alternative diagnosis. Br J Surg. 1990;77(10):1167–70.

34. Thomson H, Francis DM. Abdominal-wall tenderness: a useful sign in the acute abdomen. Lancet. 1977;2(8047):1053–4.
35. Young S, Levsky M, DeKay K, Dunn J. A prospective evaluation of Carnett's test for acute abdominal pain in the Emergency Department. Ann Emerg Med. 2007;50(3):A244.
36. Manier JW. Rectus sheath hematoma. Am J Gastroenterol. 1972;57(5):443–52.
37. Smithson A, Ruiz J, Perello R, Valverde M, Ramos J, Garzo L. Diagnostic and management of spontaneous rectus sheath hematoma. Eur J Intern Med. 2013;24(6):579–82.
38. Khan MI, Medhat O, Popescu O, Rastogi A, Thompson T. Rectus sheath haematoma (RSH) mimicking acute intra-abdominal pathology. N Z Med J. 2005;118(1217):U1523.
39. Segev Y, Orron D, Alon R, Graif M. Pseudoaneurysm of the inferior epigastric artery mimicking abdominal wall hematoma. J Ultrasound Med. 1994;13(6):483–4.
40. Cherry WB, Mueller PS. Rectus sheath hematoma: review of 126 cases at a single institution. Medicine (Baltimore). 2006;85(2): 105–10.
41. Heinz GJ, Zavala DC. Slipping rib syndrome. JAMA. 1977;237(8):794–5.
42. Stojadinovic A, Hoos A, Karpoff HM, Leung DH, Antonescu CR, Brennan MF, et al. Soft tissue tumors of the abdominal wall: analysis of disease patterns and treatment. Arch Surg. 2001;136(1): 70–9.
43. Jørgensen LS, Fossgreen J. Back pain and spinal pathology in patients with functional upper abdominal pain. Scand J Gastroenterol. 1990;25(12):1235–41.
44. Ashby EC. Abdominal pain of spinal origin. Value of intercostal block. Ann R Coll Surg Engl. 1977;59(3):242–6.
45. Xiong Y, Lachmann E, Marini S, Nagler W. Thoracic disk herniation presenting as abdominal and pelvic pain: a case report. Arch Phys Med Rehabil. 2001;82(8):1142–4.
46. Rohde RS, Kang JD. Thoracic disc herniation presenting with chronic nausea and abdominal pain. A case report. J Bone Joint Surg Am. 2004;86-A(2):379–81.
47. Yelland MJ. Back, chest and abdominal pain. How good are spinal signs at identifying musculoskeletal causes of back, chest or abdominal pain? Aust Fam Physician. 2001;30(9):908–12.
48. Sparkes V, Prevost AT, Hunter JO. Derivation and identification of questions that act as predictors of abdominal pain of musculoskeletal origin. Eur J Gastroenterol Hepatol. 2003;15(9):1021–7.
49. Kanakarajan S, High K, Nagaraja R. Chronic abdominal wall pain and ultrasound-guided abdominal cutaneous nerve infiltration: a case series. Pain Med. 2011;12(3):382–6.
50. Slocumb JC. Chronic somatic, myofascial, and neurogenic abdominal pelvic pain. Clin Obstet Gynecol. 1990;33(1):145–53.
51. Orr RK, Porter D, Hartman D. Ultrasonography to evaluate adults for appendicitis: decision making based on meta-analysis and probabilistic reasoning. Acad Emerg Med. 1995;2(7):644–50.
52. Kotagal M, Richards MK, Chapman T, Finch L, McCann B, Ormazabal A, et al. Improving ultrasound quality to reduce computed tomography use in pediatric appendicitis: the Safe and Sound campaign. Am J Surg. 2015;209(5):896–900.
53. Rao PM, Rhea JT, Novelline RA, Mostafavi AA, McCabe CJ. Effect of computed tomography of the appendix on treatment of patients and use of hospital resources. N Engl J Med. 1998;338(3):141–6.
54. Wang PI, Chong ST, Kielar AZ, Kelly AM, Knoepp UD, Mazza MB, et al. Imaging of pregnant and lactating patients: part 2, evidence-based review and recommendations. AJR Am J Roentgenol. 2012;198(4):785–92.
55. Soffer E, Kunze G, Conwell D, Yap E, Mekhail N. Chronic abdominal pain: the diagnostic value of a combined clinical and pain management approach. Gastroenterology. 1998;114 suppl 1:A42.
56. Gray AT. Atlas of ultrasound-guided regional anesthesia. 2nd ed. Philadelphia: Elsevier Saunders; 2013.
57. Wang PI, Chong ST, Kielar AZ, Kelly AM, Knoepp UD, Mazza MB, et al. Imaging of pregnant and lactating patients: part 1, evidence-based review and recommendations. AJR Am J Roentgenol. 2012;198(4):778–84.
58. de Jose MB, Gotzens V, Mabrok M. Ultrasound-guided umbilical nerve block in children: a brief description of a new approach. Paediatr Anaesth. 2007;17(1):44–50.
59. Mehta M, Ranger I. Persistent abdominal pain. Treatment by nerve block. Anaesthesia. 1971;26(3):330–3.
60. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
61. Batistaki C, Saranteas T, Adoni A, Kostopanagiotou G. Ultrasound-guided anterior abdominal cutaneous nerve block for the management of bilateral abdominal cutaneous nerve entrapment syndrome (ACNES). Pain Physician. 2013;16(6):E799–801.
62. El-Dawlatly AA, Turkistani A, Kettner SC, Machata AM, Delvi MB, Thallaj A, et al. Ultrasound-guided transversus abdominis plane block: description of a new technique and comparison with conventional systemic analgesia during laparoscopic cholecystectomy. Br J Anaesth. 2009;102(6):763–7.
63. Guirguis MN, Abd-Elsayed AA, Girgis G, Soliman LM. Ultrasound-guided transversus abdominis plane catheter for chronic abdominal pain. Pain Pract. 2013;13(3):235–8.
64. Azar FM, Lake JE, Grace SP, Perkinson B. Ethyl chloride improves antiseptic effect of betadine skin preparation for office procedures. J Surg Orthop Adv. 2012;21(2):84–7.
65. Richards RN. Ethyl chloride spray for sensory relief for botulinum toxin injections of the hands and feet. J Cutan Med Surg. 2009;13(5):253–6.
66. Birthi P, Calhoun D, Grider JS. Pulsed radiofrequency for chronic abdominal pain. Pain Physician. 2013;16(4):E443–5.
67. Doouss TW, Boas RA. The abdominal cutaneous nerve entrapment syndrome. N Z Med J. 1975;81(540):473–5.
68. Rosendal F, Moir L, de Pennington N, Green AL, Aziz TZ. Successful treatment of testicular pain with peripheral nerve stimulation of the cutaneous branch of the ilioinguinal and genital branch of the genitofemoral nerves. Neuromodulation. 2013;16(2):121–4.
69. Cairns KD, McRoberts WP, Deer T. Peripheral nerve stimulation for the treatment of truncal pain. Prog Neurol Surg. 2011;24:58–69.
70. Paicius RM, Bernstein CA, Lempert-Cohen C. Peripheral nerve field stimulation in chronic abdominal pain. Pain Physician. 2006;9(3):261–6.

Lumbar Plexus Entrapment: Abdominal

43

Susan R. Anderson-Jones and Tiffany Zhang

Introduction

The lumbosacral plexus provides the nerve supplies to lower back, abdomen, pelvis, and lower extremities. As it travels from the lower spine to the lateral pelvic walls, there are characteristic areas for compression and entrapment. These compressions may manifest clinically with predictable symptoms, specific to the nerve or nerves involved. However, more often, they present as vague and poorly localized symptoms that make it difficult to distinguish from more common causes of pain. These plexopathies can be under-recognized and underdiagnosed. They may be mistaken for more proximal lesions, such as compression from a disk within the canal or foramen. The pain may be located in the low back, pelvic, sciatic, and any part of lower extremities. Careful history taking, physical examination, and relevant diagnostic studies, such as electrophysiological techniques and imaging studies, are required for a correct diagnosis. Lumbar and sacral plexus entrapments are described in the abdominal (this chapter), pelvic (Chap. 49), and lower extremity (Chap. 66) plexus chapters, with specific regional emphasis in each respective chapter, although overlapping information is inevitable.

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Clinical Presentation (Tables 43.1, 43.2, and 43.3)

The clinical picture of lumbosacral plexus entrapment varies and depends on the location and degree of involvement. Patients may present with significant and debilitating pain, proximal muscle weakness and wasting, sensory deficits, and loss of deep tendon reflexes. Bowel and bladder incontinence may occur, though relatively rare, and clinically may mimic *cauda equina syndrome*.

In addition to nonspecific abdomen (Fig. 43.1), low back, or pelvic pain, patients with lumbosacral plexus pathology often present with asymmetric motor deficits (weakness), sensory changes (decreased sensation, numbness, dysesthesia,

Table 43.1 Occupation/exercise/trauma history relevant to lumbosacral plexus entrapment

Mechanical compression (cysts, aneurysms, tumor, hematoma, etc.)	Fallopian and ovarian cysts [1]
	Intra-abdominal or pelvic aneurysm [2]
	Lymphoma or enlarged lymph node [3]
	Neurofibromatosis [4]
	Retroperitoneal or psoas hematoma or abscess [5–8]
Tumor compression or invasion	Malignant psoas syndrome
	Adjacent tumor (colorectal, ovarian, uterine, or cervical) [9, 10]
	Metastatic tumor (breast, sarcoma, lymphoma, multiple myeloma) [11]
Trauma (plexus traction)	Pelvic or sacral fracture [12–14]
Intraoperative compression or traction	From intraoperative patient positioning or instruments leading to stretching or compressing the plexus [15, 16]
Infection	Psoas abscess [5]
Endometriosis	Pelvic endometriosis [17]
Pregnancy and/or delivery	3rd trimester pregnancy
	Labor/delivery when lumbosacral trunk is compressed against less cushioned pelvic rim [18, 19]

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Table 43.2 Lumbar plexopathy clinic presentations

	Ilioinguinal/iliohypogastric neuropathy	GFN neuropathy	Femoral neuropathy	Obturator neuropathy	LFC neuropathy
Motor findings	Possible weakness of abdominal wall	Cremasteric	Weakness of the quadriceps and iliopsoas	Weakness of hip adductors	None
Sensory findings	Deficits in the lower abdomen and groin	Deficits in the lower abdomen and groin	Deficits in the anterior and medial thigh and anteromedial leg	Deficits in the upper medial thigh	Deficits in the anterolateral thigh

GFN genitofemoral neuralgia, *LFC* lateral femoral cutaneous neuralgia

Table 43.3 Sacral plexopathy clinic presentations

	Sciatic nerve	Posterior cutaneous nerve of the thigh	Superior and inferior gluteal nerve	Pudendal nerve
Motor findings	Weakness of hamstrings; dorsal and plantar flexion of ankle and toes	None	Weakness of gluteal muscles (maximus, medius, and minimus)	Weakness of anal and urethral sphincters, erectile dysfunction
Sensory changes	Posterior calf; sole and dorsum of foot	Lower buttock and posterior thigh	None	Lower anal canal and perineal skin

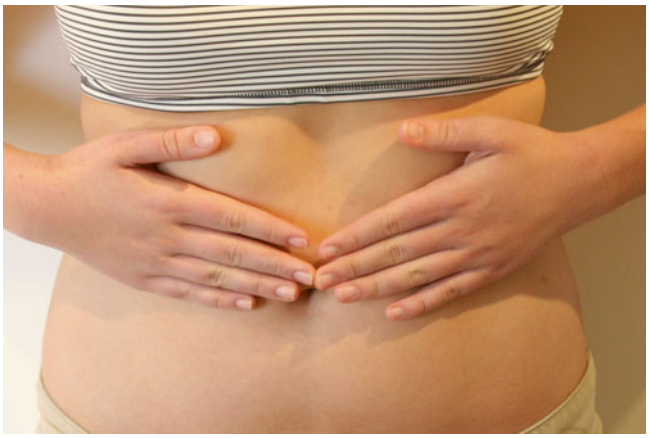

Fig. 43.1 Pattern of nonspecific abdominal pain due to lumbosacral plexus entrapment (Image courtesy of Andrea Trescot, MD)

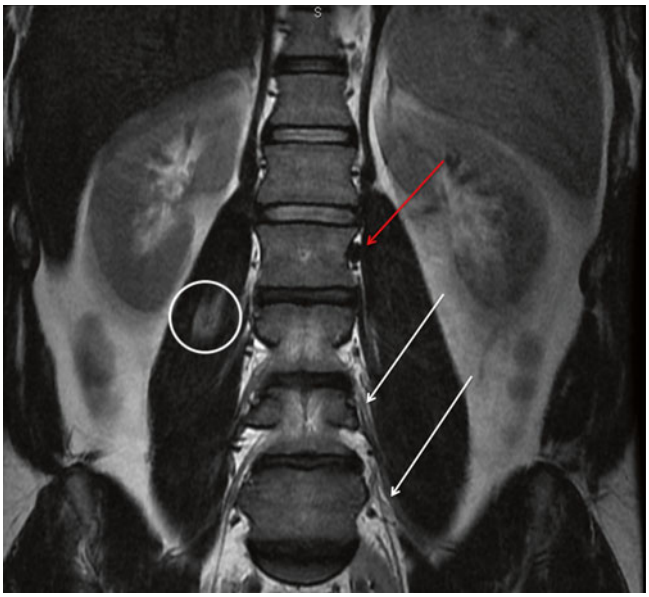

Fig. 43.2 MRI showing psoas lesion; *circle* shows lesion. *White arrows* show lumbar plexus; *red arrow* shows an enlarged blood vessel compressing the nerve root (Image courtesy of Andrea Trescot, MD)

and/or paresthesia), and pain distribution involving multiple consecutive nerve levels, which is a key element differentiating from more proximal spinal radiculopathy. Careful examination may help localizing the lesion within the plexus. The neurological findings are often related to the levels and peripheral nerves involved (Tables 43.2 and 43.3).

Femoral neuropathy (Chap. 57) causes weakness of the *quadriceps* and *iliopsoas major* muscles, with or without sensory deficits over the anterior and medial thigh and anteromedial leg, whereas *obturator neuropathy* (Chap. 48) causes weakness in the hip adductors and sensory changes in the upper medial thigh [20, 21]. *Lateral femoral cutaneous nerve* (Chap. 61) entrapment causes pain and sensory changes in the anterolateral thigh; *genitofemoral* nerve entrapment (Chap. 45) causes symptoms in the *scrotum* or *mons* and *labia majora*. Entrapment of the *ilioinguinal* or *iliohypogastric nerves* (Chap. 40) manifests with burning pain and sensory changes in the lower abdomen, groin, upper medial thigh, or pelvic area [22]. *Sacral plexus entrapment* may present with debilitating pain in the lower back and buttocks, radiating down the legs. In some patients, the clinical picture may be further complicated by foot drop, sensory changes of the foot, a loss of tendon reflexes, and, rarely, bowel and bladder incontinence, as well as sexual dysfunction (Tables 43.2 and 43.3) [23–25].

Typically, nerve entrapment presents with unilateral symptoms, whereas bilateral symptoms are more likely to indicate a systemic process, such as *diabetes* or *neurofibromatosis* [23].

Table 43.4 Lumbar plexus anatomy

Origin	Anterior rami T12 to L5
General route	Passes anterior to transverse process from L2 to L5 within the psoas muscle. Exits at lateral psoas muscle. 6 nerves:
	Iliohypogastric (Chap. 40), ilioinguinal (Chap. 40), genitofemoral (Chap. 41), lateral femoral cutaneous (Chap. 60), femoral (Chap. 56), obturator (Chap. 63)
Sensory distribution	*Iliohypogastric/ilioinguinal nerves* (from L1) – suprapubic and inguinal regions
	Genitofemoral nerve (L1 and L2) – inguinal region
	Lateral femoral cutaneous nerve (L2 and L3) – lateral thigh region
	Femoral nerve (L2–L4) – anterior and medial thigh, medial leg distal to the knee
	Obturator nerve (L2–L4) – medial leg proximal to the knee
Motor innervation	*Genitofemoral nerve* (L1 and L2) – cremaster muscle
	Femoral nerve (L2–L4) – rectus femoris, vastus medialis, vastus intermedius, and vastus lateralis muscles of the thigh
	Obturator nerve (L2–L4) – adductor thigh muscles

Table 43.5 Sacral plexus anatomy

Origin	Anterior rami of L4 to L5, S1 to S4
General route	Located on the posterolateral wall of the lesser pelvis, where it is closely related to the anterior surface of the piriformis. Most branches leave the pelvis through the greater sciatic foramen
	Two main nerves: sciatic (Chap. 55) and pudendal (Chap. 47) nerves, plus posterior femoral cutaneous nerve of the thigh (Chap. 56) and superior gluteal (Chap. 53) and inferior gluteal (Chap. 54) nerves
Sensory distribution	*Sciatic nerve* (L4–S3) – the posterior calf, sole and dorsum of the foot
	Posterior femoral cutaneous nerve of the thigh (S1–S3) – the posterior thigh and lower posterior buttock
	Pudendal nerve (S2–S4) – the lower anal canal and perineal skin
Motor innervation	*Sciatic nerve* – hamstrings, dorsi, and plantar flexors of the ankle and toes; intrinsic foot muscles
	Superior and inferior gluteal nerves (L5–S2) – gluteus maximus, medius and minimus, tensor fasciae latae muscles
	Pudendal nerve – sphincters (external anal and urethral), erectile vessels
Anatomic variability	The sacral plexus may arise higher or lower

Onset of entrapment syndrome can be acute (e.g., hematoma, trauma), subacute, or insidious (e.g., tumor). Careful history taking may help define the underlying pathology. Clinical assessment is focused to identify the pattern of motor and sensory changes. Associated pain and sensation changes are also important clues in the lumbosacral plexopathies. If the weakness seems to be confined to the plexus distribution, but the sensory loss seems to follow a more dermatomal distribution, this may raise the concern for a primary root involvement (i.e., radiculopathy); other associated systemic features should be considered too, such as weight loss, night sweats, or history of malignancy, which might support neoplastic infiltration or a paraneoplastic cause. A rash associated with the neuropathic symptoms may indicate vasculitis. The majority of psoas abscesses are usually secondary to local gastrointestinal, urinary, or spinal infection [26]. If symptoms occur in an anticoagulant patient, or shortly after an invasive procedure, evidence of bleeding disorders or hematoma should be immediately sought after [27].

Psoas muscle disease, whether related to surgery, trauma, hematoma, abscess, or tumor infiltration (*neoplastic plexopathy*) (Fig. 43.2), can directly compress the lumbar plexus. Painful neoplastic plexopathy due to tumor infiltration of the psoas muscle is known as *malignant psoas syndrome*, most commonly following direct invasion of the psoas muscle by adjacent tumors of colorectal, ovarian, uterine, or cervical origin, though they can also be the result of metastatic cancer. Direct invasion of the lumbosacral plexus from perineural tumors is less common [17, 21, 22]. *Malignant psoas syndrome* can mimic *cauda equina syndrome*, though it typically involves a *proximal lumbosacral plexopathy* (L1–4).

Endometriosis is an infrequent cause of lumbosacral plexopathy; there are only a few case reports in the literature. Patients may present with cyclic symptoms, or symptoms associated with menses (*catamenial*) [12].

Anatomy (Tables 43.4 and 43.5)

The lumbosacral plexus lies within the psoas muscle in front of the lumbar transverse process and may be divided functionally and anatomically into a lumbar and sacral plexus (Fig. 43.3). The *lumbar plexus* is formed from the anterior rami of the T12–L5 nerve roots. The *sacral plexus* is formed from the anterior rami of the L4–L5 and S1–S4 nerve roots [28]. The lumbar nerve roots are located in the posterior portion of the psoas muscle; they are arranged from medial at L5

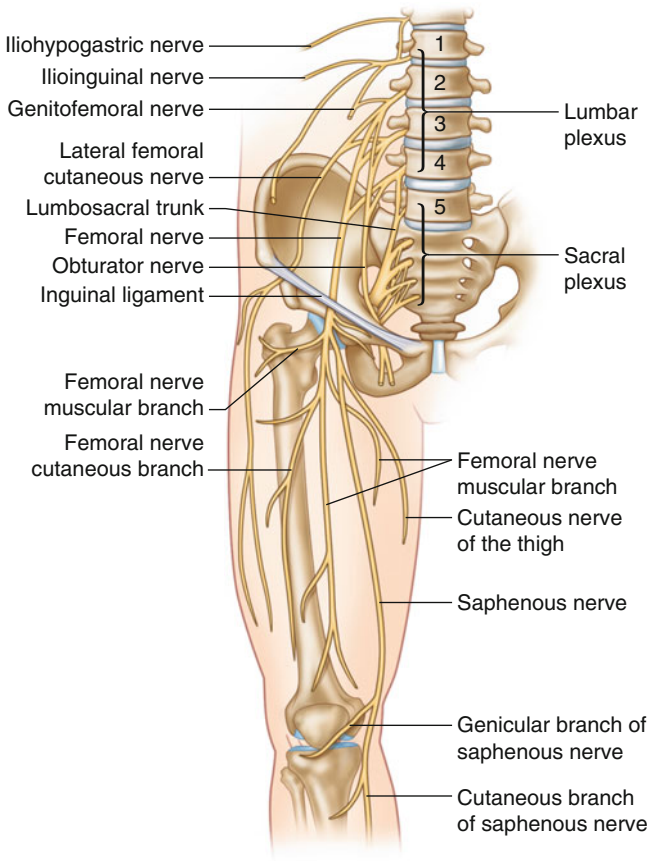

Fig. 43.3 Anatomy of the lumbosacral plexus (Image by Springer)

to lateral at L2 and from ventral at L2 to dorsal at L5 [29]. The roots then pass anterior to the transverse processes from L2 to L5 (ventral rami of L1–L4) and lie within the psoas major muscle (Figs. 43.4 and 43.5). The lumbar plexus exits at the lateral border of the psoas muscle (Fig. 43.6) and consists of six nerves – *iliohypogastric*, *ilioinguinal*, *genitofemoral*, *lateral femoral cutaneous*, *femoral*, and *obturator*.

The *iliohypogastric* and *ilioinguinal* nerves (see Chap. 40) are primarily sensory nerves from L1 and supply innervation to the skin of the suprapubic and inguinal regions. The *genitofemoral* nerve (see Chap. 41) arises from L1 and L2 to supply motor innervation to the cremaster muscle and additional sensory innervation to the inguinal area. The *lateral femoral cutaneous nerve* (see Chap. 60) is a sensory nerve from L2 and L3 and provides sensation to the lateral aspect of the thigh.

The *femoral nerve* (see Chap. 56) (L2–L4) is the major motor nerve of the thigh. It emerges from the lateral psoas border before coursing along the groove between the iliacus and psoas muscles, lateral to the external iliac artery before reaching the thigh. The femoral nerve innervates the knee extensors (*rectus femoris*, *vastus medialis*, *vastus intermedius*, and *vastus lateralis*). It also provides sensory innervation to much of the anterior and medial thigh, as well as the medial portion of the leg. The *obturator nerve* (see Chap. 63) (L2–L4) emerges from the medial border of the psoas and provides sensory innervation to a portion of the medial lower thigh, as well as motor innervation to the adductor muscles of the thigh.

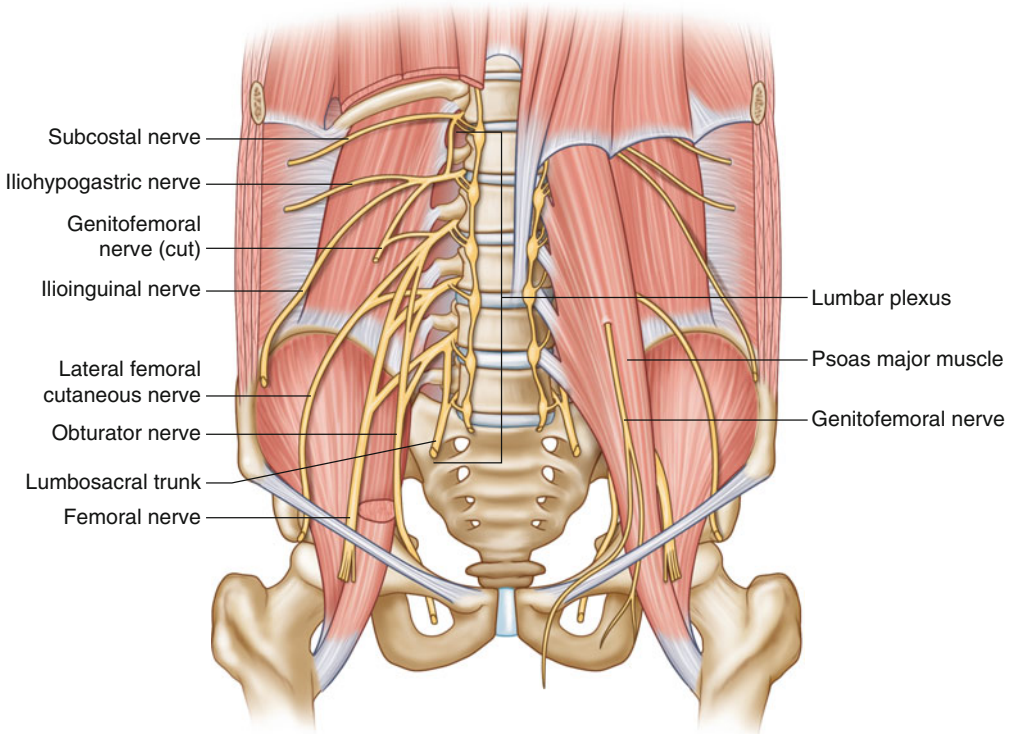

Fig 43.4 Anatomy of the psoas muscle and lumbar plexus (Image by Springer)

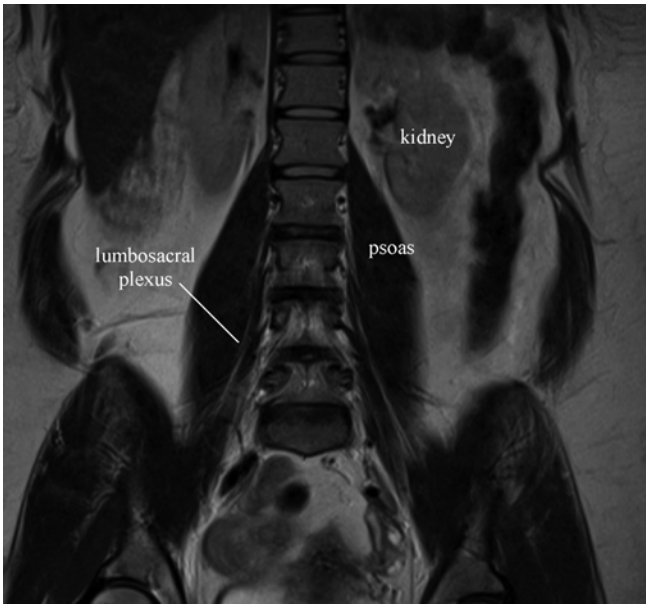

Fig. 43.5 MRI sagittal image of the lumbosacral plexus (Image courtesy of Andrea Trescot, MD)

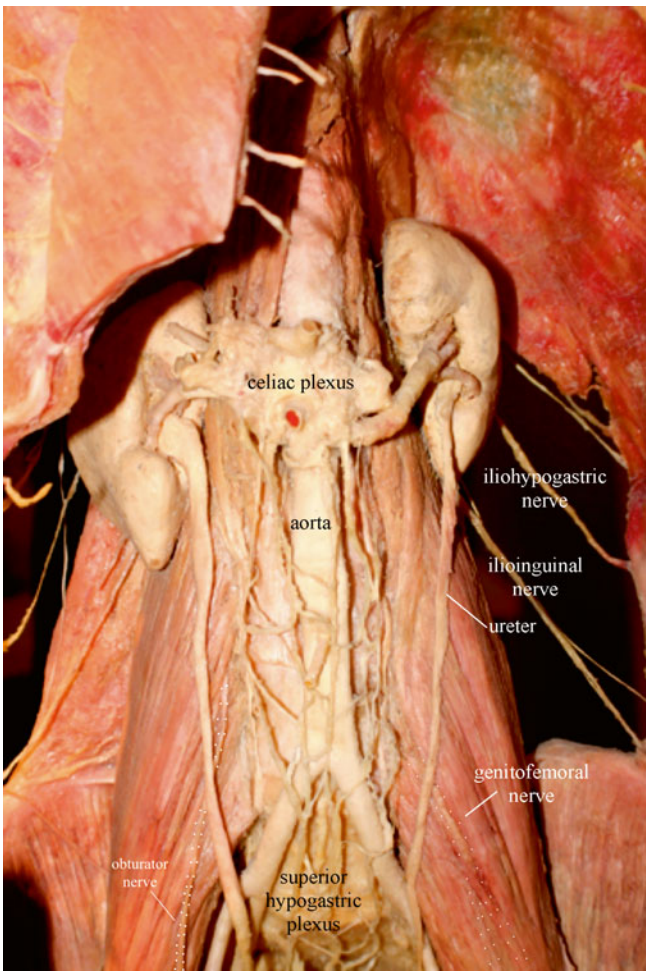

Fig. 43.6 Dissection of the lumbosacral plexus, from *Bodies, The Exhibition*, with permission (Image courtesy of Andrea Trescot, MD)

The lumbosacral trunk joins with the upper sacral roots anterior to the piriformis, behind the internal iliac vessels. It passes through the *greater sciatic foramen* deep to the *piriformis muscle* and continues as the *sciatic nerve* (see Chap. 64). The sacral plexus also gives off *superior gluteal nerve* and *inferior gluteal nerve*, the *posterior cutaneous nerve of the thigh*, and the *pudendal nerve*. The *superior gluteal nerve* (see Chap. 52) exits the *greater sciatic foramen* above the *piriformis*, while *inferior gluteal nerve* (see Chap. 53) leaves the greater sciatic foramen below the piriformis; both then enter the gluteus muscles (maximus, medius, and minimus). The *posterior femoral cutaneous nerve of the thigh* (see Chap. 62) also leaves the pelvis through the greater sciatic foramen inferior to piriformis, supplying the skin of buttock and posterior thigh. Finally, the *pudendal nerve* (see Chap. 47) passes out of the greater sciatic foramen close to the ischial spine and then reenters the pelvis through the *lesser sciatic foramen*, terminating in the perineum [28, 30].

Entrapment

Lumbosacral plexus entrapment may occur in various intra-abdominal or pelvic conditions that produce mechanical mass effect, or it may be related to trauma, surgery, or even systemic disease (Table 43.1). Aneurysmal dilatation of the distal aorta, iliac arteries, and their branch vessels has been implicated in compressing the lumbosacral plexus (Fig. 43.5). This partly explains the low back pain experienced by patients with aneurysms, where there is compression of the iliohypogastric, ilioinguinal, or obturator nerves. Aneurysms of the intrapelvic iliac or hypogastric arteries may also compress the lumbosacral plexus [2]. Such patients may present with abrupt sciatic pain and impaired straight leg raising, initially suggestive of a prolapsed intervertebral disk. Similarly, although rare, sciatic artery aneurysms or arteriovenous malformations have been reported to cause direct lumbosacral plexus compression. Psoas muscle abscess or hematoma, retroperitoneal hematoma, and lymphoma have all been implicated in compressing the lumbosacral plexus.

Trauma causing pelvic or sacral fracture may cause a lumbosacral plexus traction injury [14, 24]. The lumbosacral plexus may be directly compressed at the pelvic brim during pregnancy and delivery, causing postpartum foot drop [18, 19]. Entrapment can also occur after prolonged supine position, direct nerve compression, or damage during surgery [15, 16].

Lumbar and sacral plexopathies can be induced by extrinsic compression of a mass (such as tumor) or diffuse infiltration by inflammatory processes [10, 30]. Retroperitoneal processes are more likely to affect the lumbar plexus. Pelvic disease is more likely to affect the sacral plexus.

Physical Exam

There is wide array of signs and symptoms associated with lumbosacral plexus entrapment syndrome, often related to the cause and location of the compression or entrapment. The signs and symptoms can be vague and nonspecific; however, it often presents with asymmetric and focal weakness, as well as sensory changes involving multiple lumbosacral nerve levels. A thorough neurological exam often helps in localizing the nerve injury; a careful sensory and motor function assessment may identify the individual spinal level or nerve that is involved or injured. Lumbar plexopathy tends to cause weakness of hip flexion and adduction and/or knee extension, whereas sacral plexopathy often leads to weakness of knee flexion or foot drop. Lumbar plexus entrapment may affect anteromedial thigh and leg sensation, whereas the sacral plexus collectively provides sensation to posterior thigh and leg, as well most of the foot.

Refer to Tables 43.2 and 43.3 for specific patterns of the lumbosacral plexus and its branches. Because contraction of the psoas muscle will increase compression of the plexus, resistance against hip flexion (Fig. 43.7) may provoke or exacerbate the symptoms. Pain from distal tendonitis of the psoas at its attachment onto the lesser trochanter (Fig. 43.8) may also worsen with this maneuver.

Fig. 43.7 Physical exam of resistance to hip flexion (Image courtesy of Andrea Trescot, MD)

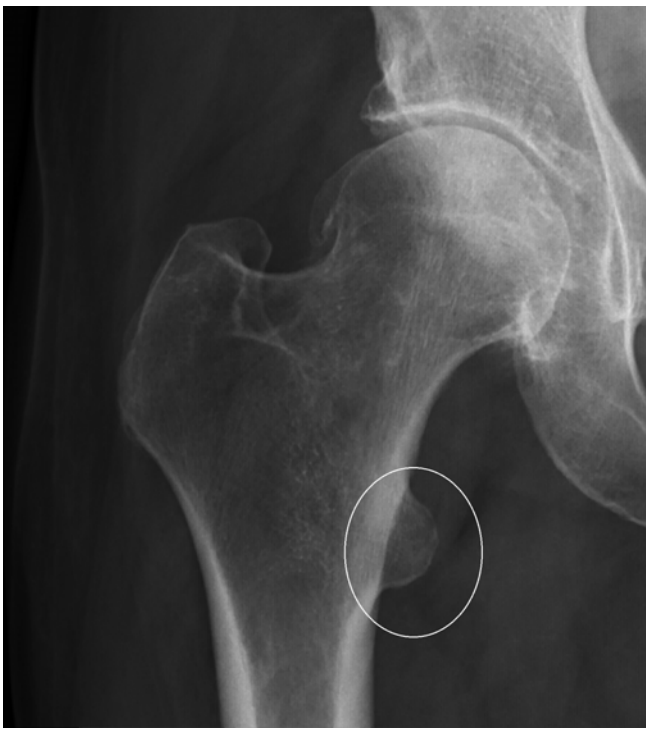

Fig. 43.8 X-ray showing the lesser trochanter, site of distal attachment of the psoas muscle (Image courtesy of Andrea Trescot, MD)

Differential Diagnosis (Tables 43.6 and 43.7)

Because the causes and presentation of lumbosacral plexus entrapment can be vast and vague, the differential diagnosis can be extensive. Table 43.6 lists some of common conditions that should be considered or ruled out during the workup, and Table 43.7 lists some of the diagnostic tests.

It is important to include lumbosacral plexopathy in the differential diagnosis of patients presenting with nonspecific abdominal, pelvic, and back pain, especially when combined with lower extremity pain and asymmetric motor and/or sensory changes of lower extremities [30]. However, certain conditions may cause bilateral symptoms, such as diabetic neuropathy or pelvic radiation for cancer treatment [23, 31]. Pertinent history and comorbidities may give a clue as to the etiology (e.g., history of trauma, diabetes, cancer, etc.). Imaging, mainly MRI, and electrodiagnostic testing (EMG) are valuable in detecting the lesions or localizing the nerve involvement [10, 32]. It should, however, also be stressed that many conditions may not be obvious on imaging or EMG [30].

Electrodiagnostic Testing

Electrodiagnostic studies with nerve conduction studies and needle *electromyography* (EMG) are very helpful for diagnosing and localizing the nerve injury and underlying

Table 43.6 Differential diagnosis of pertinent abdominal pain

	Potential distinguishing features
Neuropathy after radiation therapy	Insidious bilateral leg weakness, history of radiation therapy [31]
Diabetic peripheral neuropathy	History of diabetes, other symptoms and signs of diabetes and diabetic peripheral neuropathy (DPN)
Tumor compression or invasion	History of malignancy, especially pelvic or abdominal malignancies MRI may show tumor invasion [10]
Lumbosacral radiculopathy	Sensory and motor symptoms follow single nerve root; NCV/EMG may be diagnostic
Caudal equina syndrome	Urinary and/or bowel incontinence, saddle anesthesia, often acute onset
Lumbar facet pathology	Paravertebral tenderness, lumbar spondylosis
Hematoma	Anticoagulant therapy, recent invasive procedure
Systemic inflammatory disease	Widespread and symmetrical symptoms and signs
Abscess	GI, pelvic, or abdominal infection

Table 43.7 Diagnostic tests for lumbar plexus entrapment

	Potential distinguishing features
Physical exam	Sensory and motor changes often involve overlapping contiguous spinal levels
Diagnostic injection	Psoas plexus block
Ultrasound	Less value in diagnosis due to the deep location of lumbosacral plexus
MRI	Valuable for detailed nerve, psoas muscle, and surrounding lesions. Increased T2 intensity of the nerves; may enhance with contrast (infection, tumor invasion, inflammatory)
X-ray	Rule out other bony causes
Electrodiagnostic studies	EMG abnormalities in at least 2 different peripheral nerves in at least 2 nerve roots with no paravertebral abnormalities

process. The diagnosis of lumbosacral plexopathy is indicated when electrophysiologic abnormalities are present in the distribution of at least two different peripheral nerves in at least two different nerve root distributions. EMG may be the most important component of the electrodiagnostic evaluation of lumbosacral plexopathies, both for localization as well as determination of the severity of the disease. In this case, a good examination and history to decide which muscles to test is a critical step [32]. Sparing of the paraspinal muscles on needle examination is helpful in determining a pure lumbosacral plexopathy; the sensory studies are likely to be most helpful for localization of the injury, because it helps differentiating a preganglionic process (i.e., radiculopathy) from a postganglionic process (i.e., plexopathy or mononeuropathy). Reduced *sensory nerve action potential* (SNP) amplitudes imply a postganglionic process and can help to exclude a radiculopathy [32].

The femoral nerve is the only motor nerve conduction study likely to give meaningful information about the possibility of a pure lumbar plexopathy (vs. lumbosacral). The most common method is to stimulate the nerve in the inguinal region and record from a quadriceps muscle [32]. This method may be contraindicated in patients on anticoagulation, given the proximity to the femoral artery [33].

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is a valuable diagnostic tool in the evaluation of lumbosacral plexopathy, because it provides detailed anatomic information about the nerves and surrounding soft tissues. The plexus nerves can be seen as fascicular structures surrounded by fat within or posterior to psoas muscles (Fig. 43.5). Terminal branches of the lumbar plexus are seen at the lateral border of the psoas muscle (iliohypogastric, ilioinguinal, genitofemoral, lateral femoral cutaneous, and femoral nerves). The value of MRI also lies on identifying causes or origins of the entrapment or compression, for which axial images are often the most useful imaging plane. MRI may show injured nerves as structures with increased signal intensity on T2-weighted images, though this is a nonspecific response [21]. If the perineurial blood-nerve barrier is disrupted, there may also be neural enhancement with gadolinium contrast, though the sensitivity and necessity of gadolinium contrast have not been defined. Other valuable MRI findings of neural injuries include size and morphological changes, such as neuronal enlargement, loss of the normal fascicular appearance, or blurring of the perifascicular fat [21]. These findings are more easily appreciated in the larger nerves. Intra-psoas lesions may also cause psoas spasms, causing lumbar plexus entrapment (Fig. 43.3).

Identification and Treatment of Contributing Factors

The anatomical restriction of the lumbosacral plexus makes it susceptible to entrapment by variety of conditions. Any occupying lesion intrinsic or external of the psoas muscle may cause lumbar plexus entrapment [34], including retroperitoneal processes [23] and muscle invasion [35]. Its

proximity to bones and major blood vessels, such as the abdominal aorta, the iliac artery, and their branches, also makes it prone to compression from aneurysms of those arteries or metastatic lesions. The sacral plexus trunk passes over the sacral ala and lies over the posterior lateral wall of the pelvis and is therefore easily injured from sacral and pelvic trauma or during delivery.

Leg-length discrepancies, including improper use of a heel lift, might irritate the psoas muscle causing muscle spasm, therefore leading to lumbar plexus entrapment. Proper correction of the leg length and hip level, or osteopath manipulation, may prevent recurrent lumbar plexopathy [36].

Injection Techniques

The lumbar plexus block is most frequently used for surgical anesthesia of the lower extremity. It is occasionally used for the treatment of inflammatory conditions of the lumbar plexus such as *idiopathic lumbosacral plexitis* or when a tumor has invaded the tissues innervated by the lumbar plexus or the plexus itself [35, 37]. It can be utilized as a diagnostic maneuver when performing a differential neural blockade for evaluation of groin and lower extremity pain. It may be used palliatively for acute pain emergencies, including groin and lower extremity trauma or fracture, acute herpes zoster, and cancer pain, including tumor invasion while waiting for pharmacologic, surgical, and anticancer therapies to become effective [38].

Landmark-Guided Technique

The patient is placed in the lateral decubitus position, with the painful or operative side up and a slight forward tilt. The foot on the side to be blocked is positioned over the dependent leg so that twitches of the quadriceps muscle can be easily seen. Although several needle insertion sites have been suggested, locating the transverse process of the lumbar vertebral body with the needle tip after insertion is common to all techniques. The two surface anatomic landmarks of importance for determining the insertion point are the iliac crest and the midline spinous processes. The top of the iliac crest correlates with the body of the L4 vertebral body or the L4–L5 interspace in most patients. Draw a line between the iliac crests (*Tuffier's line*) and then a vertical line marking the midline (Fig. 43.9). A second vertical line is made 5 cm lateral to the midline parasagittally on the side to be anesthetized. On this second line, a mark is made 3 cm caudal to Tuffier's line, which identifies the needle entry point [39]. A 20- or 22-gauge, 15-cm needle is then inserted to the depth of the transverse process of the fifth lumbar vertebra. Once the transverse process is located, the needle is partially withdrawn and redirected cephalad until it slides past the transverse process. Attach a 5 cc saline-filled syringe to the needle and slowly advance it until loss of resistance is achieved. The needle tip is then within the psoas compartment. The loss of resistance typically occurs at a depth of 12 ± 2 cm. This is followed by 30 cc of local anesthetic for a surgical block or 5 cc of local anesthetic for a diagnostic injection. Keep the patient in the lateral position for 5 min following completion of the local anesthetic injection.

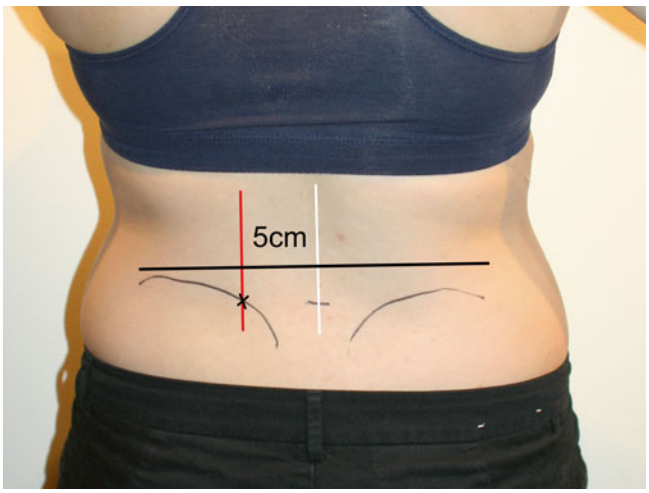

Fig. 43.9 Landmark-guided psoas injection site. *Black line* top off iliac crests (Tuffier's line); *white line* midline; *red line* paravertebral line 5 cm lateral of midline; *X* injection site (Image courtesy of Andrea Trescot, MD)

Using a Nerve Stimulator

A nerve stimulator is set to an initial current of 1.5 mA. The needle is advanced at an angle perpendicular to all skin planes. As the needle is advanced, local twitches of the paravertebral muscles are first obtained. As the needle is further advanced, the transverse process may be encountered. Contact with the transverse process is not routinely sought but, when present, it provides a consistent landmark to avoid excessive needle penetration during the lumbar plexus block [40]; the distance from the skin to the lumbar plexus ranges from 6.1 to 10.1 cm in men and 5.7–9.3 cm in women [40], with the distance correlating to gender and body mass index (BMI). The distance from the transverse process to the lumbar plexus is usually less than 2 cm, independent of BMI or gender. Contraction of the quadriceps muscle is usually obtained at a depth of 6–8 cm. The nerve stimulator current is then reduced to between 0.5 and 1.0 mA to produce stimulation of the quadriceps muscle, and 25–30 mL of local anesthetic is injected incrementally with negative aspiration every 5 cc [41]. It is important to avoid too deep a needle penetration and the resultant complications that may arise, such as renal hematoma and total spinal anesthesia [42].

Fluoroscopically Guided Technique

The patient is positioned prone, and the transverse processes at L3 or L4 are identified; the psoas shadow should be visible (Fig. 43.10). The injection site should correlate with the lateral aspect of the transverse process to avoid the nerve roots and the epidural space (Fig. 43.11). Using a 22-gauge, 5-in., B-bevel needle, insert the needle with a "gun-barrel" technique until the needle is approximately at the anterior one third of the vertebral body in the lateral view. One cubic centimeter of nonionic contrast is then injected, which should show the oblique flow of contract cephalad and caudad (Fig. 43.12). In the lateral view, the psoas major muscle spreads vertically over the anterior one third of the lumbar vertebral body when the contrast is injected. Note that it is always anterior to the foramina. After the correct needle placement is confirmed, 8–10 mL of a local anesthetic-steroid mixture (e.g., with 0.25 % bupivacaine or 0.2 % ropivacaine) is injected into the psoas muscle on one side. Pain relief should occur approximately 30 min after injection of the local anesthetic. On examination, pain should be gone on flexion and extension of the hip [43].

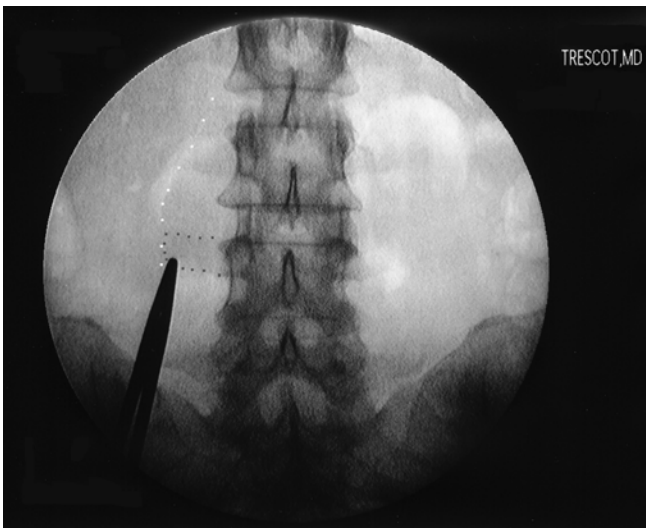

Fig. 43.10 Fluoroscopic image of the lumbar spine. Marker is on the transverse process of L4; note the distortion of the psoas shadow due to cancer mass (Image courtesy of Andrea Trescot, MD)

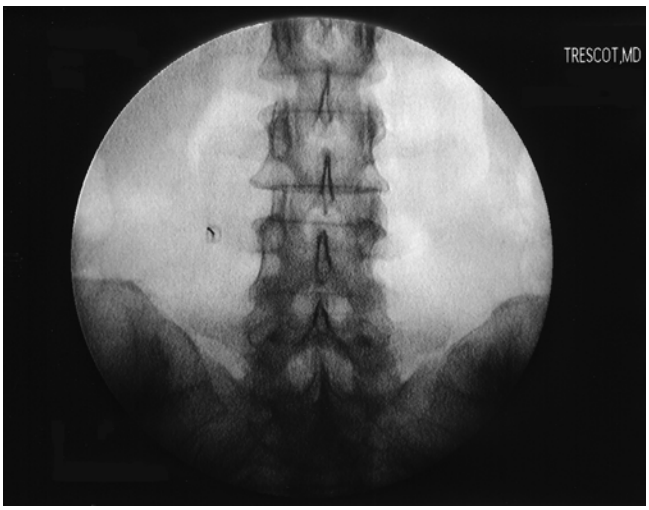

Fig. 43.11 Fluoroscopic psoas injection, needle on the transverse process of L4. *White line* represents psoas shadow (distorted by tumor); *brown line* represents the outline of the transverse process (Image courtesy of Andrea Trescot, MD)

Inguinal Perivascular Injection or Compartment Block (Three-in-One Block)

The *inguinal perivascular block* is based on the concept of injecting local anesthetic near the femoral nerve in an amount sufficient to track proximally along fascial planes to anesthetize the lumbar plexus [44]. The three principal nerves of the lumbar plexus pass from the pelvis anteriorly: the lateral femoral cutaneous, the femoral, and the obturator nerves. The theory behind this block presumes that the local anesthetic will track in the fascial plane between the iliacus and psoas muscles to reach the region of the lumbar plexus roots, so that the only anatomy one needs to visualize is the extension of sheathlike fascial planes that surround the femoral nerve. The patient should be placed supine on the operating table, with the anesthesiologist standing at the patient's side in position to palpate the ipsilateral femoral artery. After local anesthetic infiltration, a short-beveled, 22-gauge, 5-cm needle is inserted immediately lateral to the femoral artery, caudad to the inguinal ligament (Fig. 43.13) and advanced with cephalic angulation until a femoral paresthesia is obtained. At this point, the needle is firmly fixed, and, while the distal femoral sheath is digitally compressed, the entire volume of local anesthetic is injected.

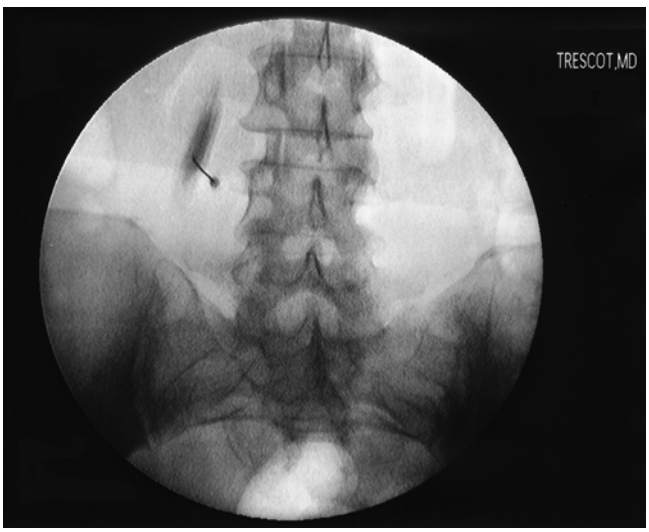

Fig. 43.12 Fluoroscopic psoas injection, contrast pattern (Image courtesy of Andrea Trescot, MD)

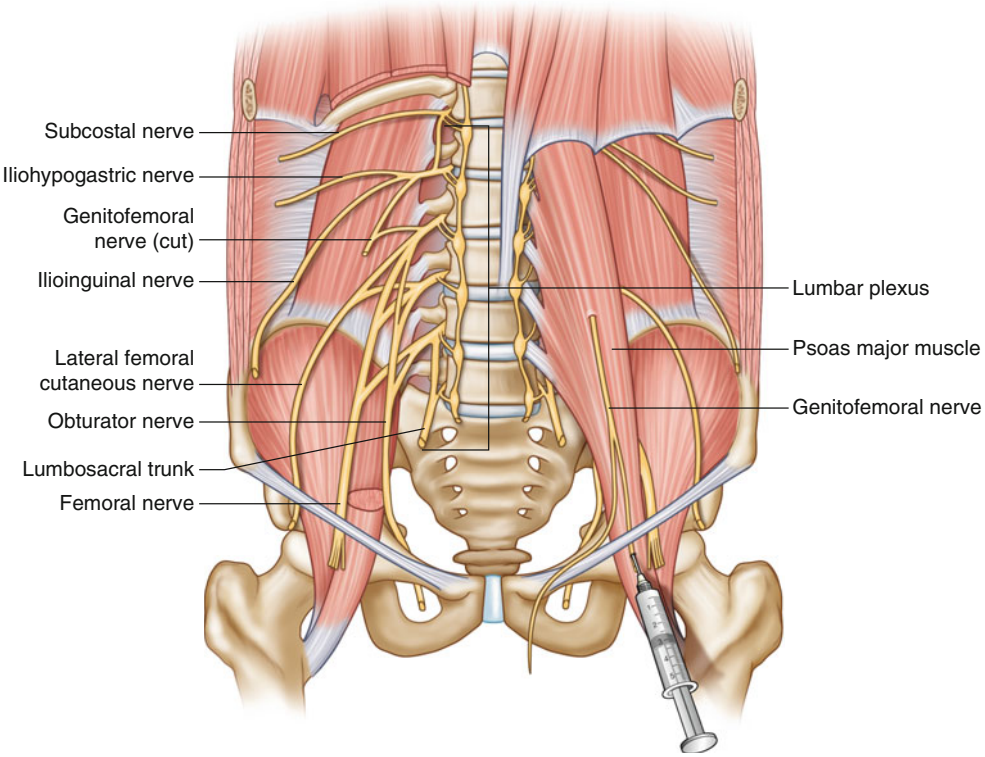

Fig. 43.13 Location for "3-in-one" lumbar plexus injection (Image courtesy of Springer)

Ultrasound-Guided Technique

The patient is placed in the lateral decubitus position so that contractions of the quadriceps muscle are visible. Identify the iliac crests and draw a line as in the landmark-guided technique. This time, however, the target is the paraspinal area at the level of L3/L4. Ultrasound is used to confirm the correct vertebral level and to guide the needle tip over the top of the transverse process. A low-frequency (2–5 MHz) curved array probe is used, placed in the paravertebral longitudinal position. Firm pressure is required to obtain good quality images. Identify the sacrum and then L5, and by counting cephalad, identify L3. Going from the midline and moving the probe laterally, the articular processes are seen, with the adjoining superior and inferior articular processes of the facets forming a continuous "sawtooth" hyperechoic line (Fig. 43.14). As the probe is moved further laterally, the transverse processes are seen with the psoas muscle lying between them. The image is of a "trident" (Fig. 43.15), with the transverse processes causing bony shadows and the psoas muscle lying in between. A color Doppler image is then obtained to identify adjacent vasculature, to avoid inadvertent intravascular injection.

At this point, the US probe is usually 3–5 cm off the midline. The lumbar plexus is not usually directly visualized, but lies within the posterior third of the psoas muscle (i.e., the closest third of the psoas muscle seen with the US probe). The distance from the skin to the psoas muscle can be measured using the caliper function of the ultrasound machine. This gives an estimate of the depth of the lumbar plexus

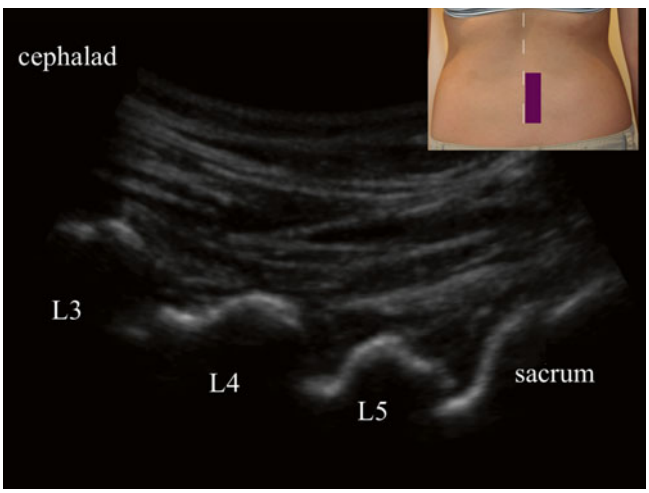

Fig. 43.14 Ultrasound image of a paramedian view of the sacrum, L5, L4, and L3 from a longitudinal approach, forming a "sawtooth" pattern (Image courtesy of Agnes Stogicza, MD)

before needle insertion. Note that anterior to the psoas muscle (further away from the skin in this US view) lie the peritoneal cavity, the great vessels, and the kidneys. Thus, care with needle tip placement should be maintained at all times.

In an alternative view, the lumbar plexus and psoas muscle can be viewed in the transverse plane. The L3 vertebral body is identified as above, in the longitudinal plane, and the probe is then rotated horizontally (Fig. 43.16) to visualize the midline spinous process and moved laterally (Fig. 43.17) to identify the psoas muscle and lumbar plexus underneath the transverse process.

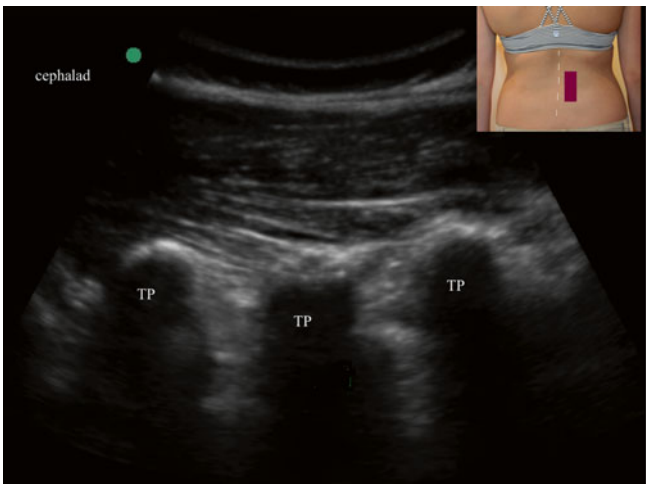

Fig. 43.15 Ultrasound image of the transverse processes from a longitudinal approach, forming a "trident" pattern. *TP* transverse process (Image courtesy of Agnes Stogicza, MD)

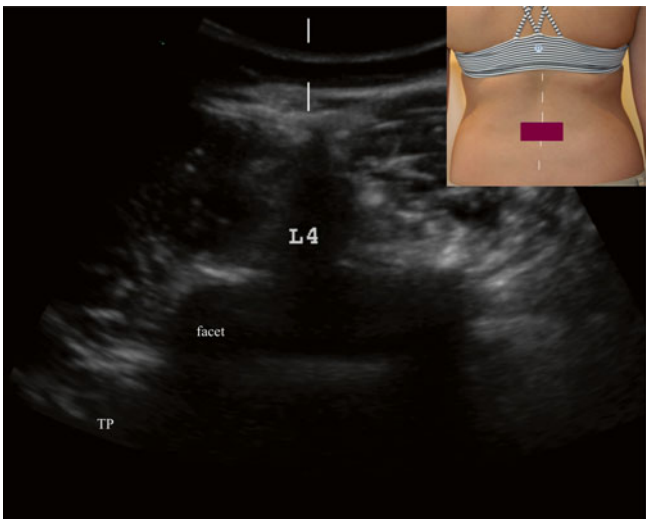

Fig. 43.16 Midline ultrasound visualization of the spinous process in a horizontal or transverse orientation (Image courtesy of Andrea Trescot, MD)

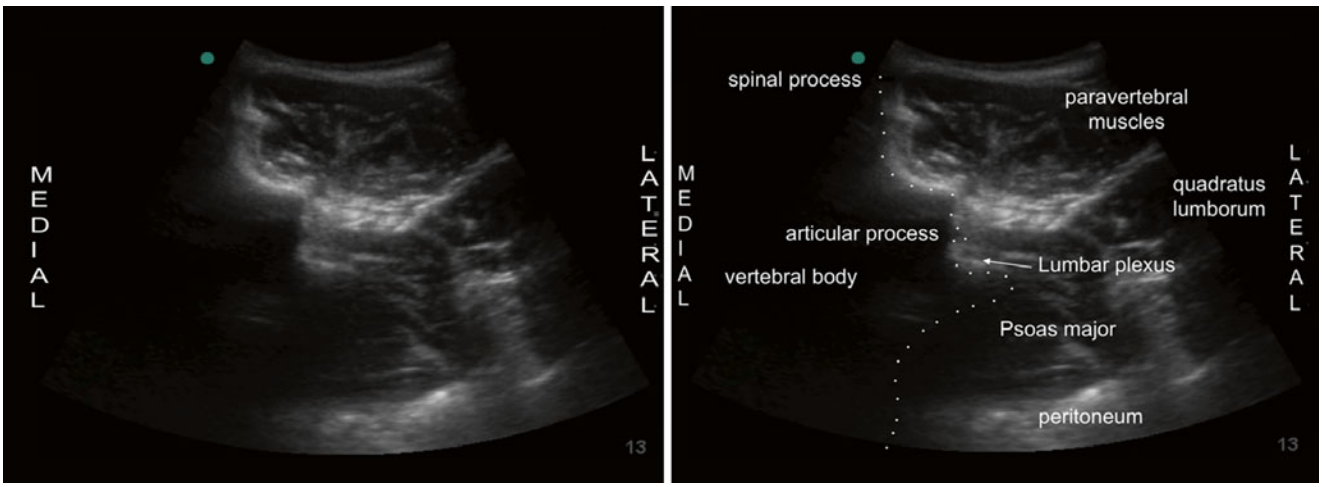

Fig. 43.17 Paramedian ultrasound visualization of the lumbar plexus (Image courtesy of Fernando Mauad, MD and modified by Charles Oliveira, MD)

The depth of the plexus is most often between 50 and 100 mm from the skin surface. An in-plane or out-of-plane technique may be used. If an in-plane approach is used with the longitudinal view, the usual direction for insertion is from caudad to cephalad. For the out-of-plane approach, the site for the block needle is on the medial side of the US probe (which is maintained in its longitudinal position) (Fig. 43.18). Advancing the needle from a medial to a lateral direction is also preferred to avoid insertion into the dural cuff, which can extend laterally beyond the neural foramina. A 13-cm stimulating needle may be utilized for specificity of location. The needle needs to be placed at the center of the probe, directed slightly laterally, such that its path comes directly under the US beam. Lidocaine is infiltrated into the skin and subcutaneous tissue at the point where the block needle is to be inserted. The needle is observed in real time and targeted toward the posterior third of the psoas muscle bulk. For the

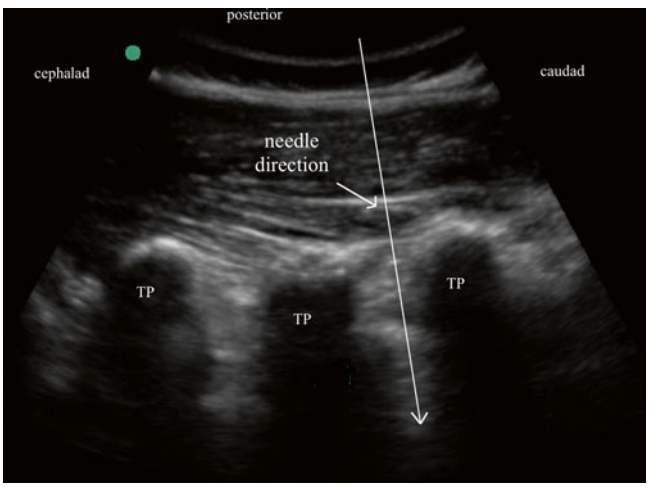

Fig. 43.18 Out-of-plane needle position for longitudinal lumbar plexus injection (Image courtesy of Agnes Stogicza, MD)

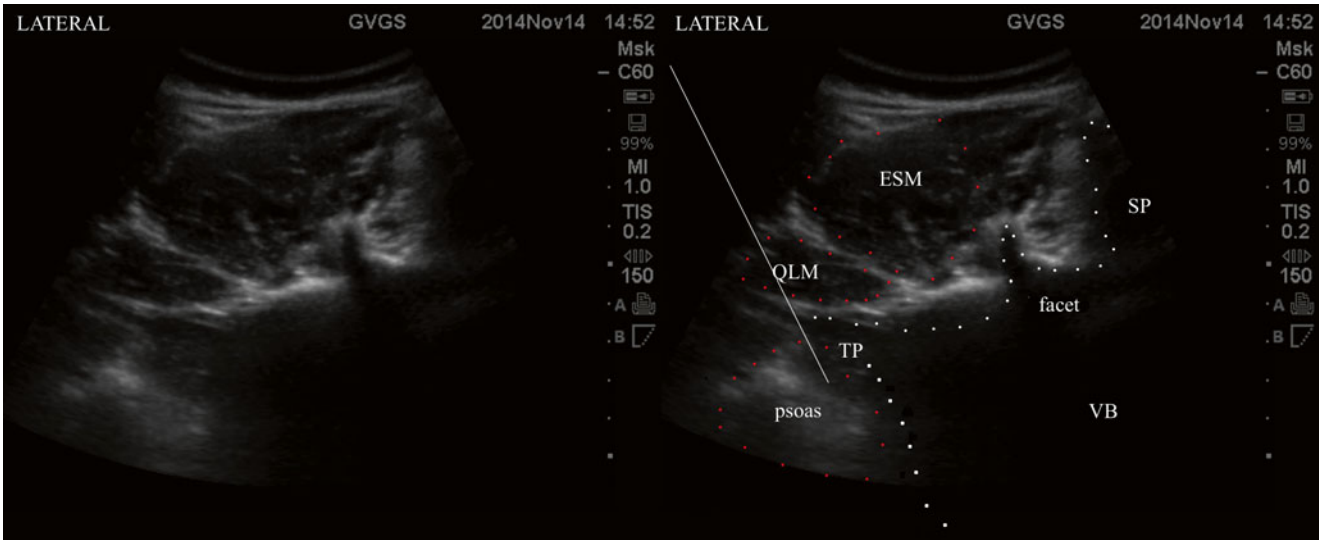

Fig. 43.19 In-plane horizontal lumbar plexus injection. *A* ultrasound image, *B* line drawing, *ESM* erector spinae muscle, *QLM* quadratus lumborum muscle, *SP* spinous process, *VB* vertebral body, *TP* transverse process (Image courtesy of Agnes Stogicza, MD, modified by Andrea Trescot, MD)

in-plane horizontal view, the needle is advanced lateral to medial under the edge of the transverse process (Fig. 43.19).

Electrical stimulation is commonly used to confirm proximity to the lumbar plexus. The target is to elicit quadriceps muscle contraction. In addition, contraction of the psoas muscle will be readily apparent under ultrasound imaging. When satisfied with the needle tip position, local anesthetic is injected incrementally (with frequent aspiration to monitor for blood or CSF), and its spread observed, looking for fluid and tissue expansion in the psoas muscle bulk [45]. The needle is removed, and pressure is placed on the injection site to avoid hematoma formation [38].

Neurolytic/Surgical Technique

Cryoneuroablation

Because the plexus is a large structure, it is not amenable to a precise technique like cryoneuroablation.

Radiofrequency Lesioning (RF)

There is no literature on using the RF technique on this entrapment, but the same issues as above are involved. The lumbar plexus is not amenable to a precise technique like radiofrequency.

Alcohol/Phenol

To provide longer-term relief, injections of neurolytic substances (6 % aqueous phenol or absolute alcohol) into the psoas sheath were described by Calava et al. [46] for a *malignant psoas syndrome* (metastatic lipoma) for whom a prior psoas compartment anesthetic block helped only temporarily. Depending on life expectancy, a lumbar plexus catheter could also be considered for continuous infusion of medication [47].

Surgery

Surgical dorsal rhizotomy may provide pain relief from lumbar plexopathy, especially in tumor patients with whom other conservative measures failed [13]. Surgical nerve repair and nerve grafting may lead to partial recovery of plexopathy from trauma or fractures [14].

Complications

The most serious complications associated with lumbar plexus block are related to the close proximity to the spinal cord and exiting nerve roots; the needle can cause trauma to the exiting lumbar nerve roots. If the needle is directed too dorsally and medially, there may be an inadvertent subarachnoid, subdural, and/or epidural injection. Epidural spread of local anesthetic has been reported to occur in up to 16 % of cases [48]. While inadvertent dural puncture is rare, unintentional dural or subdural injections can result in immediate total spinal anesthesia with associated loss of consciousness, hypotension, and apnea. This must be recognized immediately. Intravascular injections can lead to local anesthetic toxicity with loss of consciousness, hypotension, apnea, or cardiac arrest. This risk can be decreased using digital subtraction

(fluoroscopy) or color mode for Doppler (ultrasound). There can also be the risk of bleeding, which, because it may be retroperitoneal, can be difficult to recognize [49]. Patients may complain of a temporary pain exacerbation from the injection or postprocedural lumbar paravertebral muscle spasms.

Summary

Lumbar plexus entrapment can present in a myriad of ways, including abdominal pain, which makes diagnosis difficult if there is not a high index of suspicion. A careful history and physical exam, along with a diagnostic injection, can help to elucidate the cause.

References

1. Yeo PT, Grice AC. Lumbosacral nerve plexus compression by ovarian-fallopian cysts. Clin Exp Neurol. 1986;22:139–43.
2. Batchelor TJ, Vowden P. A persistent sciatic artery aneurysm with an associated internal iliac artery aneurysm. Eur J Vasc Endovasc Surg: Off J Eur Soc Vasc Surg. 2000;20(4):400–2.
3. Vinnicombe SJ, Norman AR, Nicolson V, Husband JE. Normal pelvic lymph nodes: evaluation with CT after bipedal lymphangiography. Radiology. 1995;194(2):349–55.
4. Tonsgard JH, Kwak SM, Short MP, Dachman AH. CT imaging in adults with neurofibromatosis-1: frequent asymptomatic plexiform lesions. Neurology. 1998;50(6):1755–60.
5. Griffiths EA, Kalbassi MR, Cunliffe WJ, Browell DA. Psoas abscess as a secondary complication of gastrointestinal disease: report of five cases. Case Rep Clin Pract Rev. 2003;4(2):73–6.
6. Ozcakar L, Sivri A, Aydinli M, Tavil Y. Lumbosacral plexopathy as the harbinger of a silent retroperitoneal hematoma. South Med J. 2003;96(1):109–10.
7. Ozkan OF, Guner A, Cekic AB, Turan T, Kaya U, Reis E. Iliopsoas haematoma: a rare complication of warfarin therapy. J Coll Physicians Surg Pak: JCPSP. 2012;22(10):673–4.
8. Aydogmus S, Sen Selim H, Eris S, Kelekci S. A rare complication of cesarean delivery: iliopsoas abscess. Acta Obstet Gynecol Scand. 2014;93(10):1071–2.
9. Jaeckle KA, Young DF, Foley KM. The natural history of lumbosacral plexopathy in cancer. Neurology. 1985;35(1):8–15.
10. Taylor BV, Kimmel DW, Krecke KN, Cascino TL. Magnetic resonance imaging in cancer-related lumbosacral plexopathy. Mayo Clin Proc. 1997;72(9):823–9.
11. Jaeckle KA. Nerve plexus metastases. Neurol Clin. 1991; 9(4):857–66.
12. Hughes MS, Burd TA, Allen WC. Post-traumatic catamenial sciatica. Orthopedics. 2008;31(4):400.
13. Lang EM, Borges J, Carlstedt T. Surgical treatment of lumbosacral plexus injuries. J Neurosurg Spine. 2004;1(1):64–71.
14. Kutsy RL, Robinson LR, Routt Jr ML. Lumbosacral plexopathy in pelvic trauma. Muscle Nerve. 2000;23(11):1757–60.
15. Alsever JD. Lumbosacral plexopathy after gynecologic surgery: case report and review of the literature. Am J Obstet Gynecol. 1996;174(6):1769–77; discussion 77–8.
16. Myers MA, Harmon RL. Sacral plexopathy and sciatic neuropathy after total knee arthroplasty. Electromyogr Clin Neurophysiol. 1998;38(7):423–6.
17. Cottier JP, Descamps P, Sonier CB, Rosset P. Sciatic endometriosis: MR evaluation. AJNR Am J Neuroradiol. 1995;16(7):1399–401.
18. Katirji B, Wilbourn AJ, Scarberry SL, Preston DC. Intrapartum maternal lumbosacral plexopathy. Muscle Nerve. 2002;26(3):340–7.
19. Feasby TE, Burton SR, Hahn AF. Obstetrical lumbosacral plexus injury. Muscle Nerve. 1992;15(8):937–40.
20. Irvin W, Andersen W, Taylor P, Rice L. Minimizing the risk of neurologic injury in gynecologic surgery. Obstet Gynecol. 2004;103(2):374–82.
21. Soldatos T, Andreisek G, Thawait GK, Guggenberger R, Williams EH, Carrino JA, et al. High-resolution 3-T MR neurography of the lumbosacral plexus. Radiographics. 2013;33(4):967–87.
22. Whiteside JL, Barber MD, Walters MD, Falcone T. Anatomy of ilioinguinal and iliohypogastric nerves in relation to trocar placement and low transverse incisions. Am J Obstet Gynecol. 2003;189(6):1574–8.
23. Petchprapa CN, Rosenberg ZS, Sconfienza LM, Cavalcanti CF, Vieira RL, Zember JS. MR imaging of entrapment neuropathies of the lower extremity. Part 1. The pelvis and hip. Radiographics. 2010;30(4):983–1000.
24. Gibbons KJ, Soloniuk DS, Razack N. Neurological injury and patterns of sacral fractures. J Neurosurg. 1990;72(6):889–93.
25. Hacker HS, King JC. Plexopathy: lumbosacral. In: Frontera WR, Silver SK, Rizzo Jr TD, editors. Essentials of physical medicine and rehabilitation: musculoskeletal disorders, pain, and rehabilitation. Philadelphia: Sanders Elsevier; 2008. p. 779–86.
26. Paley M, Sidhu PS, Evans RA, Karani JB. Retroperitoneal collections – aetiology and radiological implications. Clin Radiol. 1997;52(4):290–4.
27. Chevalier X, Larget-Piet B. Femoral neuropathy due to psoas hematoma revisited. Report of three cases with serious outcomes. Spine (Phila Pa 1976). 1992;17(6):724–6.
28. Whitaker R, Borley N. Chapter 6. Instant anatomy. 1st ed. Oxford: Blackwell Scientific; 1994. p. 114–23.
29. Lu S, Chang S, Zhang YZ, Ding ZH, Xu XM, Xu YQ. Clinical anatomy and 3D virtual reconstruction of the lumbar plexus with respect to lumbar surgery. BMC Musculoskelet Disord. 2011;12:76.
30. Planner AC, Donaghy M, Moore NR. Causes of lumbosacral plexopathy. Clin Radiol. 2006;61(12):987–95.
31. Thomas JE, Cascino TL, Earle JD. Differential diagnosis between radiation and tumor plexopathy of the pelvis. Neurology. 1985;35(1):1–7.
32. Laughlin RS, Dyck PJ. Electrodiagnostic testing in lumbosacral plexopathies. Phys Med Rehabil Clin N Am. 2013;24(1):93–105.
33. Boon AJ, Gertken JT, Watson JC, Laughlin RS, Strommen JA, Mauermann ML, et al. Hematoma risk after needle electromyography. Muscle Nerve. 2012;45(1):9–12.
34. Donaghy M. Lumbosacral plexus lesions. In: Dyk PJ, Thomas PK, editors. Peripheral neuropathy, vol. 2. 4th ed. Philadelphia: Elsevier Saunders; 2005. p. 1375–90.
35. Hammersted JP. Plexopathy syndromes. In: Goetz CG, editor. Textbook of clinical neurology. 2nd ed. Philadelphia: Saunders; 2003. p. 271–2.
36. Rancont CM. Chronic psoas syndrome caused by the inappropriate use of a heel lift. J Am Osteopath Assoc. 2007;107(9):415–8.
37. Waldman SD. Lumbar plexus block: psoas compartment technique. In: Waldman SD, editor. Atlas of interventional pain management. 2nd ed. Philadelphia: Saunders; 2004. p. 443–8.
38. Waldman SD. Ultrasound-guided lumbar plexus nerve block. In: Waldman SD, editor. Comprehensive altas of ultrasound-guided pain management injection techniques. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2014. p. 719–26.
39. Brown DL. Lumbar plexus block. In: Brown DL, editor. Atlas of regional anesthesia. Philadelphia: W. B. Saunders; 1992. p. 76–7.
40. Capdevila X, Macaire P, Dadure C, Choquet O, Biboulet P, Ryckwaert Y, et al. Continuous psoas compartment block for postoperative analgesia after total hip arthroplasty: new landmarks, technical guidelines, and clinical evaluation. Anesth Analg. 2002;94(6):1606–13, table of contents.

41. Hadzic A, Vioka JD. Lumar plexus block. In: Hadzic A, editor. Hadzic's peripheral nerve blocks and anatomy for ultrasound-guided regional anesthesia. 2nd ed. New York: McGraw-Hill; 2012. p. 217–28.
42. Enneking FK, Chan V, Greger J, Hadzic A, Lang SA, Horlocker TT. Lower-extremity peripheral nerve blockade: essentials of our current understanding. Reg Anesth Pain Med. 2005;30(1):4–35.
43. Paradise LA, Raj PP, Fryer JR. Lumbar myofascial blocks. In: Raj PP, Lou L, Erdine S, Staats PS, Waldman SD, Racz GB, et al., editors. Interventional pain management image-guided procedures. 2nd ed. Philadelphia: Saunders; 2008. p. 382–90.
44. Lang SA, Yip RW, Chang PC, Gerard MA. The femoral 3-in-1 block revisited. J Clin Anesth. 1993;5(4):292–6.
45. Mulchandani H, Awad I, McCaetney C. Ultrasound-guided nerve blocks of the lower limb. In: Narouze SM, editor. Atlas of ultrasound-guided procedures in interventional pain management. New York: Springer; 2010. p. 249–50.
46. Calava JM, Patt RB, Reddy S, Varma DG, Chiang J. Psoas sheath chemical neurolysis for management of intractable leg pain from metastatic liposarcoma. Clin J Pain. 1996;12(1):69–75.
47. Douglas I, Bush D. The use of patient-controlled boluses of local anaesthetic via a psoas sheath catheter in the management of malignant pain. Pain. 1999;82(1):105–7.
48. Deschner B, Robards C, Xu D, Hadzic A. Lower extremity peripheral nerve block. In: Benzon HT, Rathmell JP, Turk DC, Argoff CE, editors. Raj's practical management of pain. 4th ed. Philadelphia: Mosby Elsevier; 2008. p. 891–2.
49. Aveline C, Bonnet F. Delayed retroperitoneal haematoma after failed lumbar plexus block. Br J Anaesth. 2004;93(4):589–91.

Part VII

Pelvic Pain

Daniel Krashin

Introduction

Chronic pelvic pain is a serious medical issue affecting as much as 15–20 % of the female population ages 18–50 [1], as well as a less well-defined number of males. In addition, significant pain after inguinal surgery occurs in about 15–35 % of surgeries [2], leading to potential disability.

Damage to the nerves causing pelvic pain can be the result of either direct trauma, postsurgical trauma, vascular compression, malignancy, infection, or any other form of insult-causing neuropathy that results in hyperexcitability and inappropriate firing of these nerves, causing constant or intermittent pain and dysfunction at the innervated area. Pelvic pain from neuropathy may be due to injury of the pudendal nerve, which supplies sensation to the genital area, causing discomfort during intercourse, sitting, and elimination. Pelvic pain may also result from neuropathies that arise as complications of surgeries of the lower abdominal wall. Often, the initiating factor is difficult to identify. Frequently, the cause of chronic pelvic pain is undiagnosed, although it has high prevalence [1].

For each nerve disorder, we describe the anatomy, with particular emphasis on the various sites of potential entrapment; then we describe the clinical entrapment syndromes and their associated physical examination findings. Finally, we will outline the various available treatment approaches with an emphasis on injection techniques, their benefits, and their side effects.

After reading these chapters, we hope that you will develop increased knowledge of pelvic pain, be aware of its most common causes and impact on quality of life, and pay increased medical attention to this problem. You will also learn the best options of evaluation and treatment.

References

1. Mathias SD, Kuppermann M, Liberman RF, Lipschutz RC, Steege JF. Chronic pelvic pain: prevalence, health-related quality of life, and economic correlates. Obstet Gynecol. 1996;87(3):321–7.
2. Franneby U, Sandblom G, Nordin P, Nyren O, Gunnarsson U. Risk factors for long-term pain after hernia surgery. Ann Surg. 2006;244(2):212–9.

Ilioinguinal Nerve Entrapment: Pelvic

44

Natalia Murinova, Daniel Krashin, and Andrea M. Trescot

Introduction

Ilioinguinal nerve (IIN) entrapment causing chronic pelvic pain syndrome is considered part of the "border nerve syndrome" [1] (*ilioinguinal*, *iliohypogastric*, and *genitofemoral nerve* neuropathy) [2]. The role of the ilioinguinal, iliohypogastric, and genitofemoral nerves in abdominal wall pain is discussed in Chaps. 40 and 41. The IIN has several areas of entrapment and has a different pain pattern based on each of the sites of entrapment.

Clinical Presentation (Table 44.1)

The pain of ilioinguinal nerve entrapment is described as burning, sometimes accompanied by paresthesias or altered sensation, in the inguinal area and ipsilateral scrotum and testis/labia (Figs. 44.1, 44.2, and 44.3). It may present in either sex, and, while it is commonly associated with surgical injury, it may occur spontaneously. In a study of 46 women with abdominal and pelvic pain who were diagnosed with IIN entrapment, only 6 were found to have a clear cause for this condition, suggesting that this condition may frequently go unsuspected and undiagnosed. These women were noted to have dysesthesia, pain with pressure at the nerve exit, and hyperesthesia in the nerve distribution [10].

IIN entrapment is easily mistaken for other pain syndromes involving the lower GI tract and the genitourinary tract, as well as orthopedic conditions of the pelvis and hip. The sensory distribution of this nerve is wide, ranging from the inferior margin of the abdominal muscles to the cutaneous innervation of the inguinal crease, as well as the scrotum or labia majora and superior-medial thigh (Figs. 44.2 and 44.3). Therefore, depending on the location of the entrapment, the clinical picture may vary. The pain pattern of the IIN can also mimic the pattern of the genitofemoral nerve (Fig. 44.2). Patients with ilioinguinal neuralgia will complain of groin pain, which becomes worse with sitting, lifting, and bending. Benes et al. [11] suggested using the term *abdominoinguinal pain syndrome*, to describe the combination of these nerve pathologies.

Anatomy (Table 44.2)

The IIN arises from the anterior ramus of L1 with some contributions from T12 and L2, similar to the *iliohypogastric nerve* (IHN) (Fig. 44.4), as part of the *lumbar plexus* (see Chap. 49). Emerging from the lateral border of the psoas major muscle, both nerves run subperitoneally in front of *quadratus lumborum* before piercing the *transverse abdominis muscle* above the iliac crest to become superficial [12]. The IIN shares dermatomes with the proximal *fallopian tubes* and the *uterine fundus*, and therefore IIN entrapment can mimic uterine and ovarian pathology. It pierces the internal oblique muscle mediocaudally to the anterior superior iliac spine (ASIS). The nerve enters the inguinal canal approximately 2 cm medial to the ASIS and exits through the superficial inguinal ring to function as a sensory nerve for the overlying skin (Fig. 44.5). From

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Table 44.1 Occupation/exercise/trauma history relevant to ilioinguinal and iliohypogastric nerve entrapment

Surgery	Inguinal hernia repair [3]
	Pelvic open and laparoscopic surgeries [4]
	Trochar trauma from laparoscopic surgery [5]
	Appendectomy, hysterectomy [6], abdominoplasty
	Pfannenstiel incision
	Orchiectomy
Trauma	Blunt abdominal trauma [7]
	Femoral catheter placement
	Tearing of the lower external oblique aponeurosis (reported in hockey players) [8]
Stretch injury	Pregnancy
Entrapment	At the rectus border (ACNE)
	At the iliac crest
Spontaneous	Variations in the musculo-aponeurotic connective tissue [9]

Fig. 44.1 Patient pain complaint from ilioinguinal nerve entrapment (Image courtesy of Andrea Trescot, MD)

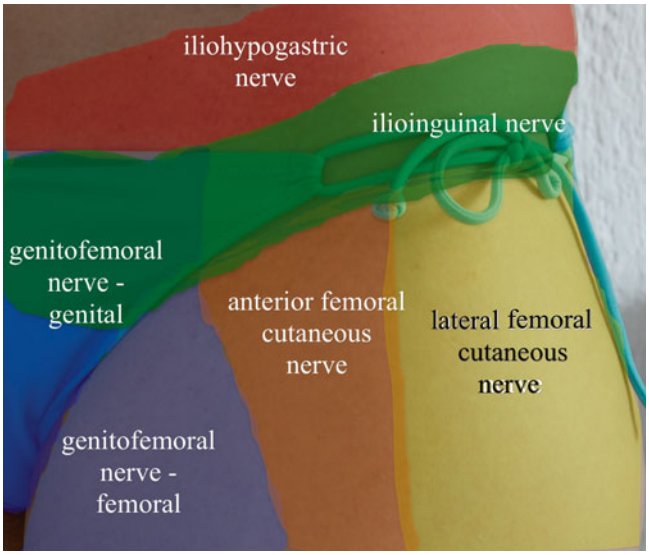

Fig. 44.2 Pain patterns from iliohypogastric, ilioinguinal, genitofemoral, lateral femoral cutaneous, and anterior femoral cutaneous nerves (Image courtesy of Andrea Trescot, MD)

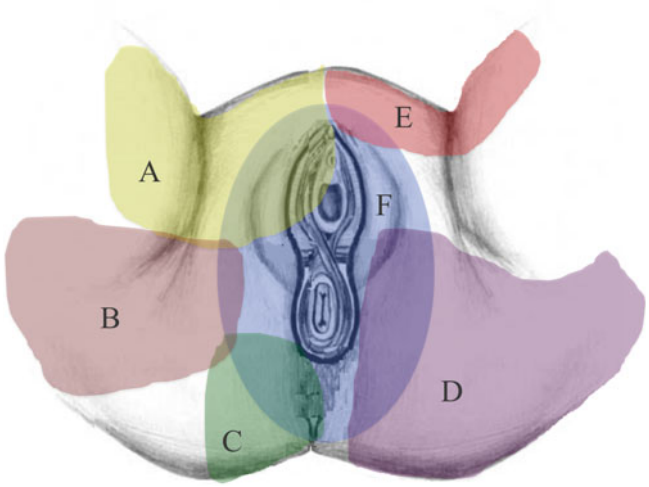

Fig. 44.3 Innervation of perineum: *A* genitofemoral nerve, *B* obturator nerve, *C* inferior cluneal nerve, *D* peroneal branch of the posterior femoral cutaneous nerve, *E* ilioinguinal nerve, and *F* pudendal nerve (Image inspired by Hibner et al. [39], courtesy of Andrea Trescot, MD)

there, it supplies innervation to the inner thigh and either scrotum or labia. The IIN usually terminates approximately 3 cm lateral to the midline and 2 cm superior to the pubic symphysis.

The IIN is highly variable in its course and innervation, with multiple potential connections with the IHN and the genitofemoral nerves (see Chap. 45), and it sometimes assumes the role of the genital branch of the genitofemoral nerve. The site where the IIN penetrates the different layers of abdominal muscle is also highly variable [5]. It generally pierces the lower border of the internal oblique medially and below the ASIS and then passes through the superficial inguinal ring in front of the spermatic cord.

Rab et al. [13] described the dissection of 32 cadavers and identified 4 different types of branching of the ilioinguinal nerve. Type A (43.7 %) had no sensory contribution in the groin from the IIN, with sensation to the groin supplied instead by the genitofemoral nerve. Type B (28.1 %) had a dominant IIN with motor fibers to the cremaster muscle but no sensation to the groin. Type C (20.3 %) had a dominant genitofemoral nerve, with an IIN providing sensory branches to the inguinal crease and the mons pubis, as well as the root of the penis/labia majora; the IIN also shared a branch with the IHN in this pattern. Type D (7.8 %) had cutaneous branches from both the ilioinguinal and genitofemoral nerves, with the IIN

Table 44.2 Ilioinguinal nerve anatomy

Origin	Anterior ramus of L1 (occasionally L2)
General route	Exits L1 (sometimes L2), passes through psoas, leaves internal oblique, past ASIS, travels below aponeurosis of external oblique along spermatic cord/round ligament through superficial abdominal ring
Sensory distribution	Ribbon-shaped area over inguinal region up to iliac crest, over symphysis, root of the penis, proximal scrotum/labia, and small area of the anterior and medial thigh
Motor innervation	Transverse abdominis and external oblique muscles
Anatomic variability	There is an inverse size of the IIN and IHN; they may be joined or one may be absent
Other relevant structures	ASIS, inguinal canal, spermatic cord/round ligament

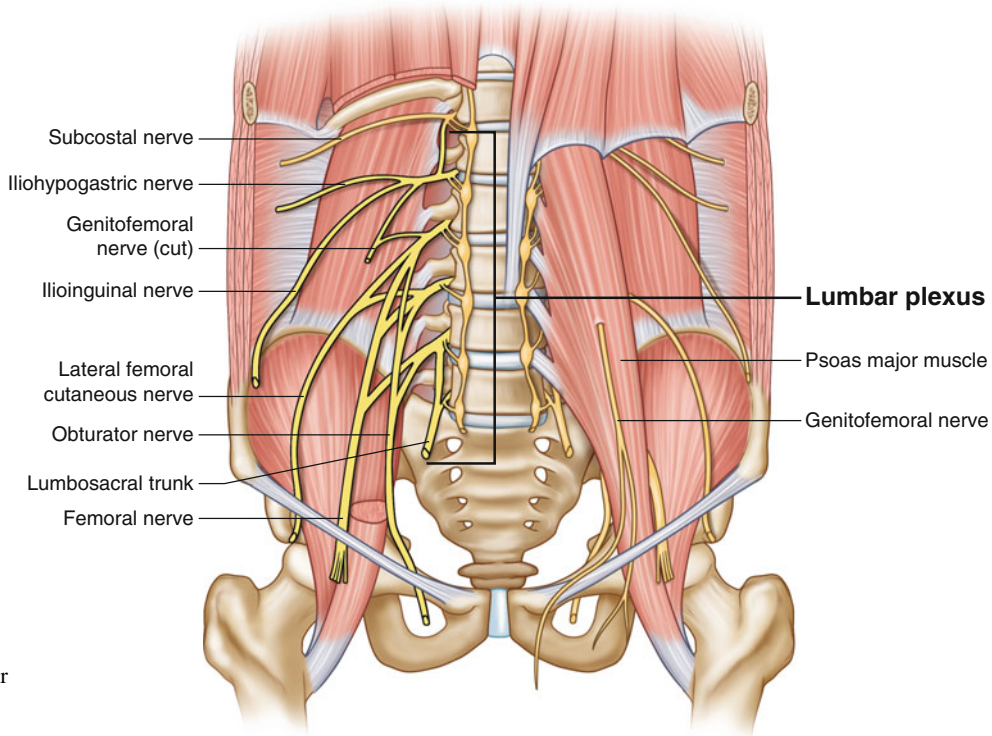

Fig. 44.4 Anatomy of the lumbar plexus (Image by Springer)

innervating the inguinal crease and the mons, as well as the most anteroproximal portion of the root of the penis/labia majora.

Entrapment

The IIN is commonly trapped at the rectus border, the iliac crest, and the paravertebral area. However, it can also be entrapped at the inguinal region, and patients with *Pfannenstiel incisions* or *inguinal hernia repairs* are at an increased risk for IIN entrapment anywhere between the ASIS and the pubis, due to tissue scarring or fibrosis [14]. Hernia repair using *mesh* has a risk factor for entrapment of the IIN in the fibrotic tissue surrounding and adhering to the mesh.

Physical Exam

For the physical exam of the rectus border entrapment, position the patient supine or standing. Patients will often walk flexed at the hips to avoid tension on the abdominal

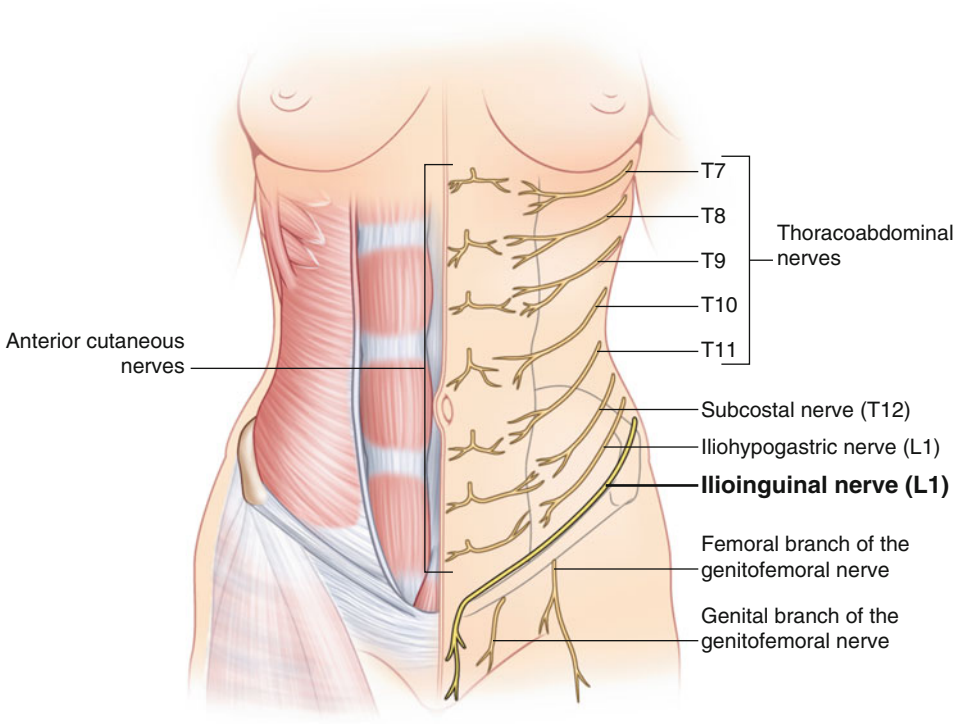

Fig. 44.5 Anatomy of the ilioinguinal nerve (Image by Springer)

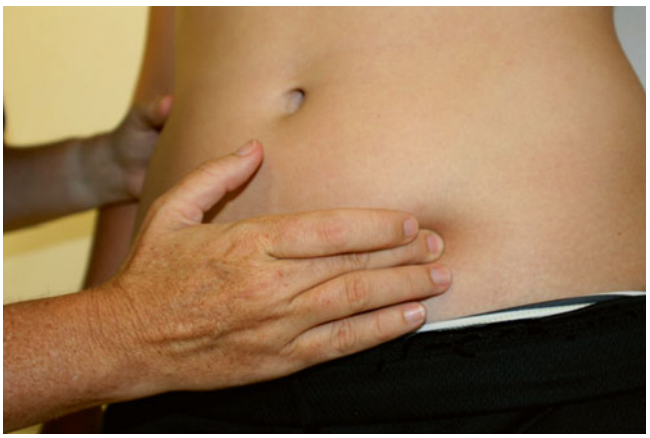

Fig. 44.6 Ilioinguinal nerve exam at the rectus border (Image courtesy of Andrea Trescot, MD)

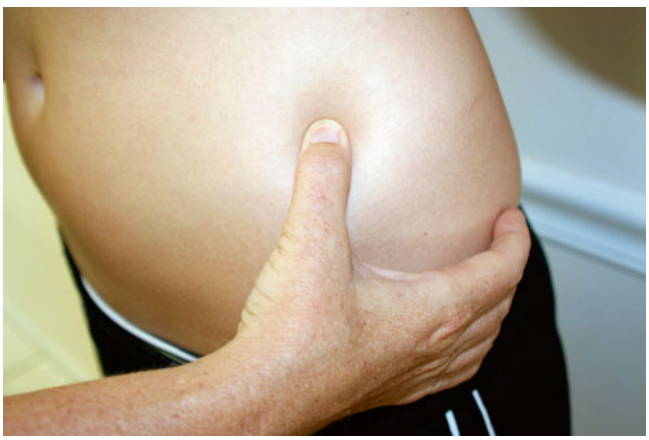

Fig. 44.7 Ilioinguinal nerve exam at the iliac crest (Image courtesy of Andrea Trescot, MD)

wall. Check for point tenderness at the rectus border (Fig. 44.6). This occurs approximately 10 cm inferior to the umbilicus, in a position similar to anterior cutaneous nerve entrapment (see Chap. 42). To check for iliac crest entrapment, stand in front of the patient and place hands on the iliac crest laterally (Fig. 44.7). Feel the edge of the external oblique with your thumbs (Video 44.1). Tenderness at the external oblique tendon attachment can indicate ilioinguinal nerve entrapment (Fig. 44.8). For paravertebral entrapment, palpate the paravertebral L1 vertebrae. Tenderness here can indicate ilioinguinal entrapment. This exam is helpful if the treatment needs to be more proximal. A focal bulging of the abdominal wall due to loss of muscle tone in the external and internal oblique muscles may be visible in some cases of ilioinguinal neuropathy (Fig. 44.9).

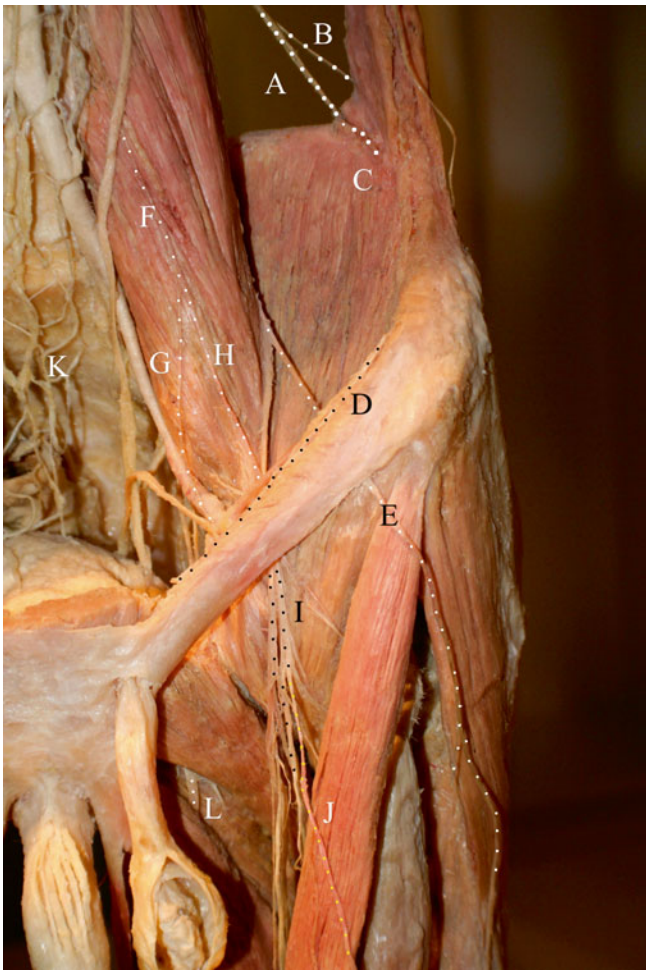

Fig. 44.8 Ilioinguinal nerve entrapment at the iliac crest (modified from an image from *Bodies*, *The Exhibition*, with permission). *A* ilioinguinal nerve, *B* iliohypogastric nerve, *C* site of ilioinguinal nerve entrapment at the external oblique, *D* ilioinguinal nerve over the inguinal ligament, *E* lateral femoral cutaneous nerve, *F* genitofemoral nerve, *G* genital branch of the genitofemoral nerve, *H* femoral branch of the genitofemoral nerve, *I* femoral nerve, *J* saphenous nerve, *K* inferior hypogastric plexus, *L* obturator nerve (Image courtesy of Andrea Trescot, MD)

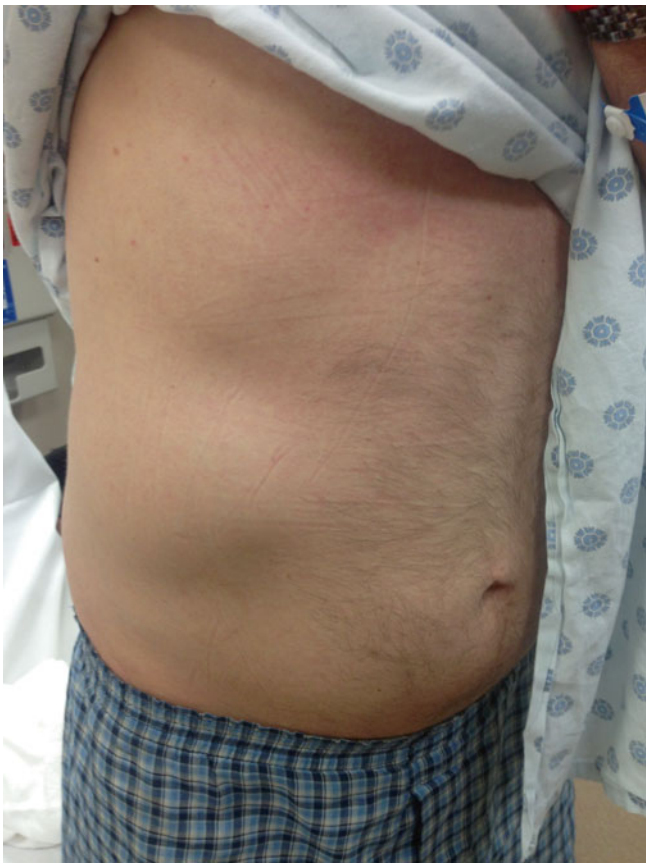

Fig. 44.9 Abdominal bulge due to proximal ilioinguinal entrapment (Image courtesy of Andrea Trescot, MD)

Differential Diagnosis (Table 44.3)

There are many conditions resulting in groin pain that mimic ilioinguinal entrapment, including inguinal hernia, tumor, varicocele, hydrocele, spermatocele, transient testicular or ovarian torsion, and myofascial injury. Entrapment of the iliohypogastric or genitofemoral nerve (see Chap. 45) needs to be included in the differential diagnosis as well. Other diseases that can clinically present with chronic inguinal pain (including hip osteoarthritis, disk hernia, recurrent inguinal hernia, and tumor) can be ruled out by selective radiological examinations including ultrasonography (US), computed tomography (CT), and magnetic resonance imaging (MRI). Diagnostic laparoscopy can also be considered for further diagnosis [15]. If a diagnostic nerve injection in the region of the digital exam did not provide relief, then one needs to consider the possible aberrant innervation of the nerve and using ultrasound for better localization for improved results of the nerve block. The diagnostic tests for IIN entrapment are found on Table 44.4.

Table 44.3 Differential diagnosis of lower abdominal pain

	Potential distinguishing features
Inguinal hernia	Palpable abdominal wall defect
Varicocele, hydrocele	Scrotal venous distension or fluid collection
Transient testicular or ovarian torsion	Testicular tenderness (male), bimanual exam (female)
Myofascial injury	History, examination with local tenderness
Entrapment of iliohypogastric or genitofemoral nerves	Injection of individual nerves at the ASIS
Osteoarthritis	Symphysis pubis osteophytes on X-ray

Table 44.4 Diagnostic tests for ilioinguinal nerve entrapment

	Potential distinguishing features
Physical exam	Tenderness over the lateral rectus border (ACNE syndrome – see Chap. 42), ASIS, external oblique
Diagnostic injection	At rectus border, ASIS, external oblique
Ultrasound	Localization of IN at ASIS
MRI	Not useful
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies	Not useful

Identification and Treatment of Contributing Factors

Perimenstrual fluid retention can lead to monthly episodes of abdominal pain. Pfannenstiel incisions [16] can lead to ilioinguinal entrapment at the lateral corner of the incision. Inguinal hernia repairs, especially with mesh, can lead to early (immediately postoperatively) as well as late (sometimes years after the surgery) entrapment. *Ilioinguinal nerve entrapment* (INE) is one of the most significant complications following inguinal hernia repair. In 2003, Poobalan et al. [14] reviewed the literature and found that chronic groin pain after inguinal herniorrhaphy was as high as 54 %, at least in the first 3 months. The incidence of postoperative ilioinguinal neuralgia ≥1 year reported after inguinal herniorrhaphy ranges from 6 to 29 % [17–20].

Desensitization exercises have been reported to provide relief in some patients. Unfortunately, this nerve entrapment is often triggered by abdominal wall exercises. Giving diuretics (HCTZ or furosemide) 5 days before scheduled menses can sometimes prevent the perimenstrual edema that may trigger entrapment.

Injection Techniques

Landmark-Guided Technique

For entrapment that occurs at the rectus border, advance a 25-gauge 2-in. needle to the lateral rectus border (Fig. 44.10) (Video 44.2). Inject 2 cc maximum volume of local anesthetic with or without deposteroid. Using a peripheral nerve stimulator to identify the nerve may help. Relying on a "fascial click" as indication that the needle has pierced the fascia is associated with increased complications [12].

For an iliac crest entrapment, position the patient in either the supine or lateral decubitus position. After identifying the lateral edge of the external oblique muscle at the iliac crest, advance a 25 g 2-in. needle onto the tendon attachment (Fig. 44.8 site C). Inject a 2 cc maximum volume of local anesthetic with or without deposteroid. In a third technique, the anterior superior iliac spine (ASIS) is palpated in the supine position; the injection point is 2 in. medial and 2 in. inferior to that point (Fig. 44.11). If the patient's physique makes body landmarks difficult to identify by palpation, consider using ultrasound or fluoroscopy for the iliac crest injection.

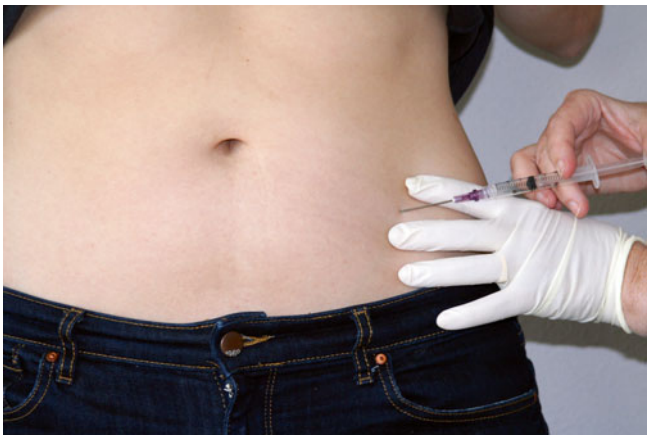

Fig. 44.10 Injection of the ilioinguinal nerve at the rectus border (Image courtesy of Andrea Trescot, MD)

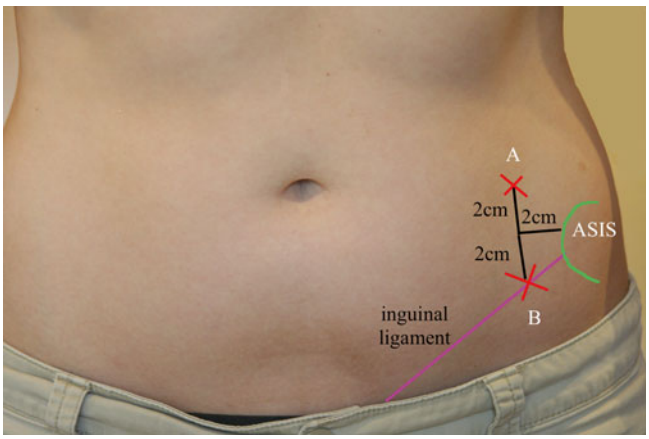

Fig. 44.11 Injection of the ilioinguinal nerve at the iliac crest (Image courtesy of Andrea Trescot, MD)

Fluoroscopic-Guided Technique

There are no specific bony landmarks for IIN entrapment, but fluoroscopy may help to identify the iliac crest and the ASIS in the morbidly obese (Fig. 44.12). The proximal approach at T12, L1, and L2 can be done under fluoroscopic control. The procedure is done in a manner very similar to a transforaminal epidural. Position the patient prone under fluoroscopy and identify the foramen (Fig. 44.13). A peripheral nerve stimulator is used to confirm the level of pathology, since the origin of this nerve is sometimes variable. Stimulation should allow

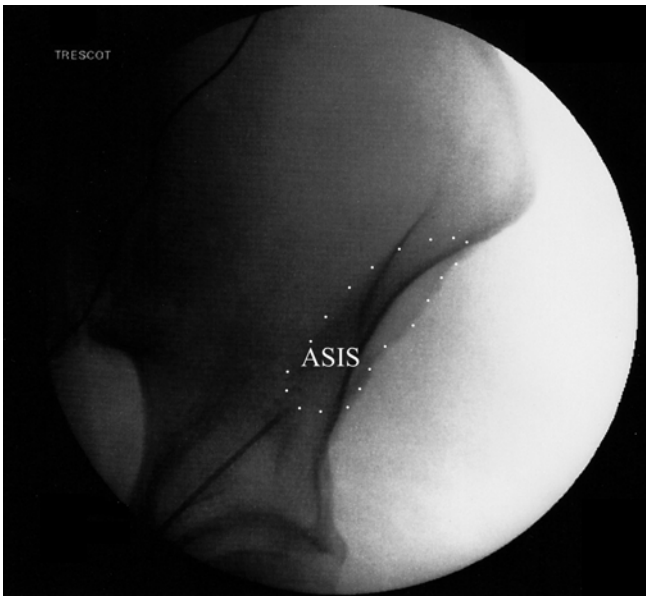

Fig. 44.12 Fluoroscopic identification of the anterior superior iliac spine (Image courtesy of Andrea Trescot, MD)

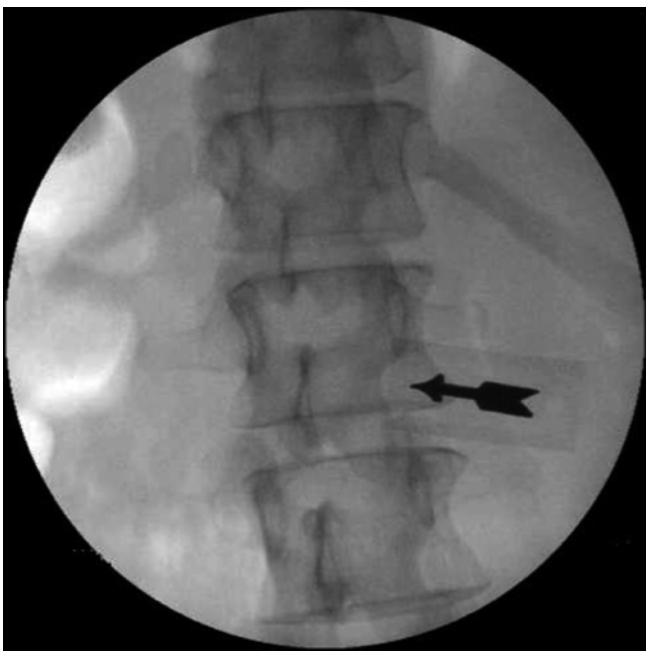

Fig. 44.13 Location of the proximal injection of the ilioinguinal nerve at its origin at T12 and L1 (Image courtesy of Andrea Trescot, MD)

the patient to identify the "that's it" site. If you notice twitching in the patient's lower back (representing the cluneal nerve) instead of the groin, reposition the needle slightly more anteriorly. After injection of contrast under live fluoroscopy, inject a 1 cc maximum volume of local anesthetic with or without steroid. Because of concerns regarding possible anterior spinal artery infarcts from particulate steroids, it may be prudent to use non-particulate steroids.

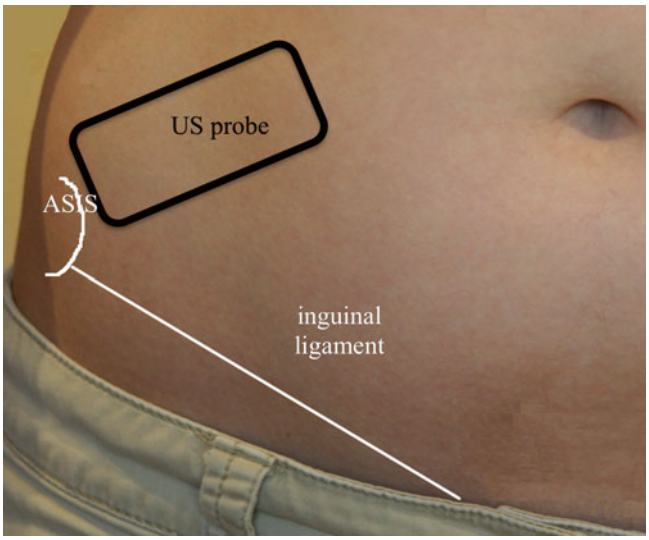

Fig. 44.14 Location of the ultrasound probe to evaluate the ilioinguinal nerve (Image courtesy of Andrea Trescot, MD)

Ultrasound-Guided Technique

The ultrasound localization of the ilioinguinal and iliohypogastric nerves is best seen at the anterior superior iliac spine (ASIS). Eichenberger et al. [21] described one of the first US approaches to the ilioinguinal and iliohypogastric nerves. The high-frequency linear probe is placed slightly rotated from a transverse to an oblique plane to be perpendicular to the anatomical course of both the IIN and the IHN, with the lateral/caudal part of the transducers brought into contact with the iliac crest (Fig. 44.14). The fascial layers between the external oblique, the internal oblique, and the transverse abdominus should be visible as bright hypoechoic white lines; the IHN and IIN will be hypoechoic oval structures between the internal oblique and the transverse abdominus muscles (Fig. 44.15).

Bischoff et al. [22] described a randomized, double-blind, placebo-controlled crossover trial in 12 patients with severe, persistent inguinal pain after herniorrhaphy and 12 normal controls. The authors felt that ultrasound-guided lidocaine injections of the ilioinguinal and iliohypogastric nerves, at the level of the ASIS, were not useful in diagnosis and management of persistent inguinal post-herniorrhaphy pain. The authors offered several possible explanations for these findings: first, the sensory innervation of the groin is complex, consisting of branches of the ilioinguinal, iliohypogastric, and genitofemoral nerves, which share origins from the T12 to L2 spinal nerves. Communicating branches between these nerves and anatomic variations is common, meaning that interruption of one nerve may not abolish sensory transmission from a specific area [13]. Second, in some patients, sensory branches to the painful area may leave the main nerves proximal to the ASIS, so that even a properly performed

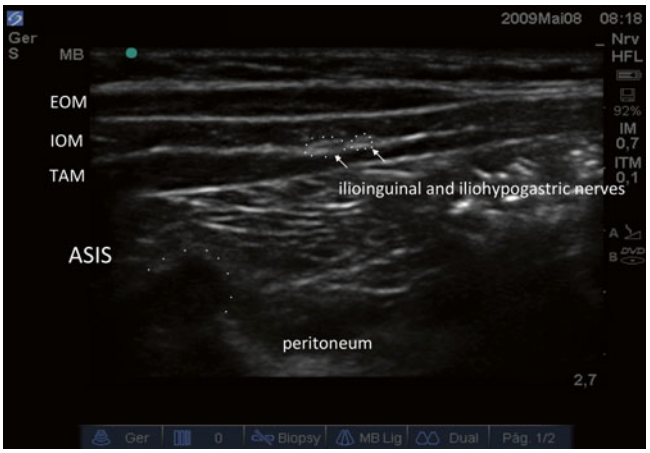

Fig. 44.15 Ultrasound image of ilioinguinal and iliohypogastric nerves at the iliac crest. *ASIS* anterior superior iliac spine, *EOM* external oblique muscle, *IOM* internal oblique muscle, *TAM* transverse abdominis muscle (Image courtesy of Thiago Nouer Frederico, MD, modified by Charles de Oliveira, MD)

nerve block in that area will have no effect on the pain. Third, a more prominent role of the genitofemoral nerve in persistent post-herniorrhaphy pain has recently been suggested (see Chap. 45) [15]. Fourth, a peripheral nerve block of short duration may not be adequate to affect persistent postsurgical pain, which is thought to be maintained by central sensitization [23].

Neurolytic Technique

Cryoneuroablation

For the ilioinguinal, iliohypogastric, and genitofemoral nerves (Chap. 45), cryoneuroablation is a common further therapy. After superficial infiltration with local anesthetic and deep infiltration with saline containing epinephrine 1:200,000, a 12-gauge intravenous catheter is used as the introducer for the 2.0 mm cryoprobe, usually trying to place the probe parallel to the nerve from lateral to medial. Location may be optimized with the use of US guidance (Fig. 44.16) as well as with the peripheral nerve stimulator.

Fanelli et al. [24] described ten patients with ilioinguinal, genitofemoral, or combined neuralgia after inguinal hernia repair treated with cryoneuroablation. After cryotherapy, patients reported overall pain reduction of 0–100 % (mean, 77.5 %; median, 100 %), 80 % reported decreased analgesic use, and 90 % reported increased physical capacity. Two patients underwent additional cryotherapy, one for incomplete relief and one for recurrent pain, both with 100 % efficacy. Wound infection ($n = 1$) was the only complication.

Phenol/Alcohol

Phenol has been used [25] to treat persistent ilioinguinal neuralgia, but there is the possibility of tissue necrosis around the nerve and a significant risk of neuritis. Absolute alcohol provides good relief with the possibility of nerve and surrounding tissue necrosis and damage [25].

Radio-Frequency Lesioning

Standard, neurodestructive heat *radio-frequency ablation* (RFA) has been evaluated for the treatment of ilioinguinal neuralgia. Kastler et al. [26] compared local anesthetic blockade to radio-frequency ablation. CT guidance was utilized at the level of the ASIS. A 22-gauge 5 mm active tip needle was used. There was no mention of threshold, but stimulation was used to finalize needle position. Three ablations were performed, each for 90 s, at 70, 80, and 90 °C. Results showed 12.5 months relief in the RFA arm, compared to 1.6 months in the local anesthetic/steroid arm.

Several groups have used pulsed radio frequency (PRF) for ilioinguinal neuralgia. For instance, Mitra et al. [27] described PRF for the treatment of chronic ilioinguinal neuropathy. PRF has been used at the vertebral level (dorsal root ganglion); Rozen and Ahn [28] described five patients treated with PRF at T12, L1, and L2, noting that four of the five patients had relief for 4–9 months.

Kastler et al. [26] compared local anesthetic blockade to pulsed radio-frequency ablation for the treatment of 42 patients with postsurgical ilioinguinal neuralgia after hernia repair. A total of 18 PRF procedures (14 patients) and 28 steroid/local anesthetic injections (28 patients) were performed under CT guidance at the ASIS. Injections contained 1.5 mL of *cortivazol* (a high-affinity glucocorticoid) and 3 mL of a lidocaine-ropivacaine mixture. Mean duration of pain relief in the RF group was 12.5 months, compared to 1.6 months in the injection group.

Rozen and Parvez in 2006 [29] described a case series of PRF at the DRG, while in 2015 Makharita and Amr [30] performed a randomized, double-blind controlled trial of PRF at the DRG. Both groups noted significant improvement.

Neurostimulation

Peripheral nerve stimulation has been used for chronic groin pain [30, 31]. The trial electrodes are placed percutaneously through introducers, and, if there is significant temporary relief, the leads can be placed permanently. Banh et al. described the use of a peripheral nerve stimulator for a

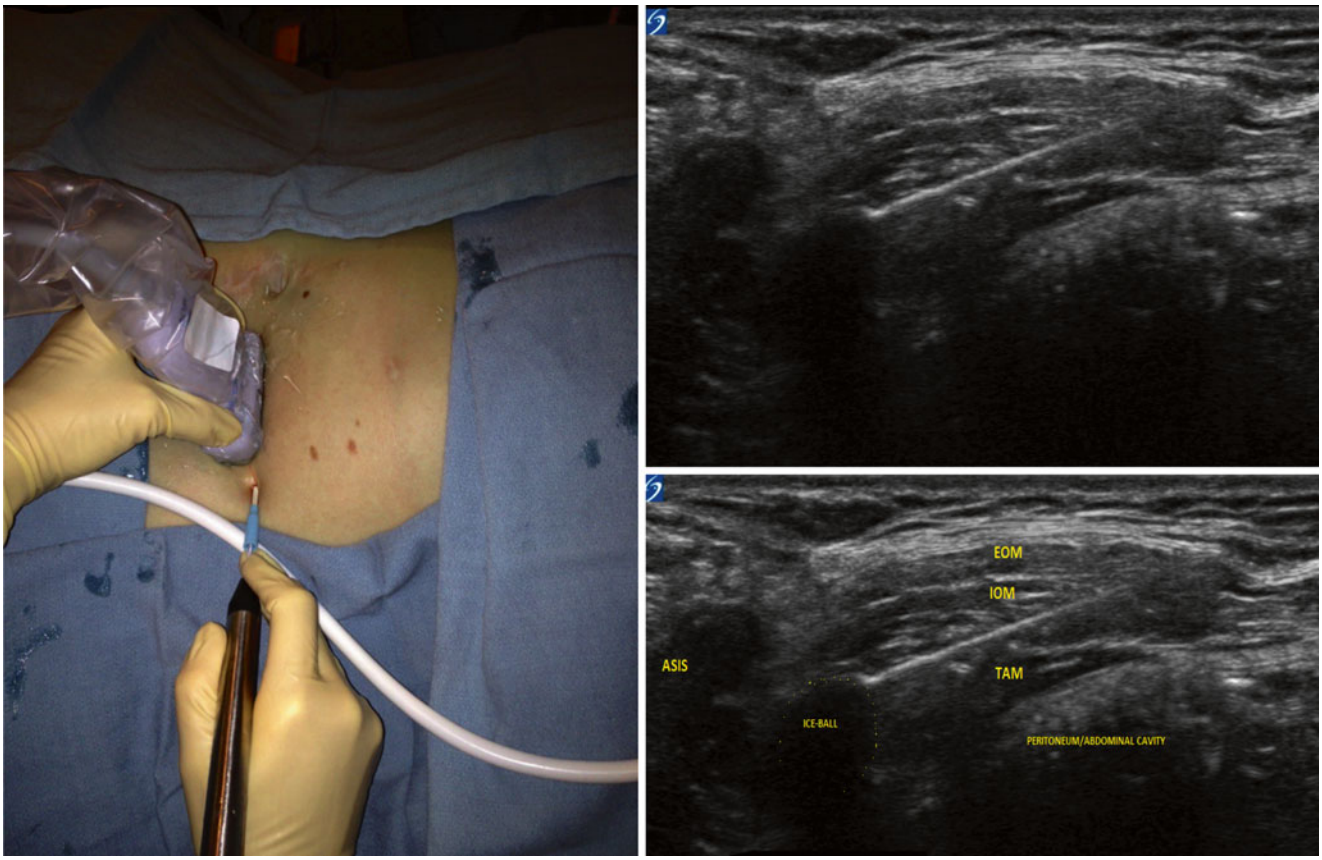

Fig. 44.16 Cryoneuroablation of the ilioinguinal nerve. *EOM* external oblique muscle, *IOM* internal oblique muscle, *TAM* transverse abdominus muscle, *ASIS* anterior superior iliac spine (Image courtesy of Agnes Stogicza, MD

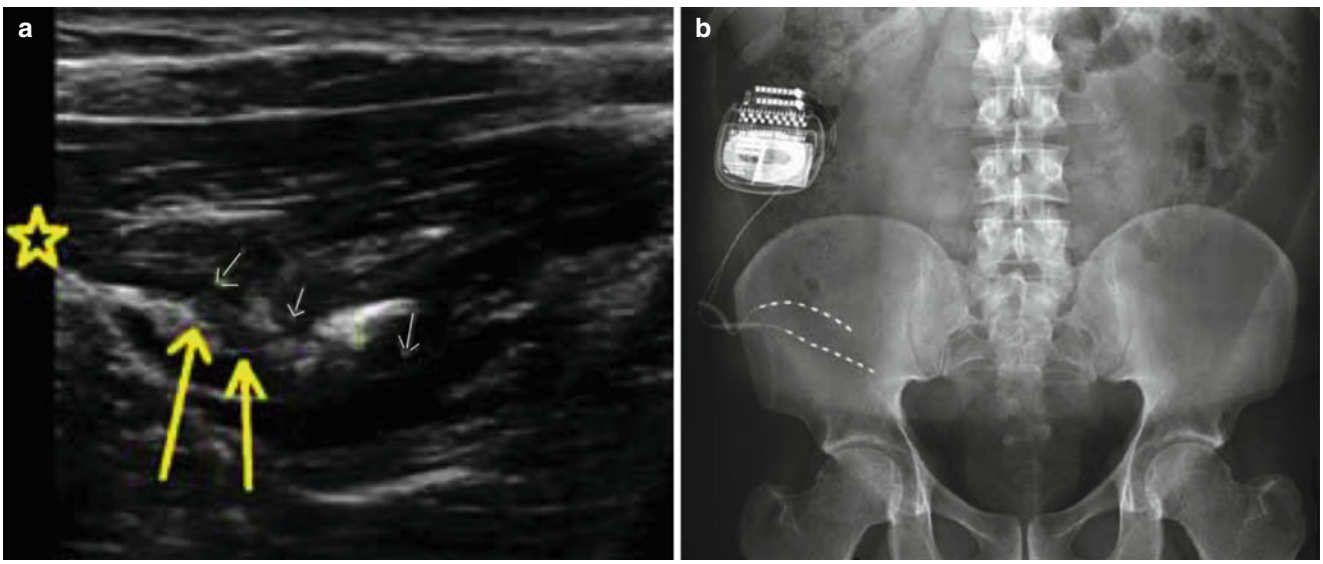

Fig. 44.17 Peripheral nerve stimulation of the ilioinguinal nerve. (**a**) Ultrasound image: anterior superior iliac spine (*yellow star*), ilioinguinal/iliohypogastric nerves (*thick yellow arrows*), and the percutaneous nerve stimulator lead (*thin white arrows*). (**b**) Fluoroscopic image (Images from Elahi et al. [33]. Reprinted with permission from American Society of Interventional Pain Physicians)

patient with combat injury-related ilioinguinal damage [32], and Elahi et al. [33] described a peripheral nerve stimulator placed under US guidance to treat ilioinguinal neuralgia after an inguinal herniorrhaphy (Fig. 44.17).

Surgical Technique

Entrapment neuralgia arising from the ilioinguinal nerve is also often treated by the surgical release of the entrapped nerve. Entrapment neuralgia arising from the ilioinguinal nerve can be treated by the surgical release of the entrapped nerve. Kim et al. [6] were able to retrospectively review charts on 33 patients; 23 had ilioinguinal neuralgia, while 10 had combined ilioinguinal and iliohypogastric neuralgia. All had a positive preoperative local anesthetic injection; 30 of the 33 had "considerable" pain relief after surgical release. Zacest and colleagues [34] noted complete or partial pain relief in 13 of 19 (66.7 %) patients undergoing ilioinguinal nerve release, with a mean follow-up of 35 months. Hahn [35], in a crossover prospective study, randomized 19 women to medication management or surgical release of the ilioinguinal nerve. The surgery patients noted good relief. Nine of the ten women initially randomized to the medication arm discontinued the medications due to side effects; they were then offered surgery and also noted good relief.

A prerequisite for any operation should be a positive result of a block with local anesthesia. Excision of the scar neuroma can give relief, but the neuroma is often difficult to localize. One technique that has been successful (Trescot, personal correspondence) is mixing local anesthetic with *methylene blue*. If the local anesthetic abolishes the pain, then the neuroma is within the dye tissue, and the surgeon is instructed to "take out everything blue."

Complications

Because proper performance of ilioinguinal/iliohypogastric blocks requires small volume injections, the possibility of local anesthetic toxicity is remote. Because the injection is limited to the lower abdominal wall and inguinal region, any hemodynamic changes would be unusual. As with other injections, the patient is advised to protect the anesthetized area from trauma. Even properly performed ilioinguinal/iliohypogastric blocks can result in transient femoral anesthesia, with a reported incidence of 3.7–5 % [36]. The mechanism of femoral anesthesia with these methods is the tracking of local anesthetic along the iliac fascia. Perforations of the bowels [37] and pelvic hematomas [38] have been reported after ilioinguinal/iliohypogastric blocks. This illustrates the importance of using blunt needles to appreciate the loss of resistance as the needle traverses the layers of the abdominal wall. Damage to the L1 nerve root can leave an abdominal wall weakness (Fig. 44.9).

Summary

Ilioinguinal entrapment can cause abdominal pain, as seen in Chap. 40, as well as pelvic pain. The variable location of the ilioinguinal nerve and the variable pattern of sensation can make diagnosis difficult. Recognition of the ilioinguinal nerve as a cause of pelvic pain can save patients from unnecessary surgery.

References

1. Bardeen CR. A statistical study of the abdominal and border nerves in man. Am J Anat. 1902;1(1):203–28.
2. Peng P, Tumber PS. Ultrasound-guided interventional procedures for patients with chronic pelvic pain – a description of techniques and review of literature. Pain Physician. 2007;11(2):215–24.
3. Ndiaye A, Diop M, Ndoye JM, Konate I, Ndiaye AI, Mane L, et al. Anatomical basis of neuropathies and damage to the ilioinguinal nerve during repairs of groin hernias. (about 100 dissections). Surg Radiol Anat. 2007;29(8):675–81.
4. Stulz P, Pfeiffer KM. Peripheral nerve injuries resulting from common surgical procedures in the lower portion of the abdomen. Arch Surg. 1982;117(3):324–7.
5. Whiteside JL, Barber MD, Walters MD, Falcone T. Anatomy of ilioinguinal and iliohypogastric nerves in relation to trocar placement and low transverse incisions. Am J Obstet Gynecol. 2003;189(6):1574–8.
6. Kim DH, Murovic JA, Tiel RL, Kline DG. Surgical management of 33 ilioinguinal and iliohypogastric neuralgias at Louisiana State University Health Sciences Center. Neurosurgery. 2005;56(5):1013–20; discussion – 20.
7. Starling JR, Harms BA, Schroeder ME, Eichman PL. Diagnosis and treatment of genitofemoral and ilioinguinal entrapment neuralgia. Surgery. 1987;102(4):581–6.
8. Hollis MH, Lemay DE. Nerve entrapment syndromes of the lower extremity: Medscape; 2012. Available from: http://emedicine.medscape.com/article/1234809-overview.
9. Toussaint CP, Perry 3rd EC, Pisansky MT, Anderson DE. What's new in the diagnosis and treatment of peripheral nerve entrapment neuropathies. Neurol Clin. 2010;28(4):979–1004.
10. Hahn L. Clinical findings and results of operative treatment in ilioinguinal nerve entrapment syndrome. BJOG Int J Obstet Gynaecol. 1989;96(9):1080–3.
11. Benes J, Nádvornik P, Dolezel J. Abdominoinguinal pain syndrome treated by centrocentral anastomosis. Acta Neurochir (Wien). 2000;142(8):887–91.
12. Soneji N, Peng PW. Ultrasound-guided pain interventions – a review of techniques for peripheral nerves. Korean J Pain. 2013;26(2):111–24.
13. Rab M, Ebmer, Dellon AL. Anatomic variability of the ilioinguinal and genitofemoral nerve: implications for the treatment of groin pain. Plast Reconstr Surg. 2001;108(6):1618–23.
14. Poobalan AS, Bruce J, Smith WC, King PM, Krukowski ZH, Chambers WA. A review of chronic pain after inguinal herniorrhaphy. Clin J Pain. 2003;19(1):48–54.

15. Giger U, Wente M, Büchler M, Krähenbühl S, Lerut J, Krähenbühl L. Endoscopic retroperitoneal neurectomy for chronic pain after groin surgery. Br J Surg. 2009;96(9):1076–81.
16. Luijendijk RW, Jeekel J, Storm RK, Schutte PJ, Hop W, Drogendijk AC, et al. The low transverse Pfannenstiel incision and the prevalence of incisional hernia and nerve entrapment. Ann Surg. 1997;225(4):365.
17. Douek M, Smith G, Oshowo A, Stoker D, Wellwood J. Prospective randomised controlled trial of laparoscopic versus open inguinal hernia mesh repair: five year follow up. BMJ. 2003;326(7397):1012–3.
18. Bringman S, Ramel S, Heikkinen T-J, Englund T, Westman B, Anderberg B. Tension-free inguinal hernia repair: TEP versus mesh-plug versus Lichtenstein: a prospective randomized controlled trial. Ann Surg. 2003;237(1):142.
19. Nordin P, Bartelmess P, Jansson C, Svensson C, Edlund G. Randomized trial of Lichtenstein versus Shouldice hernia repair in general surgical practice. Br J Surg. 2002;89(1):45–9.
20. Vrijland W, van Den Tol M, Luijendijk R, Hop W, Busschbach J, de Lange D, et al. Randomized clinical trial of non-mesh versus mesh repair of primary inguinal hernia. Br J Surg. 2002;89(3):293–7.
21. Eichenberger U, Greher M, Kirchmair L, Curatolo M, Moriggl B. Ultrasound-guided blocks of the ilioinguinal and iliohypogastric nerve: accuracy of a selective new technique confirmed by anatomical dissection. Br J Anaesth. 2006;97(2):238–43.
22. Bischoff JM, Koscielniak-Nielsen ZJ, Kehlet H, Werner MU. Ultrasound-guided ilioinguinal/iliohypogastric nerve blocks for persistent inguinal postherniorrhaphy pain: a randomized, double-blind, placebo-controlled, crossover trial. Anesth Analg. 2012;114(6):1323–9.
23. Kehlet H, Jensen TS, Woolf CJ. Persistent postsurgical pain: risk factors and prevention. Lancet. 2006;367(9522):1618–25.
24. Fanelli RD, DiSiena MR, Lui FY, Gersin KS. Cryoanalgesic ablation for the treatment of chronic postherniorrhaphy neuropathic pain. Surg Endosc. 2003;17(2):196–200.
25. Hakeem A, Shanmugam V. Current trends in the diagnosis and management of post-herniorraphy chronic groin pain. World J Gastrointest Surg. 2011;3(6):73–81.
26. Kastler A, Aubry S, Piccand V, Hadjidekov G, Tiberghien F, Kastler B. Radiofrequency neurolysis versus local nerve infiltration in 42 patients with refractory chronic inguinal neuralgia. Pain Physician. 2012;15(3):237–44.
27. Mitra R, Zeighami A, Mackey S. Pulsed radiofrequency for the treatment of chronic ilioinguinal neuropathy. Hernia. 2007;11(4):369–71.
28. Rozen D, Ahn J. Pulsed radiofrequency for the treatment of ilioinguinal neuralgia after inguinal herniorrhaphy. Mt Sinai J Med N Y. 2006;73(4):716–8.
29. Rozen D, Parvez U. Pulsed radiofrequency of lumbar nerve roots for treatment of chronic inguinal herniorraphy pain. Pain Physician. 2006;9(2):153–6.
30. Makharita MY, Amr YM. Pulsed radiofrequency for chronic inguinal neuralgia. Pain Physician. 2015;18(2):E147–55.
31. Rosendal F, Moir L, de Pennington N, Green AL, Aziz TZ. Successful treatment of testicular pain with peripheral nerve stimulation of the cutaneous branch of the ilioinguinal and genital branch of the genitofemoral nerves. Neuromodulation. 2013;16(2):121–4.
32. Banh DP, Moujan PM, Haque Q, Han TH. Permanent implantation of peripheral nerve stimulator for combat injury-related ilioinguinal neuralgia. Pain Physician. 2013;16(6):E789–91.
33. Elahi F, Reddy C, Ho D. Ultrasound guided peripheral nerve stimulation implant for management of intractable pain after inguinal herniorrhaphy. Pain Physician. 2015;18(1):E31–8.
34. Zacest AC, Magill ST, Anderson VC, Burchiel KJ. Long-term outcome following ilioinguinal neurectomy for chronic pain. J Neurosurg. 2010;112(4):784–9.
35. Hahn L. Treatment of ilioinguinal nerve entrapment – a randomized controlled trial. Acta Obstet Gynecol Scand. 2011;90(9):955–60.
36. Rosario DJ, Jacob S, Luntley J, Skinner PP, Raftery AT. Mechanism of femoral nerve palsy complicating percutaneous ilioinguinal field block. Br J Anaesth. 1997;78(3):314–6.
37. Johr M, Sossai R. Colonic puncture during ilioinguinal nerve block in a child. Anesth Analg. 1999;88(5):1051–2.
38. Vaisman J. Pelvic hematoma after an ilioinguinal nerve block for orchialgia. Anesth Analg. 2001;92:1048–9.
39. Hibner M, Desai N, Robertson LJ, Nour M. Pudendal neuralgia. J Minim Invasive Gynecol. 2010;17(2):148–53.

Genitofemoral Nerve Entrapment: Pelvic

45

Thais Khouri Vanetti, Alexandra Tavares Raffaini Luba, Fabrício Dias Assis, and Charles Amaral de Oliveira

Introduction

Despite the paucity of literature on the *genitofemoral nerve* (GFN), impairment of this nerve, which may result in chronic pain, is not uncommon but rather likely under-recognized. Blunt trauma or pelvic operations such as inguinal herniorrhaphy, C sections, and others are often accompanied by chronic inguinal and perineal pain and paresthesias, which may be due to GFN pathology [1].

The GFN, *iliohypogastric nerve* (IHN), and *ilioinguinal nerve* (IIN) originate from similar levels of the spinal nerves; therefore, it is often clinically difficult to determine which nerve is causing the pain. The major differential diagnosis of GFN is *ilioinguinal neuralgia*. Diagnostic blocks can aid in making this diagnosis. The femoral branch of the GFN is also known as the *lumboinguinal nerve*.

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Clinical Presentation (Table 45.1)

Chronic inguinal and perineal pain and paresthesias may be due to GFN pathology [1]. Postoperative neuropathies after major pelvic surgeries are rare, with a total estimated incidence of 1.9 %; postoperative GFN neuropathy is estimated at 0.3 % [6]. Magee first reported the syndrome of genitofemoral neuralgia in 1942 [7], but it remains a rarely encountered (or diagnosed) clinical entity. The most common clinical presentation of GFN entrapment consists of intermittent or constant pain, burning dysesthesia, and sensory changes in the inguinal region (Fig. 45.1), with radiation of the pain to the skin of the genitalia (scrotum, vagina, labia majora) and upper middle thigh (Fig. 45.2) [1]. Female patients usually present with dyspareunia and pelvic pain, and GFN neuralgia can be misdiagnosed as interstitial cystitis or pudendal neuralgia (Fig. 45.3). The pain is exacerbated by activities such as walking and hyperextension of the thigh and is ameliorated by recumbent position and thigh flexion [1]. Paresthesia and persistent pain in the lower abdomen or pelvic region, including the lateral scrotum or labia majora and the anterior proximal thigh, may also represent GFN

Table 45.1 Occupation/exercise/trauma history relevant to genitofemoral nerve entrapment

Compression	Hematoma and adhesions associated with surgery
Lumbar plexus abnormalities (see Chap. 49)	Psoas abscess, psoas entrapment [2]
Mechanical	Pubic symphysis irritation
	Late pregnancy
Trauma	Lumbar sympathetic neurolytics
	Celiac plexus neurolytics
Surgery	Trans-obturator "sling" surgery for incontinence [3]
	Inguinal hernia repair [4]
	Hysterectomy and cesarean section [5]
Entrapment	Surgical scar, adhesions

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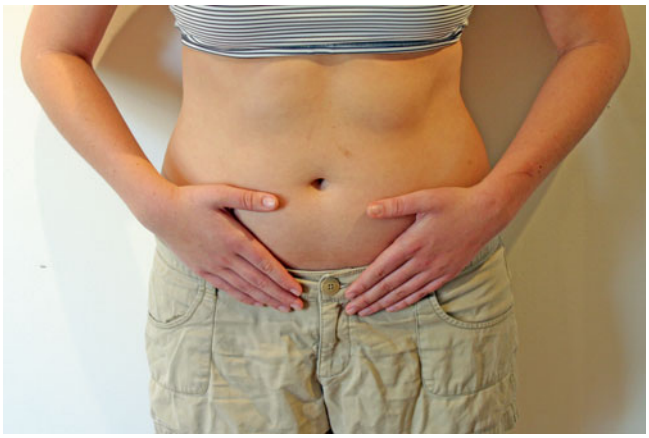

Fig. 45.1 Patient complaints of genitofemoral pain (Image courtesy of Andrea Trescot, MD)

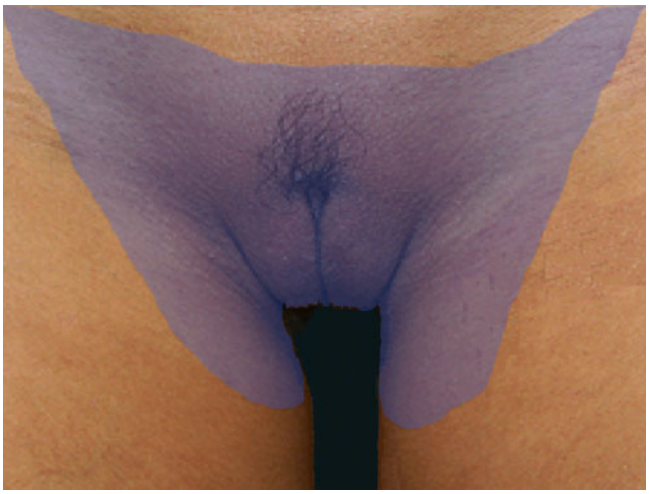

Fig. 45.2 Pain pattern from genitofemoral neuralgia (Image courtesy of Andrea Trescot, MD)

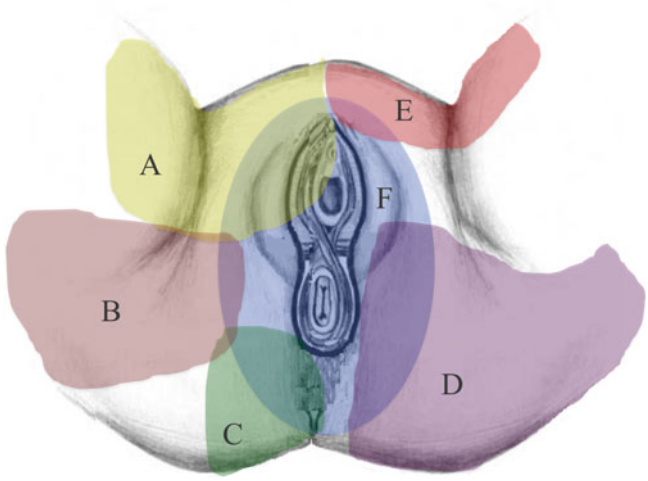

Fig. 45.3 Innervation of perineum: *A* genitofemoral nerve, *B* obturator nerve, *C* inferior cluneal nerve, *D* peroneal branch of the posterior femoral cutaneous nerve, *E* ilioinguinal nerve, and *F* pudendal nerve (Image courtesy of Andrea Trescot, MD)

Table 45.2 Genitofemoral nerve anatomy

Origin	L1 and L2 anterior (ventral) rami
General route	Perforates the psoas at L3 and L4, descends along the medial psoas border, divides into genital and femoral branches just above the inguinal ligament
	Genital branch
	Males: inside internal inguinal ring with spermatic cord to scrotum
	Females: accompanies the round ligament
	Femoral branch
	Located caudally and laterally to the genital branch, travelling caudally with external iliac artery, behind inguinal ligament through fascia lata to femoral sheath
Sensory distribution	Pelvic region, groin, scrotum or labia majora, and anterior proximal thigh area; femoral branch: anterior proximal thigh in the femoral triangle
Motor innervation	Cremaster muscle and cremasteric reflex
Anatomical variability	Ilioinguinal occasionally provides genital branch of GFN
Other relevant structures	Pubic tubercle, Poupart's ligament

neuropathy. There can be a great deal of overlap between the ilioinguinal (see Chap. 44), genitofemoral, and lateral femoral cutaneous nerves (see Chap. 61). Benes et al. [8] suggested using the term *abdomino-inguinal pain syndrome*, to describe the combination of these nerve pathologies.

Anatomy (Table 45.2)

The *iliohypogastric* (IHN), *ilioinguinal* (IIN) (see Chap. 44), and *genitofemoral* (GFN) nerves are known as "border nerves" because they innervate the transitional area between the abdomen and lower extremity: the lower abdominal wall, groin, labia majora, and medial thigh. These nerves arise from the *lumbar plexus* (see Chap. 49), whose cutaneous branches include the IHN, IIN, GFN, *lateral femoral cutaneous nerve* (LFCN), and *obturator nerve* (Fig. 45.4).

The GFN arises from L2 with occasional L1 contributions, which unite within the psoas muscle, while the IHN and IIN arise from the L1 anterior rami with contributions from T12 and L2. The GFN is mainly sensory, with a small motor component that innervates the cremaster muscle and provides the motor component of the *cremasteric muscle* (*cremasteric reflex*). The GFN emerges from the psoas muscle anteriorly at the L3 and L4 level and descends to the medial border of the psoas muscle, where it divides into a genital and femoral branch just above the inguinal ligament (Fig. 45.5). The GFN innervates, and thus may refer pain to,

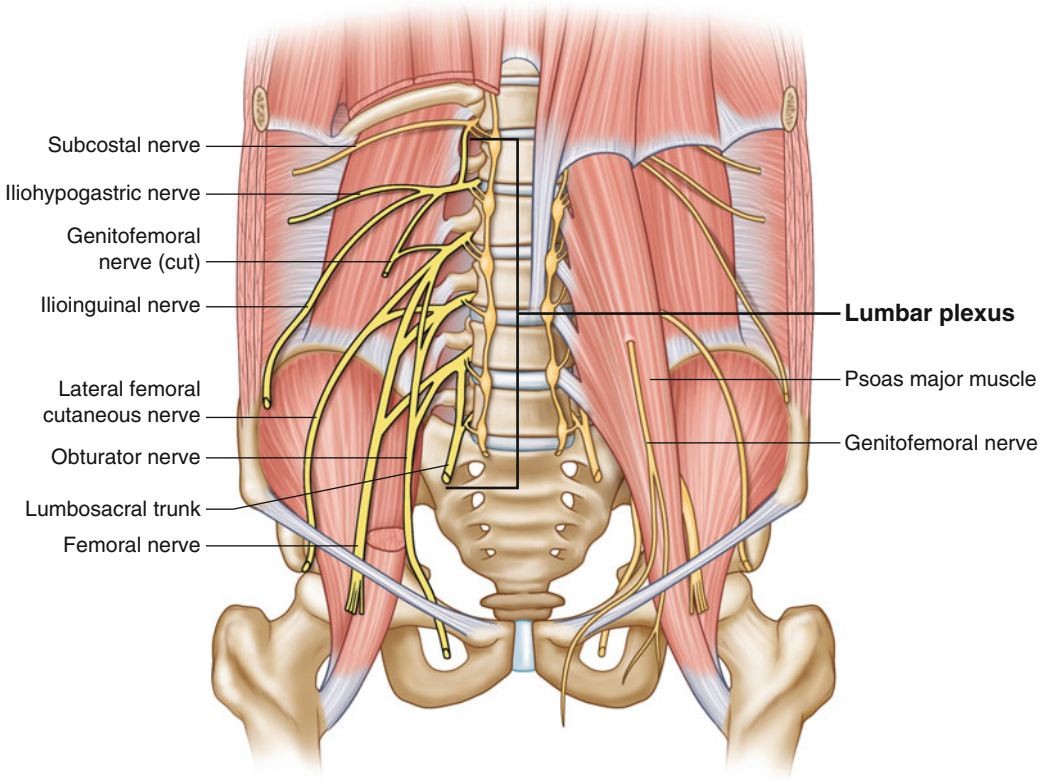

Fig. 45.4 Lumbar plexus (Image courtesy of Springer)

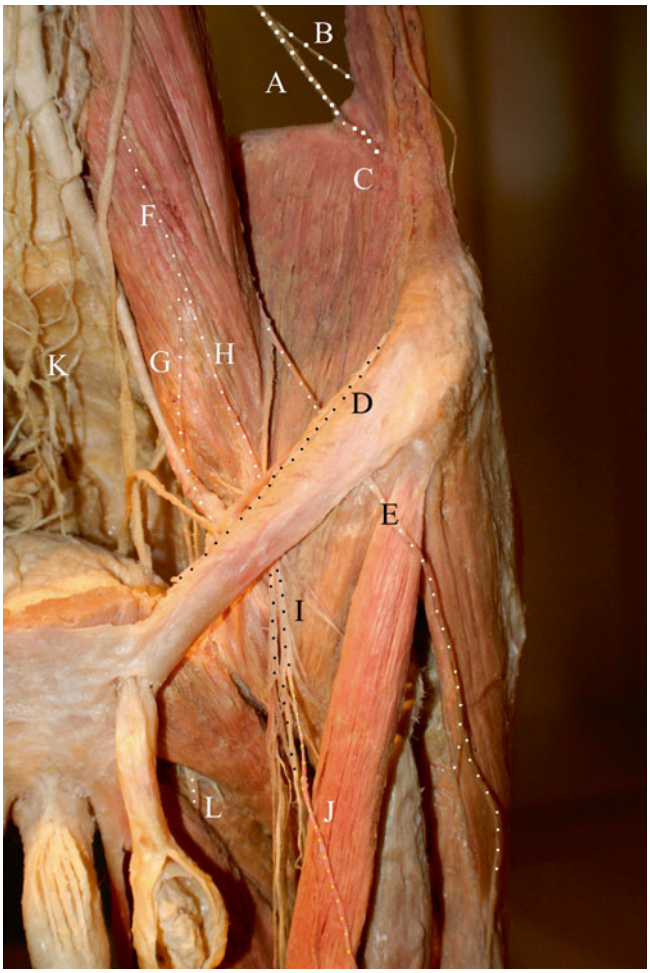

Fig. 45.5 Genitofemoral nerve anatomy, modified from an image from *Bodies*, *The Exhibition*, with permission. *A* ilioinguinal nerve, *B* iliohypogastric nerve, *C* site of ilioinguinal nerve entrapment at the external oblique, *D* ilioinguinal nerve over the inguinal ligament, *E* lateral femoral cutaneous nerve, *F* genitofemoral nerve, *G* genital branch of the genitofemoral nerve, *H* femoral branch of the genitofemoral nerve, *I* femoral nerve, *J* saphenous nerve, *K* inferior hypogastric plexus, *L* obturator nerve (Image courtesy of Andrea Trescot, MD)

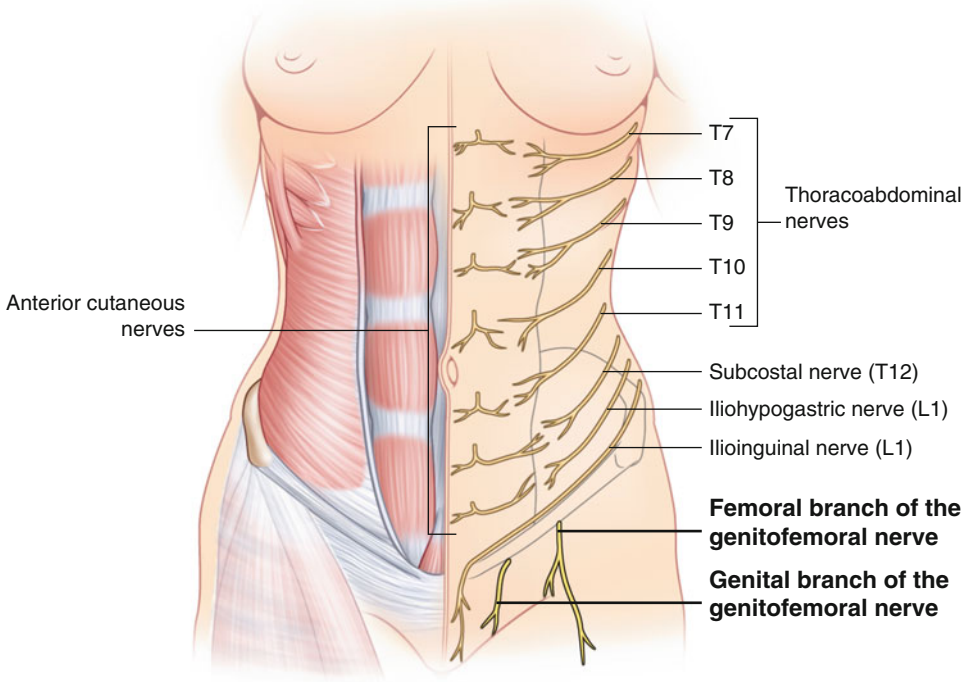

Fig. 45.6 Genital and femoral branches of the genitofemoral nerve (Image courtesy of Springer)

areas including the pelvic region, groin, scrotum or labia majora, and anterior proximal thigh area.

The *genital branch of the GFN* accompanies the psoas muscle. In males, it passes inside the internal inguinal ring together with the spermatic cord, supplying motor fibers to the cremaster muscle and sensation to the lateral scrotum. In females, it accompanies the round ligament, innervating the mons pubis and the labia majora. The femoral branch (also known as the *lumboinguinal nerve*) is located caudally and laterally to the genital branch and travels with the external iliac artery beneath the *inguinal ligament*, piercing the *fascia latae* and entering the *femoral sheath* to innervate the skin of the anterior proximal thigh in the *femoral triangle* (Fig. 45.6) [9].

Though the course of this nerve and its branches is similar in men and women, anatomical studies suggest great variability among individuals, with only about 37 % of individual innervation patterns conforming to the conventional description [10]. The location where the genital and femoral branches split is typically reported just superior to the inguinal ligament, but variations are common [11]. Additionally, in males, the relation between the genital branch and the spermatic cord varies considerably; it can travel outside the spermatic cord, dorsally, ventrally, or inferiorly [12]. In a cadaver dissection study, the genital branch of the GFN was found in 28 % of subjects to arise from the IIN nerve and hence from T12, L1, and L2 [10].

Special attention to the great variation of the nerves in the groin region (ilioinguinal, iliohypogastric, and genitofemoral) is warranted, because of the free communication between these branches. According to a cadaver study, the ilioinguinal nerve was solely responsible for cutaneous innervation of the genital branch of the genitofemoral nerve in 28 % of the dissections and shared innervation with the genital branch of the genitofemoral nerve in 8 % [10].

Entrapment

The GFN may be entrapped during its association with the psoas muscle and in the pelvis, by a spasm of the muscle, or by the presence of intramuscular hematoma, abscess, or adhesions. During the retroperitoneal course of this nerve, it may also become entrapped by a retroperitoneal hematoma or lymphoma [13]. A more common cause of entrapment is surgery involving the pelvis or inguinal area, including C section, appendectomy, and inguinal hernia repair, particularly when done laparoscopically [4]. Pain may begin months or years after surgery, due to gradual scar tissue formation at the surgical site [10]. On its passage above the *pubic ramus*, the genitofemoral nerve is vulnerable to surgical trauma. Albeit less common, this nerve can also be compressed and injured during the final stages of pregnancy [14].

Physical Examination

Careful neurological sensory examination may demonstrate sensory changes in the "border zone" between the abdomen and thigh, groin, anterior proximal thigh, and lateral scrotum or labia majora. Tender points may be found on the internal

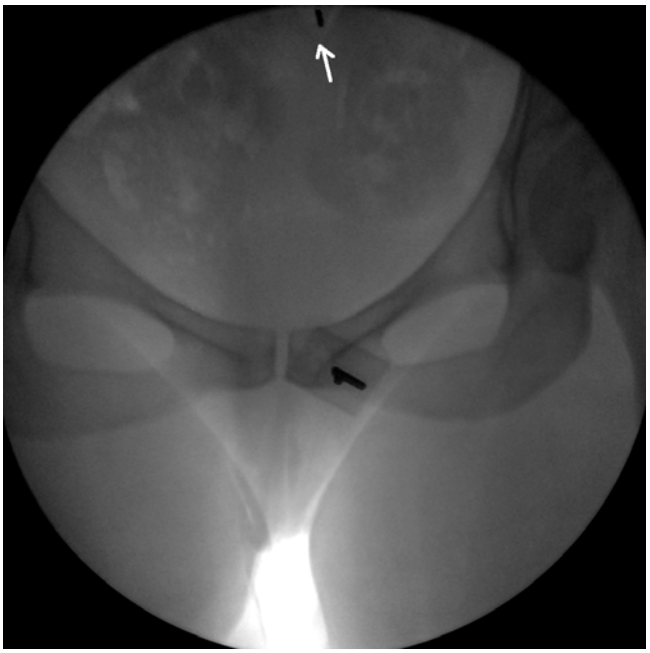

Fig. 45.7 Location of tenderness to palpation, which is consistent with genitofemoral nerve pain. Note the *white arrow* showing the InterStim® placed for "interstitial cystitis" pain that offered no relief (Image courtesy of Andrea Trescot, MD)

part of the *inguinal ring*, with pain on palpation of the lower pubic area and pain on vaginal palpation [3].

Sensory changes can include hypoesthesia, paresthesias, and allodynia. Sensory changes in the upper thigh and the groin region occur when the femoral branch of the GFN has been affected [3].

Palpation of the psoas muscle and maneuvers to activate or tighten the psoas muscle may aggravate the pain. Pain may be exacerbated by Valsalva maneuvers, such as coughing or rising from a seated position. Hip mobilization or peristaltic movements, such as during defecation, may also be pain provoking [3]. The cremasteric reflex in male patients is lost with more severe neuropathy, particularly neuropathy involving the genital branch. In females, there may be tenderness with palpation of the lateral vagina and vulva [3]. This pain may also be exacerbated in women by the menstrual period and by intercourse [14]. There will be tenderness over the pubic tubercle, palpation of which will replicate the pain (Fig. 45.7).

Differential Diagnosis (Table 45.3)

The challenge of diagnosing GFN neuropathy involves ruling out the other conditions of the lumbar spinal nerves, lumbar plexus, and tributary nerves that can produce a similar clinical picture. Lumbar plexopathy in particular may present with a complex pattern of motor and sensory symptoms that may resist clarification [2].

Table 45.3 Differential diagnosis of inguinal and perineal pain

	Potential distinguishing features
Lumbar plexopathy	EMG testing, as genitofemoral nerve does not have significant motor component
IIN and IHN neuropathy	First perform a block of the ilioinguinal nerve; if this does not relieve the pain, perform blocks of the ipsilateral L1 and L2 nerves, as with a paravertebral block
Iliopectineal bursitis	Lack of neuropathic features, calcification on X-ray
Inguinal hernia	Palpable abdominal wall defect
Spermatic cord disorders	Palpable spermatic cord mass
Pelvic tumor	Mass on bimanual exam or on MRI
Endometriosis	Endometrial implants on laparoscopy

Table 45.4 Diagnostic tests for genitofemoral nerve entrapment

Physical exam	Pain more prominent around pelvic tubercle
Diagnostic injection	Ilioinguinal nerve block should not relieve pain, while L1/L2 nerve block should relieve pain
Ultrasound	Scarring or neuroma may be visible in nerve course
MRI	May show abscess or hematoma in retroperitoneal section of nerve
Arteriography	Not useful
X-ray	May detect urolithiasis as a cause of pain
Electrodiagnostic studies	Not useful

The innervation patterns of the genitofemoral, ilioinguinal, and iliohypogastric nerves can be very challenging to distinguish, particularly as there is considerable anatomical variation and overlap in the innervation of these three nerves [11]. A methodical approach to diagnosis, including a detailed exam and sequential diagnostic blocks, can usually clarify the pain generator involved in a specific patient. While specific techniques for blocking the GFN will be discussed below, many experts suggest a process of exclusion: first performing a block of the ilioinguinal nerve, if the patient has pain and sensory changes in the inguinal region; then, if this does not relieve the pain, performing blocks of the GFN, followed by the ipsilateral L1 and L2 nerves [15]. See Table 45.4.

Identification and Treatment of Contributing Factors

Strenuous exercise and overstretching of the psoas muscle or injury of the psoas; infra-abdominal incision (Pfannenstiel) or laparoscopic operations [4,14]; past abdominal or pelvic surgeries, particularly repeated surgeries; and open or laparoscopic inguinal hernia repair,

appendectomy, and C-section hernia repairs with mesh placement or done laparoscopically are all causes of GFN entrapment. Retroperitoneal surgery and pregnancy are also causes.

Treatment of the underlying conditions that caused the nerve entrapment should be incorporated into treatment. Physical therapy can provide mobilization and strengthening to decrease spasm and dysfunction; desensitization therapy can be used for treating hyperpathic or allodynic areas. If the perpetuating factor is nerve compression by a surgical scar, it is possible to remove the scar tissue, thereby releasing the nerve. If compression is caused by muscle hypertrophy, the treatment of choice is to inject local anesthetic to enable muscle relaxation and, if necessary, botulinum toxin for longer-lasting relaxation. Early recognition and treatment of surgical complications such as hematoma will also help limit the extent of nerve injury.

Injection Technique

As noted above, it can be challenging to distinguish genitofemoral from ilioinguinal and iliohypogastric neuralgia, due to the adjacent areas of innervation and the considerable anatomical variation. One common diagnostic approach is to first perform diagnostic blocks of the ilioinguinal and/or iliohypogastric nerves as appropriate, given the patient's symptoms, preferably using ultrasound guidance for maximum accuracy. If these blocks successfully create appropriate areas of numbness without decreasing the pain, then a follow-up block may be performed of L1 and L2 as a selective nerve root block or paravertebral block. If this block relieves the pain, it is likely a result of an entrapment of the GFN. This process has the disadvantage of requiring two or three distinct procedures with the attendant time and risk. A block which successfully relieves the pain symptoms may be followed up with cryoneuroablation or pulsed radiofrequency treatment (see below) [11].

Landmark-Guided Technique

Trescot described a landmark-guided ("blind") technique to inject the genital branch of the GFN [16]. The patient is placed in the supine position with a pillow under the knees if extending the lower limbs evokes pain. After a sterile skin prep, the pubic tubercle is palpated, and 1 cc of local anesthetic and deposteroid is injected via a 25-gauge needle, just superior and lateral to the tubercle. The use of a PNS can facilitate the accuracy of the injection. The accuracy of this technique has not been studied in comparison to ultrasound-guided approaches. When performing the blind technique, the utmost care must be taken in relation to important spermatic cord structures (testicular artery) and peritoneal cavity transgression.

Fluoroscopy-Guided Technique

With the patient in the supine position, the area of maximal tenderness (just lateral to the pubic tubercle) is identified by fluoroscopy (Fig. 45.7). After a sterile skin prep and local anesthetic infiltration subcutaneously, a 22-gauge needle is advanced to the periosteum. The use of a PNS will facilitate identification of the nerve (Fig. 45.8). One cc of local anesthetic and deposteroid is then injected.

Another technique for GFN diagnosis and treatment is the dorsal root ganglion (DRG) local anesthetic block at T12, L1, and L2, ipsilateral to the pain (Fig. 45.9). The T12 DRG should be included because it is common for the ilioinguinal and genitofemoral nerves to communicate. If the injection results in significant pain relief (at least 50 % improvement), but is short-lived, cryoneuroablation or pulsed radio frequency may be applied posteriorly to these ganglia (see below) [11].

Ultrasound-Guided Technique

Another approach is to perform a selective injection of the genital branch of the GFN under ultrasound guidance.

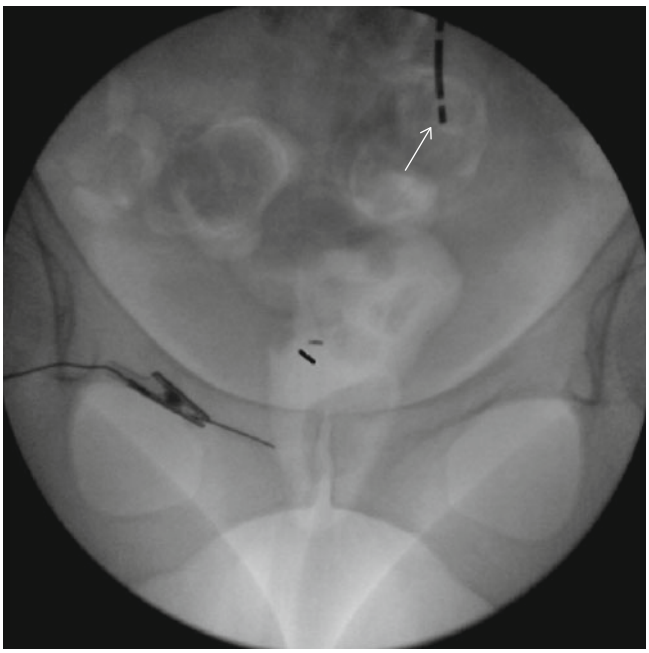

Fig. 45.8 Peripheral nerve stimulation identification of the genitofemoral nerve. Note the *white arrow* showing the InterStim® placed for "interstitial cystitis" pain that offered no relief (Image courtesy of Andrea Trescot, MD)

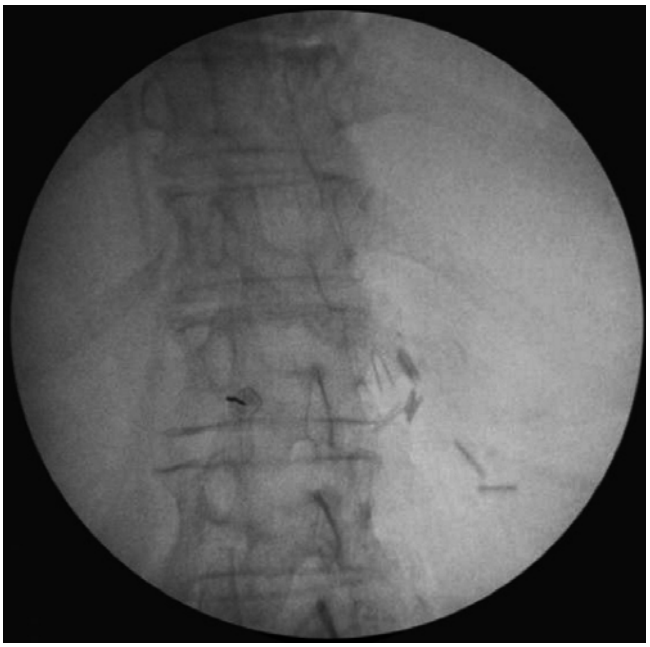

Fig. 45.9 Injection of the dorsal root ganglia at L1 (Image courtesy of Fabrício Assis, MD)

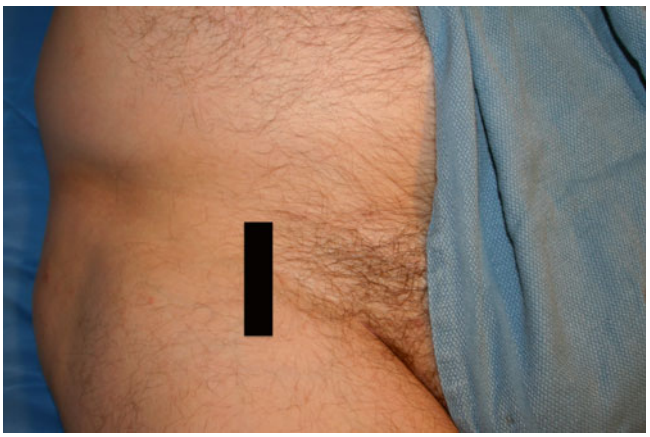

Fig. 45.10 Location of ultrasound probe for genitofemoral identification (Image courtesy of Andrea Trescot, MD)

The genital branch is slender and cannot be directly visualized, but the external iliac artery is easily found at the inguinal canal, and the genital branch is immediately medial to it, within the internal inguinal ring [5,17]. A high-frequency linear probe is utilized, oriented perpendicularly to the inguinal ligament (Fig. 45.10), with its tip about one fingerbreadth lateral to the pubic tubercle. It is suggested that the practitioner start in the internal inguinal ring, where it is possible to visualize the longitudinal (lengthwise) section of the artery. When the probe is advanced cephalad, the artery is seen to penetrate deep inside the inguinal ligament. At this point, an oval or round structure (the cremaster in males, the round ligament in females) superficial to the femoral artery can easily be visualized (Fig. 45.11). The probe is then advanced in the medial direction, slowly, moving away from the femoral artery. The needle may be inserted in-plane (Fig. 45.12) or out-of-plane, injecting local anesthetic without vasoconstrictors, to avoid the adverse effects of testicular artery vasoconstriction. Because of anatomical variability, it is recommended to use 5 mL inside and 5 mL outside the spermatic cord [18]. Shanthanna [19] described the use of US to identify and inject the GFN in a patient with disabling groin pain after testicular cancer; the patient noted excellent relief after the US-guided GFN injection, lasting >12 months.

CT-Guided Technique

A fourth approach is to perform a block of the GFN proximal to the genital-femoral split, while it is still retroperitoneal. This requires CT guidance to avoid inadvertently violating the peritoneum or perforating bowel, as well as the use of a nerve stimulator, since the GF nerve is not visible on CT [9]. The needle entry point is just above the L4 transverse process and, utilizing the nerve stimulator, stimulation radiating to the groin and the upper ipsilateral thigh should be achieved [9]. This technique is limited by the availability of CT-guided technology and is discussed more fully in the GFN abdominal section (Chap. 41).

Neurolytic Techniques

Cryoneuroablation

Cryoneuroablation at the pubic tubercle can be performed by either fluoroscopy-guided or ultrasound-guided techniques. Because the tissue at the pubis is usually relatively thin, it is conceivable that one could identify the GFN by just landmarks and the built-in nerve stimulator on the cryoprobe, but this is not a recommended technique. Trescot [14] described placement of the cryoprobe onto the pubic tubercle, using fluoroscopy and the nerve stimulator to find the nerve (Fig. 45.13). In that same publication, Trescot also described cryoneuroablation at the L1 foramen to treat the GFN proximally (Fig. 45.14). Campos et al. [20] described cryoneuroablation of the femoral branch of the GFN under US guidance. The details of that approach are described more fully in the GFN lower extremity section (Chap. 59).

Over a 2-year time frame, Agnes Stogicza, MD (personal communication), described treating 18 post-herniorrhaphy patients with persistent groin pain using cryoneuroablation placed under US guidance (Fig. 45.15). She often lesioned

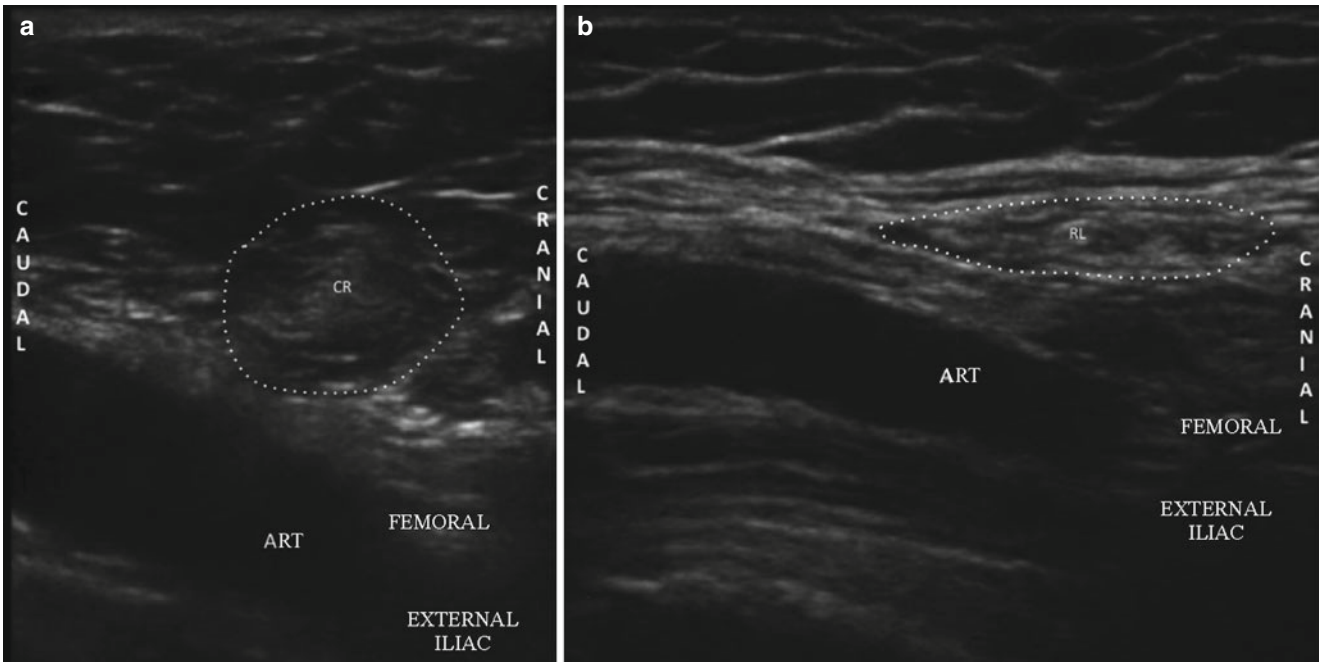

Fig. 45.11 Composite cross-sectional ultrasound image of the internal inguinal ring. *Dotted line* represents the internal inguinal ring; (**a**) male, (**b**) female, *CR* cremaster, *RL* round ligament, *ART* femoral artery (superficial) and external iliac artery (deep) (Image courtesy of Thiago Nouer Frederico, MD)

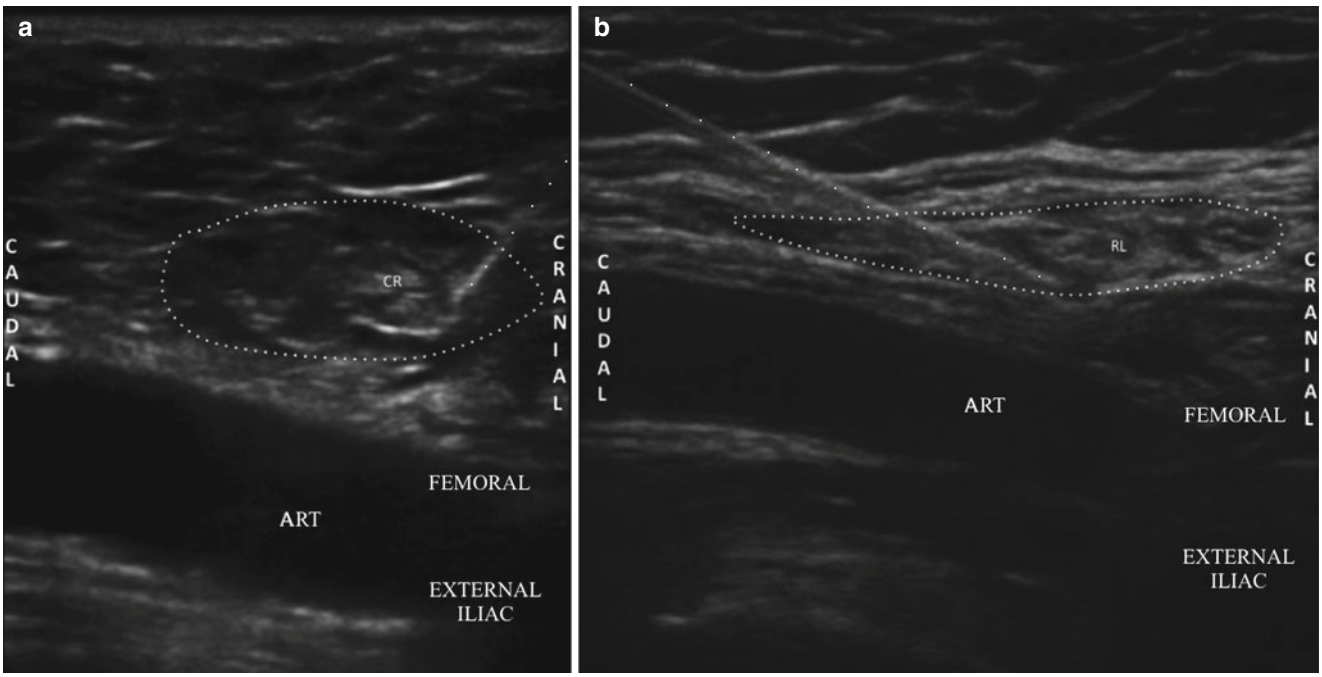

Fig. 45.12 Cross-sectional ultrasound image of the internal inguinal ring filled with local anesthetic and needle tip near the cremaster or round ligament. *Dotted line* represents the internal inguinal ring; (**a**) male, (**b**) female, *CR* cremaster, *RL* round ligament, *ART* femoral artery (superficial) and external iliac artery (deep), (Image courtesy of Thiago Nouer Frederico, MD)

multiple sites, primarily at the GFN but occasionally also at the ilioinguinal nerve. All patients had post-procedure numbness of the scrotum, with increased function and increased ability to lift and carry objects; three patients underwent repeat cryoneuroablation, and these consecutive cryoneuroablations yielded longer-lasting results. Interestingly, she has observed that the pain does not return to the same area that was treated, suggesting an element of "unmasking" (see Chap. 1).

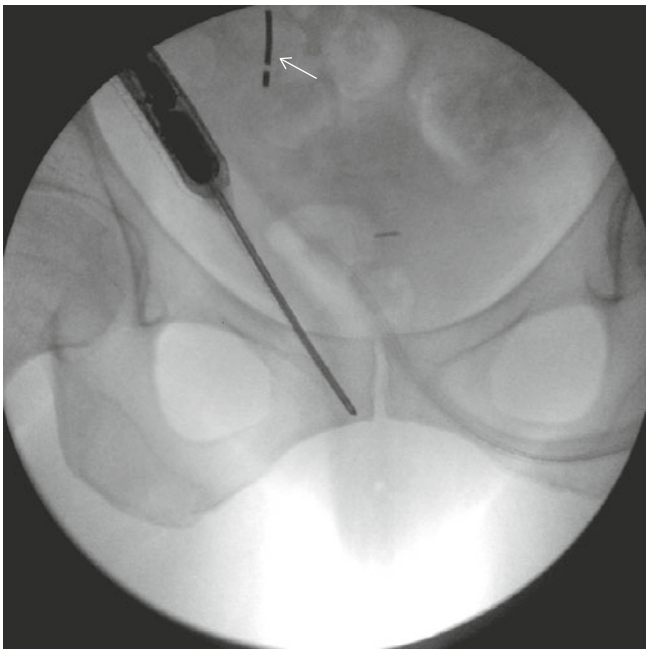

Fig. 45.13 Cryoneuroablation of the genitofemoral nerve at the pubic tubercle. Note the *white arrow* showing the InterStim® placed for "interstitial cystitis" pain that offered no relief (Image courtesy of Andrea Trescot, MD)

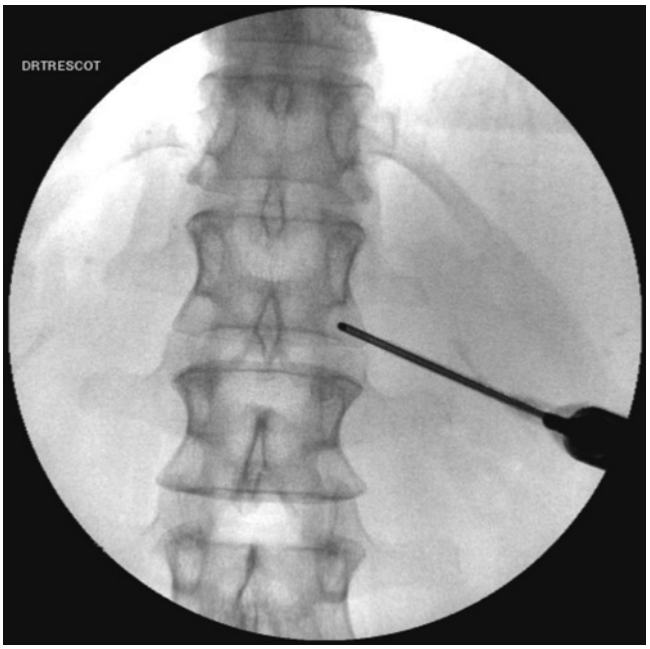

Fig. 45.14 Cryoneuroablation probe positioned on the proximal genitofemoral nerve at L1 (Image courtesy of Andrea Trescot, MD)

Radiofrequency Lesioning

Pulsed radiofrequency treatment of the GFN has been described. Terkawi and Romdhane [21] treated a young man suffering from chronic orchialgia; a diagnostic injection of the genital branch under US guidance (using a technique similar to that described above) gave excellent but only temporary relief. The patient underwent a pulsed RF treatment, again using the same ultrasound technique. At 7 months, the patient was still noting excellent relief.

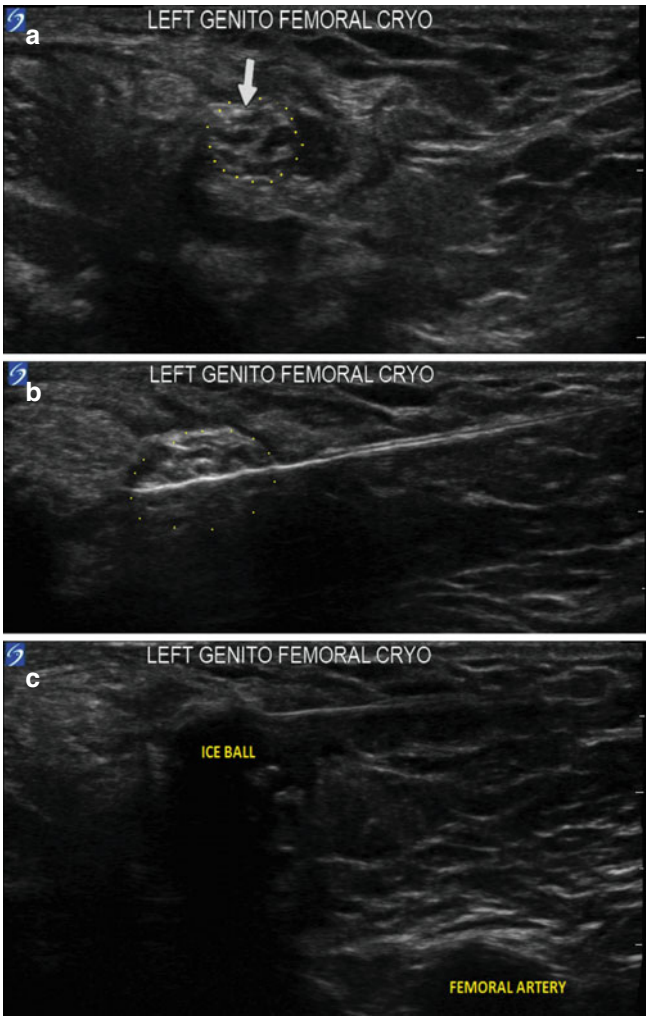

Fig. 45.15 Cryoneuroablation of the genitofemoral nerve at the internal inguinal ring under ultrasound guidance. (**a**) Ultrasound identification of the internal inguinal ring and the genitofemoral nerve (*white arrow*); (**b**) cryoprobe placement; (**c**) cryo-ice ball (Image courtesy of Agnes Stogicza, MD)

Phenol

Weksler and colleagues [22] described injecting 4 % phenol on to a variety of painful structures (including the GFN) in 35 patients; they noted good relief and no complications.

Neurostimulation

Peripheral nerve stimulation has been used for chronic groin and genital pain (Fig. 45.16) [23,24]. The trial electrodes are

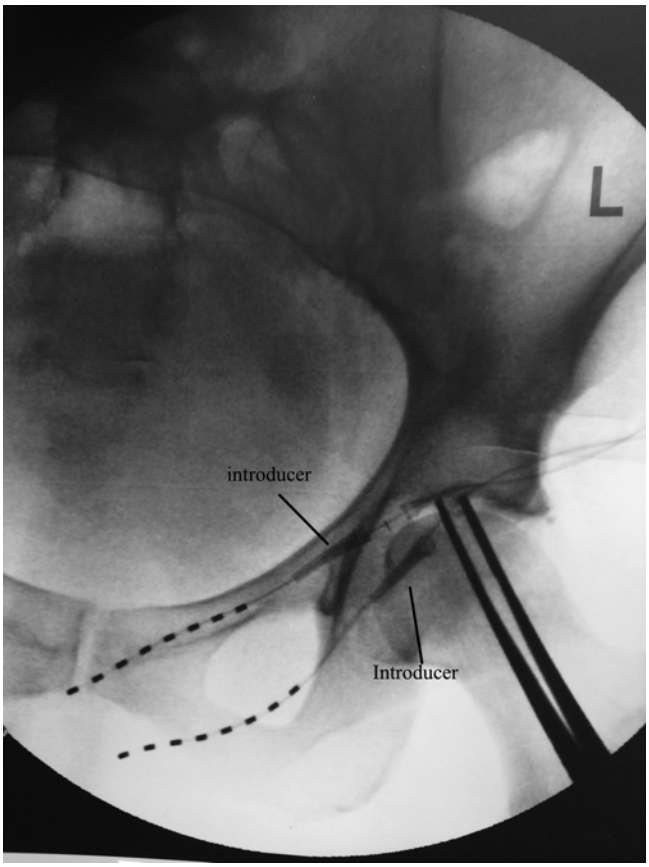

Fig. 45.16 Peripheral nerve stimulation of abdominal wall (Image courtesy of Gladstone MacDowell, MD)

placed percutaneously through introducers, and, if there is significant temporary relief, the leads can be placed permanently. Rosendal et al. [23] described the successful use of a dual lead peripheral nerve stimulator over the ilioinguinal and genitofemoral nerves to treat persistent testicular pain.

Surgical Techniques

Excision of scar neuroma can give relief, but the neuroma is often difficult to localize. One technique that has been successful (Trescot, personal correspondence) is mixing local anesthetic with methylene blue. If the local anesthetic abolishes the pain, then the neuroma is within the dyed tissue, and the surgeon is instructed to "take out everything blue."

Another surgical option is to locate the genitofemoral nerve in the inguinal region, near the round ligament or spermatic cord. Dissection is performed under sedation and local anesthesia, which can prove very useful to identify these little nerve branches, especially in obese patients, by allowing the patient to report when a determined structure reproduces the pain sensations.

Endoscopic retroperitoneal neurectomy (ERN) is a surgical technique involving the insufflation of the retroperitoneal space, allowing neurectomy before the GFN becomes embedded in the psoas muscle [25]. This technique has been used successfully in the treatment of postsurgical groin pain [26]. *Triple neurectomy* (IIN, IHN, and GFN) has also been advocated, with a reported 80 % success rate in relieving postsurgical groin pain [27].

Complications

Any injection may cause the usual complications of bleeding, infection, and nerve damage. Psoas hematoma is a potentially serious complication of nerve blocks but is usually encountered in the setting of anticoagulation. A retroperitoneal technique also runs the risk of peritoneal injury, retroperitoneal hematoma, and lumbar plexus injury. It is thought that CT- or ultrasound-guided techniques have lower rates of complications, but this has not been conclusively proven. ERN may rarely result in genital anesthesia in women [25].

Summary

The GFN is an under-recognized cause of pelvic pain, as well as abdominal pain (see Chap. 41) and lower extremity pain (see Chap. 60). A careful history and physical, as well as a high index of suspicion, will help the clinician to begin to recognize and then treat GFN entrapments.

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References

1. Starling JR, Harms BA. Diagnosis and treatment of genitofemoral and ilioinguinal neuralgia. World J Surg. 1989;13(5):586–91.
2. Alsever JD. Lumbosacral plexopathy after gynecologic surgery: case report and review of the literature. Am J Obstet Gynecol. 1996;174(6):1769–78.
3. Parnell BA, Johnson EA, Zolnoun DA. Genitofemoral and perineal neuralgia after transobturator midurethral sling. Obstet Gynecol. 2012;119(2 Pt 2):428–31.
4. Reinpold WMJ, Nehls J, Eggert A. Nerve management and chronic pain after open inguinal hernia repair: a prospective two phase study. Ann Surg. 2011;254(1):163–8.
5. Recker DC, Perry PM. Postsurgical pain syndromes: chronic pain after hysterectomy and cesarean section. Tech Reg Anesth Pain Manag. 2011;15(3):133–9.
6. Cardosi RJ, Cox CS, Hoffman MS. Postoperative neuropathies after major pelvic surgery. Obstet Gynecol. 2002;100(2):240–4.
7. Magee RK. Genitofemoral Causalgia: (a new syndrome). Can Med Assoc J. 1942;46(4):326–9.
8. Benes J, Nádvornik P, Dolezel J. Abdominoinguinal pain syndrome treated by centrocentral anastomosis. Acta Neurochir (Wien). 2000;142(8):887–91.
9. Parris D, Fischbein N, Mackey S, Carroll I. A novel CT-guided transpsoas approach to diagnostic genitofemoral nerve block and ablation. Pain Med. 2010;11(5):785–9.

10. Rab M, Ebmer J, Dellon LA. Anatomic variability of the ilioinguinal and genitofemoral nerve: implications for the treatment of groin pain. Plast Reconstr Surg. 2001;108(6):1618–23.
11. Rozen D, Parvez U. Pulsed radiofrequency of lumbar nerve roots for treatment of chronic inguinal herniorraphy pain. Pain Physician. 2006;9(2):153–6.
12. Anloague PA, Huijbregts P. Anatomical variations of the lumbar plexus: a descriptive anatomy study with proposed clinical implications. J Man Manip Ther. 2009;17(4):107E–14.
13. Lewit K. Manipulative therapy in rehabilitation of the locomotor system. Oxford: Butterworth-Heinemann; 1991.
14. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
15. Harms BA, DeHaas DR, Starling JR. Diagnosis and management of genitofemoral neuralgia. Arch Surg. 1984;119(3):339–41.
16. Trescot AM. Cryoneurolysis and the healing response. Annual Meeting American Academy of Pain Management; San Diego, 2005.
17. Peng PW, Narouze S. Ultrasound-guided interventional procedures in pain medicine: a review of anatomy, sonoanatomy, and procedures: part I: nonaxial structures. Reg Anesth Pain Med. 2009;34(5):458–74.
18. Peng PW, Tumber PS. Ultrasound-guided interventional procedures for patients with chronic pelvic pain – a description of techniques and review of literature. Pain Physician. 2008;11(2):215–24.
19. Shanthanna H. Successful treatment of genitofemoral neuralgia using ultrasound guided injection: a case report and short review of literature. Case Rep Anesthesiol. 2014;2014:371703.
20. Campos NA, Chiles JH, Plunkett AR. Ultrasound-guided cryoablation of genitofemoral nerve for chronic inguinal pain. Pain Physician. 2009;12(6):997–1000.
21. Terkawi AS, Romdhane K. Ultrasound-guided pulsed radiofrequency ablation of the genital branch of the genitofemoral nerve for treatment of intractable orchalgia. Saudi J Anaesth. 2014;8(2):294–8.
22. Weksler N, Klein M, Gurevitch B, Rozentsveig V, Rudich Z, Brill S, et al. Phenol neurolysis for severe chronic nonmalignant pain: is the old also obsolete? Pain Med. 2007;8(4):332–7.
23. Rosendal F, Moir L, de Pennington N, Green AL, Aziz TZ. Successful treatment of testicular pain with peripheral nerve stimulation of the cutaneous branch of the ilioinguinal and genital branch of the genitofemoral nerves. Neuromodulation. 2013;16(2):121–4.
24. Banh DP, Moujan PM, Haque Q, Han TH. Permanent implantation of peripheral nerve stimulator for combat injury-related ilioinguinal neuralgia. Pain Physician. 2013;16(6):E789–91.
25. Muto C, Pedana N, Scarpelli S, Galardo R, Guida G, Schiavone V. Inguinal neurectomy for nerve entrapment after open/laparoscopic hernia repair using retroperitoneal endoscopic approach. Surg Endosc Interv Tech. 2005;19(7):974–6.
26. Giger U, Wente M, Büchler M, Krähenbühl S, Lerut J, Krähenbühl L. Endoscopic retroperitoneal neurectomy for chronic pain after groin surgery. Br J Surg. 2009;96(9):1076–81.
27. Amid P. Causes, prevention, and surgical treatment of postherniorrhaphy neuropathic inguinodynia: triple neurectomy with proximal end implantation. Hernia. 2004;8(4):343–9.

Posterior Femoral Cutaneous Nerve Entrapment: Pelvic

46

Natalia Murinova, Daniel Krashin, and Andrea M. Trescot

Introduction

Posterior femoral cutaneous nerve (PFCN) neuropathy is an uncommon, potentially under-recognized cause of posterior and lateral thigh pain and numbness. The PFCN provides sensation to the posterior thigh and perineal region, mimicking sciatic and pudendal nerve pathologies. It is rare for PFCN entrapment to present as an isolated neuropathy. In the literature, the most common reported causes of PFCN injury have been intramuscular injections or pressure. Alternate names for this nerve and syndrome include *lesser sciatic nerve entrapment* and *posterior cutaneous nerve of the thigh*. The perineal branch of the PFCN has been called the *inferior pudendal nerve*, *pudendal longus inferior*, *long pudendal nerve*, or *nerve of Soemmering*. It is common to have injury of the inferior pudendal nerve along with injury of the PFCN neuropathy. Consider PFCN entrapment neuropathy in a patient presenting with posterior and lateral thigh pain.

Clinical Presentation (Table 46.1)

PFCN entrapment presents as posterior thigh pain, from the gluteal fold to the back of the knee down to a variable amount of the posterior calf (Fig. 46.1). There can be sensory abnormalities localized to the lower buttock and the posterior thigh, as well as the posterior thigh of the upper leg (Fig. 46.2). The PFCN, through its perineal branch, also innervates the perineal region (Fig. 46.3). PFCN injury has been reported due to trauma from intramuscular gluteal injections [1]. This nerve is traumatized by prolonged bicycle rides, pelvic tumors, venous malformation, trauma, and idiopathic causes (Table 46.1) [6, 7]. Other cases may be due to pressure on the nerve at the inferior margin of the gluteus maximus due to sitting on hard surfaces [5]. It can also present as perineal pain, including the rectum, scrotum or labia majora, and penis or clitoris. Isolated lesions of the PFCN are described in a small number of individual case studies in the literature (Table 46.2).

Table 46.1 Occupation/exercise/trauma history relevant to posterior femoral cutaneous nerve entrapment

Trauma	Intramuscular gluteal injection [1–3]
	Pelvic fracture
Compression	Pelvic tumor [4]
	Venous malformation
	Sitting on a hard surface [5]
	Prolonged bicycle rides [5]
Stretch injury	Gymnastics [5]

Fig. 46.1 Patient-described pattern from posterior femoral cutaneous nerve entrapment (Image courtesy of Andrea Trescot, MD)

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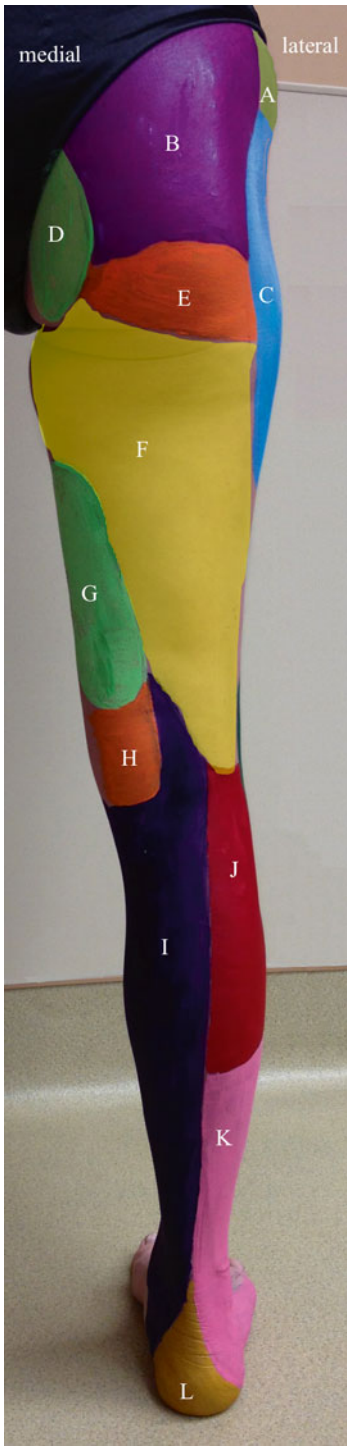

Fig. 46.2 Pain pattern from nerves of posterior leg. *A* lateral branch iliohypogastric nerve, *B* superior cluneal nerve, *C* lateral femoral cutaneous nerve, *D* middle cluneal/sacral nerve, *E* inferior cluneal nerve, *F* posterior femoral cutaneous nerve, *G* obturator nerve, *H* femoral nerve, *I* saphenous nerve, *J* lateral sural cutaneous nerve, *K* superficial peroneal nerve, *L* medial calcaneal nerve (Image courtesy of Terri Dallas-Prunskis, MD)

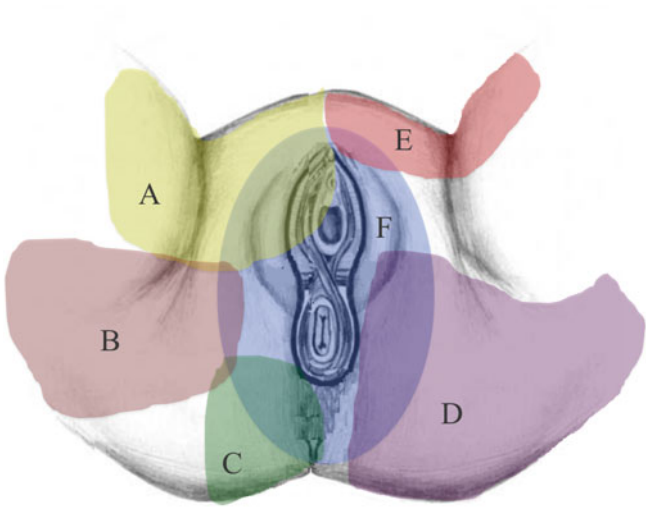

Fig. 46.3 Innervation of perineum: (*A*) genitofemoral nerve; (*B*) obturator nerve; (*C*) inferior cluneal nerve; (*D*) peroneal branch of the posterior femoral cutaneous nerve, (*E*) ilioinguinal nerve, and (*F*) pudendal nerve (Image courtesy of Andrea Trescot, MD)

Anatomy (Table 46.3)

The PFCN is a pure sensory nerve, which originates from the posterior rami of S1–S3 [10] (Table 46.3). The anatomy of PFCN is very variable, and it may involve branches as high as L4 or as low as S4 [11]. The PFCN exits the pelvis beneath the gluteus maximus with the inferior gluteal artery, through the sciatic foramen, below the piriformis muscle, and passes down the buttock and thigh on the medial aspect of the sciatic nerve; it is sometimes referred to as the "*lesser sciatic nerve*" (Fig. 46.4). The *sciatic foramen* is divided into two parts by the piriformis muscle (*suprapiriformis foramen* and *infrapiriformis foramen*). The PFCN reaches the dorsal part of the pelvis in close proximity to the ischial tuberosity as it passes though the infra-piriformis foramen. Deep to the gluteus maximus muscle, the PFCN gives off the *inferior cluneal nerve* (Chap. 51) and the *perineal branch of the PFCN* (PBPFCN). The inferior cluneal nerve provides the cutaneous innervation of the inferior buttocks, while the perineal branch innervates the lateral perineum, the proximal medial thigh, the posterolateral scrotum or labium majora, and part of the penis or clitoris (Fig. 46.3) (Table 46.4) [12].

The perineal branch courses medially, staying about 4 cm inferior to the attachment of the sacrotuberous ligament onto the ischial tuberosity, parallel to the ischial ramus [12]. However, Bergman et al. [13] described the perineal branch piercing the sacrotuberous ligament,

Table 46.2 Cases in literature regarding different causes of neuropathy of posterior femoral cutaneous nerve, most of them are case presentations

Different causes of PFCN neuropathy	Symptoms of patients presenting with PFCN	Article
1. Injury of the PFCN after gluteal injection	54-year-old man developed hyperalgesia of posterior thigh and lateral scrotum	Iyer, VG et al. Isolated injection injury. 1989. [2]
2. Injury of the PFCN after gluteal injection – describes complete syndrome of the infra-piriformis foramen	137 cases of sciatic nerve injury, including two with PFCN lesions	Obach, J et al. The infra-piriformis foramen syndrome from intragluteal injection. 1983. [8]
3. Injury of PFCN from compression of the nerve by the ischial tuberosity, thought to arise during exercise, especially sitting and biking, as per speculation of Arnoldussen et al.	37-year-old female compressing her PFCN with gymnastics; 47-year-old sedentary male with bilateral hyperpathia; 54-year-old male riding bicycle 30 miles daily with pain in the posterior thigh and paresthesias in lower buttocks and lateral scrotum	Arnoldussen, WJ. Pressure neuropathy. 1980. [5]
4. Unknown etiology	40-year-old woman with decreased sensation in the right posterior thigh after a left putamen (thalamic) hemorrhage 4 years prior	Dumitru, D et al. 1988. [9]
5. Injury of PFCN after gluteal intramuscular injections (after two right gluteal intramuscular injections)	25-year-old woman with posterior/lateral thigh decreased sensation	Tong, H. 2000. [1]
6. Injury of PFCN after gluteal intramuscular injection	22-year-old woman with pain, lack of sensation in posterior thigh and lower half of buttock; had abnormal nerve conduction study	Kim, JE. 2009. [3]

Table 46.3 Posterior femoral cutaneous nerve anatomy

Origin	Posterior rami S1–S3 (but may include up to L4 and down to S4)
General route	Exits the pelvis anterior to the piriformis but posterolateral to the sciatic nerve; gives off inferior cluneal nerve and perineal branch of the PFCN. Travels down the posterior thigh between the medial and lateral hamstring muscles, joining the sural nerve, occasionally down to calcaneus
Sensory distribution	Inferior buttocks, lateral perineum, proximal medial thigh, posterolateral scrotum or labia, and part of the penis or clitoris
Motor innervation	None
Anatomic variability	Perineal branch can come from the inferior cluneal
Other relevant structures	Sacrotuberous ligament, piriformis muscle, pudendal nerve

which would place it close to the *pudendal nerve* in *Adcock's canal* (Chap. 47).

The rest of the nerve then continues inferiorly from the lower edge of the gluteus maximus in a muscular groove formed by the medial and lateral hamstring muscles, with branches providing the sensation to the posterior thigh. The PFCN extends a variable distance into the calf, becoming superficial near the popliteal fossa, joining with the *lateral sural* (Chap. 72) and the *saphenous nerves* (Chap. 58) to innervate the posterior calf. Kosinski [14] described the PFCN as occasionally extending all the way to the calcaneal region.

The *main trunk of the PFCN* to the back of the thigh and leg consists of numerous filaments derived from both sides of the nerve, and it distributes to the skin covering the back and medial side of the thigh, the popliteal fossa, and the upper part of the back of the leg.

A study by Nakamishi et al. [15] described the dissection of 37 Japanese subjects, which showed that the origin of PFCN was variable. They found that the PFCN may receive root components from segments of S1 through S4, and it does not arise from S1 alone.

Tubbs et al. [12] dissected 20 cadavers and found that the perineal branch of the PFCN (PBPFCN) arose directly from the PFCN in 55 % of the sides; in 30 % of the sides, it arose from the inferior cluneal nerve; and it was absent in 15 % of sides (Table 46.5). The PBPFCN is described to provide two to three branches to the medial thigh and then innervate the scrotum or labia major. In males, one nerve branch traveled inferior to the corpora cavernosa and anterior to the spermatic cord to cross the midline. Communications between the PBPFCN and the perineal branch of the pudendal nerve are common.

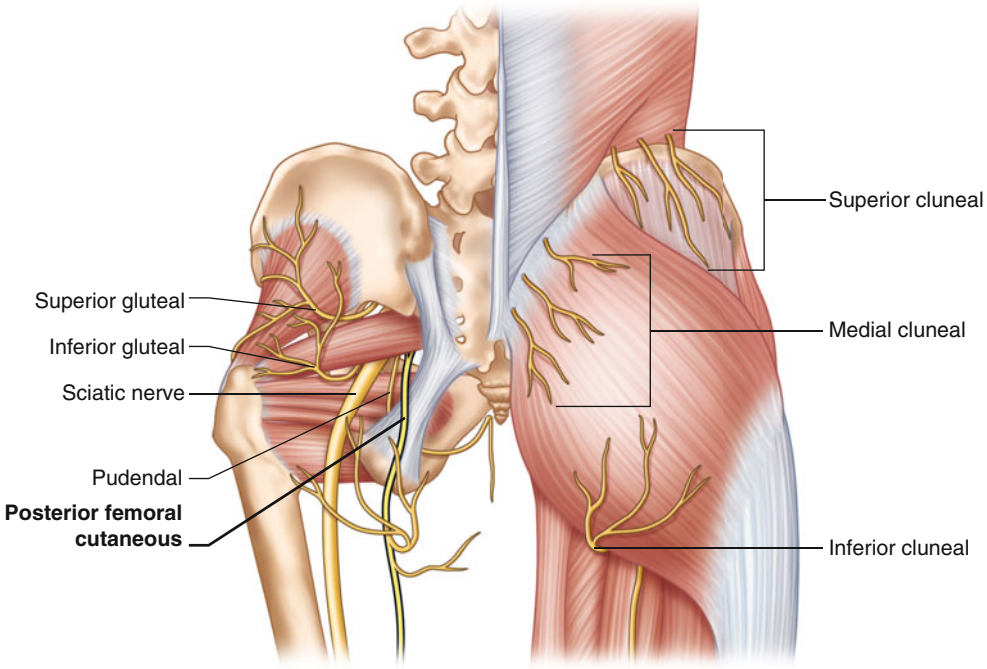

Fig. 46.4 Anatomy of the buttocks and pelvis (Image by Springer)

Table 46.4 Sensory innervation of the branches of the posterior femoral cutaneous nerve

Branches of PFCN	Sensory innervation
Major branch of PFCN	Back and medial side of the thigh
	Popliteal fossa
	Upper part of the back of the leg
Inferior cluneal nerve of PFCN	Inferior buttocks
Perineal branch of PFCN	Lateral perineum
	Upper and medial thigh
	Posterolateral scrotum
	Labium majora
	Part of the penis or clitoris
Gluteal branches of PFCN	Lower and lateral part of gluteus maximus
Other name: inferior cluneal nerves	
Collateral branches from the anterior divisions	Quadratus femoris
	Inferior gemmelus muscles (L4, L5, S1)
	Internal obturator
	Superior gemmelus muscles (L5, S1, S2)

Entrapment

The posterior femoral cutaneous nerve lies adjacent to the sciatic nerve and can be injured by prolonged tourniquet time or, like the sciatic nerve, may be compressed by the *piriformis muscle*. Compression of the nerve can occur at the ischial tuberosity, such as by prolonged sitting on the edge of a chair [5]. Gluteal intramuscular injections have been reported to injure the PFCN and present as thigh and scrotal or labial pain [2].

Table 46.5 Origin of the perineal branch of the posterior femoral cutaneous nerve (PBPFCN)

From PFCN directly	55 %
From inferior cluneal nerve	30 %
Absent	15 %

Data from Tubbs et al. [12] study of 20 dissected adult cadavers (40 sides)

Both the pudendal nerve and the PFCN leave the pelvis together through the greater sciatic foramen after passing through the infra-piriformis canal, so they can be trapped together along that path. They also share innervation of the perineal region. As early as 1900, Cushing [16] suggested that pain after ligation of testicular veins might be due to trauma of the perineal branch of PFCN.

Physical Exam

Because the nerve lies deep in the thigh, it is relatively hard to examine the PFCN in the thigh by palpation. However, at the level of the ischium, the nerve can be palpated between the heads of the hamstring muscles (Fig. 46.5). In the case reports, only two mentioned that there was numbness in the region of the PFCN [5, 9]. Two cases had decreased sensation in the posterior thigh and lower buttock. This suggests that in a significant number of posterior femoral cutaneous

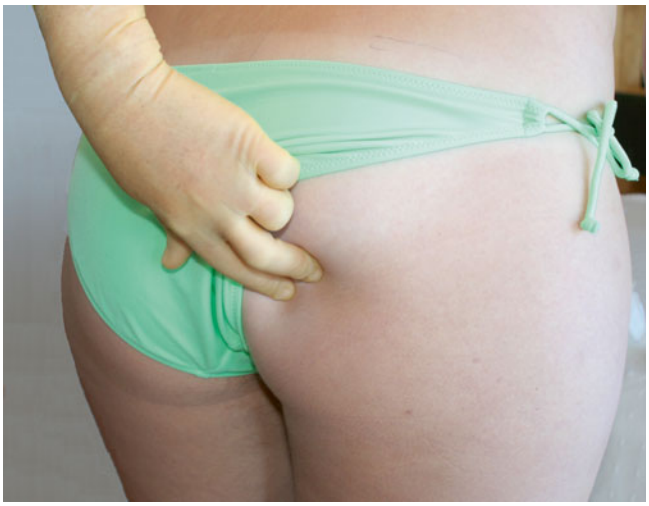

Fig. 46.5 Palpation of the posterior femoral cutaneous nerve at the ischium (Image courtesy of Andrea Trescot, MD)

neuropathies, the inferior cluneal nerve is also affected. On physical examination, look for distribution of pain and sensory abnormality, localized in the area corresponding to the particular branch of PFCN that is injured, such as the lower buttock, the posterior thigh, and the dorsal surface of the upper leg. The rest of the physical examination should be normal, including motor strength and reflexes.

Differential Diagnosis (Table 46.5)

The PFCN may be responsible for many symptoms that are attributed to sciatic nerve and "*piriformis syndrome*" (Table 46.6). Piriformis syndrome presents with pain in the gluteal area, which may mimic PFCN neuropathy. However, with piriformis syndrome, the pain can be worsened by internal rotation and flexion at the hip. Pressure over the greater sciatic foramen also elicits local pain in the piriformis syndrome. Often, there is tenderness to palpation along the course of the nerve at the level of the thigh. The *perineal branch of the PFCN* can mimic *pudendal nerve* pathology, and it shares much of the same innervation. Table 46.6 discusses the distinguishing features of the differential diagnosis, and Table 46.7 describes the diagnostic tests.

Dumitru and Marquis [9] described somatosensory evoked potential (SSEP) evaluation of the PFCN. A recording electrode is placed 6 cm proximal to the midpopliteal fossa, and the nerve is stimulated supramaximally 12 cm proximally on a line between the active electrode and the ischial tuberosity. A ground electrode is placed just proximal to the active recording electrode. The lower extremities of 40 individuals with a mean age of 34 years (20–78 years) were examined. The mean peak latency of the response is 2.8 (2.3–3.4) ms ± 0.2 ms, with a mean amplitude of 6.5 (4.1–12.0) microV ± 1.5 microV.

Table 46.6 Differential diagnosis of low back and perineal pain

S1	Lower back pain
S1 only	Buttock pain
	Pain, numbness, or weakness in various parts of the leg and foot
	Pain may radiate below the knee, but not always
Pudendal neuropathy	Pelvic pain
S2, S3, and S4 responsible for perineal innervation	Pain worse with sitting and driving
	Pain reduced by sitting on a toilet seat
	Vaginal pain with intercourse
	Bladder pain during micturition
	Rectal pain during defecation
	Bowel, bladder, and sexual dysfunction
PFCN neuropathy	Back and medial side of the thigh
S1, S2, and S3/S4	Popliteal fossa
	Upper part of the back of the leg
	Inferior buttocks
	Normal motor exam
	No problem with urination/defecation/sex function
Piriformis syndrome	Pain worse with internal rotation and flexion of the hip

Table 46.7 Diagnostic tests for posterior femoral cutaneous nerve

	Potential distinguishing features
Physical exam	Tenderness between the heads of the hamstring muscles at the ischium
Diagnostic injection	At the ischium
Ultrasound	Not described
MRI	Not useful
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies	SSEP

Identification and Treatment of Contributing Factors

Contributing factors for PFCN entrapment include:

- Iatrogenic trauma from gluteal injections
- Pressure on the ischial area from prolonged bicycle riding or prolonged sitting
- Anatomic variations
- Smoking
- Diabetes
- Poor circulation
- Cachexia or excessive muscle loss that functions as cushion for the nerve
- Obesity

Iatrogenic trauma can be prevented with careful intramuscular injections in the gluteal area. Tourniquet times and pressures should be closely monitored to prevent ischemia of the PFCN. Prevent pressure into the ischial area from prolonged bicycle riding or prolonged sitting, especially in people who are prone to peripheral nerve injuries due to genetic and other medical conditions such as diabetes, poor circulation, and smoking. It is important to educate patients that if they experience recurrent pain in the gluteal area due to exercise, they might be overstretching the PFCN or causing entrapment of PFCN. Also for bikers with gluteal pain, the recommendation is to use a cushion that can prevent compression due to excessive pressure. It is likely that athletes such as rowers, bikers, and other athletes with excessive compression of the gluteal area are especially prone to this injury, which might be misdiagnosed as pudendal neuralgia (Chap. 47) and other causes of pelvic pain.

Injection Technique

The choice of approach to the PFCN depends on the proposed site of entrapment; in other words, entrapment in the sciatic foramen would be targeted differently than pathology at the ischium, perineum, or distal thigh. The types of PFCN injections are summarized in Table 46.8.

Landmark-Guided Technique

There was only one study found on PubMed describing the landmark-guided injection technique of PFCN. Hughes et al. [17] described blocking the PFCN at the point where its branches come from below the medial border of gluteus maximus. This location is found by Hughes to be a quarter of the distance from the ischial tuberosity to the greater trochanter in the gluteal fold (Fig. 46.6). They suggested feeling two distinct losses of resistance as superficial and deep fascia are penetrated with a short-beveled needle. The perineal branch of the PFCN is usually injected at the ischial tuberosity [12].

Table 46.8 Types of posterior femoral cutaneous nerve blocks

Type of nerve block and comments	Study
Landmark-guided technique	There was only one study describing using the landmark-guided injection technique of PFCN searching PubMed [17]
Fluoroscopy-guided technique	No literature found
Ultrasound-guided technique	No literature found
CT-guided PFCN injection – see *CT-Guided Technique* section	Block of PFCN in two patients [19]; no improvement of their symptoms
MRI-guided PFCN block – see *MRI-Guided Technique* section	Fritz et al. performed MRI-guided PFCN injections technically successful in 12/12 cases (100 %) with uniform perineural distribution of the injectant
	No comment on improvement of pain [18]

Fig. 46.6 Landmark-guided injection of the posterior femoral cutaneous nerve at the ischium (Image courtesy of Andrea Trescot, MD)

The choice of approach to the PFCN depends on the proposed site of entrapment. For patients presenting with pain and paresthesia in the perineal area, consider injecting the PFCN 2 cm below the ischial tuberosity, proximal to the PBPFCN [18]. In patients with inferior lateral buttock area pain and numbness, target the cluneal nerves under the gluteus maximus muscle. In patients presenting with pain in the posterior thighs, the most likely area of involvement is the distal portion of the PFCN [18]. This area is located between the gluteus maximus and the long head of the biceps femoris, and it is the area that should be considered for injection [18]. Based on cadaver studies, Tubbs et al. suggested that the PBPFCN was, on average, located 4 cm inferior to the ischial tuberosity; they recommend injecting this area with anesthetic to block this nerve [12].

Fluoroscopy-Guided Technique

There are no good fluoroscopic landmarks other than the ischium (Fig. 46.7).

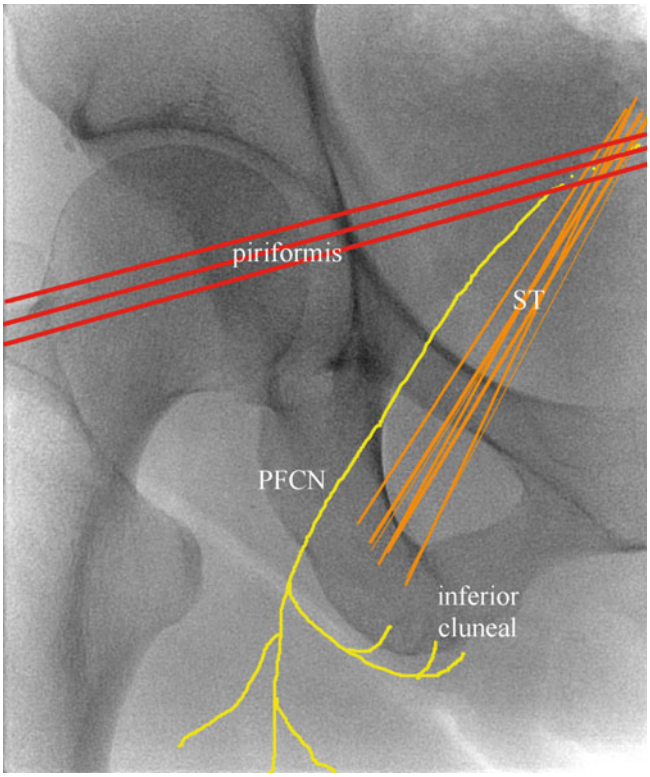

Fig. 46.7 Fluoroscopic landmarks of the posterior femoral nerve (Image courtesy of Andrea Trescot, MD)

Ultrasound-Guided Technique

The sciatic nerve is well visualized by ultrasound, but there are no reported US approaches of the PFCN.

CT-Guided Technique

Kasper et al. [19] describe CT-guided PFCN block in two patients. The PFCN neurovascular bundle was traced from the inferior margin of the ipsilateral gluteus maximus muscle, in between the hamstring tendon origin and the sciatic nerve. After aseptic precautions, a 22-gauge needle was advanced to the above-described position, and the location was documented by injecting 1 cc of 50 % diluted nonionic water-soluble contrast. One patient had decreased sharp sensation in the inferior gluteal region, as well as the posterior thigh (inferior cluneal nerve and PFCN distribution) [19]. Neither of the two patients had improvement of their pain intensity.

MRI-Guided Technique

Fritz et al. [18] performed MRI-guided PFCN injections technically successfully in 12 out of 12 cases (100 % success), with uniform perineural distribution of the injectate (Table 46.8).

All procedures were performed using a clinical, wide-bore 1.5-T MR imaging system with patients in prone position and the table landmark centered at the inferior buttocks area. The course of the PFCN was mapped. Superficial local anesthesia was used, and a 20-gauge needle of 10- or 15 cm length was used in the immediate vicinity of the PFCN. In all patients and locations, a total amount of 4 mL was injected around the PFCN, consisting of 1 mL of 1 % preservative-free lidocaine, 1 mL of 0.5 % bupivacaine, and 1 mL of non-particulate dexamethasone (10 mg/mL).

Neurolytic Technique

There are no described neurolytic techniques, although cryoneuroablation or pulsed radiofrequency lesioning would theoretically be reasonable options.

Surgical Technique

Mobbs et al. [20] described one case of successful surgery of PFCN entrapment in a 51-year-old healthy male that presented with a 12-month history of shooting pains down the posterior and lateral aspect of the thigh and buttock. The pain extended to the knee and to the lateral thigh; lower limb power was intact. The somatosensory evoked potentials of the PCFN demonstrated response differences consistent with entrapment neuropathy. Tubbs et al. [12] performed an open exploration of the PFCN. After identification of the sciatic nerve, they identified the PFCN, as it lies superficial to the sciatic nerve in its proximal course. Although no site of entrapment was obvious, release of the fascia over the nerve was successful in their patient, with complete resolution of his symptoms at a 12-month follow-up.

Complications

There were no complications of the blind technique described in the literature; however, since there are multiple nerves in this area, the main concerns are neurological. There were no complications using CT-guided or MRI-guided injections of the PFCN during the procedure or during follow-up [18].

Summary

PFCN is rarely considered as a cause of pelvic pain, which probably contributes to the limited available literature. PFCN entrapment should be considered in the patient with pelvic pain and posterior thigh pain.

References

1. Tong HC, Haig A. Posterior femoral cutaneous nerve mononeuropathy: a case report. Arch Phys Med Rehabil. 2000;81(8):1117–8.
2. Iyer VG, Shields CB. Isolated injection injury to the posterior femoral cutaneous nerve. Neurosurgery. 1989;25(5):835–8.
3. Kim JE, Kang JH, Chol Choi J, Seok Lee J, Kang SY. Isolated posterior femoral cutaneous neuropathy following intragluteal injection. Muscle Nerve. 2009;40(5):864–6.
4. LaBan MM, Meerschaert JR, Taylor RS. Electromyographic evidence of inferior gluteal nerve compromise: an early representation of recurrent colorectal carcinoma. Arch Phys Med Rehabil. 1982;63(1):33–5.
5. Arnoldussen W, Korten J. Pressure neuropathy of the posterior femoral cutaneous nerve. Clin Neurol Neurosurg. 1980;82(1):57–60.
6. Schuchmann JA. Sural nerve condition: a standardized technique. Arch Phys Med Rehabil. 1977;58:166–8.
7. Chutkow JG. Posterior femoral cutaneous neuralgia. Muscle Nerve. 1988;11:1146–8.
8. Obach J, Aragones JM, Ruano D. The infrapiriformis foramen syndrome resulting from intragluteal injection. J Neurol Sci. 1983;58(1):135–42.
9. Dumitru D, Marquis S. Posterior femoral cutaneous nerve neuropathy and somatosensory evoked potentials. Arch Phys Med Rehabil. 1988;69(1):44.
10. Enneking FK, Chan V, Greger J, Hadzic A, Lang SA, Horlocker TT. Lower-extremity peripheral nerve blockade: essentials of our current understanding. Reg Anesth Pain Med. 2005;30(1):4–35.
11. Hollinshead WH. Anatomy for surgeons: the back and limbs. 3rd ed. Philadelphia: Harper & Row; 1982.
12. Tubbs RS, Miller J, Loukas M, Shoja MM, Shokouhi G, Cohen-Gadol AA. Surgical and anatomical landmarks for the perineal branch of the posterior femoral cutaneous nerve: implications in perineal pain syndromes. Laboratory investigation. J Neurosurg. 2009;111(2):332–5.
13. Bergman RA, Afifi AK, Miyauchi R. Posterior femoral cutaneous nerve 2014 [Jan 4, 2014]. Available from: http://www.anatomyatlases.org/AnatomicVariants/NervousSystem/Text/PosteriorFemoralCutaneous.shtml.
14. Kosinski C. Course, mutual relations and distribution of the cutaneous nerves of the metazonal regions of leg and foot. J Anat. 1926;60:274–97.
15. Nakanishi T, Kanno Y, Kaneshige. Comparative morphological remarks on the origin of the posterior femoral cutaneous nerve. Anat Anz. 1976;139(1–2):8–23.
16. Cushing HI. The employment of local anaesthesia in the radical cure of certain cases of hernia, with a note upon the nervous anatomy of the inguinal region. Ann Surg. 1900;31(1):1–34.
17. Hughes P, Brown T. An approach to posterior femoral cutaneous nerve block. Anaesth Intensive Care. 1986;14(4):350–1.
18. Fritz J, Bizzell C, Kathuria S, Flammang AJ, Williams EH, Belzberg AJ, et al. High-resolution magnetic resonance-guided posterior femoral cutaneous nerve blocks. Skeletal Radiol. 2013;42(4):579–86.
19. Kasper JM, Wadhwa V, Scott KM, Chhabra A. Clunealgia: CT guided therapeutic posterior femoral cutaneous nerve block. Clin Imaging. 2014;38(4):540–42.
20. Mobbs R, Szkandera B, Blum P. Posterior femoral cutaneous nerve entrapment neuropathy: operative exposure and technique. Br J Neurosurg. 2002;16(3):309–11.

Pudendal Nerve Entrapment

47

Susanti K. Chowdhury and Andrea M. Trescot

Introduction

The *pudendal nerve* (PN) is a mixed sensory, motor, and autonomic nerve, supplying innervation to much of the pelvis. *Pudendal neuralgia* (PNa) (also called *Alcock's syndrome*) is a painful condition caused by PN entrapment that can often be difficult to diagnose and is essentially a clinical diagnosis. The majority of patients suffering from this are female; chronic pelvic pain affects approximately one in seven women [1]. Frequently, these patients have seen multiple doctors with a complaint of chronic pelvic pain, which usually earns them multiple diagnoses and treatments without resolution of symptoms [2]. Finally, this elusive diagnosis has been recognized as valid, and multiple modalities exist for treatment. *The International Pudendal Neuropathy Association* (TIPNA.org) estimates the incidence of PNa to be 1:100,000, but, because it is often overlooked as a diagnosis, the incidence may be much higher [3]. There are multiple sites of *pudendal nerve entrapment* (PNE), and the presentation may be different with different entrapments.

Clinical Presentation (Table 47.1)

The PN is a mixed sensory and motor nerve with numerous potential entrapment sites. Therefore, the occupational and trauma history (Table 47.1) varies. In 2008, a group of clinicians (the *Nantes group*) [13] set up diagnostic criteria for PN entrapment (PNE) (see below). Patients with PNa typically describe pain in the labia, penis, scrotum, perineum, or anorectal region. Dr. Jack McDonald, chairman of anesthesiology and OB-GYN at the University of California in Los Angeles (Harborview), proposed in 2005 [14] that PNa is an entrapment, analogous to median nerve compression at the wrist. He described PNE as a pelvic pain that is aggravated by sitting, relieved by standing, and absent when lying down or sitting on the toilet [15]. The pain of PNa typically waxes and wanes and is often described as burning, tearing, stabbing, sharp, electrical, and shooting, along with feelings of "a lump" or foreign body in the vagina or rectum. The concept of entrapment is further supported by a questionnaire of 160 male long-distance amateur cyclists participating in a 540-km bicycle race; symptoms of impotence from pudendal neuralgia were described by 22 %, while hand numbness (consistent with median or ulnar entrapment) was reported by 30 % [16].

Symptoms include abnormal temperature sensations, constipation, pain and straining with bowel movements, burning when urinating, painful intercourse, and sexual dysfunction (including uncomfortable arousal, decreased sensation, or impotence). There may also be voiding dysfunction [17].

Table 47.1 Occupation/exercise/trauma history relevant to pudendal nerve entrapment

Direct compression	Bicycle riders [4]
	Prolonged sitting
Mechanical trauma	Posterior sciatic nerve block [5]
	Pelvic trauma [3, 6]
	Chronic constipation [4]
Surgery	Gynecological surgery [3]
	Hip arthroscopy [7]
	Laser prostatectomy [8]
Stretch pathology	Childbirth [3, 9]
	Proctalgia fugax [9]
Viral infection	Postherpetic neuralgia [10]
Immunologic condition	Inflammatory demyelinating polyneuropathy [11]
Entrapment	Pelvic floor spasm
	Scar tissue
Vascular compromise	Diabetes
	Radiation damage [12]

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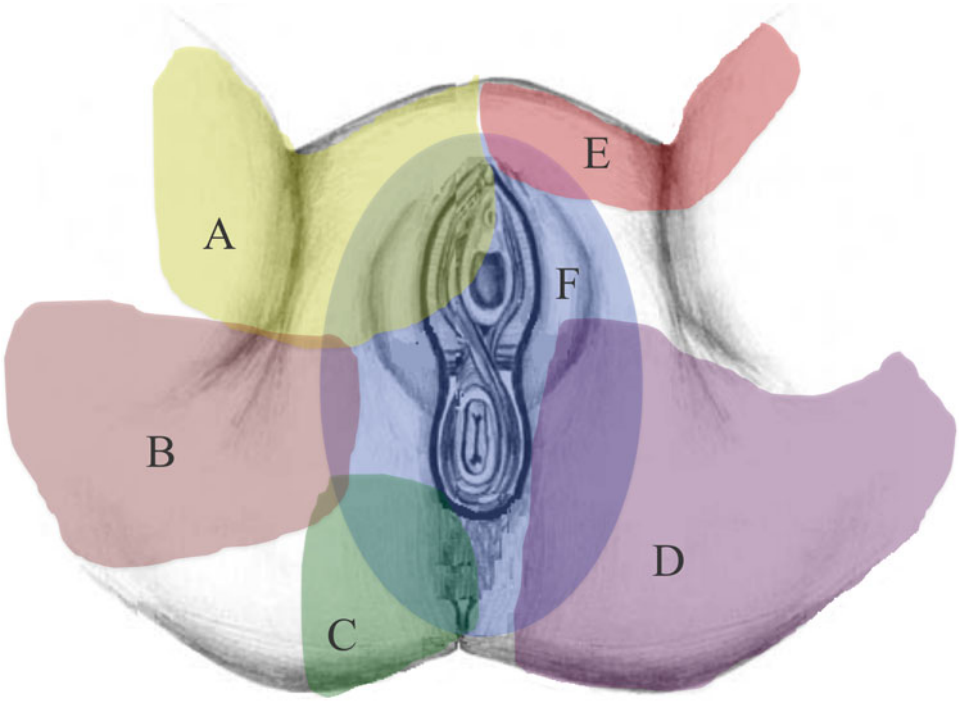

Fig. 47.1 Innervation of the perineum: *A* genitofemoral nerve, *B* obturator nerve, *C* inferior cluneal nerve, *D* peroneal branch of the posterior femoral cutaneous nerve, *E* ilioinguinal nerve, and *F* pudendal nerve (Image inspired by Hibner et al. [3], courtesy of Andrea Trescot, MD)

Additionally, patients may experience hyperesthesia and allodynia so intense that they avoid wearing certain clothing that may irritate the area. There can be overlap between the ilioinguinal (Chap. 44), genitofemoral (Chap. 45), inferior cluneal (Chap. 51), and obturator nerve (Chap. 48) entrapments (Fig. 47.1). PNE may be one of the causes of *vulvodynia*, the name for chronic vulvar and vaginal introitus burning, often associated with pelvic floor dysfunction [18], as well as a cause of rectal pain [19]. PNa presents most commonly as a unilateral pain.

Nantes Criteria for Pudendal Neuralgia

Essential Criteria

- Pain in the distribution of the pudendal nerve (Fig. 47.1).
- Pain predominately with sitting.
- Pain does not wake the patient at night.
- No objective sensory deficit on clinical exam.
- Pain relieved by diagnostic pudendal nerve block.

Additional Diagnostic Signs

- Burning, shooting, and stabbing pain; *subjective* numbness
- Allodynia or hyperpathia
- Rectal or vaginal sensation of the presence of a foreign body
- Worsening of pain during the day
- Predominately unilateral pain
- Increased pain with defecation
- Exquisite tenderness on palpation of the ischial spine

Exclusion Criteria

- *Exclusively* coccygeal, gluteal, suprapubic, or hypogastric pain
- *Exclusively* paroxysmal pain
- Excessive pruritus
- Imaging abnormalities able to account for the pain

Associated Signs That Do Not Exclude the Diagnosis

- Buttock pain on sitting
- Referred "sciatic" pain
- Medial thigh pain
- Suprapubic pain
- Urinary frequency or pain on bladder distension
- Pain after ejaculation or erectile dysfunction
- Dyspareunia (modified from Labat [13] with permission from John Wiley and Sons)

Anatomy (Table 47.2)

The PN arises from the sacral plexus and is formed from contributions of the first, second, third, and fourth sacral nerve roots (mainly S2 and S3) (Fig. 47.2). Huang et al. [20] mapped the contribution of each sacral nerve to the PN and found that the distribution of the contributions was

Table 47.2 Pudendal nerve anatomy

Origin	S1–S4 (mostly S2 and S3, rarely S4)
	Of 105 patients, 56 % were asymmetric [20]. Nerve root contributions:
	S1 = 4 %
	S2 = 60.5 %
	S3 = 35.5 %
General route	From ventral piriformis through the greater sciatic foramen, over the ischial spine, through the lesser sciatic foramen, over the sacrospinous ligament, into Alcock's canal. Rectal branch comes off just before Alcock's canal and continues as perineal and dorsal nerve of the penis or clitoris
Sensory distribution	Anal, perineal, and genital sensation
Motor innervation	Anal and urethral sphincters, pelvic floor muscles
Anatomic variability	Sacral contributions vary; rectal nerve may separate outside the pudendal canal
Other relevant structures	Sacrospinous ligament, sacrotuberous ligament, and pudendal artery

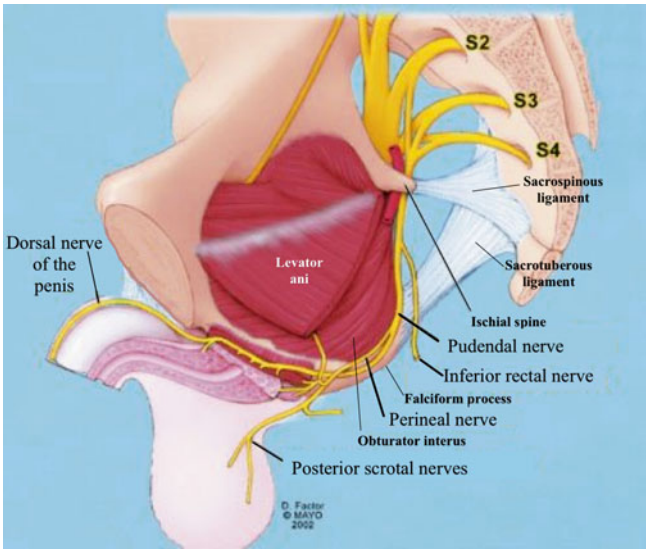

Fig. 47.2 Schematic anatomy of the intrapelvic path of the pudendal nerve (From Popeney et al. [21]. Reprinted with permission from John Wiley and Sons and used with permission of Mayo Foundation for Medical Education and Research. All rights reserved)

asymmetric in 56 % of the 105 patients they tested, with the S2 roots providing 60.5 % of the overall pudendal sacral nerve root contributions. The pudendal nerve is a mixed nerve (motor 20 %, sensory 50 %, and autonomic 30 %); it gives rise to three branches on each side of the body: a *rectal branch*, a *perineal branch*, and a *penile or clitoral branch* (Table 47.2) [21]. PNa occurs when the nerve or one of its branches becomes damaged, inflamed, or entrapped.

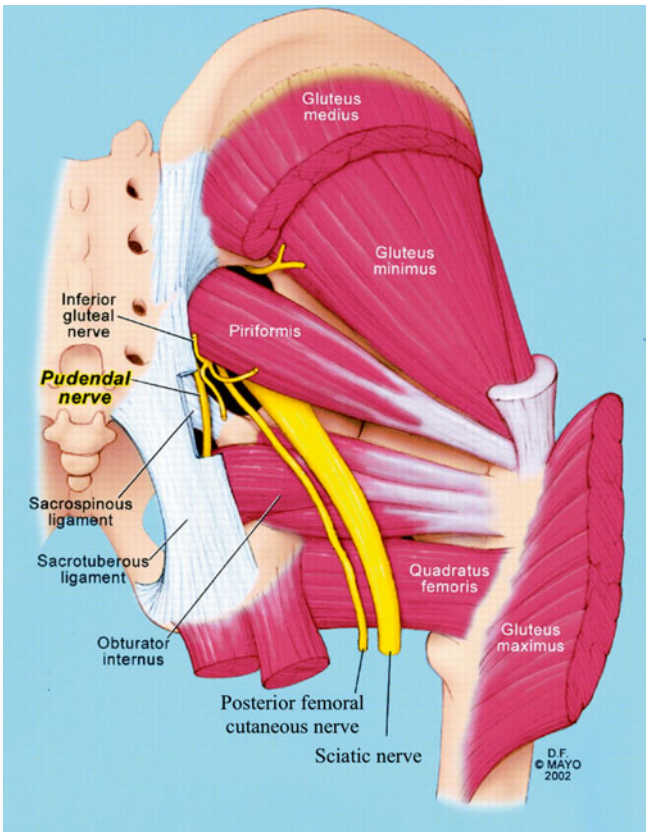

Fig. 47.3 Schematic anatomy at the level of the piriformis (From Popeney et al. [21]. Reprinted with permission from John Wiley and Sons and used with permission of Mayo Foundation for Medical Education and Research. All rights reserved)

The PN has a very tortuous course, traveling through the pelvis, buttocks, and perineum. After the nerve roots leave their sacral foramen, they join together on the ventral surface of the piriformis muscle to form the PN, which travels into the gluteal region with the *internal pudendal artery* by passing through the *greater sciatic foramen*; the neurovascular bundle hooks around the *sacrospinous ligament* (SS) on the ischial spine to pass into the perineum through the lesser sciatic notch (Fig. 47.3), underneath the *sacrotuberous ligament* (ST). The nerve and vessels then pass through the *ischioanal fossa* into the *pudendal* canal (also known as *Alcock's canal*), which is formed by a duplication of the *obturator fascia* on the lateral wall of the ischioanal fossa, deep to the ST attachment on the ischium (Fig. 47.4). Either just before entering the pudendal canal or just within it, the pudendal neurovascular bundle gives rise to the *inferior rectal nerve* (*inferior anal nerve*), which crosses the ischioanal fossa toward the *anal canal* and the *external anal sphincter muscle*. According to Montoya et al. [22], the dissection of 18 female cadavers showed that the inferior rectal nerve did not enter the pudendal canal in 44 % of the specimens.

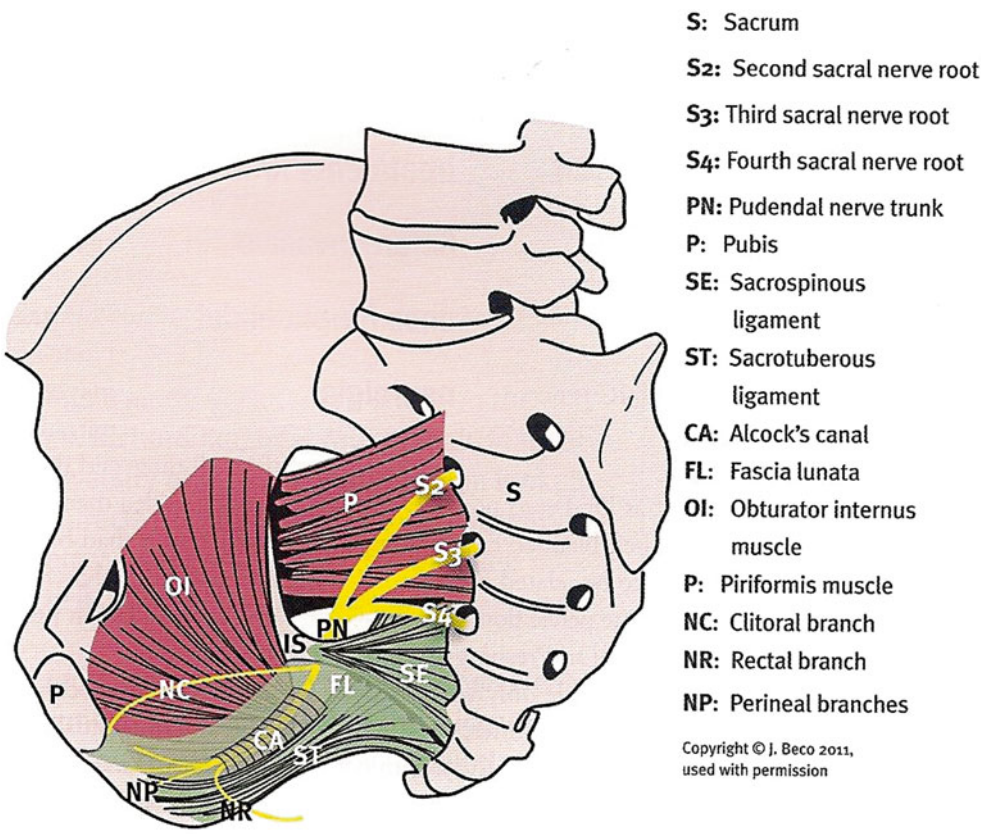

Fig. 47.4 Schematic anatomy of the pudendal canal (©2011 Jacques Beco M.D., used with permission)

Within the pudendal canal, the pudendal nerve divides into two terminal branches, the *perineal nerve* and the *dorsal nerve of the penis or clitoris* [23]. Along with the *inferior rectal nerve* and the *peroneal branch of the posterior femoral cutaneous nerve* (PFCN) (see Chap. 62), these nerves innervated the majority of the perineum and genitals (Fig. 47.5). The dorsal nerve crosses the *inferior pubic ramus* inferior to the pubic symphysis an average of 27.6 mm in males and 29.1 mm in women [6]. Montoya et al. [22] found that the clitoral and perineal nerves traveled caudad to the ventral portion of the *perineal membrane* (bilateral transverse fibrous sheets that attach the lateral wall of the vagina and perineal body to the ischiopubic ramus) and therefore should be at low risk during urethral sling surgeries.

The PN innervates the *external anal sphincter*, the *external urethral sphincter*, the perineal musculature, the clitoris or penis, and the skin of the perineum. However, Barber et al. [24] performed 12 pelvic dissections and could not find pudendal innervation of the *levator ani*.

Entrapment (Table 47.3)

Butler [25] described the anatomic sites of nerve injuries, which include soft tissue tunnels, areas where nerves branch (especially at acute angles), sites where the nerve is relatively fixed, and sites where the nerve is subject to friction because of close proximity to an unyielding interface. Each of these mechanisms can be potentially involved in the multiple sites of entrapment of the pudendal nerve (Table 47.3). The first site occurs as the pudendal nerve passes under the piriformis muscle (Fig. 47.3). A second site of entrapment is at the ischial spine, where the pudendal nerve can be compressed between the SS and ST (Figs. 47.3, 47.4, 47.5, and 47.6). The nerve can also be trapped by the *falciform process of the sacrotuberous ligament* (the broad attachment of the ST on the *ischial tuberosity*) (Fig. 47.2) or by the *obturator fascia* in *Alcock's canal* (Fig. 47.4). The pudendal nerve can also be entrapped upon exiting Alcock's canal and as it branches in the perineal area [21] (Figs. 47.4 and 47.5). According to Sedý et al., there can also be compression of the dorsal nerve of the penis or clitoris against the lower border of the pubic bone, in a groove called the *sulcus nervi dorsalis* penis or clitoris [26].

Báča et al. [6] reviewed 225 pelvic fracture cases; they concluded that the pudendal nerve could be injured or entrapped as the nerve left the sacral foramen, as well as along the inferior pubic ramus. They noted that the pudendal bundle is unprotected as it travels from the ischial spine to the ischial tuberosity and therefore is at risk for injury by dislocated fractures of the posterior pelvis. In addition, because the dorsal nerve of the penis has a slightly longer course along the

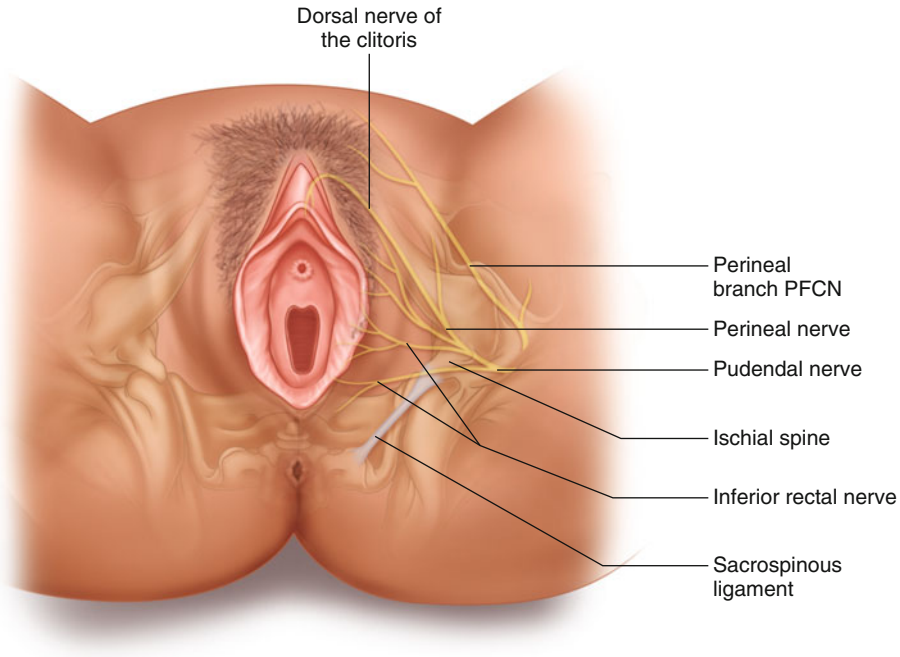

Fig. 47.5 Schematic image of the perineal view of the pudendal nerves. *PFCN* posterior femoral cutaneous nerve (Image courtesy of Springer)

Table 47.3 Incidence of pudendal nerve entrapment at various sites (may be more than one entrapment)

Site of entrapment	Incidence
Sacrospinous ligament (SS)	58 %
Sacrotuberous ligament (ST)	69 %
Falciform process on the ischial tuberosity	42 %
Ischial spine	11 %
Piriformis/sacrospinous ligament	17 %
Obturator fascia	48 %

Modified from Butler [25]

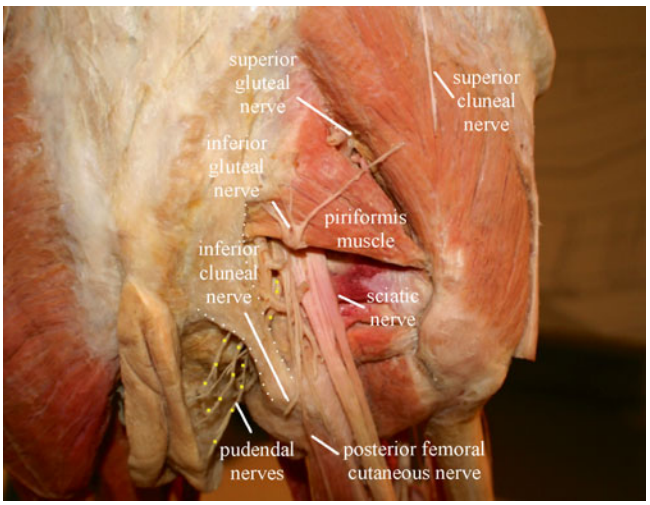

Fig. 47.6 Gluteal dissection modified from an image from *Bodies*, *The Exhibition*, with permission. *Yellow dotted lines* identify the pudendal nerve and branches. *White dotted line* outlines the sacrotuberous ligament (Image courtesy of Andrea Trescot, MD)

inferior pubic ramus than does the dorsal nerve of the clitoris (due to the sharper angle between the male inferior rami than the female rami), males are at greater risk for injury from inferior pubic rami fractures. In their series, 74 % of the fractures were of the inferior pubic ramus, and there was unilateral entrapment in 70 % of patients. Determination of the area of entrapment is crucial to the treatment of pudendal entrapment by injection therapy or surgical release.

Physical Exam

The physical exam findings in patients with entrapment may be subtle. Pain may be replicated with application of pressure on the pudendal nerve at the ischial spine (during a bimanual exam) or the inferior pubic ramus (percutaneously). The symptoms may worsen with passive internal and external rotation of the hip and resisted abduction/adduction of the hip flexed to 90°. However, there are many causes of pelvic pain that might mimic pudendal neuralgia (Fig. 47.7). It is not uncommon for PN entrapment to be present concurrently with musculoskeletal pain in other regions such as the sacroiliac joint, piriformis muscle, greater trochanteric bursa, and the coccyx.

Differential Diagnosis (Table 47.4)

Since there is no confirmatory test available, pudendal neuralgia is a diagnosis of exclusion. Unfortunately, proper diagnosis can take many years. Other conditions to consider in the differential diagnosis include *levator ani syndrome*,

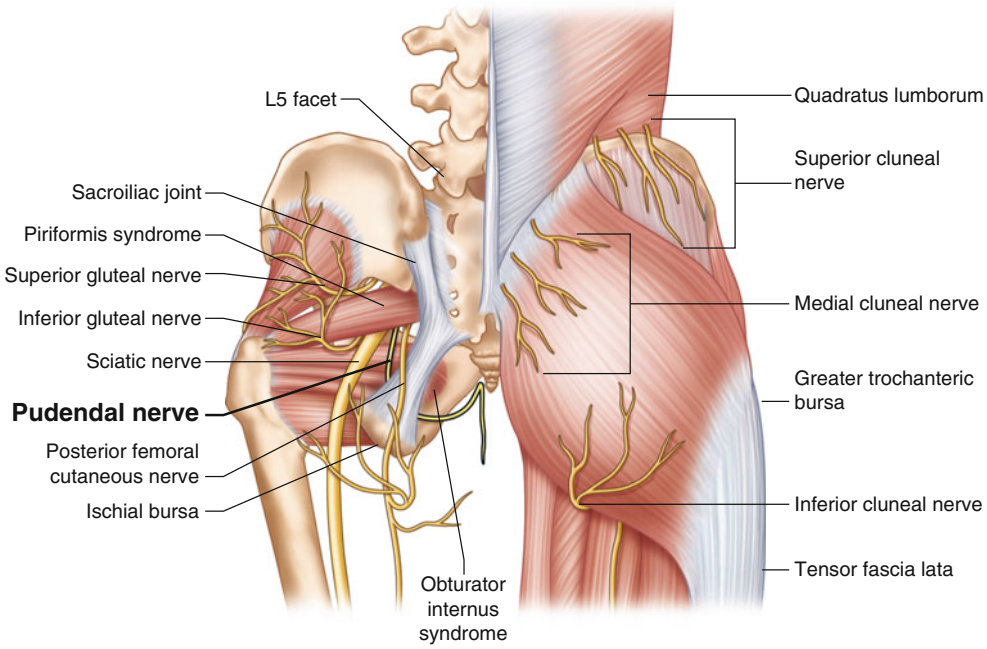

Fig. 47.7 Schematic displaying points of a physical examination of the posterior pelvis (Image courtesy of Springer)

Table 47.4 Differential diagnosis of pelvic pain

	Potential distinguishing features
Levator ani syndrome	Muscle tenderness on rectal exam
Proctalgia fugax	Pelvic floor dysfunction (which may be related to PNE) [9]
Interstitial cystitis	Cystoscopy showing bladder mucosal irritation
Vulvodynia/vestibulitis	Pudendal neuralgia may be one of the causes
Hemorrhoids	Rectal exam
Piriformis syndrome	US/fluoroscopic injection of the muscle
Coccydynia	Bimanual (vaginal or rectal) pelvic exam of the coccyx
Ischial bursitis	Edema or enlarged bursa on MRI; tenderness at the apex of the ischium, not along the medial aspect
Prostatitis	Tenderness on palpation of the prostate; inflammatory cells in prostate fluid

proctalgia fugax, *interstitial cystitis*, *vulvodynia*, *vestibulitis*, chronic pelvic pain syndrome, *hemorrhoids*, piriformis syndrome, *coccydynia*, or *ischial bursitis*.

Popeney et al. [21] evaluated 58 consecutive patients (32 males and 26 females) with unilateral or bilateral pudendal entrapment; all had a history of chronic, intractable pelvic pain, with pain in the testicles, penis, or rectum in the males and labia, clitoris, or rectum in the females. Other symptoms included urinary symptoms (hesitancy, urgency, and frequency) in 40 %, constipation and painful bowel movements in 29 %, and sexual dysfunction in 33 %. Patients presented with the following misdiagnoses: interstitial cystitis (30 %), *prostatitis* or *epididymitis* (63 % of males), vulvodynia (50 % of females), endometriosis (13 % of females), piriformis syndrome (20 %), levator ani syndrome (3 %), coccydynia (6 %), lumbosacral radiculopathy (3 %), and chronic pelvic pain syndrome (20 %). Electromyography (EMG) was abnormal in 43 %.

Table 47.5 Diagnostic tests for pudendal neuralgia

	Potential distinguishing features
Physical exam	Tenderness at the ischial spine
Diagnostic injection	Proximal at the ischial spine, distally at the ischium
Ultrasound	Shows the potential entrapment and nerve enlargement in seven of ten cases [27]
MRI	Not well visualized but can be used to rule out other causes such as tumors or lesions. See Figs. 47.9 and 47.10
Arteriography	Compression of the artery by arteriography suggests compression of the nerve as well [28]
X-ray	Pelvic fractures of the inferior pubic ramus may entrap the pudendal nerve
Electrodiagnostic studies	A greater than normal conduction delay can indicate entrapment [29]

Diagnostic Tests

The diagnostic tests for PNa are seen on Table 47.5. MRI (Figs. 47.8 and 47.9) can be particularly useful to determine potential causes of pudendal neuralgia, such as compression from a mass; ultrasound may also be useful.

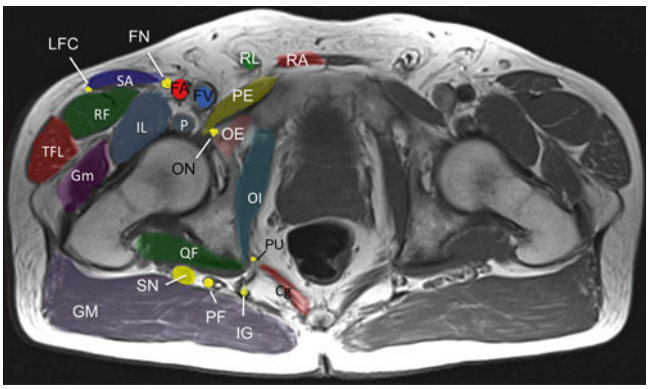

Fig. 47.8 Axial MRI of the pelvic structures. *Cg* coccygeus muscle, *FA* femoral artery, *FN* femoral nerve, *FV* femoral vein, *GM* gluteus maximus muscle, *Gm* gluteus medius, *IG* inferior gluteal nerve, *IL* iliopsoas muscle, *LFC* lateral femoral cutaneous nerve, *OI* obturator internus muscle, *ON* obturator nerve, *P* psoas muscle, *PE* pectineus muscle, *PF* posterior femoral cutaneous nerve, *PU* pudendal nerve, *QF* quadratus femoris muscle, *RA* rectus abdominis muscle, *RF* rectus femoris muscle, *RL* round ligament, *SA* sartorius muscle, *SN* sciatic nerve, *TFN* tensor fascia lata muscle (Image courtesy of Andrea Trescot, MD)

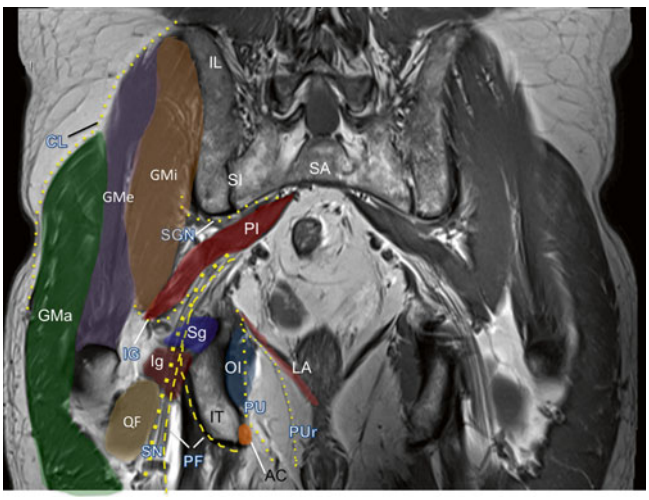

Fig. 47.9 Coronal MRI of the pelvic structures. *AC* Alcock's canal, *CL* cluneal nerve, *GMa* gluteus maximus muscle, *GMe* gluteus medius muscle, *GMi* gluteus minimus muscle, *IG* inferior gluteal nerve, *Ig* inferior gemellus muscle, *IL* iliac crest, *IT* ischial tuberosity, *LA* levator ani muscle, *OI* obturator internus muscle, *PF* posterior femoral cutaneous nerve, *PI* piriformis muscle, *PU* pudendal nerve, *PUr* rectal branch of the pudendal nerve, *QF* quadratus femoris muscle, *SA* sacrum, *Sg* superior gemellus muscle, *SGN* superior gluteal nerve, *SN* sciatic nerve (Image courtesy of Andrea Trescot, MD)

Identification and Treatment of Contributing Factors

Pudendal neuralgia can be caused by mechanical injury to the nerve, viral infection, or immunologic processes. Mechanical injury may be caused by pelvic floor spasm, pressure from surrounding ligaments, or scar tissue from trauma or surgeries involving the surrounding area. It has been seen in competitive cyclists and patients who sit on hard surfaces. In women, the most common causes are surgical injury, pelvic trauma, or childbirth [3]. The dual injuries to the pelvic floor and the pudendal nerve seen in childbirth have been implicated in stress urinary incontinence [30]. Diabetes may make the nerve more susceptible to injury [31].

Avoidance of causes of pudendal nerve entrapment can be helpful. This would include avoiding sitting on hard surfaces and using proper padding if cycling. Learning relaxation techniques for the pelvic floor can be helpful, and there are several physical therapy modalities for intrapelvic ischemic compression. Medication such as membrane stabilizers (antidepressants and anticonvulsants) and oral anti-inflammatories can be beneficial.

Injection Technique

Landmark-Guided Technique

For an intravaginal injection, the patient is placed in the lithotomy position. The procedure can be done transrectally for males and either transrectally or transvaginally for females. First palpate the ischial spine. A needle with a guide, such as an Iowa trumpet (Fig. 47.10), is recommended to limit the depth of submucosal penetration and to prevent injury to the vagina. To perform a left-sided block, palpate the ischial spine with the index finger of the left hand, hold the syringe in the right hand, and guide the needle between the index and middle fingers of the left hand toward the ischial spine (Fig. 47.11). Place the end of the guide beneath the tip of the ischial spine. Push the needle into the mucosa; aspirate to ensure that the injection is not intravascular. Raise a mucosal wheal with 1 cc of local anesthetic and then advance the needle through the vaginal mucosa until it touches the sacrospinous ligament, usually 1 cm medial and posterior to the ischial spine. Infiltrate the tissue with 3 cc of local anesthetic and then advance the needle further through the sacrospinous ligament for a distance of about 1 cm until a loss of resistance is felt. The tip now lies in the area of the pudendal nerve; at this point, the pudendal vessels lie just lateral to the pudendal nerve, so care must be taken to avoid intravascular administration. Aspirate to confirm the needle placement is not intravascular prior to injecting local anesthetic. Subsequently, withdraw the needle into the guide and move the tip of the guide to just above the ischial spine. Reinsert the needle though the mucosa and again inject [32].

Because patients tend to dislike transvaginal or transrectal injections (and because of the increased risk of infection with these techniques), a *transgluteal approach* can be used; the use of a peripheral nerve stimulator (PNS) helps to

Fig. 47.10 Iowa trumpet (Image courtesy of Andrea Trescot, MD)

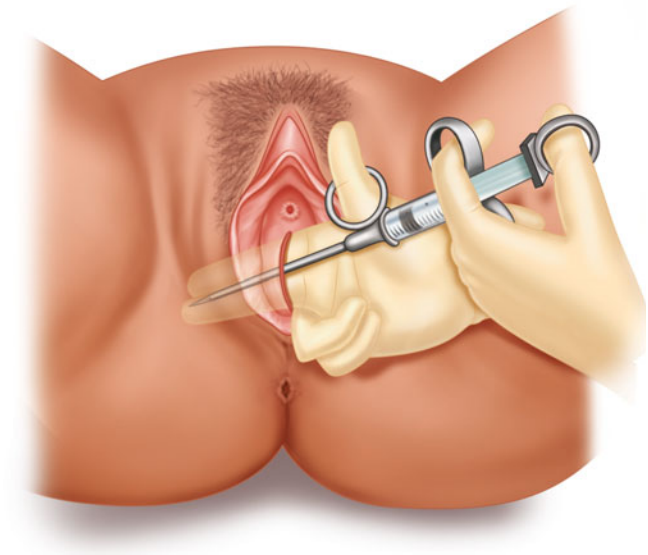

Fig. 47.11 Landmark-guided technique – intrapelvic pudendal nerve injection (Image inspired by Prendergast et al. [15], courtesy of Springer)

identify the nerve (Fig. 47.12). For patients with primarily vaginal or urethral pain, a modified lithotomy position (with the patient's feet on the exam table) will suffice; for patients with primarily rectal pain, placing the patient in a prone jackknife position can aid in the access to the rectal branch.

Fluoroscopy-Guided Technique

Fluoroscopy provides the advantage of direct visualization of structures that surround the pudendal nerve, most specifically the ischial spine. Abdi et al. [33] describe a fluoroscopic approach to the pudendal nerve proximally at the ischial spine. The patient is placed in the prone position, with the fluoroscope at the level of the femoral heads, angled cephalad or caudad until the pelvic inlet is visualized. The ischial spine is then visualized by an ipsilateral oblique angulation of the fluoroscope about 5–15° (Fig. 47.13). A needle is then advanced to the tip of the ischial spine, where the pudendal nerve leaves the pelvis (Fig. 47.14). The use of a peripheral nerve stimulator and contrast (Figs. 47.15, 47.16, and 47.17) can aid in the identification of the pudendal nerve.

There is also a fluoroscopic approach to the pudendal nerve on the ischium, as the nerve leaves Alcock's canal (Fig. 47.18). Using a peripheral nerve stimulator allows for selective identification and injection of the individual rectal, perineal, or penile or clitoral nerves.

Ultrasound-Guided Technique

Ultrasound offers visualization of important landmarks: the *ischial spine*, *pudendal artery*, *sacrospinous ligament*, *sacrotuberous ligament*, and PN. It also allows real-time needle advancement and confirmation of injectate spread within the interligamentous plane. With the patient in the prone position, scanning is performed in a transverse plane to visualize the ischium forming the lateral border of the sciatic notch. The ischium is initially seen as a curved line as it forms the posterior aspect of the acetabulum. When the probe is at the ischial spine level, the ischium will appear as a straight line. At this level, a color Doppler is used to localize the *internal pudendal artery* pulsations in close proximity to the ischial spine (Fig. 47.19). Another arterial pulsation is often seen lateral to the tip of the ischial spine and is accompanied by the sciatic nerve. This is the *inferior gluteal artery*. Mistaking this artery for the pudendal artery will result in sciatic nerve block. The sacrospinous ligament appears as a hyperechoic line in continuity with the ischial spine, with lower echogenicity than the bone. Similarly, the sacrotuberous ligament is seen as a light hyperechoic line deep within the *gluteus maximus muscle* and appears parallel and superior to the sacrospinous ligament in ultrasound images. Localization of the pudendal nerve is targeted in the plane between these two ligaments. A 22- or 25-gauge needle is inserted from the medial aspect of the probe. It is advanced in line with the ultrasound probe to the medial aspect of the internal pudendal artery. Once the needle passes through the sacrotuberous ligament, a "click" is usually felt and a small volume (1–2 mL) of D5W or normal saline can be injected as a contrast, especially if the syringe is briefly shaken to create tiny bubbles. The solution appears as a hypoechoic collection, which helps to identify the plane between the sacrotuberous and the sacrospinous ligaments and to accentuate the pudendal nerve appearance. Although visualization of the pudendal nerve is not possible in all cases, the two ligaments and the internal pudendal artery can usually be easily identified. The needle is inserted medially toward the pudendal artery, since the pudendal nerve is principally located medial to this artery [34].

CT-Guided Technique

A CT-guided technique may be more accurate than fluoroscopically guided or blind injection techniques. The needle tip is positioned adjacent to the pudendal nerve at the

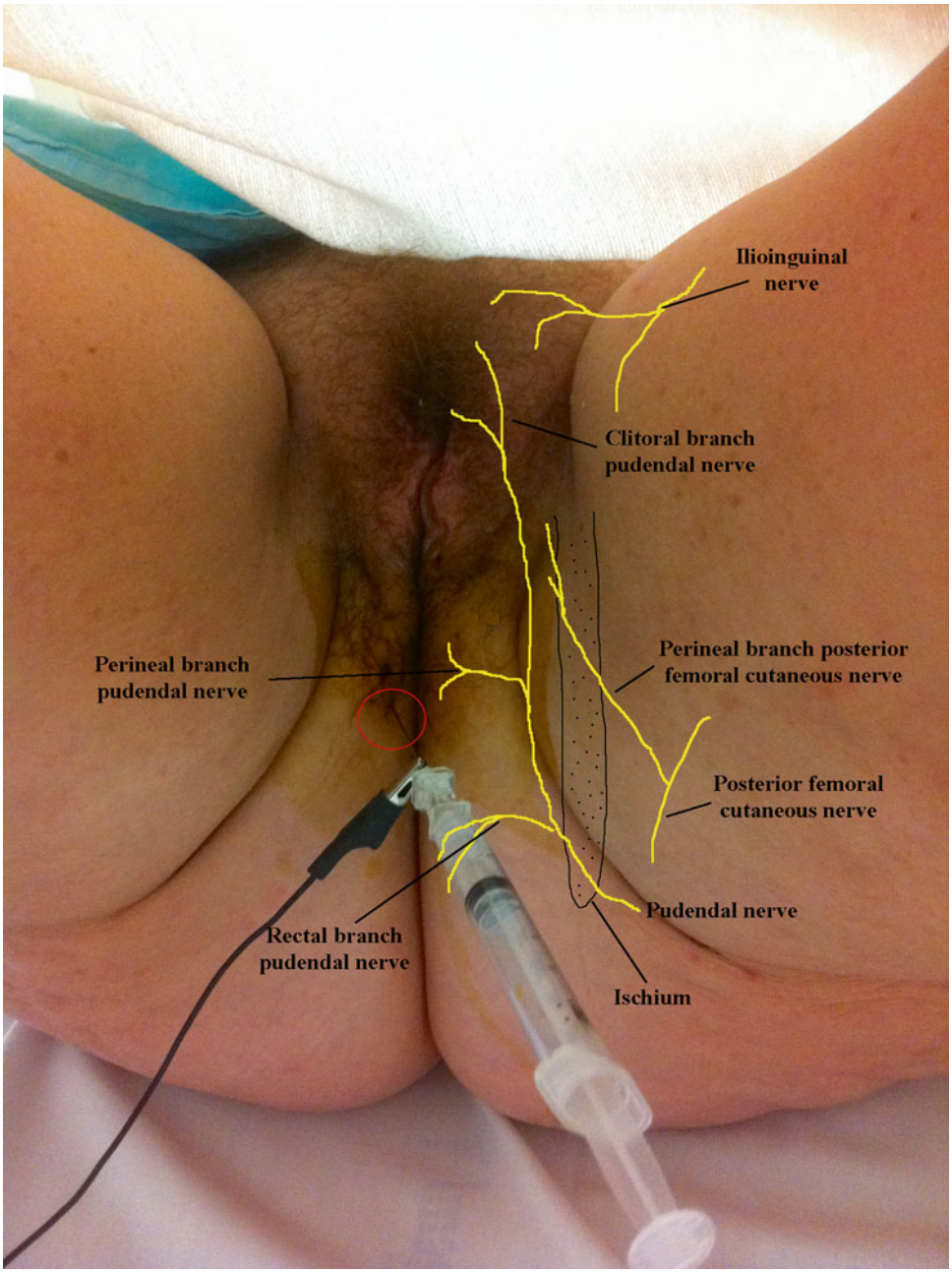

Fig. 47.12 Transgluteal pudendal nerve block with peripheral nerve stimulator; *red circle* demonstrates needle entry (Image courtesy of Andrea Trescot, MD)

ischial spine in the interligamentous space or at the pudendal canal (Fig. 47.20) [35]. A long-acting local anesthetic (such as bupivacaine) and a corticosteroid (such as methylprednisolone) are injected to provide immediate and potentially long-term, pudendal neuralgia relief [35]. CT-guided injections have also been described at Alcock's canal (Fig. 47.21) [36, 37].

MRI-Guided Technique

Fritz et al. [38] described the MRI neurography of pelvic pain structures, including the pudendal nerve. They performed a variety of injections under MRI guidance, including the obturator nerve, lateral femoral cutaneous nerve, pudendal nerve, posterior femoral cutaneous nerve, sciatic nerve, ganglion impar, and sacral spinal nerve, and injection into the piriformis muscle.

Neurolytic/Surgical Technique

Cryoneuroablation

Trescot [39] described the use of cryoneuroablation for the treatment of pudendal neuralgia. Freezing the pudendal nerve can be performed intravaginally (personal communication,

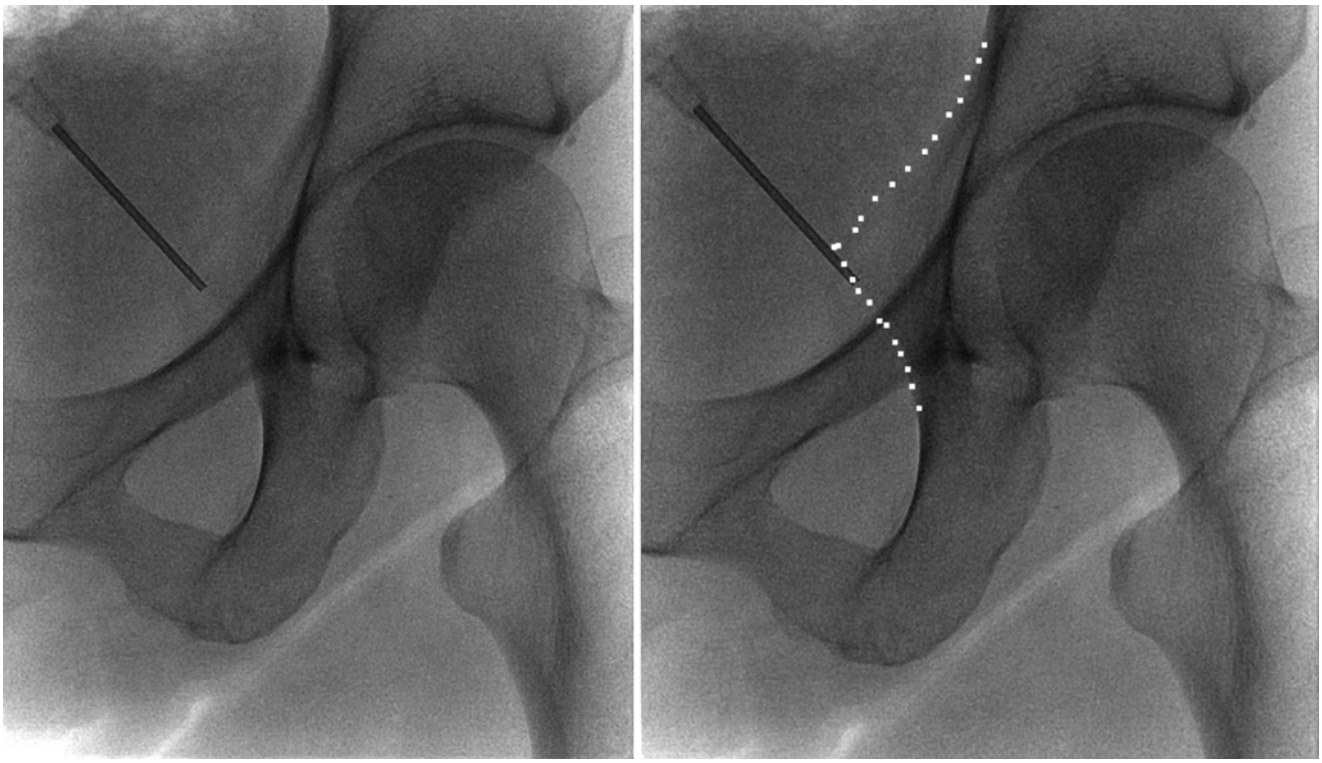

Fig. 47.13 Ischial spine seen after an ipsilateral oblique imaging of the pelvis (Image courtesy of Andrea Trescot, MD)

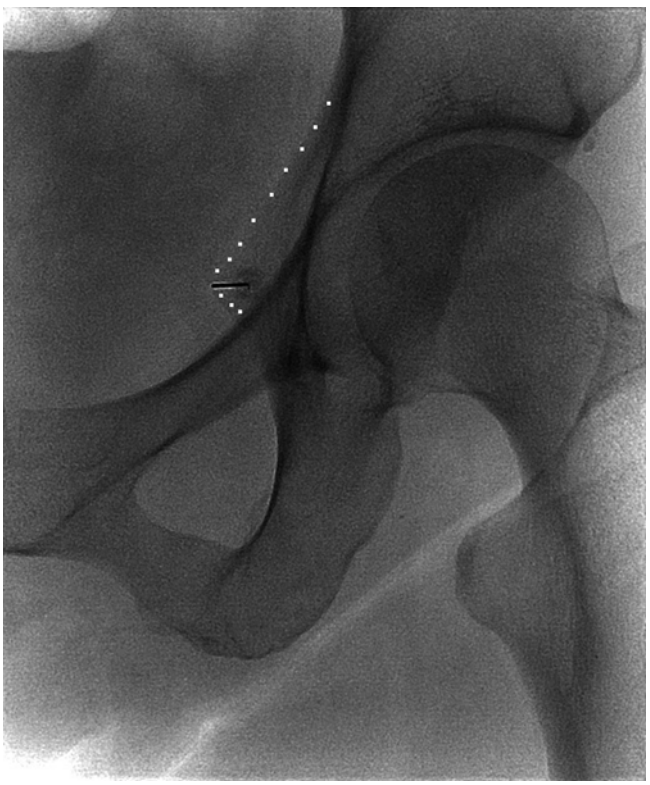

Fig. 47.14 Pudendal nerve block at the ischial spine (Image courtesy of Andrea Trescot, MD)

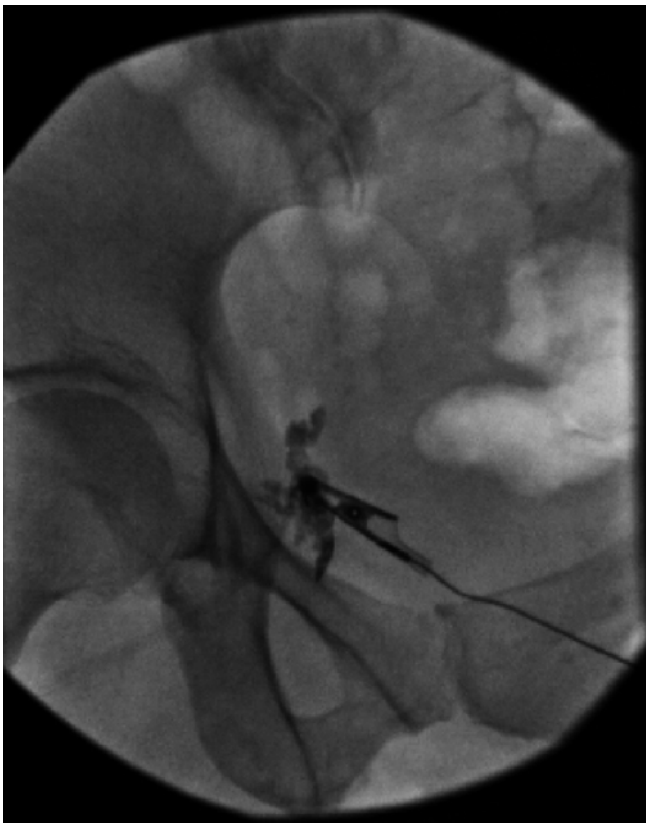

Fig. 47.15 Pudendal nerve block at the ischial spine with peripheral nerve stimulator and contrast (Image courtesy of Andrea Trescot, MD)

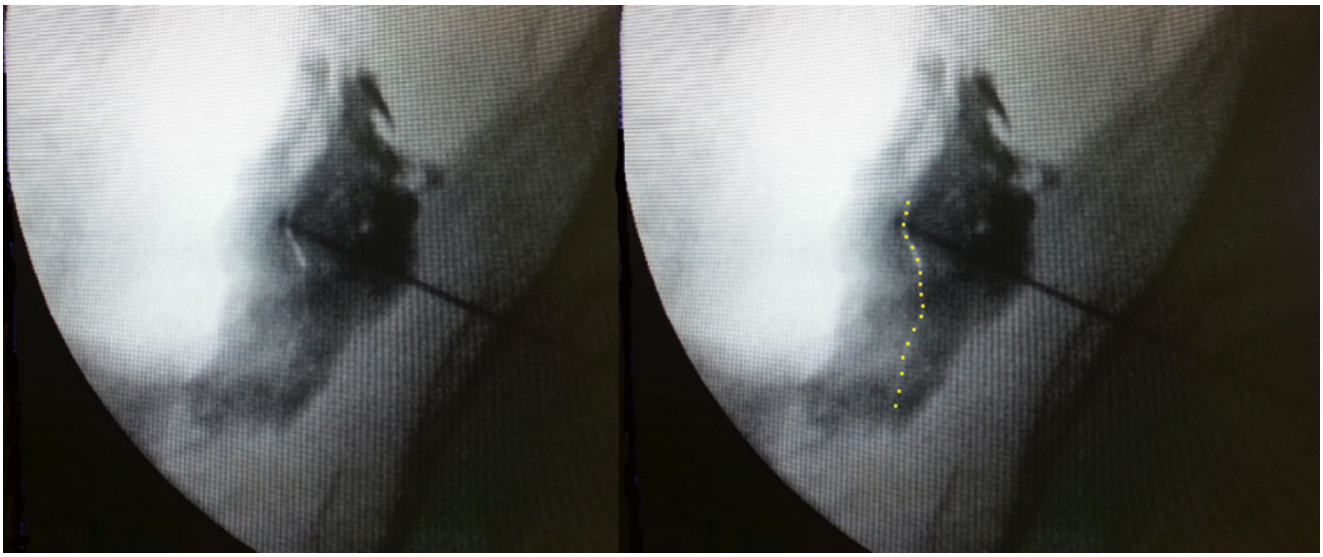

Fig. 47.16 Pudendal nerve block at the ischial spine, with contrast outlining the nerve (Image courtesy of Eric Wilson, MD)

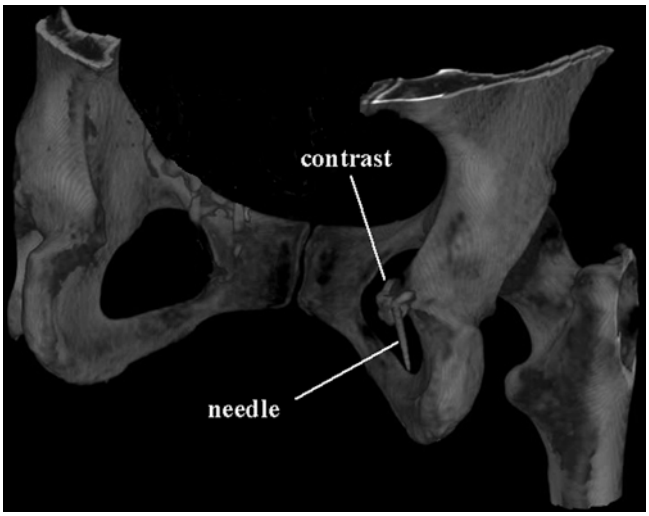

Fig. 47.17 Three-dimensional pudendal nerve injection at the ischial spine (Image courtesy of Andrea Trescot, MD)

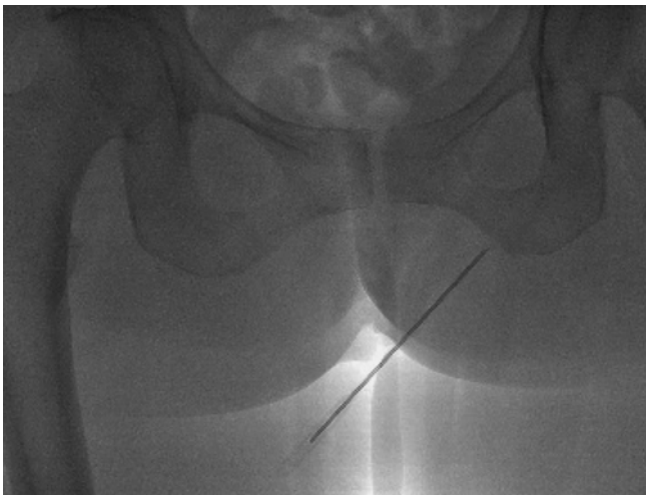

Fig. 47.18 Pudendal nerve block at the ischium (Image courtesy of Andrea Trescot, MD)

Dr. Jack McDonald, UCLA), at the ischial spine (Fig. 47.22), at Alcock's canal [40], or at the individual branches of the pudendal nerve (Fig. 47.23). Trescot cautioned against neurolysis of the pudendal nerve proximally bilaterally because of concerns regarding denervation of the clitoris. The location of the nerve is identified by palpation, fluoroscopy, or US, and after infiltration of the tissues with saline and epinephrine 1:200,000, a 12-gauge catheter is advanced to the target area. The probe is then advanced to the nerve location, and sensory stimulation is used to confirm the placement, while motor stimulation will confirm adequate distance from important motor structures (such as the sciatic nerve).

Prologo et al. [40] described 11 patients with pudendal neuralgia who were treated with cryoneuroablation at the pudendal canal under CT guidance; there was an immediate improvement that was sustained at 6 months.

Radiofrequency (RF)

Masala et al. [41] studied pudendal neuralgia patients who were unresponsive to "conservative approaches." Thirty patients with PNa were prospectively enrolled in a study (26 patients finished the study) to evaluate CT-guided pulsed

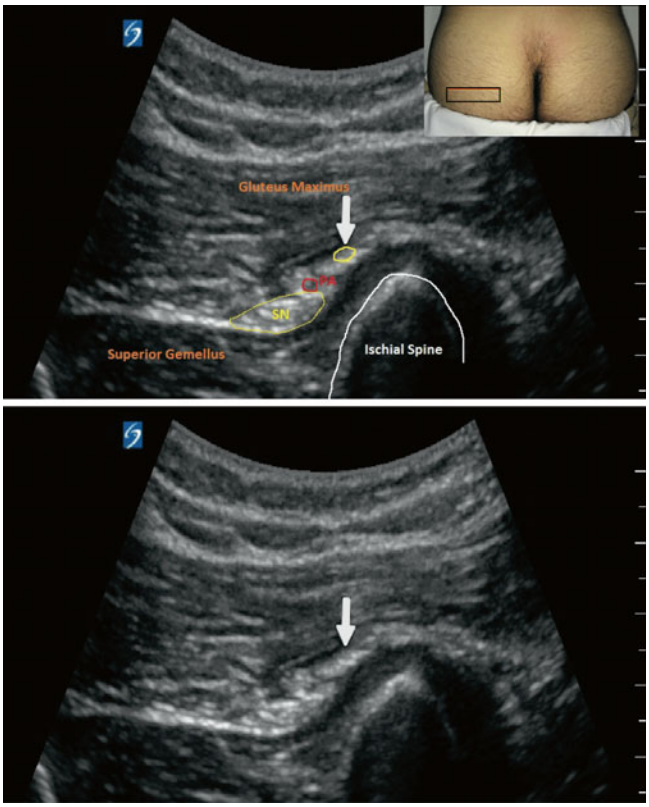

Fig. 47.19 Ultrasound image of the pudendal nerve. *SN* sciatic nerve, *PA* pudendal artery; *white arrow* pudendal nerve (Image courtesy of Agnes Stogicza, MD)

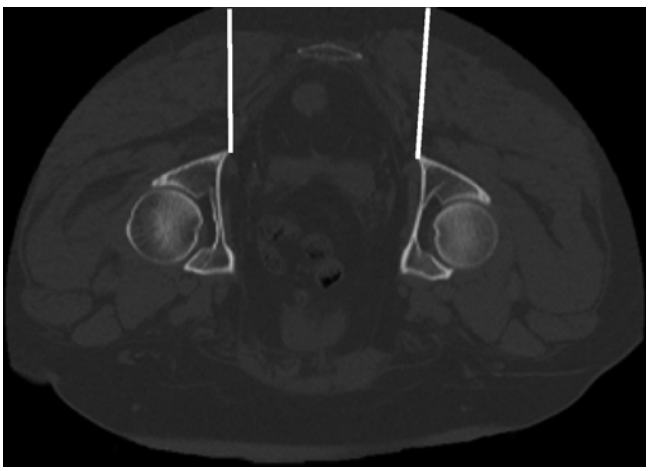

Fig. 47.20 Axial CT scan with simulated injection bilaterally at the ischial spine (Image courtesy of Andrea Trescot, MD)

radiofrequency (PRF) lesioning. A 20-gauge cannula was placed on the pudendal nerve at Alcock's canal; position was confirmed with contrast, and the nerve was treated with 1,200 pulses at high voltage (45 V) with 20 ms duration. Patients noted "excellent" results both initially and after 1 year.

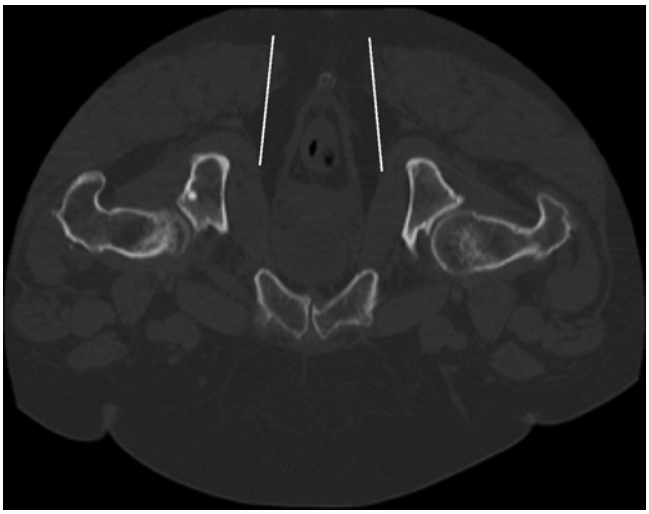

Fig. 47.21 Axial CT scan with simulated injection bilaterally at Alcock's canal (Image courtesy of Andrea Trescot, MD)

PRF was also used to treat the PN from a transvaginal approach in a modified lithotomy position; Rhames et al. identified the ischial spine and sacrospinous ligament transvaginally, and the nerve was pulsed at 42 °C for 120 s, with the patient noting 1.5 years of at least partial relief [42].

Neurolytic Injections

Phenol has been used on the pudendal nerve to treat voiding dysfunction due to hypertonicity of the pudendal nerve. Ko and Kim [43] described 13 pudendal nerve injections on seven patients with PNa who received a pudendal block with a peripheral nerve stimulator at the ischial tuberosity with 7 % phenol. The procedure dramatically improved the voiding pattern.

Calabrò et al. [44] treated a patient with pudendal neuralgia by injecting *palmitoylethanolamide (PEA*), which apparently works on the neuropathy by decreasing inflammation and mast cell degranulation.

Central and Peripheral Nerve Stimulation

Standard thoracic spinal cord stimulation has not considered very useful for pudendal neuralgia, because of the need to stimulate the conus, though Martin described the placement of a dual electrode system at the conus with good relief [45] (Fig. 47.24). Retrograde (cephalocaudal implantation) stimulation has been used for many years to stimulate the sacral nerve roots to treat idiopathic overactive bladder, urgency-frequency syndromes, interstitial cystitis, pudendal neuralgia, vulvodynia, prostadynia, and coccygodynia (Fig. 47.25) [46]. Trans-sacral stimulation,

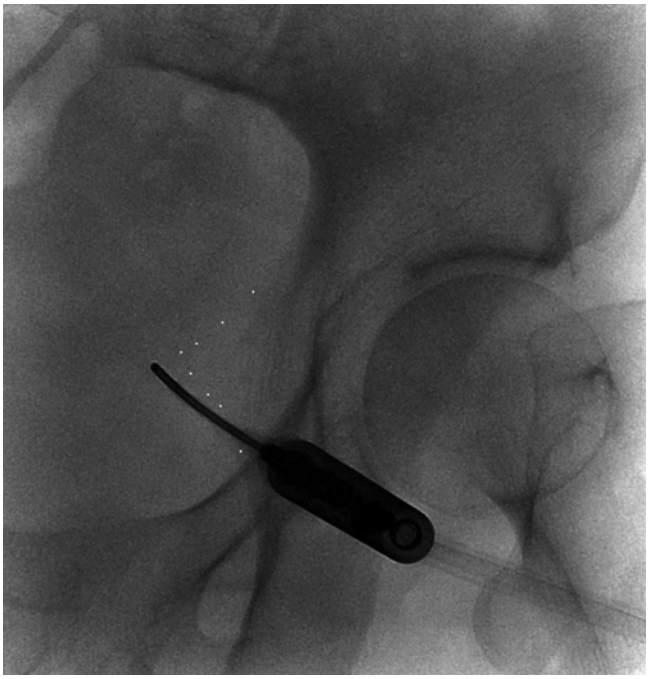

Fig. 47.22 Cryoneuroablation of the pudendal nerve at the ischial spine (Image courtesy of Agnes Stogicza, MD)

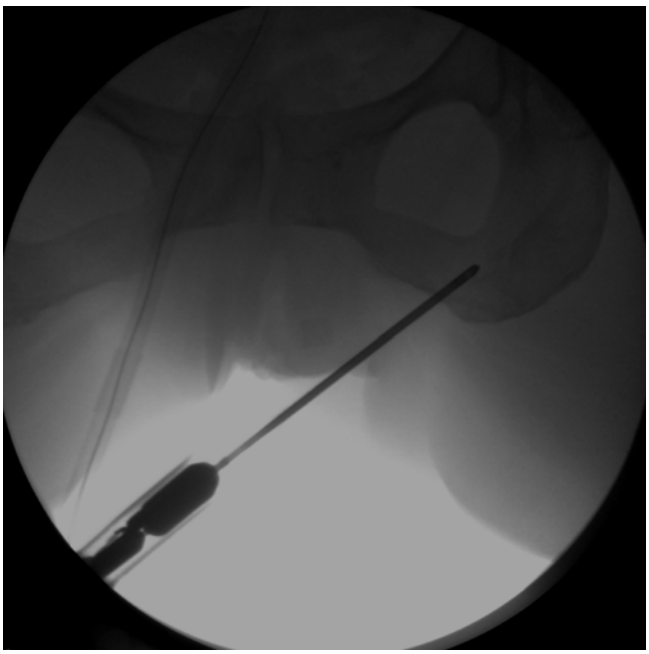

Fig. 47.23 Cryoneuroablation of the pudendal nerve at the ischium (Image courtesy of Andrea Trescot, MD)

using a tined InterStim® (Medtronic) lead placed through the bilateral S3 and S4 sacral foramen for pudendal neuralgia, was first described by Valovska et al. in 2014 [47] (Fig. 47.26). Another option could include an antegrade spinal cord stimulator (SCS) catheter through the sacral hiatus.

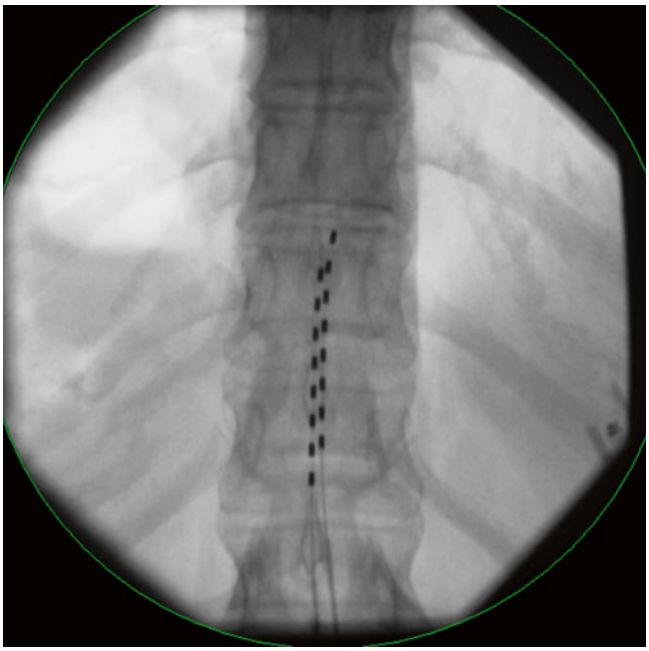

Fig. 47.24 Spinal cord stimulator placed at the conus to stimulate the pudendal nerve (Image courtesy of Robert Martin, MD)

According to Buffenoir et al. [48], 30 % of patients with pudendal neuralgia due to entrapment noticed little to no relief from decompressive surgery (see below). They performed a prospective study evaluating the efficacy of intraspinal stimulation of the conus medullaris. Patients underwent a percutaneous trial for an average of 13 days, followed by permanent implantation. Twenty of the 27 patients trialed noted relief from the trial, and "100 %" of the implanted patients (followed for up to 15 months) noted long-term relief, with increased sitting tolerance and 55.5 % estimated percent improvement.

Motor cortex stimulation is usually reserved for the most severe pains; Louppe et al. [49] described successful motor cortex stimulation in two patients with severe, intractable pelvic and perineal pain.

Recently, Marc [50] described the laparoscopic implantation of a pudendal stimulator to treat urinary symptoms.

Surgery

Pudendal nerve decompression surgery is an option that is usually considered after more conservative therapies such as lifestyle changes, pelvic floor physical therapy, and nerve blocks have not proven successful. Outcomes from pudendal nerve decompression surgery depend on multiple factors such as length, degree, and cause of nerve injury. Approximately 40 % of patients who undergo transgluteal pudendal decompression have significant improvement in pain, 30 % of patients have some improvement in pain, and

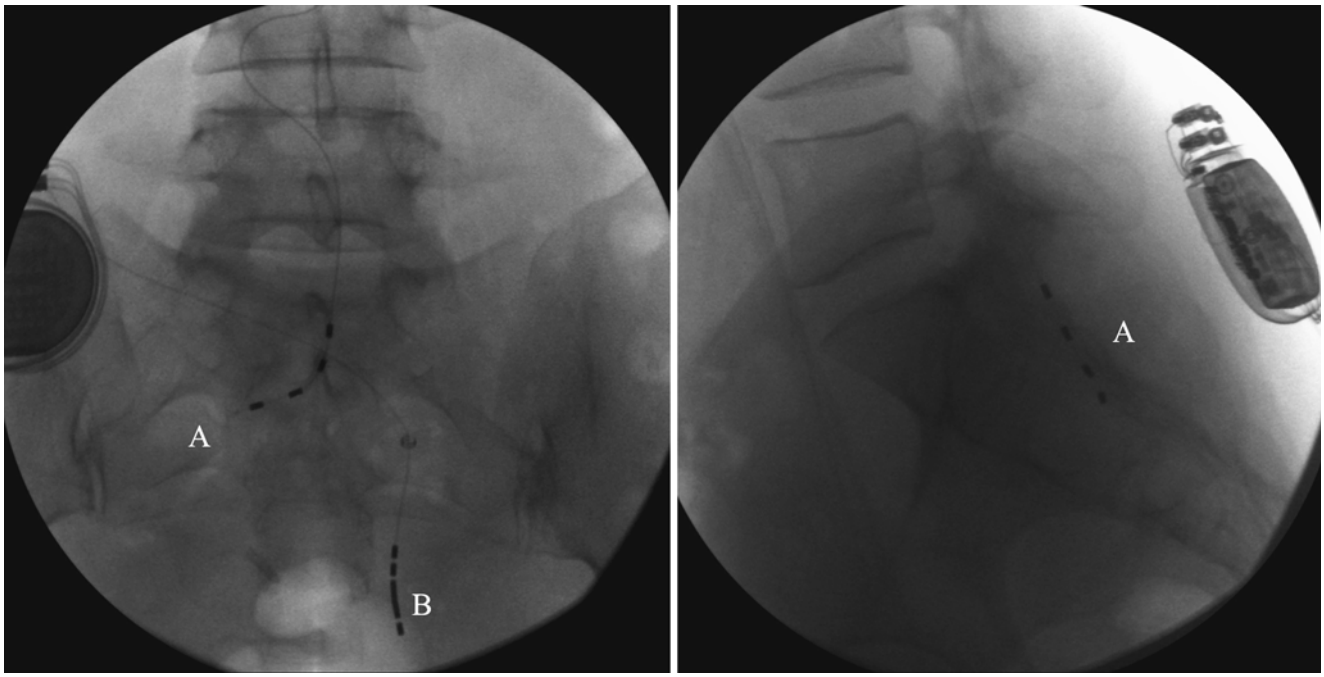

Fig. 47.25 Retrograde spinal cord stimulator lead placed in a patient with an InterStim® trans-sacral stimulator. *A* the percutaneous retrograde lead, *B* the InterStim® lead (Image courtesy of Andrea Trescot, MD)

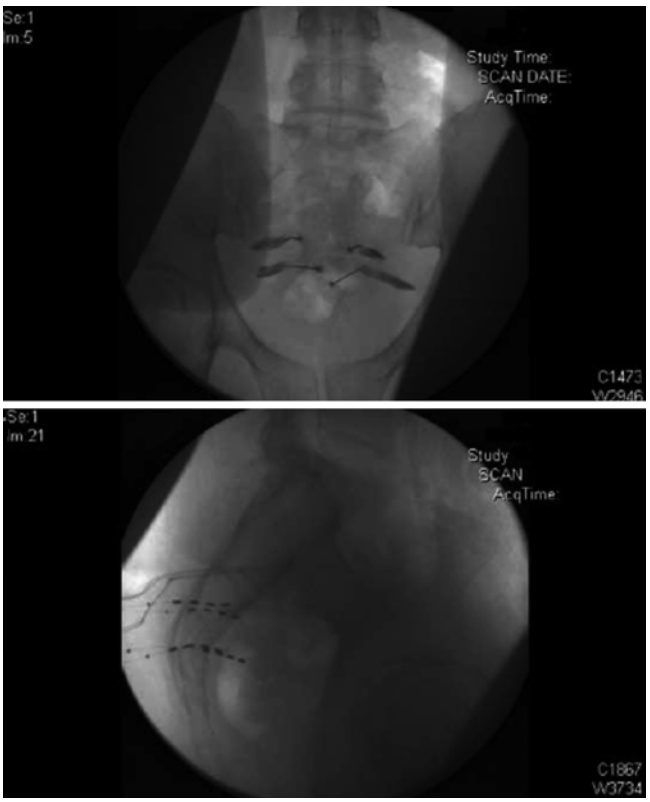

Fig. 47.26 Trans-sacral stimulation for pudendal neuralgia (From Valovska et al. [47]. Reprinted with permission from the American Society of Interventional Pain Physicians)

30 % have no change in pain. Surgery is generally considered successful if there is at least a 50 % reduction in pain and symptoms [51]. The four approaches are the *transgluteal approach*, the *trans-ischiorectal fossa approach*, the *perineal approach*, and the *laparoscopic approach*.

The *transgluteal approach* is probably the most widely used method of decompression surgery, offering the greatest visualization of the nerve during surgery. The incision is made in the buttocks, through the gluteal muscles. The sacrotuberous ligament and the sacrospinous ligament are explored, and any compression of the pudendal nerve is relieved at the ischial spine. Alcock's canal is explored, and the nerve is released from any fascia that might be tethering it.

In the *trans-ischiorectal fossa approach*, a small incision is made in the back of the vagina, about halfway up. In most cases, the surgeon severs or partially severs the sacrospinous ligament to release the compression between the ST and SS ligaments. Alcock's canal is explored by the surgeon's finger, and the nerve released from any fascia that might be tethering it.

In the *perineal approach*, a small vertical incision is made in the perineum between the anus and ischial tuberosity. The surgeon then uses a finger to free up the nerve in Alcock's canal.

The *laparoscopic approach* to the pudendal nerve has only recently been described [52]. During laparoscopic surgery,

the sacrospinous ligament is severed allowing visual access of the nerve at the ischial spine and Alcock's canal [3]. The nerve is freed from scarring, fibrotic tissue, and swollen varicose veins. A solution of heparin may be infused into the area to prevent scar tissue from forming. Manipulation is minimal, and usually patients can go home within 24 h [52].

Other Therapies

Stem cells have been used in multiple therapies, most recently for PNa. Venturi et al. [53] described the injection of adipose tissue and stem cells into Alcock's canal of 15 women, a technique they called "lipofilling." Two patients had no response, but there was an improvement in pain and nerve conduction in the remaining patients.

Complications

Laceration of the vaginal mucosa is a potential complication. The pudendal nerve is surrounded by arterial and venous blood flow; intravascular injections can cause systemic anesthetic complications, which may include palpitation, tinnitus, dysarthria, drowsiness, confusion, loss of consciousness, convulsions, hypotension, and bradycardia. Needle trauma can cause hematomas (vaginal, retroperitoneal, and ischiorectal) from injury to the pudendal artery. Infection (retro psoas and subgluteal abscess) has occasionally been reported, spreading superiorly along the psoas muscle or laterally along the obturator internus [32].

Summary

Pudendal neuralgia can cause devastating and debilitating pain if undiagnosed and untreated. Although there are many causes of pelvic pain, this is a relatively complicated but treatable etiology and should be carefully considered for all patients with pelvic pain.

References

1. Mathias SD, Kuppermann M, Liberman RF, Lipschutz RC, Steege JF. Chronic pelvic pain: prevalence, health-related quality of life, and economic correlates. Obstet Gynecol. 1996;87(3):321–7.
2. Dodson MG, Friedrich Jr EG. Psychosomatic vulvovaginitis. Obstet Gynecol. 1978;51(1 Suppl):23s–5.
3. Hibner M, Desai N, Robertson LJ, Nour M. Pudendal neuralgia. J Minim Invasive Gynecol. 2010;17(2):148–53.
4. Amarenco G, Lanoe Y, Perrigot M, Goudal H. A new canal syndrome: compression of the pudendal nerve in Alcock's canal or perineal paralysis of cyclists. Press Med (Paris, France: 1983). 1987;16:399.
5. Bondar A, Egan M, Jochum D, Amarenco G, Bouaziz H. Case report: pudendal nerve injury after a sciatic nerve block by the posterior approach. Anesth Analg. 2010;111(2):573–5.
6. Báča V, Bacova T, Grill R, Otcenasek M, Kachlik D, Bartoska R, et al. Pudendal nerve in pelvic bone fractures. Injury. 2013;44(7):952–6.
7. Pailhe R, Chiron P, Reina N, Cavaignac E, Lafontan V, Laffosse JM. Pudendal nerve neuralgia after hip arthroscopy: retrospective study and literature review. Orthop Traumatol Surg Res. 2013;99(7):785–90.
8. Dellon AL, Wright EJ, Manson PN. Chronic pelvic pain after laser prostatectomy: treatment by resection of the perineal branches of the pudendal nerve. J Reconstr Microsurg. 2014;30(8):547–50.
9. Tetzschner T, Sorensen M, Lose G, Christiansen J. Pudendal nerve function during pregnancy and after delivery. Int Urogynecol J Pelvic Floor Dysfunct. 1997;8(2):66–8.
10. Howard EJ. Postherpetic pudendal neuralgia. JAMA J Am Med Assoc. 1985;253(15):2196.
11. Bussemaker L, Schelhaas HJ, Overeem S, Hopman WP, Zwarts MJ. Treatment-responsive pudendal dysfunction in chronic inflammatory demyelinating polyneuropathy. Neurology. 2007;68(12):957–8.
12. Elahi F, Callahan D, Greenlee J, Dann TL. Pudendal entrapment neuropathy: a rare complication of pelvic radiation therapy. Pain Physician. 2013;16(6):E793–7.
13. Labat JJ, Riant T, Robert R, Amarenco G, Lefaucheur JP, Rigaud J. Diagnostic criteria for pudendal neuralgia by pudendal nerve entrapment (Nantes criteria). Neurourol Urodyn. 2008;27(4):306–10.
14. Nickel JC, Berger R, Pontari M. Changing paradigms for chronic pelvic pain: a report from the chronic pelvic pain/chronic prostatitis scientific workshop, October 19–21, 2005, Baltimore, MD. Rev Urol. 2006;8(1):28–35.
15. Nickel JC, Berger R, Pontari M. Changing Paradigms for Chronic Pelvic Pain: A Report from the Chronic Pelvic Pain/Chronic Prostatitis Scientific Workshop, October 19–21, 2005, Baltimore, MD. Reviews in Urology. 2006;8(1):28–35.
16. Andersen KV, Bovim G. Impotence and nerve entrapment in long distance amateur cyclists. Acta Neurol Scand. 1997;95(4):233–40.
17. Possover M, Forman A. Voiding dysfunction associated with pudendal nerve entrapment. Curr Bladder Dysfunct Rep. 2012;7(4):281–5.
18. Shafik A. Pudendal canal syndrome as a cause of vulvodynia and its treatment by pudendal nerve decompression. Eur J Obstet Gynecol Reprod Biol. 1998;80(2):215–20.
19. Follmar KE, Williams EH, Dellon AL. Rectal pain of neural origin: resection of sensory rectal branches of pudendal nerve. J Reconstr Microsurg. 2015;31(2):119–23.
20. Huang JC, Deletis V, Vodusek DB, Abbott R. Preservation of pudendal afferents in sacral rhizotomies. Neurosurgery. 1997;41(2):411–5.
21. Popeney C, Ansell V, Renney K. Pudendal entrapment as an etiology of chronic perineal pain: diagnosis and treatment. Neurourol Urodyn. 2007;26(6):820–7.
22. Montoya TI, Calver L, Carrick KS, Prats J, Corton MM. Anatomic relationships of the pudendal nerve branches. Am J Obstet Gynecol. 2011;205(5):504 e1–5.
23. Thoumas D, Leroi AM, Mauillon J, Muller JM, Benozio M, Denis P, et al. Pudendal neuralgia: CT-guided pudendal nerve block technique. Abdom Imaging. 1999;24(3):309–12.
24. Barber MD, Bremer RE, Thor KB, Dolber PC, Kuehl TJ, Coates KW. Innervation of the female levator ani muscles. Am J Obstet Gynecol. 2002;187(1):64–71.

25. Butler DS. Mobilization of the Nervous System. London, Churchill Livingston; 1991, pp.55–74.
26. Šedý J, Nanka O, Belisova M, Walro JM, Jarolim L. Sulcus nervi dorsalis penis/clitoridis: anatomic structure and clinical significance. Eur Urol. 2006;50(5):1079–85.
27. Tagliafico A, Bignotti B, Miguel Perez M, Reni L, Bodner G, Martinoli C. Contribution of ultrasound in the assessment of patients with suspect idiopathic pudendal nerve disease. Clin Neurophysiol. 2014;125(6):1278–84.
28. Seckin B, Kibar Y, Goktas S, Erdemir F. Alcock's canal releasing for pudendal artery syndrome resulting from gunshot injury. Int J Impot Res. 2005;17(5):471–3.
29. Ramsden CE, McDaniel MC, Harmon RL, Renney KM, Faure A. Pudendal nerve entrapment as source of intractable perineal pain. Am J Phys Med Rehabil. 2003;82(6):479–84.
30. Jiang HH, Pan HQ, Gustilo-Ashby MA, Gill B, Glaab J, Zaszczurynski P, et al. Dual simulated childbirth injuries result in slowed recovery of pudendal nerve and urethral function. Neurourol Urodyn. 2009;28(3):229–35.
31. Pan HQ, Lin DL, Strauch C, Butler RS, Monnier VM, Daneshgari F, et al. Pudendal nerve injury reduces urethral outlet resistance in diabetic rats. Am J Ren Physiol. 2010;299(6):F1443–50.
32. Satpathy HK, Chelmow D. Nerve block, transvaginal pudendal. 2010 [7 Jan 11]. Available from: http://emedicine.medscape.com/article/83078-overview.
33. Abdi S, Shenouda P, Patel N, Saini B, Bharat Y, Calvillo O. A novel technique for pudendal nerve block. Pain Physician. 2004;7(3):319–22.
34. Peng PW, Narouze S. Ultrasound-guided interventional procedures in pain medicine: a review of anatomy, sonoanatomy, and procedures: part I: nonaxial structures. Reg Anesth Pain Med. 2009;34(5):458–74.
35. Hough DM, Wittenberg KH, Pawlina W, Maus TP, King BF, Vrtiska TJ, et al. Chronic perineal pain caused by pudendal nerve entrapment: anatomy and CT-guided perineural injection technique. AJR Am J Roentgenol. 2003;181(2):561–7.
36. Mamlouk MD, van Sonnenberg E, Dehkharghani S. CT-guided nerve block for pudendal neuralgia: diagnostic and therapeutic implications. AJR Am J Roentgenol. 2014;203(1):196–200.
37. Filippiadis DK, Velonakis G, Mazioti A, Alexopoulou E, Malagari A, Brountzos E, et al. CT-guided percutaneous infiltration for the treatment of Alcock's neuralgia. Pain Physician. 2011;14(2):211–5.
38. Fritz J, Chhabra A, Wang KC, Carrino JA. Magnetic resonance neurography-guided nerve blocks for the diagnosis and treatment of chronic pelvic pain syndrome. Neuroimaging Clin N Am. 2014;24(1):211–34.
39. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
40. Prologo JD, Lin RC, Williams R, Corn D. Percutaneous CT-guided cryoablation for the treatment of refractory pudendal neuralgia. Skeletal Radiol. 2015;44(5):709–14.
41. Masala S, Calabria E, Cuzzolino A, Raguso M, Morini M, Simonetti G. CT-Guided percutaneous pulse-dose radiofrequency for pudendal neuralgia. Cardiovasc Intervent Radiol. 2014;37(2):476–81.
42. Rhame EE, Levey KA, Gharibo CG. Successful treatment of refractory pudendal neuralgia with pulsed radiofrequency. Pain Physician. 2009;12(3):633–8.
43. Ko HY, Kim KT. Treatment of external urethral sphincter hypertonicity by pudendal nerve block using phenol solution in patients with spinal cord injury. Spinal Cord. 1997;35(10):690–3.
44. Calabro RS, Gervasi G, Marino S, Mondo PN, Bramanti P. Misdiagnosed chronic pelvic pain: pudendal neuralgia responding to a novel use of palmitoylethanolamide. Pain Med. 2010;11(5):781–4.
45. Martin, R. Spinal cord stimulation for refractory pudendal neuralgia. Poster presentation, Texas Pain Foundation 7th Annual Pain Expo, Oct 2015, San Antonio.
46. Richter EO, Abramova MV, Alo KM. Percutaneous cephalocaudal implantation of epidural stimulation electrodes over sacral nerve roots – a technical note on the importance of the lateral approach. Neuromodulation. 2011;14(1):62–7.
47. Valovska A, Peccora CD, Philip CN, Kaye AD, Urman RD. Sacral neuromodulation as a treatment for pudendal neuralgia. Pain Physician. 2014;17(5):E645–50.
48. Buffenoir K, Rioult B, Hamel O, Labat JJ, Riant T, Robert R. Spinal cord stimulation of the conus medullaris for refractory pudendal neuralgia: a prospective study of 27 consecutive cases. Neurourol Urodyn. 2015;34(2):177–82.
49. Louppe JM, Nguyen JP, Robert R, Buffenoir K, de Chauvigny E, Riant T, et al. Motor cortex stimulation in refractory pelvic and perineal pain: report of two successful cases. Neurourol Urodyn. 2013;32(1):53–7.
50. Possover M. A new technique of laparoscopic implantation of stimulation electrode to the pudendal nerve for treatment of refractory fecal incontinence and/or overactive bladder with urinary incontinence. J Minim Invasive Gynecol. 2014;21(5):729.
51. Robert R, Labat JJ, Bensignor M, Glemain P, Deschamps C, Raoul S, et al. Decompression and transposition of the pudendal nerve in pudendal neuralgia: a randomized controlled trial and long-term evaluation. Eur Urol. 2005;47(3):403–8.
52. Erdogru T, Avci E, Akand M. Laparoscopic pudendal nerve decompression and transposition combined with omental flap protection of the nerve (Istanbul technique): technical description and feasibility analysis. Surg Endosc. 2014;28(3):925–32.
53. Venturi M, Boccasanta P, Lombardi B, Brambilla M, Contessini Avesani E, Vergani C. Pudendal neuralgia: A new option for treatment? Preliminary results on feasibility and efficacy. Pain Med. 2015;16(8):1475–81.

Obturator Nerve Entrapment: Pelvic

48

Andrea M. Trescot

Introduction

Groin pain is often difficult to diagnose because of the many structures that pass through that region, and *obturator nerve* (ON) entrapment is not commonly diagnosed or considered. The ON can present as groin pain, pelvic pain, and/or lower extremity pain. ON entrapment is also called *obturator tunnel syndrome*. This chapter will discuss the presentation, diagnosis, and treatment of obturator nerve (ON) entrapment as a cause of pelvic and groin pain, while Chap. 63 will discuss the obturator nerve's lower extremity presentation.

Clinical Presentation (Table 48.1)

Patients with obturator entrapment present with groin pain (Table 48.1), often related to participation in sports, especially those involving kicking or twisting [17]. Males seem to be much more affected than females, perhaps because of the sports association [3]. In fact, in one study, 81 % of athletes with ON entrapment were soccer players [17]. Patients note exercise-induced medial thigh pain, adductor muscle weakness, and pelvic pain, and they may complain of paresthesias along the lateral peroneal region (Fig. 48.1), with pain on adduction or a monopedal stance [27]. Other common symptoms include sensory loss or a deep ache in the medial thigh from the pubis to the medial knee, occasionally to the ipsilateral ASIS [3], and very rarely to the medial calf [28]. Pain is worsened by maneuvers, such as the lithotomy position, which can stretch the ON [14], and medial knee pain can be induced by forced hip abduction, extension, and internal rotation (*Howship-Romberg's sign*) [29]. There may be a sense of "lack of propulsion… during running but numbness is very rarely reported" [30]. Like with claudication, the symptoms

Table 48.1 Occupation/exercise/trauma history relevant to obturator nerve entrapment

Abdominal conditions [1, 2]	Pelvic hematomas/hemorrhage, retroperitoneal masses [3, 4]
	Endometriosis [5]
	Infection/inflammation [6, 7]
	Malignant tumor [8]
	Genitourinary pathology [5, 9, 10]
Trauma	Pregnancy and delivery [11, 12]
	Prolonged lithotomy position [13, 14]
	Gunshot wound to the abdomen [4]
	Stress fracture pubic ramus/fracture pelvis [15, 16]
	Sports injuries [3, 17]
	Hip replacement [18–22]
Groin compression	Bleeding after cardiac catheterization [23]
	Obturator hernia [4]
	Acetabular cyst/obturator foramen tumor [24, 25]

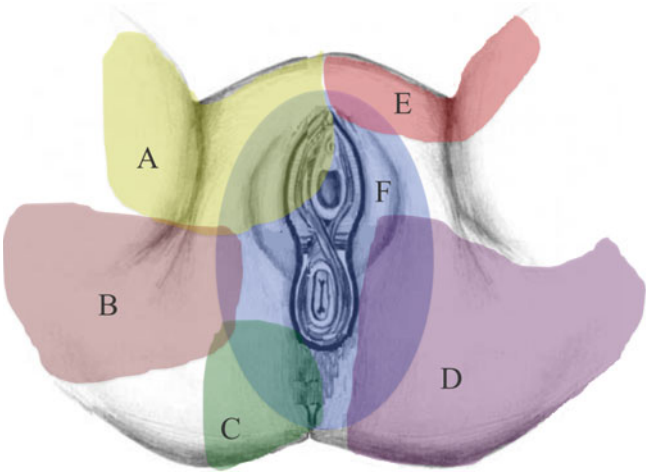

Fig. 48.1 Innervation of perineum: (*A*) genitofemoral nerve, (*B*) obturator nerve, (*C*) inferior cluneal nerve, (*D*) peroneal branch of the posterior femoral cutaneous nerve, (*E*) ilioinguinal nerve, and (*F*) pudendal nerve (Image inspired by Hibner et al. [26], courtesy of Andrea Trescot, MD)

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subside with rest but reoccur with the resumption of activity. According to Sorenson, 73 % of patients with known ON entrapment complained of groin and thigh pain [27].

Although nerve injuries after hip arthroplasty are not common (0.7–1 % of all postoperative complications), obturator nerve pathology after total hip replacement (THR) can be a cause of persistent hip or groin pain, as well as varying degrees of adductor weakness [19].

Anatomy (Table 48.2)

The ON arises from the *anterior divisions* of the ventral rami of L2, L3, and L4, which join within the *psoas muscle* to form the ON (Table 48.2), as opposed to the *femoral nerve*, which is formed by the *posterior divisions* of the ventral rami of those same nerves. Rarely, the entire ON can be positioned posterior to the psoas muscle [34]. The ON then travels over the pelvic brim at the level of the sacroiliac joint (Fig. 48.2), curving anterior inferiorly along the lateral pelvic wall through a fibro-osseous tunnel [3]. It is the only nerve from the *lumbar plexus* (Chap. 49), which does not innervate any of the intrapelvic structures. The ON passes into the obturator foramen through a fibro-osseous tunnel (the *obturator canal*), which is formed superiorly by the obturator sulcus of the pubic bone and inferiorly by the *internal and external obturator muscles* (Fig. 48.3) [35]. Within the tunnel, the nerve splits into two main branches (an anterior branch and a posterior branch), as well as a branch to the *external obturator muscle* (which crosses the *obturator artery*), and then exits through the *obturator tunnel* to enter the thigh. Kumka [35] noted that the ON could bifurcate within the pelvic cavity, at the entrance of the pelvic cavity, within the obturator canal, or at the exit of obturator canal.

The anterior branch travels anterior to the *external obturator muscle* and posterior to the pectineal muscle and gives a branch to the hip joint. It innervates the *adductors* and the *gracilis* and *pectineal* muscles, with a cutaneous branch descending in the adductor canal to innervate the skin of the distal two-thirds of the medial thigh. The larger posterior branch pierces and supplies the *external obturator* muscle and the *adductor magnus* (which is also innervated by the sciatic nerve) and then runs between the *short and great adductors*, with a sensory branch that also descends through the *adductor canal* to supply the *knee capsule*, *cruciate ligaments*, and *synovial membranes* (Table 48.3). The posterior branch occasionally innervates the *adductor brevis* and usually was found by dissection to be in immediate contact with the *musculotendinous aponeurotic arch* [35]. There is also an *accessory obturator nerve*, arising from L3 to L4, present in about 13 % of the population [32], which descends along the medial psoas and crosses the superior pubic ramus behind the *pectineal muscle*, providing the nerve supply to that muscle and the hip joint [3]. The adductor magnus and longus are also partially innervated by the sciatic and femoral nerves [36]. The adductor brevis is the only muscle solely innervated by the obturator nerve [37].

The ON can be seen by ultrasound (US) in the triangle formed by the lower border of the superior pubic ramus, posterior pectineus, and anterior surface of obturator externus [33]. The anterior division is easier to visualize by ultrasound than the posterior division, which is within fascia [38, 39] (see section "Ultrasound (US)-Guided Technique").

Table 48.2 Anatomy of the obturator nerve

Origin	Anterior divisions of ventral rami of L2–L4 nerve roots (femoral nerve is the posterior division of ventral rami of the same nerves)
General route	Formed within the psoas muscle, leaves its medial border and travels through the pelvis medial to the femoral nerve. Follows the iliopectineal line into the lesser pelvis and through the fat-filled obturator canal with the obturator vessels
	Variable anatomy. Division into the anterior and posterior branches may occur in the pelvis (23 %), in the canal (52 %), or in the thigh (25 %) [31]. An accessory ON is present in 13 % of the population [32] and may communicate with the femoral nerve
Sensory distribution	Articular branch to the anteromedial hip joint from anterior branch
	Articular branch to medial knee joint from posterior branch
	Cutaneous branch to a patch of skin on the inner thigh, just above the medial knee from anterior branch. Absent in 20 % of the population [33]
Motor innervation	Obturator externus
	Anterior division: adductor longus, brevis, gracilis, and sometimes pectineus (pectineus gets 90 % of its motor from femoral nerve)
	Posterior division: adductor magnus (also gets motor from the tibial branch of the sciatic nerve)
	Only the adductor brevis is innervated solely by ON
Sonoanatomy	In the triangle formed by the lower border of the superior pubic ramus, posterior pectineus, and anterior surface of obturator externus [33]

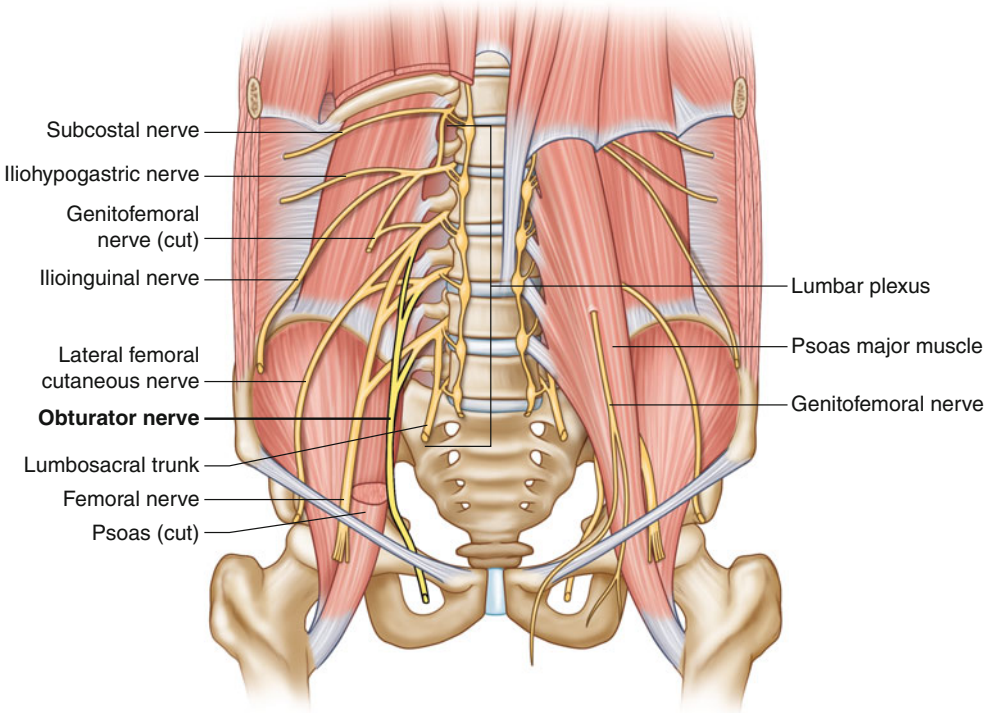

Fig. 48.2 Anatomy of the pelvic nerves (Image by Springer)

Entrapment

The ON is rarely injured in isolation. The course of the obturator nerve through the pelvis puts it at risk for entrapment, compression, and damage at many sites. Prolonged, acute hip flexion, such as seen in urologic and gynecologic surgery in the lithotomy position, has been associated with obturator palsy [10]. Litwiller et al. [14] studied various lithotomy positions and found that abduction to greater than 30° without concomitant hip flexion increased strain on the obturator nerve, both in its pelvic segment and the anterior branch. Several authors [6, 7] have described an *obturator tunnel syndrome* (Fig. 48.4). Bradshaw and McCoy [3] described a fascial entrapment of the obturator in the adductor compartment, induced by exercise, confirmed by anatomic dissection [40]. Bradshaw and McCoy [3] also suggested that the male predominance of this condition may be related to anatomy; males have higher iliac crests, a smaller transverse diameter of the pelvic inlet, and a narrower subpubic angle, which may put a greater bend in the nerve as it passes through the obturator canal.

Based on the dissections by Kumka [35] described above, the likely sites of entrapment include:

- Within the obturator canal, by the vascular bundle of the obturator vessels or by complications of gynecologic or orthopedic surgery (see above)
- In the fibromuscular canal formed by the anterior surface of the obturator membrane and the posterior surface of the obturator externus muscle
- In the muscular tunnel where the posterior division perforates the obturator externus muscle
- Within the distinct fascial plane situated deep to the pectineus and adductor brevis muscles and superficial to the obturator externus and the proximal one-third of the adductor magnus muscles

Several authors [18–22] described ON entrapment and/or heat damage of the femoral and obturator nerves caused by cement extrusion from hip arthroplasty. Haninec et al. [41] described obturator nerve injury after laparoscopic inguinal hernia mesh repair. There may also be entrapment from fibrous bands formed due to chronic adductor tendinopathy or osteitis pubis.

Physical Exam

The hallmark of obturator pathology is adductor weakness, with muscle atrophy in severe cases [28]. The *obturator externus* provides lateral rotation of the thigh, while the gracilis provides flexion and internal rotation. With loss of adduction and internal rotation, the hip will be externally rotated and abducted, which results in a wide-based, circumducting gait [42]. Ipsilateral loss of hip adductor tendon reflex sug-

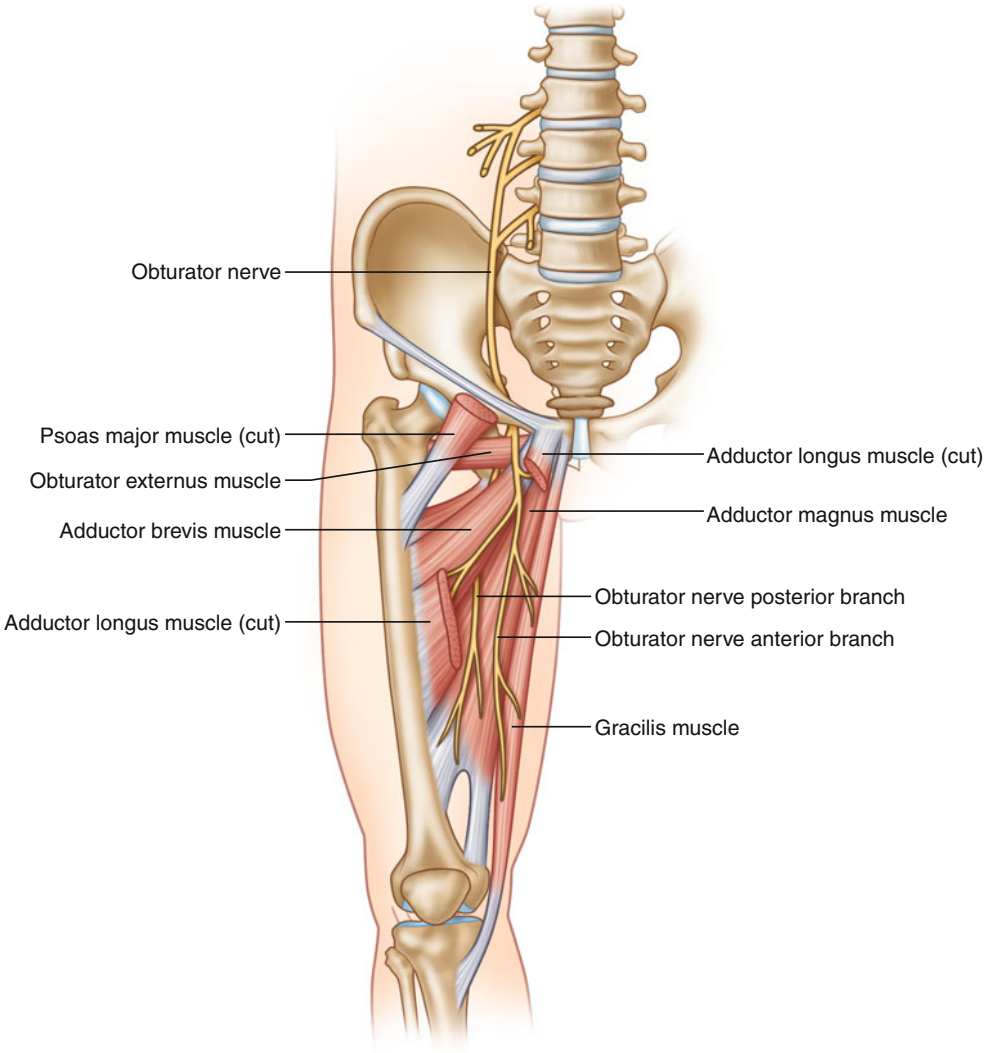

Fig. 48.3 Anatomy of the obturator nerve (Image courtesy of Springer)

Table 48.3 Kumka [35] dissected 56 lower limbs and found the following variations

Posterior division crosses over the aponeurotic arch, anterior to obturator externus	32/56
Posterior division emerges from obturator externus muscle, perforates it, and descends anterior to its distal part	22/56
Posterior division descends entirely posterior to the obturator externus muscle, emerging from its inferior border	2/56

gests but does not prove obturator pathology, since this reflex is not always present, even in healthy people [28]. There may be tenderness to deep palpation in the *adductor canal* (Fig. 48.5), as well as increased pain when the nerve is stretched from extension and/or lateral leg movement [4]. A *pectineus stretch* (where the patient actively externally rotates and abducts the hip) will stretch the obturator nerve [36]; pain may also be reproduced with internal rotation of the flexed hip against resistance (*obturator sign*) (Fig. 48.6) [37]. This test is also used on the right side to look for appendicitis.

An objective measure of adductor strength was devised by Lang [43] and used by several subsequent investigators [44, 45].

Differential Diagnosis (Table 48.4)

The innervation of the hip joint includes the femoral, sciatic, superior gluteal, and obturator nerves. Groin pain can be due to hip OA, avascular necrosis, adductor or rectus abdominus tendonitis, greater trochanter bursitis, adductor spasm, osteitis pubis, stress fracture, pregnancy (by the fetal head, forceps, or hematoma), aneurism of the hypogastric artery, lumbar radiculopathy, cardiac catheterization (from retroperitoneal hematoma), and sports hernia (obturator

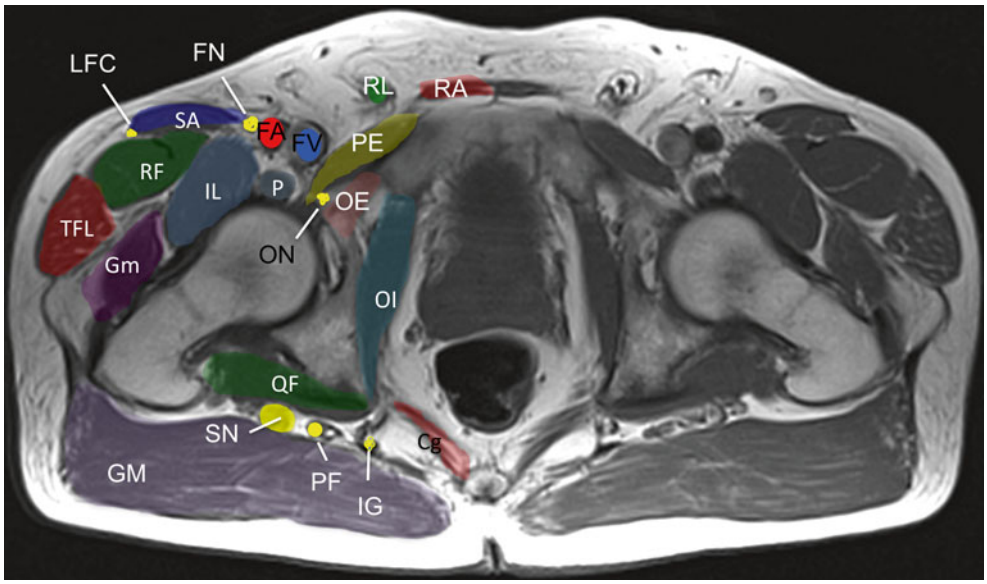

Fig. 48.4 Axial MRI of the pelvis. Axial MRI of the pelvic structures. *Cg* coccygeus muscle, *FA* femoral artery, *FN* femoral nerve, *FV* femoral vein, *GM* gluteus maximus muscle, *Gm* gluteus medius, *IG* inferior gluteal nerve, *IL* iliopsoas muscle, *LFC* lateral femoral cutaneous nerve, *OI* obturator internis muscle, *ON* obturator nerve, *P* psoas muscle, *PE* pectineus muscle, *PF* posterior femoral cutaneous nerve, *QF* quadratus femoris muscle, *RA* rectus abdominus muscle, *RF* rectus femoris muscle, *RL* round ligament, *SA* sartorius muscle, *SN* sciatic nerve, *TFN* tensor fascia lata muscle (Image courtesy of Andrea Trescot, MD)

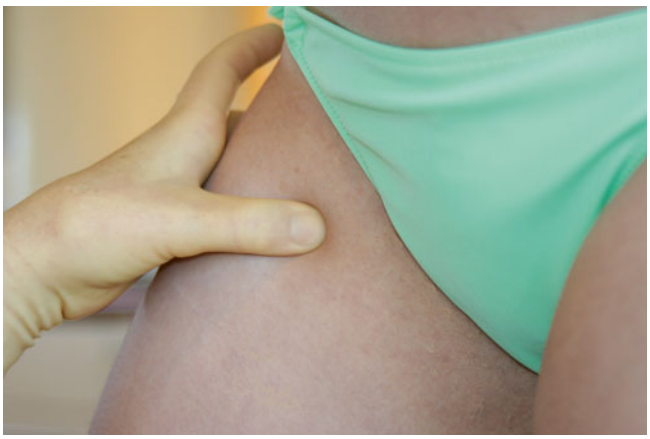

Fig. 48.5 Obturator examination (Image courtesy of Andrea Trescot, MD)

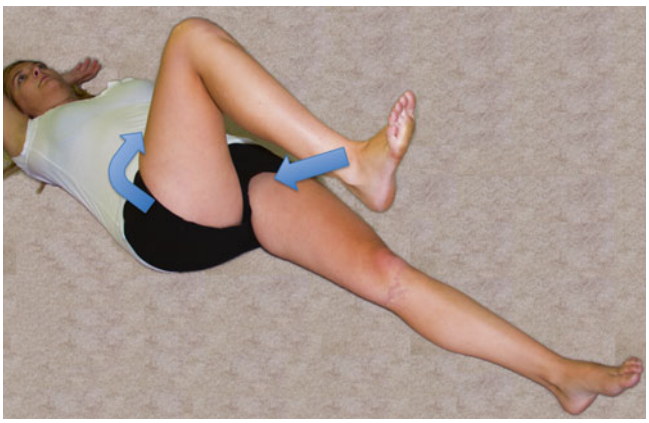

Fig. 48.6 Obturator sign – internal rotation of the flexed hip (Image courtesy of Andrea Trescot, MD)

hernia) [48]. Travell and Simons [49] described trigger points (myofascial pain) of the long adductor muscle as "perhaps the most common cause of groin pain," though Bradshaw and McCoy [3] point out that at least one of the patients described by Travell and Simons had a "clear-cut obturator block."

Diagnostic Tests (Table 48.5)

X-rays may contribute to the diagnosis by identifying pubic abnormalities, such as small bone spurs in the region of the ipsilateral pubic tubercle. In the 32 athletes with groin pain studied by Bradshaw and McCoy [3], 21 of 24 patients showed bone scan uptake on the ipsilateral pubic tubercle at the origins of the short and long adductors. MRI may show atrophy of the adductor brevis and longus or gracilis muscles [49], as well as identify intrapelvic masses that might entrap the obturator nerve [42]. EMG/NCV studies can confirm the entrapment, showing fibrillation or complex motor unit potentials of the short and long hip adductor muscles, while the iliopsoas and quadriceps are normal. Bradshaw et al. [51] reported that all 32 of the groin pain patients they studied had EMG evidence of denervation changes to the ON with normal paravertebral muscles.

Identification and Treatment of Contributing Factors

Transurethral resection of the prostate (TURP) is occasionally associated with adductor spasms, which can be relieved with obturator nerve injections.

Bradshaw and McCoy [3] noted that several of the patients in their series had symptoms of inguinal hernia prior to the

Table 48.4 Differential diagnosis of hip and groin pain

	Potential distinguishing features
Osteoarthritis hip or knee	X-ray and MRI show DJD
Adductor or rectus abdominis tendonitis/myofascial spasm	Tenderness at pubis; palpable spasm
Avascular necrosis	X-ray and MRI show femoral head collapse
Osteitis pubis or stress fracture	Bone scan is positive
Lumbar radiculopathy	EMG is positive
Articular femoral nerve branches	Femoral nerve distribution of pain and weakness (see Chap. 57)
Facet arthropathy [46]	Paravertebral tenderness, lumbar spondylosis
SI joint [46]	PSIS tenderness
Piriformis muscle syndrome [46]	Increased pain with hip internal rotation
L1–L3 radiculopathy [46]	Sensory loss and reflex changes
Hip flexor tendonitis [46]	Tenderness at the tendon attachment
Intestinal hernia [17]	Hernia on physical exam
Sports hernia [47]	Hernia on physical exam
Knee pain [46]	X-rays/MRI of the knee
Hip joint pathology [36]	Stiffness, limited range of motion, crepitus, clicking
Ilioinguinal nerve injury/entrapment [17]	Increased abdominal wall tension can result in groin pain; may be tender near ASIS; increased pain with hip hyperextension
Pubic symphysitis [17]	Pain moves from side to side
Adductor strain [36]	Tenderness over adductors. Usual site is at the muscle-tendon intersection; sometimes at tendon-bone

Table 48.5 Diagnostic tests for obturator N entrapment

Test	Potential distinguishing features
Physical exam	Adductor weakness; wide-based, circumducting gait [42]
X-ray	Pubic ramus spurring; rule out other causes
MRI	Atrophy of adductor brevis and longus or gracilis [50]; rule out other causes
Electrodiagnostic studies	Needle EMG is consistent with acute and chronic denervation of the adductor muscles with normal readings in other lower extremity muscles

development of medial thigh weakness, and they proposed the possibility of a mechanical entrapment.

Entrapment of the ON by the external obturator muscle (see Fig. 48.4) may cause a persistent pathology. Kassolik et al. [52] describe the use of massage of the obturator and piriformis muscles to treat ON entrapment.

Injection Technique

ON injections are used to help to confirm the diagnosis of obturator neuropathy. ON injections have been used to treat adductor spasms from cerebral palsy or during and after TURP [53], to supplement analgesia for knee surgery, and to treat chronic hip pain. They can also help to diagnose occult causes of groin and pelvic pain. Bouaziz et al. [54] stated that the only way to reliably confirm obturator anesthesia is to assess adductor strength.

Landmark-Guided Technique

Winnie et al. [55] introduced a "three-in-one" anesthesia injection (Fig. 48.7), designed to anesthetize the femoral, lateral femoral cutaneous, and obturator nerves at the same time. The ON can be blocked either proximal or distal to the division into the anterior and posterior branches. Proximally, only one injection is necessary, while after the division, it is necessary to injection both nerve separately.

The standard landmark-guided ON injection involves positioning the patient supine, with the leg slightly abducted and externally rotated; the needle is placed 2 cm caudad and 2 cm lateral to the pubic tubercle, advancing the needle onto the inferior border of superior pubic ramus and then dropping into the obturator canal [56] (Fig. 48.8), with or without a PNS.

The more distal, inguinal approach has the patient positioned supine with the leg slightly abducted. The patient is asked to flex the hip, and a line is drawn to mark the inguinal crease. The adductor longus tendon will be the most superficial palpable tendon in the medial thigh. A mark is made at the midpoint of the adductor longus and the femoral artery, at the site of the groove between the vascular bundle and the adductor. The needle is inserted at this site and advanced at a 30° cephalad direction with a PNS, until contractions of the gracilis or adductor are identified (anterior branch). Here, 5 cc of local anesthetic is injected, and then the needle is advanced

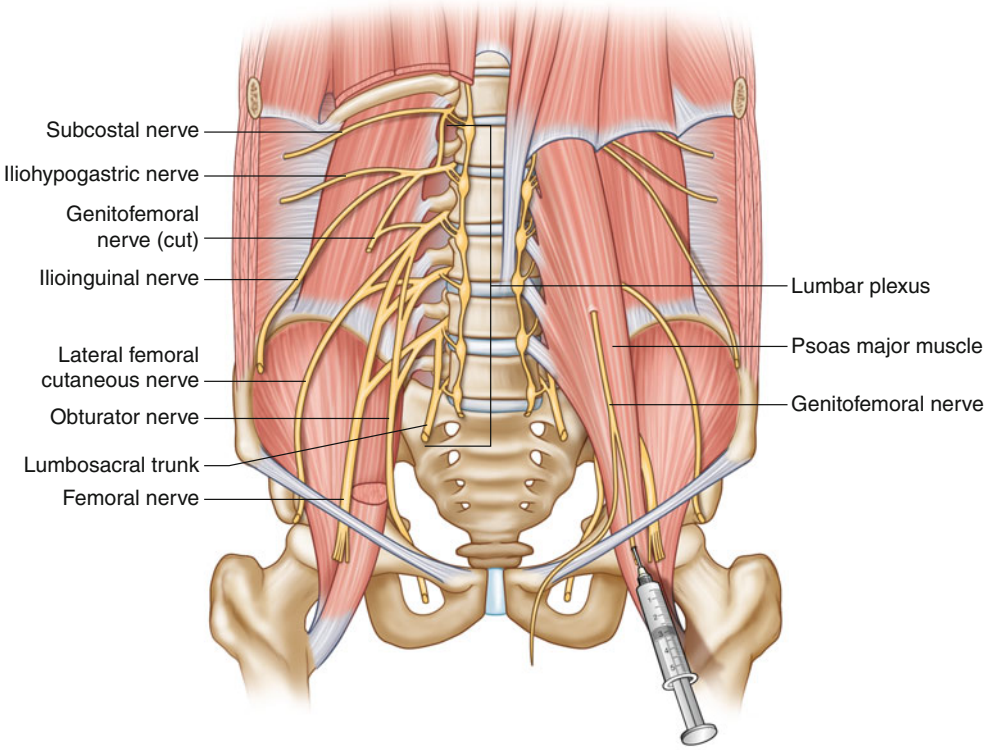

Fig. 48.7 Location of needle for a "3-in-1" injection (Image by Springer)

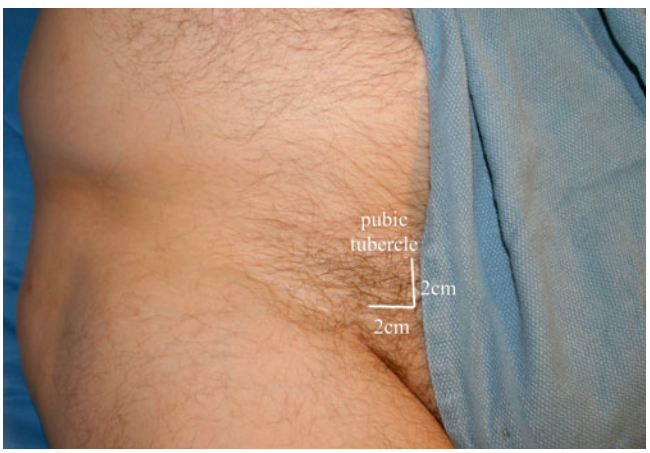

Fig. 48.8 Landmark-guided obturator injection (Image courtesy of Andrea Trescot, MD)

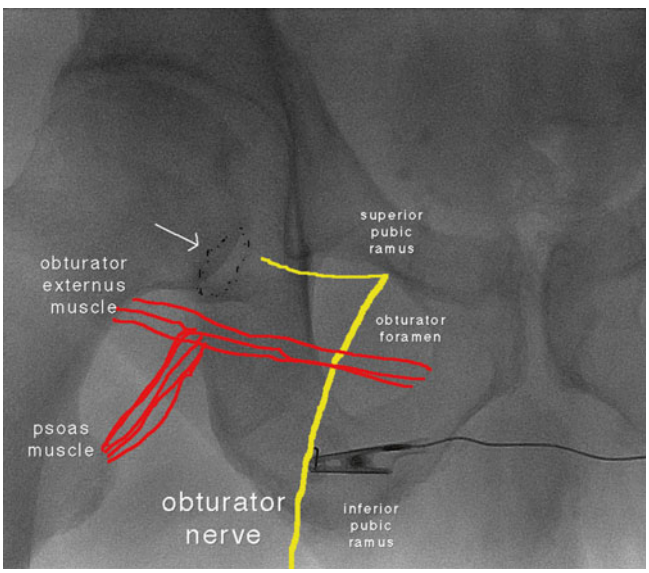

Fig. 48.9 Fluoroscopic anatomy and obturator injections sites. *White arrow* identifies the "teardrop" target for hip denervation techniques (Image courtesy of Andrea Trescot, MD)

deeper and 5° laterally, to obtain contractions of the adductor magnus (posterior branch), where another 5 cc of local is injected. In 2005, Choquet et al. [45] performed a randomized trial of 50 patients undergoing knee arthroscopy, comparing an inguinal approach versus the standard approach to the ON. Patients in the inguinal group reported less pain during the procedure, and there were no failures. This group also confirmed that the motor strength, not sensory hypoesthesia, was the only reliable way to evaluate nerve function, noting that 48 % of their patients noted no areas of sensory hypoesthesia 20 min after the injection.

Fluoroscopy-Guided Technique

Using fluoroscopy to aid the needle placement for an ON injection involves the fluoroscopic visualization of the superior and inferior pubic rami. The patient is placed supine, and the inferior portion of the obturator foramen is identified. A peripheral nerve stimulator can help to identify the nerve on the inferior pubic ramus (Fig. 48.9).

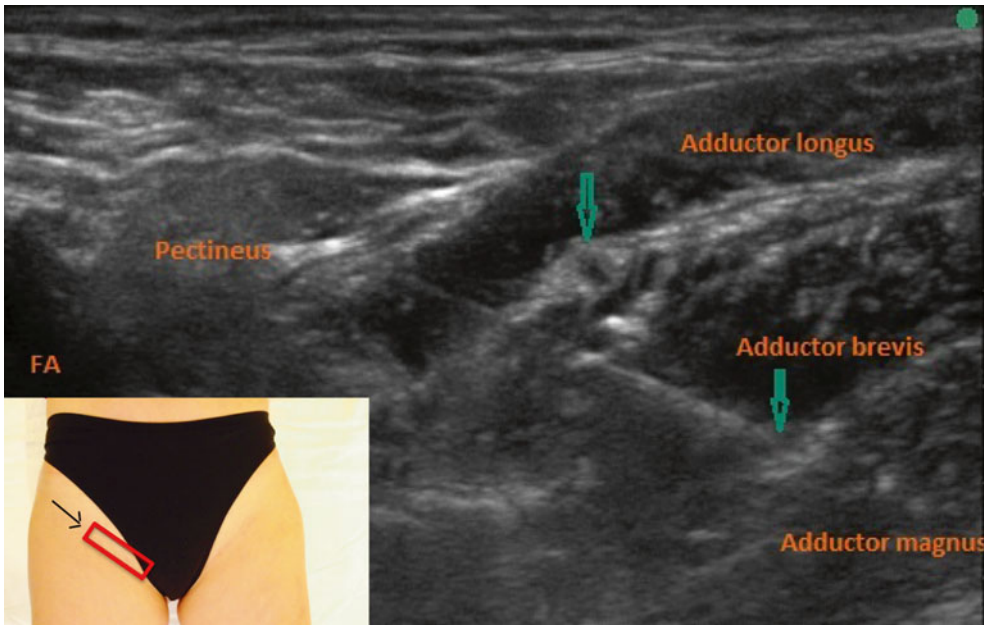

Fig. 48.10 Top green arrow marks the superficial branch of the obturator nerve, below the inguinal ligament. Needle injecting the deep branch of the obturator nerve, also marked by *green arrow. FA* femoral artery. Femoral vein compressed (Image courtesy of Agnes Stogicza, MD)

The hip joint is innervated by sensory branches of the obturator nerve as well as the femoral nerves (Chap. 56). For diagnostic ON injections of the hip joint, the patient is placed supine, and the needle is placed just medial to the femoral artery, below the inguinal ligament; the needle tip is then directed under fluoroscopy to the inferior junction of the ischium and pubis, where the "teardrop" landmark is made up of the wall of the acetabulum, the lesser pelvis, and the acetabular notch (Fig. 48.9).

Ultrasound (US)-Guided Technique

Some authors use US alone, while others use US with a nerve stimulator [44]. Soong et al. [57] described two techniques of finding the obturator nerve under US. The patient is examined supine, with the thigh slightly externally rotated. The first technique involves scanning laterally from the pubic tubercle until the three muscle layers (adductor longus, adductor brevis, and adductor magnus) were identified, moving the probe in a medial/lateral or proximal/distal direction to find the anterior and posterior divisions of the obturator nerve (Fig. 48.10). In the second technique, the femoral artery/vein/nerve is identified at the femoral crease, and the probe is moved medially toward the pubis to visualize the obturator nerve, which, unlike other nerves, appears flat instead of honeycombed.

Manassero et al. [58] compared two types of US-directed adductor injections – using either a peripheral nerve stimulator (PNS) or an intrafascial injection – on 716 patients undergoing TURP in a lithotomy position. The symptomatic leg was lowered from the lithotomy position, extended, and slightly externally rotated. An ultrasound probe for one group (interfascial) was positioned at 90-degrees to the skin, parallel to and 2 to 3 cm below the inguinal crease. The probe was positioned medially from the femoral nerve until the muscle layers of the adductor longus, adductor brevis, and adductor longus could be visualized. A 22-guage insulated needle was advanced using an in-plane approach, first to the fascial layer between the adductor longus and brevis (where 5 cc of local anesthetic was injected), and then advanced between the adductor brevis and magnus (where another 5 cc of local anesthetic was injected). For the PNS group, the US probe was positioned to visualize the anterior and posterior divisions of the ON between the adductor longus, brevis, and magnus muscles. Using an in-plane approach, the needle was advanced to the posterior division of the ON, looking for adductor magnus contractures of the posterior thigh. After 5 cc of local anesthetic was injected at this site, the needle was redirected to the anterior division; once there was adductor brevis and longus contraction at low amplitude, another 5 cc of local anesthetic was injected. They found no difference in success rates, but the PNS stimulation took slightly longer; however, when there was a failure of the initial interfascial technique, adding the PNS improved the success rate to 100 %.

Using a more proximal approach has the potential advantage of more reliable blockade of branches to the hip joint. Akkaya and colleague [33] described a more proximal approach, first dissecting the obturator structures and then identifying the structures with US. They described a "triangle," with the superior pubic ramus as the superior border, the pectineus muscle as the anterior border, and the external obturator as the posterior border. They then used eight volunteers to confirm that the structures could be identified on live patients; they were able to identify the ON in 12 out of 16

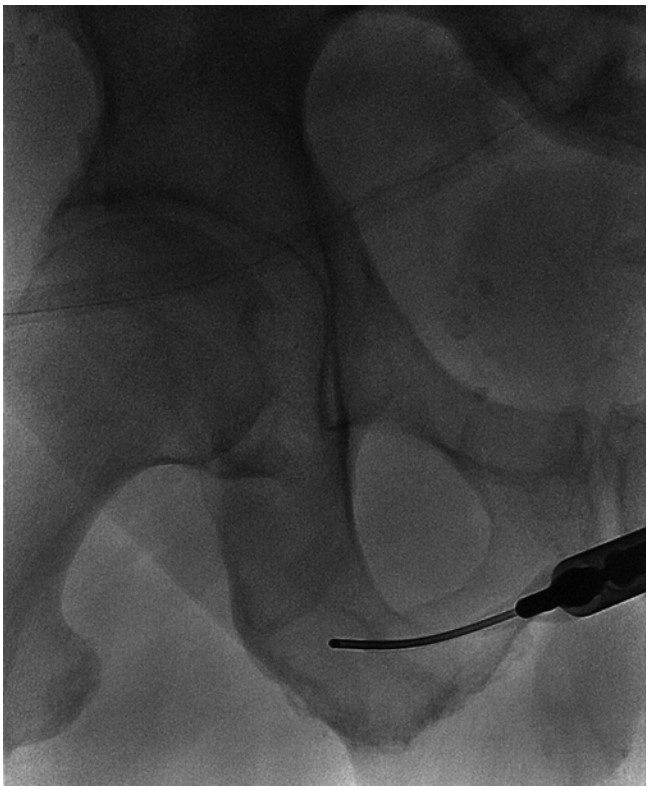

Fig. 48.11 Cryoneuroablation obturator nerve (Image courtesy of Andrea Trescot, MD)

groins. This was followed by ON injections under US guidance in 15 patients, which was deemed successful in all the patients. The area around the ON is very vascular; even with US, 1 out of 15 patients had a puncture of the obturator vein. Soong and colleagues [57] felt that it was easier to see the branches (≥85 %) with US than to see the main nerve (25 %).

Neurolytic Technique

Cryoneuroablation

The following cryoneuroablation technique for the ON was described by Trescot [59]. The patient is placed supine, with the affected limb abducted slightly. Fluoroscopy may be useful in the obese or severely spastic patient. The pubic tubercle is palpated, and local anesthetic is infiltrated subcutaneously, approximately one fingerbreadth laterally and inferiorly to the tubercle. After saline with epinephrine infiltration, the 12-gauge catheter is carefully and gently advanced to the inferior border of the ramus. If done blindly, hitting the edge of the ramus will confirm depth. If done under fluoroscopy, the catheter can be directed to just below the inferior border of the ramus (Fig. 48.11). Kim and Ferrante [60] reported cryoneuroablation of the obturator nerve for the treatment of adductor spasticity and obturator neuropathy. To treat spasticity, this is one of the few times that motor stimulation for localization is appropriate. Adduction of the thigh at low voltages (0.5–1 mV) will confirm position. Spastic muscles should relax quickly, usually during the first freeze cycle. For pain, on the other hand, localization with the sensory mode is more effective, and an effort is made to avoid strong motor simulation if repositioning is possible.

Radiofrequency Lesioning (RF)

Because the sensory branches of the obturator and femoral nerves supply feeling to the hip joint, percutaneous RF of these branches was described in 1997 [61, 62] to denervate the painful degenerated hip in patients who were not candidates for hip arthroplasty. Akatov et al. [61] looked at RF denervation of 15 hips, using a specially designed stiletto-shaped needle, introduced percutaneously, lateral to the pubic bone and below the inguinal ligament, and advanced to the obturator groove. After denervation at 80 °C for 120 s, they followed the patients for up to 3 years, noting pain relief in all but one patient. Rivera et al. [63] prospectively studied 18 patients with DJD of the hip who were not surgical candidates. After a positive diagnostic injection, each patient underwent RF of the ON and femoral sensory branches to the hip several days later. The ON was identified at the junction of the inferior ischium and the pubis (see section "Fluoroscopy-Guided Technique" for details of placement). Sensory stimulation resulted in groin and thigh pain; after negative motor stimulation, the site was lesioned at 90 °C for 90 s. The femoral sensory branch was also lesioned (Fig. 48.12). Eight patients had >50 % pain relief at a 6-month follow-up. Locher et al. [64] dissected the articular branches of the obturator nerve in 20 cadavers and compared the fluoroscopic and MRI images. They concluded that multiple lesions were necessary. Stone and Matchett [41] reported on the use of a combined ultrasound and fluoroscopic technique to denervate the hip of a patient with metastatic lesions to the hip. Because of concerns regarding conventional RF and neuroma formation, Wu and Groner [65] proposed using pulsed RF; they treated two patients, both of whom noted >50 % pain relief at 3 months.

Phenol

The above techniques of neurolysis (cryoneuroablation and radiofrequency lesioning) require special equipment, which is not available in all parts of the world. Phenol has been described for many years to treat nerves causing pain and spasticity; Akkaya and colleagues [66] retrospectively reviewed 80 phenol ON injections performed for spasticity from spinal cord injuries, cerebral palsy, and traumatic brain injury, using a

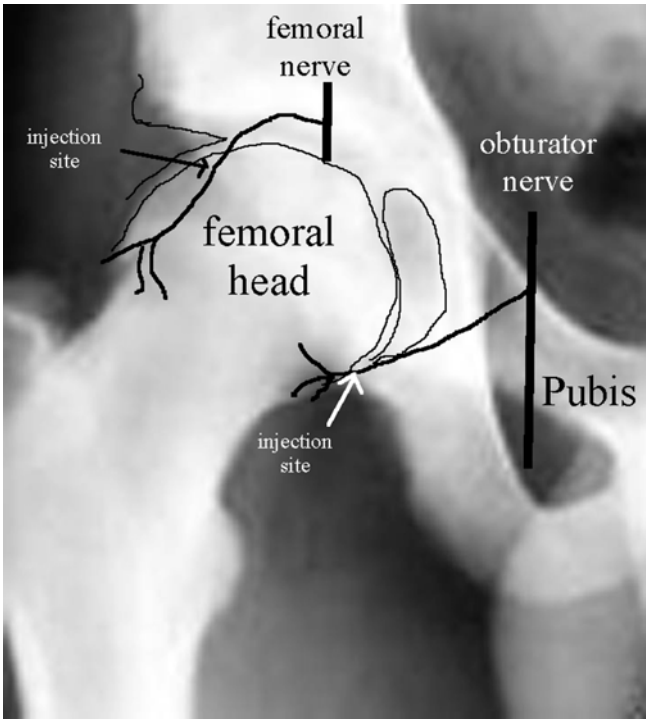

Fig. 48.12 Radiofrequency sites for denervation of the hip joint (Image courtesy of Andrea Trescot, MD)

peripheral nerve stimulator, fluoroscopy, and 6 % phenol. These patients were followed for 3 months and responded with a dramatic increase in range of motion that started to decrease by the third month; they describe "100 %" success rate, and there were no complications, though they did note that the literature describes dysesthesias in about 15 % of adults and 5 % of children undergoing neurolysis with phenol [67].

Surgical Technique

Bradshaw and McCroy [3] reported 32 cases of "obturator neuropathy" treated by surgical release using an inguinal incision; several of their patients also underwent inguinal hernia repairs at the same time. They found a fascial entrapment of the ON overlying the short adductor muscle. Rigaud et al. [68] developed a laparoscopic surgical treatment of the ON entrapment. The probability of recovery of nerve function appears to be inversely related to the length of time of the symptoms, so Tipton [69] recommended only a limited trial of conservative therapy before surgical intervention.

Complications

Zwolak et al. [22] described an obturator and femoral palsy after cement extrusion from a hip arthroplasty. Mittal and Bhandarkar [70] described temporary ON palsy after intra-abdominal local anesthetic spray during a laparoscopic hernia repair; the patient complained of difficulty getting out of bed postoperatively, with weakness of the hip adductors. Because of the vascular structures in the area, there is a significant risk of life-threatening arterial puncture, [71] as well as venous puncture [33]. Rivera et al. [63] noted three transient hematomas after hip RF.

Summary

Obturator injections have great utility in diagnosing and treating a wide variety of pelvic, hip, and groin pains. Better recognition of the pattern of pain and the physical exam should increase the awareness of this often occult pathology.

References

1. Bischoff C, Schonle PW. Obturator nerve injuries during intra-abdominal surgery. Clin Neurol Neurosurg. 1991;93(1):73–6.
2. Fishman JR, Moran ME, Carey RW. Obturator neuropathy after laparoscopic pelvic lymphadenectomy. Urology. 1993;42(2):198–200.
3. Bradshaw C, McCrory P. Obturator nerve entrapment. Clin J Sport Med. 1997;7(3):217–9.
4. Busis NA. Femoral and obturator neuropathies. Neurol Clin. 1999;17(3):633–53.
5. Redwine DB, Sharpe DR. Endometriosis of the obturator nerve. A case report. J Reprod Med. 1990;35(4):434–5.
6. Kopell HP, Thompson WA. Peripheral entrapment neuropathies of the lower extremity. N Engl J Med. 1960;262:56–60.
7. Leno C, Combarros O, Polo JM, Rebollo M, Berciano J. Bilateral obturator neuralgia secondary to osteitis of the pubis. Arch Neurobiol (Madr). 1984;47(6):347–52.
8. Sunderland S. Nerves and nerve injuries. 2nd ed. London: Churchill Livingstone; 1978.
9. Crews DA, Dohlman LE. Obturator neuropathy after multiple genitourinary procedures. Urology. 1987;29(5):504–5.
10. Pellegrino MJ, Johnson EW. Bilateral obturator nerve injuries during urologic surgery. Arch Phys Med Rehabil. 1988;69(1):46–7.
11. Hong BY, Ko YJ, Kim HW, Lim SH, Cho YR, Lee JI. Intrapartum obturator neuropathy diagnosed after cesarean delivery. Arch Gynecol Obstet. 2010;282(3):349–50.
12. Haas DM, Meadows RS, Cottrell R, Stone WJ. Postpartum obturator neurapraxia. A case report. J Reprod Med. 2003;48(6):469–70.
13. Petchprapa CN, Rosenberg ZS, Sconfienza LM, Cavalcanti CF, Vieira RL, Zember JS. MR imaging of entrapment neuropathies of the lower extremity. Part 1. The pelvis and hip. Radiographics. 2010;30(4):983–1000.
14. Litwiller JP, Wells Jr RE, Halliwill JR, Carmichael SW, Warner MA. Effect of lithotomy positions on strain of the obturator and lateral femoral cutaneous nerves. Clin Anat. 2004;17(1):45–9.
15. Pavlov H, Nelson TL, Warren RF, Torg JS, Burstein AH. Stress fractures of the pubic ramus. A report of twelve cases. J Bone Joint Surg Am. 1982;64(7):1020–5.
16. Barrick EF. Entrapment of the obturator nerve in association with a fracture of the pelvic ring. A case report. J Bone Joint Surg Am. 1998;80(2):258–61.
17. Ekberg O, Persson NH, Abrahamsson PA, Westlin NE, Lilja B. Longstanding groin pain in athletes. A multidisciplinary approach. Sports Med. 1988;6(1):56–61.

18. Melamed NB, Satya-Murti S. Obturator neuropathy after total hip replacement. Ann Neurol. 1983;13(5):578–9.
19. Schmalzried TP, Noordin S, Amstutz HC. Update on nerve palsy associated with total hip replacement. Clin Orthop Relat Res. 1997;344:188–206.
20. Siliski JM, Scott RD. Obturator-nerve palsy resulting from intrapelvic extrusion of cement during total hip replacement. Report of four cases. J Bone Joint Surg Am. 1985;67(8):1225–8.
21. Weber ER, Daube JR, Coventry MB. Peripheral neuropathies associated with total hip arthroplasty. J Bone Joint Surg Am. 1976;58(1):66–9.
22. Zwolak P, Eysel P, William-Patrick MJ. Femoral and obturator nerves palsy caused by pelvic cement extrusion after hip arthroplasty. Orthop Rev (Pavia). 2011;3(1):e6.
23. Kent KC, Moscucci M, Mansour KA, DiMattia S, Gallagher S, Kuntz R, et al. Retroperitoneal hematoma after cardiac catheterization: prevalence, risk factors, and optimal management. J Vasc Surg. 1994;20(6):905–10; discussion 910–3.
24. Yukata K, Arai K, Yoshizumi Y, Tamano K, Imada K, Nakaima N. Obturator neuropathy caused by an acetabular labral cyst: MRI findings. AJR Am J Roentgenol. 2005;184(3 Suppl):S112–4.
25. Schwabegger AH, Shafighi M, Gurunluoglu R. An unusual case of thigh adductor weakness: obturator nerve ganglion. J Neurol Neurosurg Psychiatry. 2004;75(5):775.
26. Hibner M, Desai N, Robertson LJ, Nour M. Pudendal neuralgia. J Minim Invasive Gynecol. 2010;17(2):148–53.
27. Sorenson EJ, Chen JJ, Daube JR. Obturator neuropathy: causes and outcome. Muscle Nerve. 2002;25(4):605–7.
28. Stewart JD. Obturator nerve. Focal peripheral neuropathies. 3rd ed. Philadelphia: Lippincott, Williams & Wilkins; 2000. p. 483–8.
29. Saurenmann P, Brand S. Obturator neuralgia (Howship-Romberg phenomenon). Schweiz Med Wochenschr. 1984;114(42):1462–4.
30. Brukner P, Khan KM. Groin pain. In: Bahr R, Blair S, Cook J, Crossley K, McConnell J, McCrory P, et al., editors. Brukner and Khan's clinical sports medicine. 4th ed. Sydney: McGraw-Hill; 2012. p. 545–78.
31. Anagnostopoulou S, Kostopanagiotou G, Paraskeuopoulos T, Chantzi C, Lolis E, Saranteas T. Anatomic variations of the obturator nerve in the inguinal region: implications in conventional and ultrasound regional anesthesia techniques. Reg Anesth Pain Med. 2009;34(1):33–9.
32. Katritsis E, Anagnostopoulou S, Papadopoulos N. Anatomical observations on the accessory obturator nerve (based on 1000 specimens). Anat Anz. 1980;148(5):440–5.
33. Akkaya T, Ozturk E, Comert A, Ates Y, Gumus H, Ozturk H, et al. Ultrasound-guided obturator nerve block: a sonoanatomic study of a new methodologic approach. Anesth Analg. 2009;108(3):1037–41.
34. Haninec P, Horak L, Kaiser R. Obturator nerve injury in laparoscopic inguinal hernia mesh repair. Hernia. 2012:on line.
35. Kumka M. Critical sites of entrapment of the posterior division of the obturator nerve: anatomical considerations. J Can Chiropr Assoc. 2010;54(1):33–42.
36. Tammareddi K, Morelli V, Reyes Jr M. The athlete's hip and groin. Prim Care. 2013;40(2):313–33.
37. Morelli V, Weaver V. Groin injuries and groin pain in athletes: part 1. Prim Care. 2005;32(1):163–83.
38. Saranteas T, Paraskeuopoulos T, Alevizou A, Kouskouri A, Zogojiannis J, Anagnostopoulou S, et al. Identification of the obturator nerve divisions and subdivisions in the inguinal region: a study with ultrasound. Acta Anaesthesiol Scand. 2007;51(10):1404–6.
39. Yamauchi M, Sato Y. Ultrasound-guided obturator nerve block. Masui. 2008;57(5):588–95.
40. Harvey G, Bell S. Obturator neuropathy. An anatomic perspective. Clin Orthop Relat Res. 1999;363:203–11.
41. Stone J, Matchett G. Combined ultrasound and fluoroscopic guidance for radiofrequency ablation of the obturator nerve for intractable cancer-associated hip pain. Pain Physician. 2014; 17(1):E83–7.
42. Dawson DM, Hallett M, Wilbourn AJ. Miscellaneous uncommon syndromes of the lower extremity. In: Dawson DM, Hallett M, Wilbourn AJ, editors. Entrapment neuropathies. 3rd ed. Philadelphia: Lippincott-Raven; 1999. p. 369–79.
43. Lang SA, Yip RW, Chang PC, Gerard MA. The femoral 3-in-1 block revisited. J Clin Anesth. 1993;5(4):292–6.
44. Helayel PE, da Conceicao DB, Pavei P, Knaesel JA, de Oliveira Filho GR. Ultrasound-guided obturator nerve block: a preliminary report of a case series. Reg Anesth Pain Med. 2007;32(3): 221–6.
45. Choquet O, Capdevila X, Bennourine K, Feugeas JL, Bringuier-Branchereau S, Manelli JC. A new inguinal approach for the obturator nerve block: anatomical and randomized clinical studies. Anesthesiology. 2005;103(6):1238–45.
46. Malik A, Simopolous T, Elkersh M, Aner M, Bajwa ZH. Percutaneous radiofrequency lesioning of sensory branches of the obturator and femoral nerves for the treatment of non-operable hip pain. Pain Physician. 2003;6(4):499–502.
47. Garvey JF, Hazard H. Sports hernia or groin disruption injury? Chronic athletic groin pain: a retrospective study of 100 patients with long-term follow-up. Hernia. 2013:on line.
48. Kozlowski JM, Beal JM. Obturator hernia: an elusive diagnosis. Arch Surg. 1977;112(8):1001–2.
49. Travell J, Simons D. Travell & Simmons' myofascial pain and dysfunction: the trigger point manual. In: Simmons DC, Travell JC, Simmons LS, editors. Travell & Simmons' myofascial pain and dysfunction: the trigger point manual, Lower Extremity, vol. 2. Baltimore: Williams & Wilkins; 1992. p. 289–314.
50. Marhofer P, Nasel C, Sitzwohl C, Kapral S. Magnetic resonance imaging of the distribution of local anesthetic during the three-in-one block. Anesth Analg. 2000;90(1):119–24.
51. Bradshaw C, McCrory P, Bell S, Brukner P. Obturator nerve entrapment. A cause of groin pain in athletes. Am J Sports Med. 1997;25(3):402–8.
52. Kassolik K, Kurpas D, Wilk I, Uchmanowicz I, Hyzy J, Andrzejewski W. The effectiveness of massage in therapy for obturator nerve dysfunction as complication of hip joint alloplasty-case report. Rehabil Nurs. 2014;39(6):311–20.
53. Tatlisen A, Sofikerim M. Obturator nerve block and transurethral surgery for bladder cancer. Minerva Urol Nefrol. 2007;59(2):137–41.
54. Bouaziz H, Vial F, Jochum D, Macalou D, Heck M, Meuret P, et al. An evaluation of the cutaneous distribution after obturator nerve block. Anesth Analg. 2002;94(2):445–9.
55. Winnie AP, Ramamurthy S, Durrani Z. The inguinal paravascular technique of lumbar plexus anesthesia: the "3-in-1 block". Anesth Analg. 1973;52:989–96.
56. Feigl GC, Ulz H, Pixner T, Dolcet C, Likar R, Sandner-Kiesling A. Anatomical investigation of a new vertical obturator nerve block technique. Ann Anat. 2012;195(1):82–7.
57. Soong J, Schafhalter-Zoppoth I, Gray AT. Sonographic imaging of the obturator nerve for regional block. Reg Anesth Pain Med. 2007;32(2):146–51.
58. Manassero A, Bossolasco M, Ugues S, Palmisano S, De Bonis U, Coletta G. Ultrasound-guided obturator nerve block: interfascial injection versus a neurostimulation-assisted technique. Reg Anesth Pain Med. 2011;37(1):67–71.
59. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
60. Kim PS, Ferrante FM. Cryoanalgesia: a novel treatment for hip adductor spasticity and obturator neuralgia. Anesthesiology. 1998;89(2):534–6.
61. Akatov OV, Dreval ON. Percutaneous radiofrequency destruction of the obturator nerve for treatment of pain caused by coxarthrosis. Stereotact Funct Neurosurg. 1997;69(1-4 Pt 2):278–80.

62. Kawaguchi M, Hashizume K, Iwata T, Furuya H. Percutaneous radiofrequency lesioning of sensory branches of the obturator and femoral nerves for the treatment of hip joint pain. Reg Anesth Pain Med. 2001;26(6):576–81.
63. Rivera F, Mariconda C, Annaratone G. Percutaneous radiofrequency denervation in patients with contraindications for total hip arthroplasty. Orthopedics. 2012;35(3):e302–5.
64. Locher S, Burmeister H, Bohlen T, Eichenberger U, Stoupis C, Moriggl B, et al. Radiological anatomy of the obturator nerve and its articular branches: basis to develop a method of radiofrequency denervation for hip joint pain. Pain Med. 2008;9(3):291–8.
65. Wu H, Groner J. Pulsed radiofrequency treatment of articular branches of the obturator and femoral nerves for management of hip joint pain. Pain Pract. 2007;7(4):341–4.
66. Akkaya T, Unlu E, Alptekin A, Gumus HI, Umay E, Cakci A. Neurolytic phenol blockade of the obturator nerve for severe adductor spasticity. Acta Anaesthesiol Scand. 2010;54(1):79–85.
67. Tilton AH. Injectable neuromuscular blockade in the treatment of spasticity and movement disorders. J Child Neurol. 2003;18 Suppl 1:S50–66.
68. Rigaud J, Labat JJ, Riant T, Bouchot O, Robert R. Obturator nerve entrapment: diagnosis and laparoscopic treatment: technical case report. Neurosurgery. 2007;61(1):E175; discussion E.
69. Tipton JS. Obturator neuropathy. Curr Rev Musculoskelet Med. 2008;1(3-4):234–7.
70. Mittal G, Bhandarkar D, Katara A, Udwadia TE. Temporary obturator nerve paresis after spray of local anesthetic during laparoscopic extraperitoneal inguinal hernia repair. Surg Laparosc Endosc Percutan Tech. 2010;20(5):357.
71. Akata T, Murakami J, Yoshinaga A. Life-threatening haemorrhage following obturator artery injury during transurethral bladder surgery: a sequel of an unsuccessful obturator nerve block. Acta Anaesthesiol Scand. 1999;43(7):784–8.

Lumbar Plexus Entrapment: Pelvic

49

Susan R. Anderson-Jones, Tiffany Zhang,
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Introduction

The lumbosacral plexus provides the nerve supply to the pelvis, lower back, abdomen, and legs. As it travels from the lower spine to the lateral pelvic walls, there are characteristic areas for compression and entrapment. These areas can present with confusing clinical features. These compressions would seem to manifest clinically with predictable symptoms, specific to the nerve or nerves involved. However, more often, they present as vague and poorly localized symptoms that make it difficult to distinguish from more common causes of pain. The plexopathies can go under-recognized and underdiagnosed. They may also be mistaken for more proximal lesions in the plexus such as compression from a disc within the canal or foramen. In addition, as the plexus traverses anatomically, the generator of the pain may be misdiagnosed as pelvic, low back, sciatic, hip, knee, ankle, or foot pain. Clues may be provided in the history and physical examination, electrodiagnostic, and imaging studies to provide a correct diagnosis. Lumbar and sacral plexus entrapments are described in the abdominal (Chap. 43), pelvic (this chapter), and lower extremity (Chap. 66) plexus chapters, with specific regional emphasis in each respective chapter, although overlap information is inevitable.

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Clinical Presentation (Tables 49.1, 49.2, and 49.3)

The clinical picture of lumbosacral plexopathy varies and depends on the location and degree of involvement. Patients may present with significant and debilitating pain, proximal muscle weakness and wasting, sensory deficits, and loss of deep tendon reflexes. They have difficulty getting up from a chair and climbing stairs. In rare cases, bowel and bladder incontinence can occur and clinically mimic *cauda equina syndrome*.

In addition to nonspecific pelvic (Fig. 49.1), groin (Fig. 49.2), or low back pain, the patients with lumbosacral plexus pathology often present with asymmetric motor

Table 49.1 Occupation/exercise/trauma history relevant to lumbosacral plexus entrapment

Mechanical compression (cysts, aneurysms, tumor, hematoma, etc.)	Fallopian and ovarian cysts [1]
	Intra-abdominal or pelvic aneurysm [2]
	Lymphoma or enlarged lymph node [3]
	Abscess [4]
	Retroperitoneal or psoas hematoma [5, 6]
Tumor compression or invasion	Malignant psoas syndrome
	Adjacent tumor (colorectal, ovarian, uterine, or cervical) [7, 8]
	Metastatic tumor (breast, sarcoma, lymphoma, multiple myeloma) [9]
Trauma (plexus traction)	Pelvic or sacral fracture [10]
Intraoperative compression or traction	Intraoperative patient positioning leading to stretching or compressing the plexus [11]
	Retractors [11]
Infection	Psoas abscess [4]
Endometriosis	Pelvic endometriosis, not common [12]
Pregnancy and/or delivery	Small maternal size, a large fetus, midforceps rotation, and fetal malposition [13]
Pelvic obliquity	Leg length discrepancy [14]

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Table 49.2 Lumbar plexopathy clinical presentations

	Ilioinguinal iliohypogastric neuropathy	GFN neuropathy	Femoral neuropathy	Obturator neuropathy	LFC neuropathy
Motor findings	Possible weakness of abdominal wall	Weakness cremasteric	Weakness of quadriceps and iliopsoas	Weakness of hip adductors	None
Sensory findings	Deficits of lower abdomen and groin	Deficits of lower abdomen and groin	Deficits of anterior and medial thigh and anteromedial leg	Deficits in upper medial thigh	Deficits of anterolateral thigh

Table 49.3 Sacral plexopathy clinical presentations

	Sciatic nerve	Posterior cutaneous nerve of the thigh	Superior and inferior gluteal nerve	Pudendal nerve
Motor findings	Weakness of hamstrings; also weakness dorsal and plantar flexion of ankle and toes	None	Weakness of gluteal muscles (maximus, medius, and minimus)	Weakness of anal and urethral sphincters, erectile dysfunction
Sensory changes	Posterior calf, sole, and dorsum of foot	Lower buttock and posterior thigh	None	Lower anal canal and perineal skin

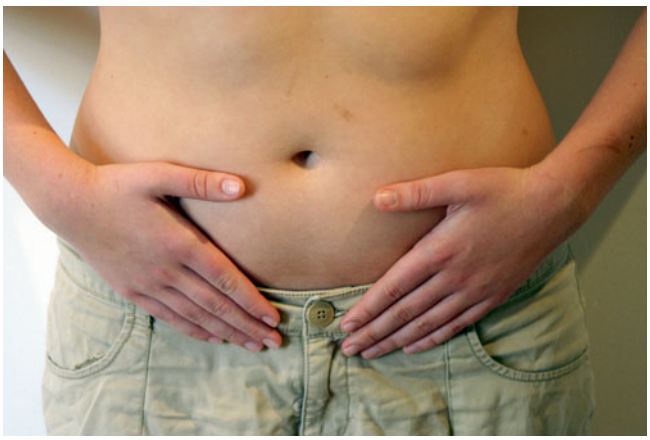

Fig. 49.1 Pattern of nonspecific abdominal pain due to lumbosacral plexus entrapment (Image courtesy of Andrea Trescot, MD)

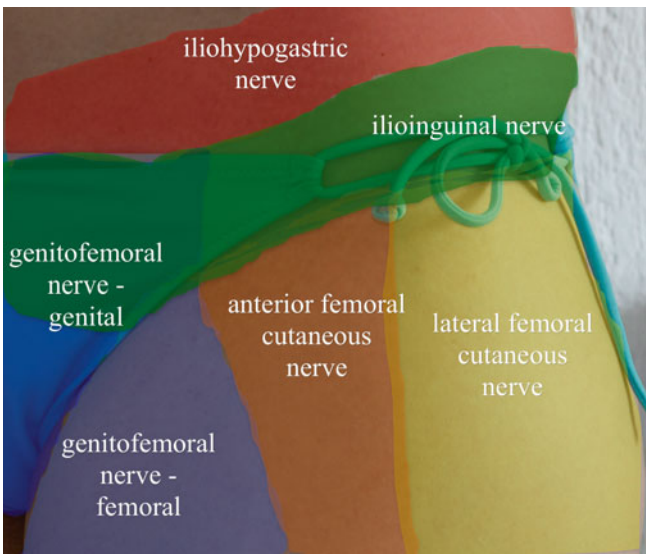

Fig. 49.2 Pain patterns from iliohypogastric, ilioinguinal, genitofemoral, lateral femoral cutaneous, and anterior femoral cutaneous nerves (Image courtesy of Andrea Trescot, MD)

(weakness) and sensory changes (numbness, dysesthesia, and/or paresthesia) and pain involving multiple consecutive nerve levels, which is a key element differentiating from more proximal spinal radiculopathy. Careful examination may help localizing the lesion within the plexus. The neurological findings are often related to the levels and the peripheral nerves involved (Tables 49.2 and 49.3).

Typically, patients with upper nerve root involvement present with symptoms in the femoral and obturator nerves. *Femoral neuropathy* causes weakness of the quadriceps and iliopsoas major muscles, with or without sensory deficits over the anterior and medial thigh and anteromedial leg, whereas *obturator neuropathy* causes weakness in the hip adductors and sensory changes in the upper medial thigh [11, 15]. Sensory symptoms may include nerve irritation (burning or dysesthesia), decreased sensation (paresthesia), or loss of sensation (1) in the area of the anterolateral thigh, a result of lateral femoral cutaneous nerve involvement; (2) in the mons and labia majora, a result of genitofemoral nerve involvement; or (3) in the upper medial thigh or pelvic area or in the inguinal area, due to damage to the ilioinguinal or iliohypogastric nerves [16]. Patients may present with debilitating leg pain that radiates to the lower back and buttocks and progresses posterolaterally down the leg, followed by numbness and weakness. In some patients, the clinical picture may be further complicated by foot drop, sensory changes to the top of the foot, a loss of tendon reflexes, and rarely bowel and bladder incontinence, as well as sexual

dysfunction (Tables 49.2 and 49.3) [10, 17, 18]. The ilioinguinal, iliohypogastric, and genitofemoral nerves also exit through the psoas muscle, causing abdominal, groin, and pelvic pain.

Typically, nerve entrapment presents with unilateral localization of symptoms, indicating a localized entity, whereas bilateral symptoms indicate a systemic process such as diabetes or neurofibromatosis [17].

Onset of entrapment syndrome can be acute (e.g., hematoma, trauma), subacute, or insidious (e.g., tumor). Careful history taking may help define the underlying pathology. Clinical assessment is focused to reveal muscle weakness and sensory disturbances, as well as pain. Most lumbosacral plexopathies have an acute to subacute onset, which is helpful in identifying the process. A slow progressive course may point to a malignant cause, whereas a relapsing course may favor an inflammatory cause. Determine if the process is unilateral or bilateral at onset. Inflammatory plexopathies are unilateral and focal in onset, but become bilateral and widespread with time. At clinical presentation, the disease may be bilateral, but it should not be assumed that symptoms were bilateral at onset, so it is important to clarify the time course with the medical history. Confirm that symptoms are confined to the lower limb; if upper limbs are involved, it makes a structural process less likely and raises concern for a more diffuse, and possibly inflammatory, process affecting cervical, thoracic, as well as lumbosacral segments.

Associated pain and sensation changes are important clues in the lumbosacral plexopathies. If the weakness seems to be confined to the plexus distribution, but the sensory loss seems to follow a more dermatomal distribution, this may encourage imaging and further workup, because of concern for a primary root-level process (i.e., radiculopathy). Weight loss, in addition to constitutional symptoms such as fever and night sweats, may support the diagnosis of neoplastic infiltration or a paraneoplastic cause. A rash associated with the neuropathic symptoms may occur in the context of certain vasculitis. The majority of abscesses are usually secondary to local gastrointestinal, urinary, or spinal infection [7]. Abscesses affecting the psoas, gluteal, and pelvic regions are uncommon but carry a significant morbidity [4]. If symptoms occurred in anticoagulant patients, or shortly after invasive procedures, bleeding or a hematoma should be immediately considered [5].

Lymphoma may cause extrinsic compression of the plexus by enlarged lymph nodes [3, 9]. There may be direct spread of the primary tumor to the plexus or metastatic deposits in the surrounding soft and bony tissues or in the plexus itself. Patients with tumor plexopathy report pain as their predominant symptom [3, 8, 19]. Many different tumors have been reported to produce tumor plexopathy including colorectal, genitourinary, and lung carcinomas, as well as a range of sarcomas [20]. Benign neurofibromas and plexiform lesions are reported to be relatively common in patients with neurofibromatosis type 1, occurring in the abdominopelvic region in up to 40 % of patients. The most common site where the lumbosacral plexus may be involved is the retroperitoneum [21].

Iliopsoas hematoma is a rare complication that occurs in patients receiving anticoagulant therapy. The clinical manifestation of iliopsoas hematoma is nonspecific. It can mimic orthopedic or neurological disorders, including paresthesia or paresis of the thigh and leg due to compression of the nerve plexus. Computed tomography is the most useful radiological method for diagnosis. If the patient is hemodynamically unstable or has active bleeding, transcatheter arterial embolization and surgical intervention may be required [6]. A *retroperitoneal hematoma* can compress the lumbar plexus diffusely within the psoas muscle, leading to weakness in both obturator and femoral nerve distributions. More often, the intrapelvic portion of the femoral nerve is compressed by relatively small hematomas within the indispensible fascia of the iliacus muscle. These syndromes usually occur during anticoagulant therapy, but they can occur with hemophilia or leaking aortic aneurysms or idiopathically [22].

Psoas muscle disease, whether related to surgery or trauma, anticoagulant therapy-related hematoma, abscess, and tumor infiltration (neoplastic pleopathy), can directly compress the lumbar plexus. Painful neoplastic plexopathy due to tumor infiltration of the psoas muscle is known as *malignant psoas syndrome*. It most commonly follows direct invasion of the psoas muscle by adjacent tumors of colorectal, ovarian, uterine, or cervical origin or in the setting of metastatic spread of disease, most commonly from breast cancer, sarcoma, lymphoma, and multiple myeloma. Direct invasion of the lumbosacral plexus from perineural tumors is less common. Malignant psoas syndrome can mimic cauda equina syndrome. It typically involves a proximal lumbosacral plexopathy (L1–L4) and presents as a painful fixed flexion of the ipsilateral hip, with pain exacerbated by attempted extension of the hip ("*positive psoas test*"). Radiological or pathological evidence will demonstrate malignant involvement of the ipsilateral psoas major muscle (Fig. 49.3).

The sacral plexus may also be affected by infection, arthritis, or compression injuries. Infectious or arthritis diseases of the sacroiliac joints, pelvic and hip fractures, and pelvic surgery may directly injure or stretch the sacral plexus. Colorectal and cervical cancers can directly involve or compress the sacral plexus. Pelvic irradiation and aortic aneurysms are less common causes of sacral plexopathy.

Endometriosis of the lumbosacral plexus has been reported in approximately 20 cases in the literature. It presents with catamenial sciatica [12]. The lesion is usually solitary and lies on the sciatic nerve within the pelvis, just as

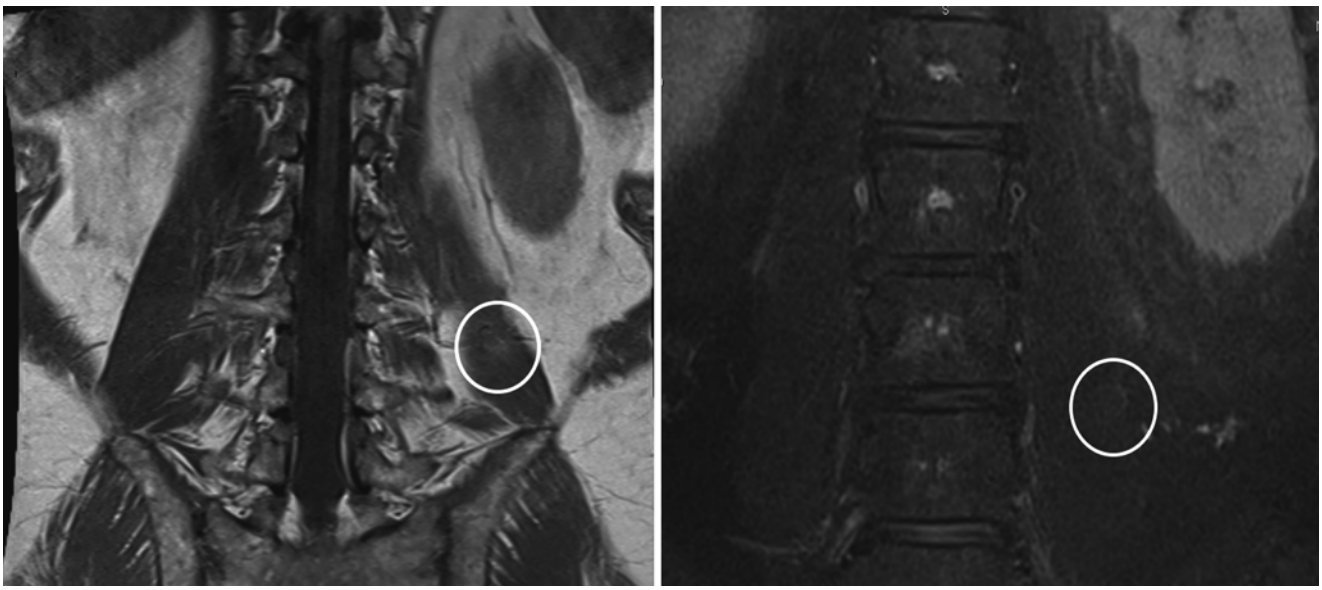

Fig. 49.3 MRI showing psoas lesion; *circle* shows lesion. (**a**) T1 sagittal image; (**b**) sagittal image with contrast (note ring enhancement of the lesion) (Image courtesy of Andrea Trescot, MD)

it passes into the gluteal compartment. The focal mass is classically of high signal intensity on both T2- and T1-weighted images, suggesting acute hemorrhage [23].

Anatomy (Tables 49.4 and 49.5)

The lumbosacral plexus may be divided functionally and anatomically to the lumbar and the sacral plexus (Fig. 49.4). The lumbar plexus is formed from the anterior rami of the T12–L5 nerve roots. The sacral plexus is the union of the lumbosacral trunk and the anterior rami of the S1–S5 nerve roots [24]. The lumbar plexus passes anterior to the transverse processes from L2 to L5 (ventral rami of L1–L4) and lies within the posterior third of the psoas major muscle. It exits at the lateral border of the psoas muscle (Figs. 49.5 and 49.6) and then separates into individual nerves that travel laterally along the abdominal wall or anteriorly into the pelvis and lower extremity (Figs. 49.7 and 49.8). The lumbar plexus consists of six nerves – *iliohypogastric*, *ilioinguinal*, *genitofemoral*, *lateral femoral cutaneous*, *femoral*, and *obturator*.

The *iliohypogastric and ilioinguinal* nerves (see Chap. 40) are primarily sensory nerves that arise from L1 and supply innervation to the skin of the suprapubic and inguinal regions. They are terminal branches that emerge from the lateral border of the psoas muscle. The *genitofemoral* nerve (see Chap. 41) arises from L1 to L2 to supply motor innervation to the cremaster muscle and additional sensory innervation to the inguinal area. The *lateral femoral cutaneous* nerve (see Chap. 61) is formed from the L2 and L3 nerve roots and is a sensory nerve. It provides sensation to the lateral aspect of the thigh. The *femoral* nerve (see Chap. 57) from L2 to L4 is the major motor nerve of the thigh. It emerges from the lateral psoas border before coursing along the groove between the iliacus and psoas muscles,

Table 49.4 Lumbar plexus anatomy

Origin	Anterior rami T12 to L5
General route	Passes anterior to transverse process from L2 to L5 within the psoas muscle. Exits at lateral psoas muscle. 6 nerves:
	Iliohypogastric (Chap. 40), ilioinguinal (Chap. 44), genitofemoral (Chap. 45), lateral femoral cutaneous (Chap. 60), femoral (Chap. 56), and obturator (Chap. 63)
Sensory distribution	*Iliohypogastric/ilioinguinal nerves* (from L1) – suprapubic and inguinal regions
	Genitofemoral nerve (L1 and L2) – inguinal region
	Lateral femoral cutaneous nerve (L2 and L3) – lateral thigh region
	Femoral nerve (L2–L4) – anterior and medial thigh, medial leg distal to the knee
	Obturator nerve (L2–L4) – medial leg proximal to the knee
Motor innervation	*Genitofemoral nerve* (L1 and L2) – cremaster muscle
	Femoral nerve (L2–L4) – rectus femoris, vastus medialis, vastus intermedius, and vastus lateralis muscles of the thigh
	Obturator nerve (L2–L4) – adductor thigh muscles

Table 49.5 Sacral plexus anatomy

Origin	Anterior rami of L4 to L5, S1 to S4
General route	Located on the posterolateral wall of the lesser pelvis, where it is closely related to the anterior surface of the piriformis. Most branches leave the pelvis through the greater sciatic foramen
	Two main nerves: sciatic (see Chap. 54) and pudendal (see Chap. 47) nerves, plus posterior cutaneous nerve of the thigh (see Chap. 55) and superior gluteal (see Chap. 52) and inferior gluteal (see Chap. 53) nerves
Sensory distribution	Sciatic nerve (L4–S3) – posterior calf, sole, and dorsum of foot
	Posterior cutaneous nerve of the thigh (S1–S3) – posterior thigh and lower posterior buttock
	Pudendal nerve (S2–S4) – lower anal canal and perineal skin
Motor innervation	Sciatic nerve – hamstrings; dorsi and plantar flexors of ankle and toes; intrinsic foot muscles
	Superior and inferior gluteal nerves (L5–S2) – gluteus maximus, medius, and minimus; tensor fasciae latae muscles
	Pudendal nerve – sphincters (external anal and urethral); erectile vessels
Anatomic variability	The sacral plexus may arise higher or lower

lateral to the external iliac artery, before reaching the thigh. The femoral nerve provides extension at the knee through innervation of the *rectus femoris*, *vastus medialis*, *vastus intermedius*, and *vastus lateralis* muscles of the thigh. It also provides cutaneous sensory innervation to much of the anterior and medial thigh, as well as the medial portion of the leg distal to the knee. The *obturator* nerve (see Chap. 64) (L2–L4), which emerges from the medial border of the psoas, provides sensory innervation to a portion of the medial leg proximal to the knee, as well as motor innervation to the adductor muscles of the thigh.

The lumbosacral trunk joins with the upper sacral roots anterior to the piriformis behind the internal iliac vessels. It passes out of the *greater sciatic foramen* deep to the *piriformis muscle* and continues as the *sciatic* nerve (see Chap. 55). The *inferior gluteal* nerve (see Chap. 54) also leaves the greater sciatic foramen below the piriformis before entering the gluteus maximus muscle, while the *superior gluteal* nerve (see Chap. 53) emerges above the piriformis. Finally, the *pudendal* nerve (see Chap. 47) passes out of the greater sciatic foramen close to the ischial spine before reentering the pelvis through the *lesser sciatic foramen*. It continues on the lower aspect of the *obturator internus* and in the lateral wall of the ischio-anal fossa before terminating in the perineum [25].

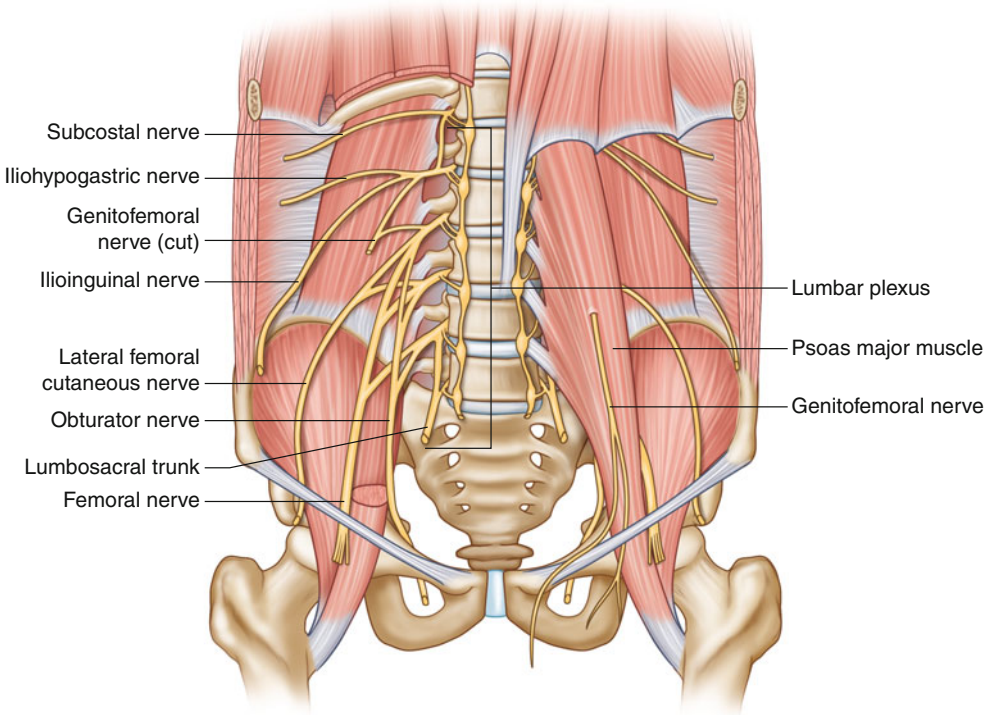

Fig. 49.4 Anatomy of the lumbosacral plexus (Image by Springer)

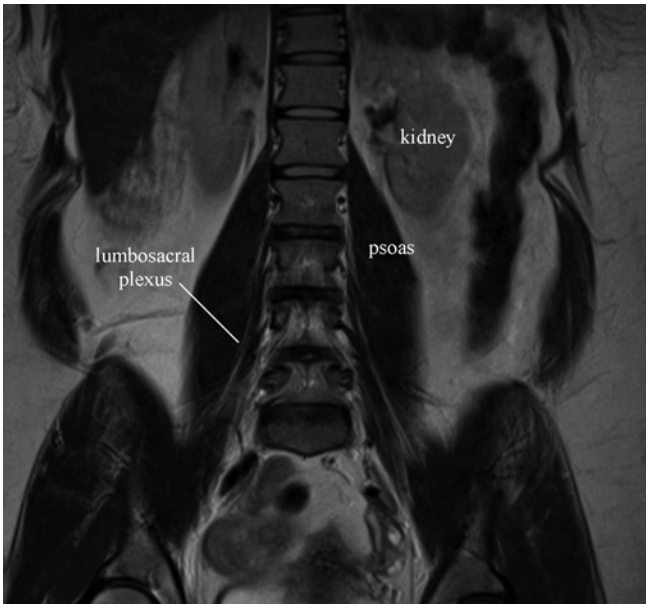

Fig. 49.5 Coronal MRI image of the lumbosacral plexus (Image courtesy of Andrea Trescot, MD)

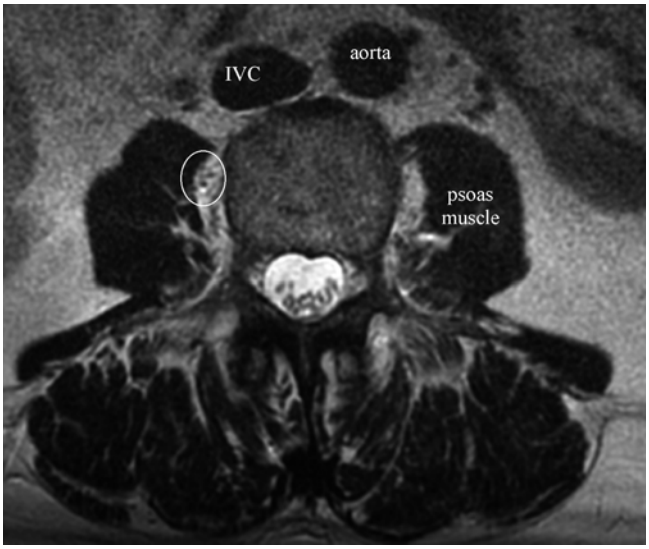

Fig. 49.6 Axial MRI image of the lumbosacral plexus (Image courtesy of Andrea Trescot, MD)

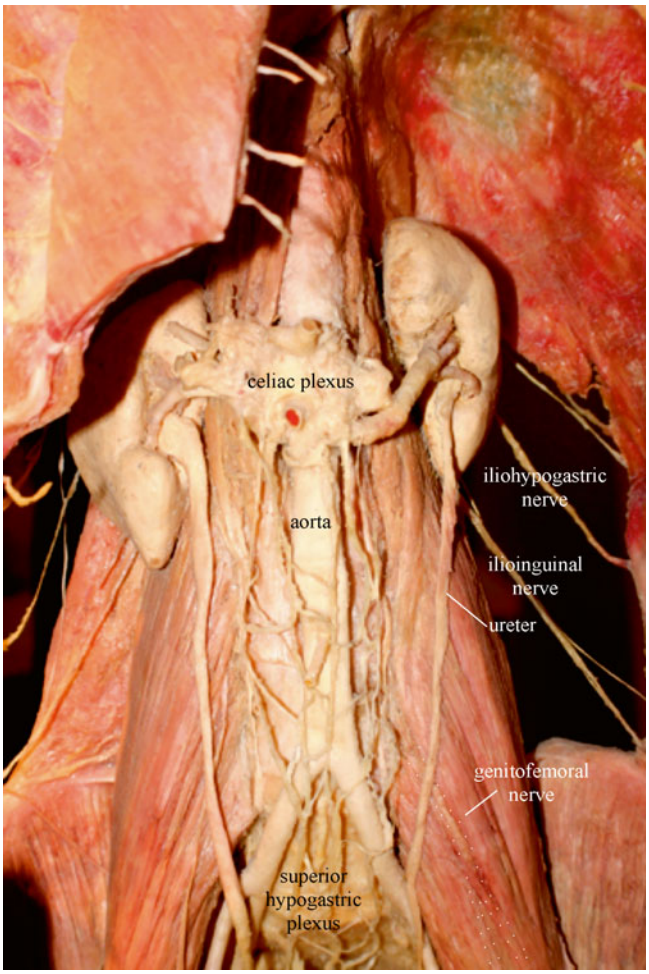

Fig. 49.7 Dissection of the lumbosacral plexus, from *Bodies*, *The Exhibition*, with permission (Image courtesy of Andrea Trescot, MD)

Entrapment

Lumbosacral plexus entrapment may occur in various intra-abdominal or pelvic conditions that produce mechanical mass effect or may be related to trauma, surgery, or even systemic disease (Table 49.1). If pelvic trauma causes double vertical fracture-dislocations of the bony pelvic ring, lumbosacral plexus traction injury may occur, most often on the same side as the sacroiliac joint injury. The lumbosacral plexus may be directly compressed at the pelvic brim, causing postpartum foot drop after protracted, cephalopelvic disproportion or midpelvic forceps delivery [13]. The intrapelvic femoral nerve may be damaged during surgical procedures involving angulation under the inguinal ligament in the lithotomy position or by compression from surgical retractor blades in the gutter between the iliacus and psoas muscles.

Lumbar and sacral plexopathies can be induced by extrinsic compression of a mass or diffuse infiltration by being secondarily involved in the setting of systemic or inflammatory processes. Retroperitoneal processes are more likely to affect the lumbar plexus. Pelvic disease is more likely to affect the sacral plexus.

Physical Exam

As opposed to injuries to peripheral nerves, where symptoms follow stereotypical innervation patterns, injuries that involve the lumbosacral plexus manifest with symptoms and skeletal muscle changes related to multiple spinal levels or multiple nerve distributions. When the symptoms are unilateral, the contralateral plexus can be used as an internal standard for comparison.

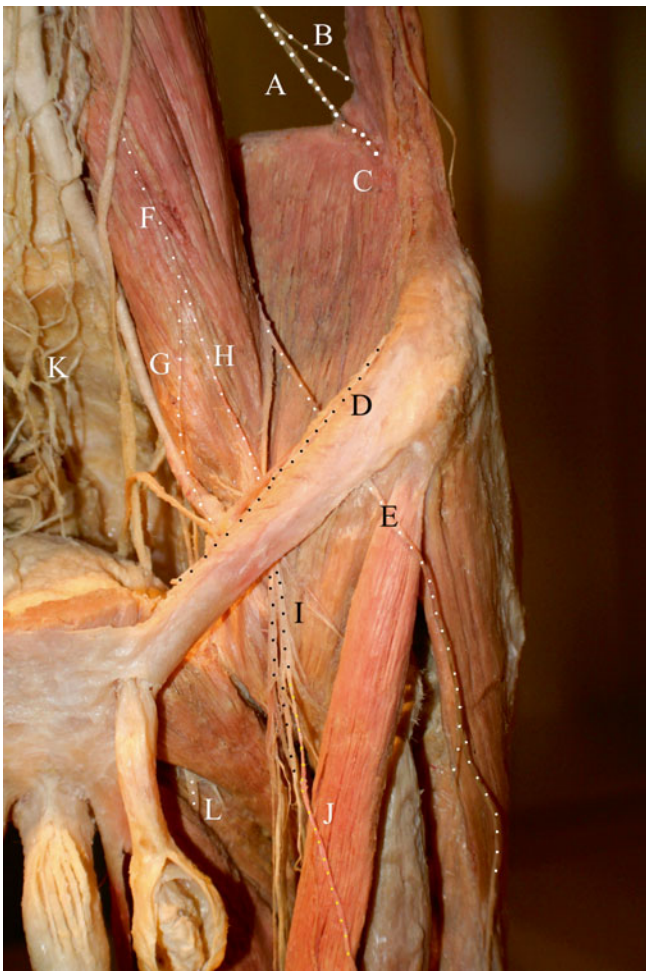

Fig. 49.8 Lumbar plexus nerves, modified from an image from *Bodies The Exhibition*, with permission. *A* ilioinguinal nerve, *B* iliohypogastric nerve, *C* site of ilioinguinal nerve entrapment at the external oblique, *D* ilioinguinal nerve over the inguinal ligament, *E* lateral femoral cutaneous nerve, *F* genitofemoral nerve, *G* genital branch of the genitofemoral nerve, *H* femoral branch of the genitofemoral nerve, *I* femoral nerve, *J* saphenous nerve, *K* inferior hypogastric plexus, *L* obturator nerve (Image courtesy of Andrea Trescot, MD)

The signs and symptoms can be vague and nonspecific; however, lumbar plexus entrapment often presents with asymmetric and focal weakness, as well as sensory changes, and involves multiple lumbosacral nerve levels. A thorough examination of the hip girdle and leg enables classification of the injury pattern. Lumbar plexopathy tends to cause weakness of hip flexion and adduction and/or knee extension, whereas sacral plexopathy often leads to weakness of knee flexion or foot drop. Lumbar plexus entrapment may affect anteromedial thigh and leg sensation, whereas the sacral plexus collectively provides sensation to the posterior thigh and leg, as well as most of the foot.

The *superior gluteal nerve* (see Chap. 53) and the *posterior femoral cutaneous nerve* (see Chap. 46) are two nerves where function can be evaluated to determine level of injury. The superior gluteal nerve emerges from the L5 spinal nerve or the lumbosacral trunk and from the S1 spinal nerve in the proximal part of the lumbosacral plexus; abolishing activity in the gluteal muscles impairing pelvic stability results in a positive *Trendelenburg test* (the inability to hold pelvis level when standing on the opposite leg) (Fig. 49.9). The posterior femoral cutaneous nerve forms at the lateral part of the sacral plexus; its intact function (sensation on the dorsal aspect of the thigh) with weakness of the muscles distal to the knee indicates a proximal sciatic nerve lesion rather than a sacral injury.

Refer to Tables 49.2 and 49.3 for specific patterns of the lumbosacral plexus and its branches. Because contraction of the psoas muscle will increase compression of the plexus, resistance against hip flexion (Fig. 49.10) may provoke or exacerbate the symptoms. Distal tendonitis of the psoas at its attachment onto the lesser trochanter (Fig. 49.11) may also worsen with this maneuver.

Differential Diagnosis (Table 49.6)

Because the causes and presentation of lumbosacral plexus entrapment can be vast and vague, the differentials are extensive. Table 49.6 lists some of common conditions that should be considered or ruled out during workup.

It should be recognized that the differential diagnosis includes many conditions invisible on imaging [27]. Patients receiving pelvic irradiation for cancer may develop insidious leg weakness [26], often bilateral, 5 or more years later; imaging is crucial to the differential diagnosis from cancer recurrence. As a distinguishing feature from tumor infiltration, pain from radiation damage tends not to be the distinguishing feature of presentation. The diagnostic tests for lumbar plexus entrapment can be found in Table 49.7.

Electrodiagnostic Testing

For cases of lumbosacral plexopathy, electrodiagnostic studies incorporating nerve conduction studies and needle electromyography (EMG) can be helpful for localization and characterization of the underlying process. The presence of lumbosacral plexopathy can be defined when there is evidence for electrophysiologic abnormalities in the distribution of at least two different peripheral nerves in at least two different nerve root distributions. Sparing of the paraspinal muscles on needle examination is also helpful in localizing a pure lumbosacral plexopathy. In cases of lumbosacral plexopathy, the sensory studies are likely to be most helpful for localization. Recall that in most spinal segments, the dorsal root ganglion lies lateral to and outside the intervertebral foramen. In electrodiagnostics, this feature

Fig. 49.9 Trendelenburg sign (Image courtesy of Andrea Trescot, MD)

is important because it helps assist in localization of a preganglionic process (i.e., radiculopathy) versus a postganglionic process (i.e., plexopathy or mononeuropathy). A reduced sensory nerve action potential amplitude implies a postganglionic process and can help to exclude a radiculopathy as the main cause for clinical symptoms [28].

The needle electromyographic examination may be the most important component of the electrodiagnostic evaluation of lumbosacral plexopathies, both for localization and for determination of the severity of disease. The femoral nerve is the only motor nerve conduction study likely to give meaningful information about the possibility of a pure lumbar plexopathy. The most common method is to stimulate the nerve high in the inguinal region and record from a quadriceps muscle. Because of the significant overlying connective tissue, needle stimulation is often necessary but may be contraindicated in some patients on anticoagulation, given the proximity to the femoral artery [29]. A thorough examination and history cannot be overstated in deciding which muscles to test.

Magnetic Resonance Imaging (MRI)

In the evaluation of lumbosacral plexopathy, magnetic resonance imaging (MRI) is a valuable adjunct to clinical examination and electrodiagnostic testing because it provides anatomic information that is not obtainable with

Fig. 49.10 Physical exam of resistance to hip flexion (Image courtesy of Andrea Trescot, MD)

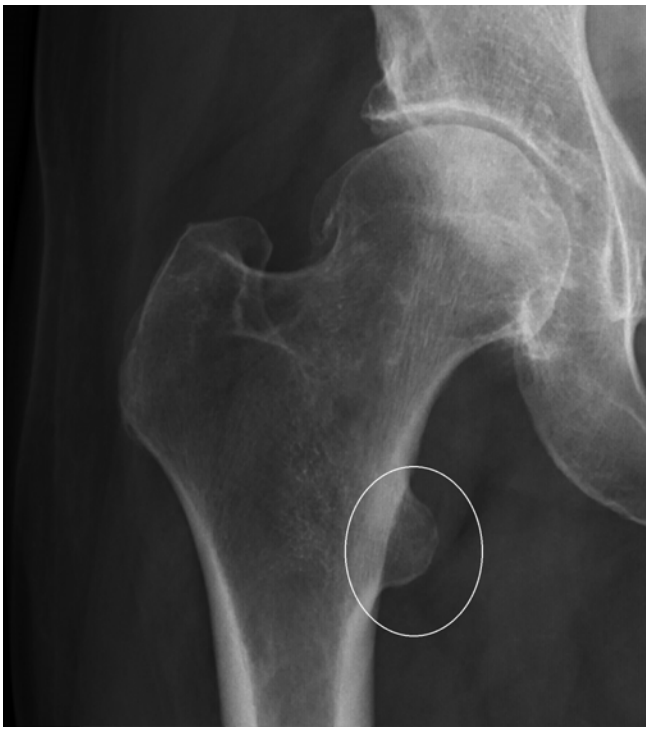

Fig. 49.11 X-ray showing lesser trochanter (*circled*) (Image courtesy of Andrea Trescot, MD)

Table 49.6 Differential diagnosis of pelvic pain

	Potential distinguishing features
Neuropathy after radiation therapy	Insidious bilateral leg weakness; history of radiation therapy [26]
Diabetic peripheral neuropathy	History of diabetes; other symptoms and signs of diabetes and DPNs
Tumor compression or invasion	History of malignancy, especially pelvic or abdominal malignancies; MRI may show tumor invasion [8]
Lumbosacral radiculopathy	Sensory and motor symptoms follow single nerve root; NCV/EMG may be diagnostic
Cauda equina syndrome	Urinary and/or bowel incontinence; saddle anesthesia; loss of rectal tone, often with acute onset
Lumbar facet pathology	Paravertebral tenderness; lumbar spondylosis
Hematoma	Anticoagulant therapy; recent invasive procedure
Systemic inflammatory disease	Widespread and symmetrical symptoms and signs
Abscess	GI, pelvic, or abdominal infection

Table 49.7 Diagnostic tests for lumbar plexus entrapment

	Potential distinguishing features
Physical exam	Sensory and motor changes often involve overlapping contiguous spinal levels (see Tables 49.2 and 49.3)
Diagnostic injection	Psoas plexus block
Ultrasound	Less value in diagnosis due to the deep location of lumbosacral plexus
MRI	Valuable for detailed nerve, psoas muscle, and surrounding lesions; increased T2 intensity of the nerves; may enhance with contrast (infection, tumor invasion, inflammatory)
X-ray	Rule out other bony causes
Electrodiagnostic studies	EMG abnormalities in at least 2 different peripheral nerves in at least 2 nerve roots with no paravertebral abnormalities

other modalities and is useful for assessing lesions. Perifascicular and perineural high signal intensity from fat makes nerves conspicuous on T1-weighted images and provides a model road map. The plexus can be seen as fascicular structures surrounded by fat within or posterior to the muscles (Figs. 49.5 and 49.6). Terminal branches of the lumbar plexus are first detected once they exit the psoas muscle at the lateral border of the muscle (the ilio-hypogastric, ilioinguinal, genitofemoral, lateral femoral cutaneous, and femoral nerves). The most useful imaging plane tends to be the axial. Disruption of the perineurial blood-nerve barrier, with nerve injury, can cause a change in the distribution of the endoneurial fluid and therefore increased perifascicular and endoneurial signal intensity on T2-weighted images, though a nonspecific response. T2-weighted neurography allows evaluation of changes in the perifascicular and endoneurial signal intensity. The disruption of the perineurial blood-nerve barrier may also allow neural enhancement with gadolinium contrast material, if the barrier is compromised. The necessity of this assessment is not yet agreed upon. Other valuable MRI findings of neural injuries include size and morphological changes, such as neuronal enlargement, loss of the normal fascicular appearance, or blurring of the perifascicular fat [15].

Treatment of Contributing Factors

The anatomic restriction of the lumbosacral plexus makes it susceptible to entrapment by variety of conditions. Any occupying lesion intrinsic or external of the psoas muscle may cause lumbar plexus entrapment [27], including retroperitoneal processes [17] and muscle invasion [30]. Its proximity to bones and major blood vessels such as the abdominal aorta and the iliac artery and its branches also make it prone to compression from aneurysms of those arteries or metastatic lesions. The sacral plexus trunk passes over the sacral ala and lies over the posterior lateral wall of the pelvis; it is therefore easily injured from sacral and pelvic trauma or during delivery.

It has been reported that leg-length discrepancies, including improper use of a heel lift, might irritate the psoas muscle and cause muscle spasm, leading to lumbar plexus entrapment. Proper correction of the leg length and hip level, or osteopath manipulation, may prevent recurrent lumbar plexopathy [14].

Injection Techniques

The lumbar plexus block is most frequently used for surgical anesthesia of the lower extremity. It is occasionally used for the treatment of inflammatory conditions of the lumbar plexus such as idiopathic lumbosacral plexitis or when a tumor has invaded the tissues subserved by the lumbar plexus or the plexus itself [30, 31]. It can be utilized as a diagnostic maneuver when performing differential neural blockade for evaluation of groin and lower extremity pain. It may be used palliatively for acute pain emergencies, including groin and lower extremity trauma or fracture, acute herpes zoster, and cancer pain, including tumor invasion while waiting for pharmacologic, surgical, or anticancer therapies to become effective [32].

Landmark-Guided Technique

The patient is placed in the lateral decubitus position, with painful or operative side up and a slight forward tilt. The foot on the side to be blocked is positioned over the dependent leg so that twitches of the quadriceps muscle can be easily seen. Although several needle insertion sites have been suggested, locating the transverse process of the lumbar vertebral body with the needle tip after insertion is common to all techniques. The two surface anatomic landmarks of importance for determining the insertion point are the iliac crest and the midline spinous processes. In most patients, the top of the iliac crest correlates with the body of the L4 vertebral body or the L4–L5 interspace. Draw a line between the iliac crests

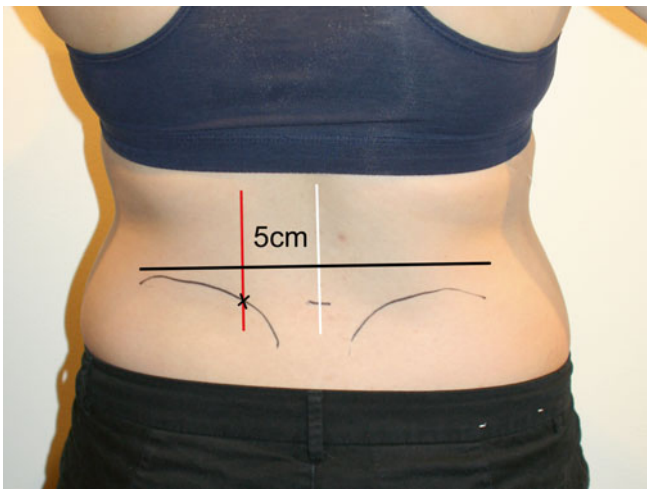

Fig. 49.12 Landmark-guided psoas injection site. *Black line* = top off iliac crests (Tuffier's line); *white line* = midline; *red line* = paravertebral line 5 cm lateral of midline; *PSIS* posterior superior iliac spine, *X* injection site (Image courtesy of Andrea Trescot, MD)

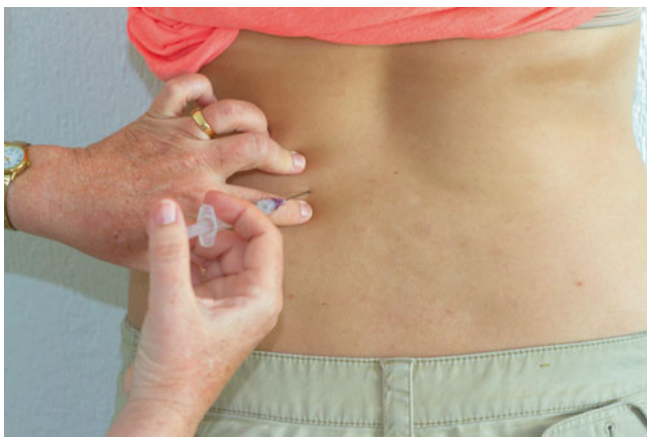

Fig. 49.13 Landmark-guided psoas injection (Image courtesy of Andrea Trescot, MD)

(*Tuffier's line*), followed by a vertical line marking the midline. A second vertical line is made 5 cm lateral to the midline parasagittally on the side to be anesthetized, which should pass through the posterior superior iliac spine (PSIS). On this second line, a mark is made 3 cm caudal to Tuffier's line, which identifies the needle entry point (Fig. 49.12) [33]. A 20- or 22-gauge, 15-cm needle is then inserted to the depth of the transverse process of the fifth lumbar vertebra (Fig. 49.13). Once the transverse process is located, the needle is partially withdrawn and redirected cephalad until it slides past the transverse process. Next, attach a 5-cc saline-filled syringe to the needle and slowly advance it until loss of resistance is achieved; the needle tip is then within the psoas compartment. The loss of resistance typically occurs at a depth of 12 ± 2 cm. This is followed by 30 cc of local anesthetic for a surgical block or 5 cc of local anesthetic for a

diagnostic injection. Keep the patient in the lateral position for 5 min following completion of the local anesthetic injection.

Using a Nerve Stimulator

After the stimulating needle is in place as described above, the nerve stimulator is set to an initial current of 1.5 mA. The needle is advanced at an angle perpendicular to all skin planes. As the needle is advanced, local twitches of the paravertebral muscles are obtained. As the needle is further advanced, the transverse process may be encountered. Contact with the transverse process is not routinely sought, but, when present, it provides a consistent landmark to avoid excessive needle penetration during the lumbar plexus block [34]. The distance from the skin to the lumbar plexus ranges from 6.1 to 10.1 cm in men and 5.7 to 9.3 cm in women [34], with the distance correlating to gender and body mass index (BMI). The distance from the transverse process to the lumbar plexus is usually less than 2 cm, independent of BMI or gender. Contraction of the quadriceps muscle is usually obtained at a depth of 6 to 8 cm. The nerve stimulator current is reduced to produce stimulation of the quadriceps muscle between 0.5 and 1.0 mA, and 25–30 mL of local anesthetic is injected incrementally with negative aspiration every 5 mL [35]. It is important to avoid too deep a needle penetration and the resultant complications that may arise, such as renal hematoma and total spinal anesthesia [36].

Ultrasound-Guided Technique

The patient is placed in the lateral decubitus position so that contractions of the quadriceps muscle are visible. Identify the iliac crests and draw a line, as in the landmark-guided technique (Fig. 49.14). This time, however, the target is the paraspinal area at the level of L3/L4. Ultrasound can be used to confirm the correct vertebral level and to guide the needle tip over the top of the transverse process. A low-frequency (2–5 MHz) curved array probe is used, placed in a paramedian longitudinal position. Firm pressure is required to obtain good quality images. Identify the transverse processes at the L3/L4 space by moving the US probe laterally from the spinous processes in the midline, staying in the longitudinal plane. Going from the midline and moving the probe laterally, the articular processes are seen, with the adjoining superior and inferior articular processes of the facets forming a continuous "sawtooth" hyperechoic line (Fig. 49.15). As the probe is moved further laterally, the transverse processes are seen with the psoas muscle lying between them. The image is of a "trident" (Fig. 49.16), with the transverse processes causing bony shadows and the psoas muscle lying in between.

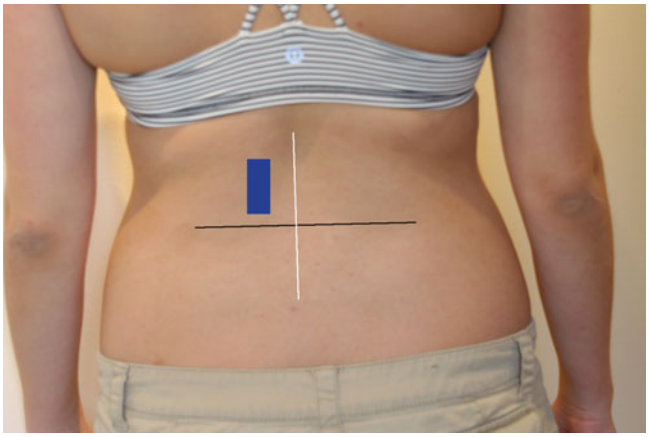

Fig. 49.14 Ultrasound-guided psoas injection site. *Black line* = top off iliac crests (Tuffier's line); *white line* = midline; *blue* = US probe (Image courtesy of Andrea Trescot, MD)

A color Doppler image is then obtained to identify adjacent vasculature, so as to avoid inadvertent intravascular injection.

At this point, the US probe is usually 3–5 cm off the midline. The lumbar plexus is not usually directly visualized, but lies within the posterior third of the psoas muscle (i.e., the closest third of the psoas muscle seen with the US probe). The distance from the skin to the psoas muscle can be measured using the caliper function of the ultrasound machine. This gives an estimate of the depth of the lumbar plexus before needle insertion. Note that anterior to the psoas muscle (further away from the skin in this US view) lie the peritoneal cavity, the great vessels, and the kidney. Thus, care with needle tip placement should be maintained at all times.

The depth of the plexus is most often between 50 and 100 mm from the skin surface. An in-plane or out-of-plane technique may be used. If an in-plane approach is used, the usual direction for insertion is from caudad to cephalad. For the out-of-plane approach, the site for the block needle is on the medial side of the US probe (which is maintained in its longitudinal position). A 13-cm stimulating needle may be utilized for specificity of location. The needle needs to be placed at the center of the probe, directed slightly laterally such that its path comes directly under the US beam. Advancing the needle from a medial to a lateral direction is also preferred to avoid insertion into the dural cuff, which can extend laterally beyond the neural foramina. Lidocaine is infiltrated into the skin and subcutaneous tissue at the point where the block needle is to be inserted. The needle is observed in real time and targeted toward the posterior third of the psoas muscle bulk. Electrical stimulation is commonly used to confirm proximity to the lumbar plexus. The target is to elicit quadriceps muscle contraction. In addition, contraction of the psoas muscle will be readily apparent under ultrasound imaging. When satisfied with needle tip position,

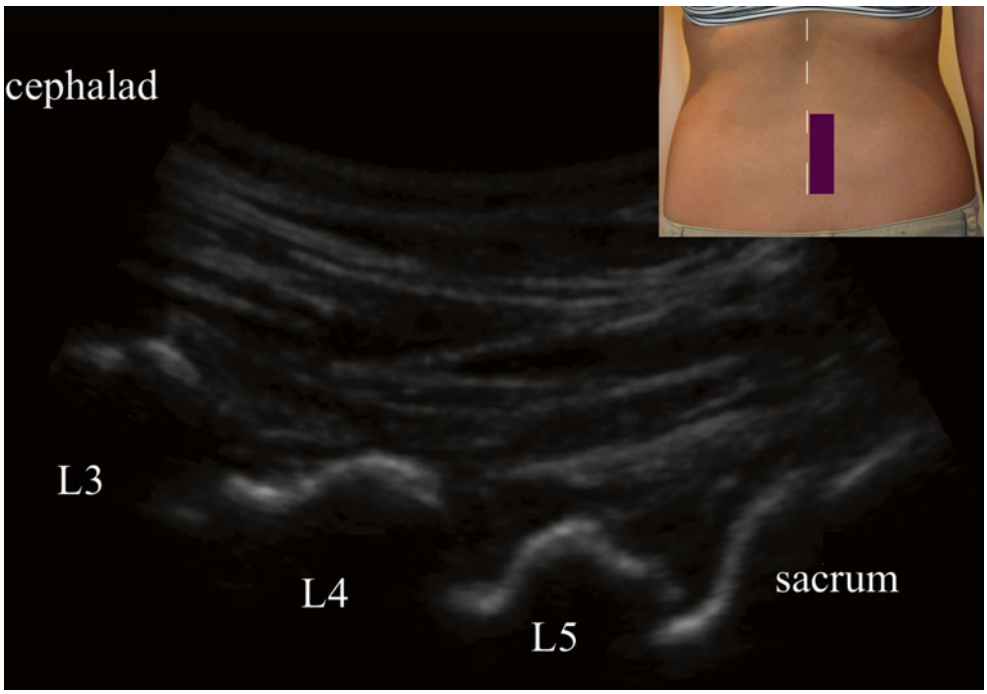

Fig. 49.15 Ultrasound image of the facets from a longitudinal approach, forming a "sawtooth" pattern (Image courtesy of Andrea Trescot, MD)

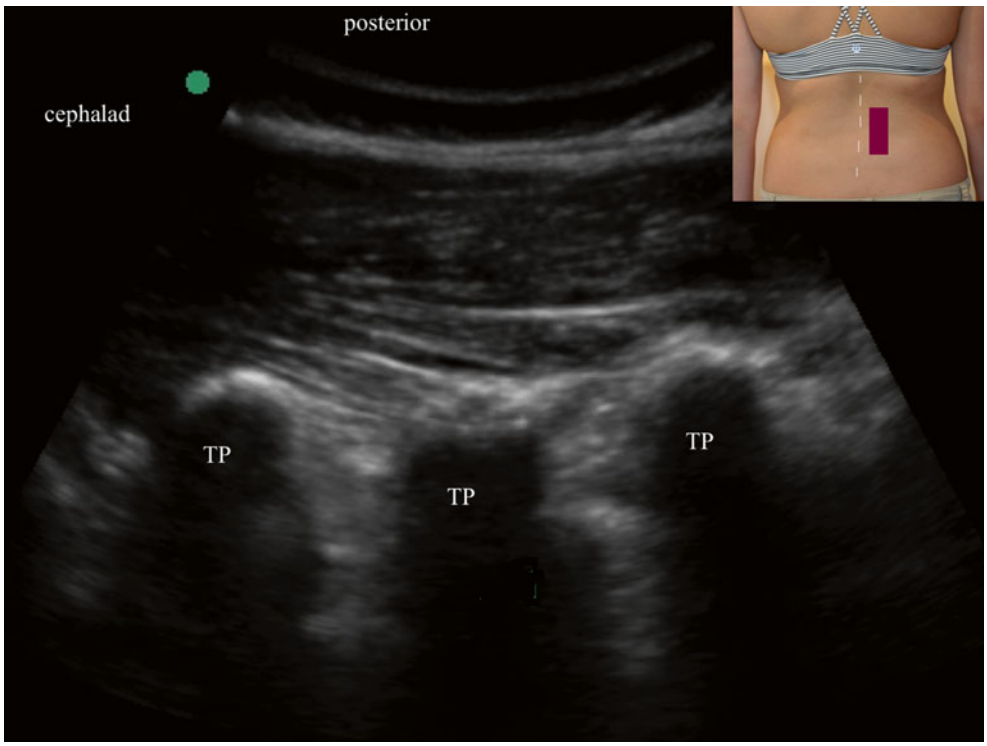

Fig. 49.16 Ultrasound image of the transverse processes from a longitudinal approach, forming a "trident" pattern (Image courtesy of Andrea Trescot, MD)

inject the local anesthetic incrementally (with frequent aspiration to monitor for blood or CSF), and observe its spread, looking for fluid and tissue expansion in the psoas muscle bulk [37]. The needle is removed and pressure is placed on the injection site to avoid hematoma formation [32].

An alternative technique uses a horizontal rather than vertical orientation. Using the same surface landmarks, the curvilinear low-frequency probe is placed parallel to the transverse processes (Fig. 49.17) and moved laterally. When the psoas muscle is visualized (which usually requires an ipsilateral oblique tilt of the transducer to "see under" the transverse process), the stimulating needle can be advanced in-plane to the target area (Fig. 49.18).

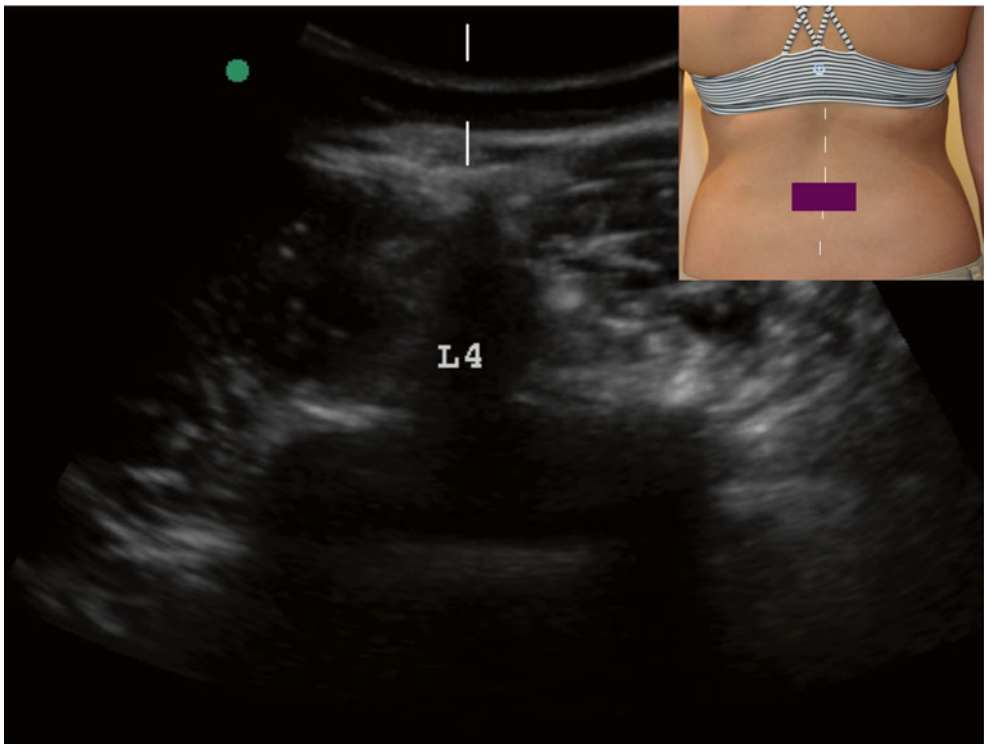

Fig. 49.17 Midline horizontal image of the ultrasound probe over the spinous process at L4 (Image courtesy of Andrea Trescot, MD)

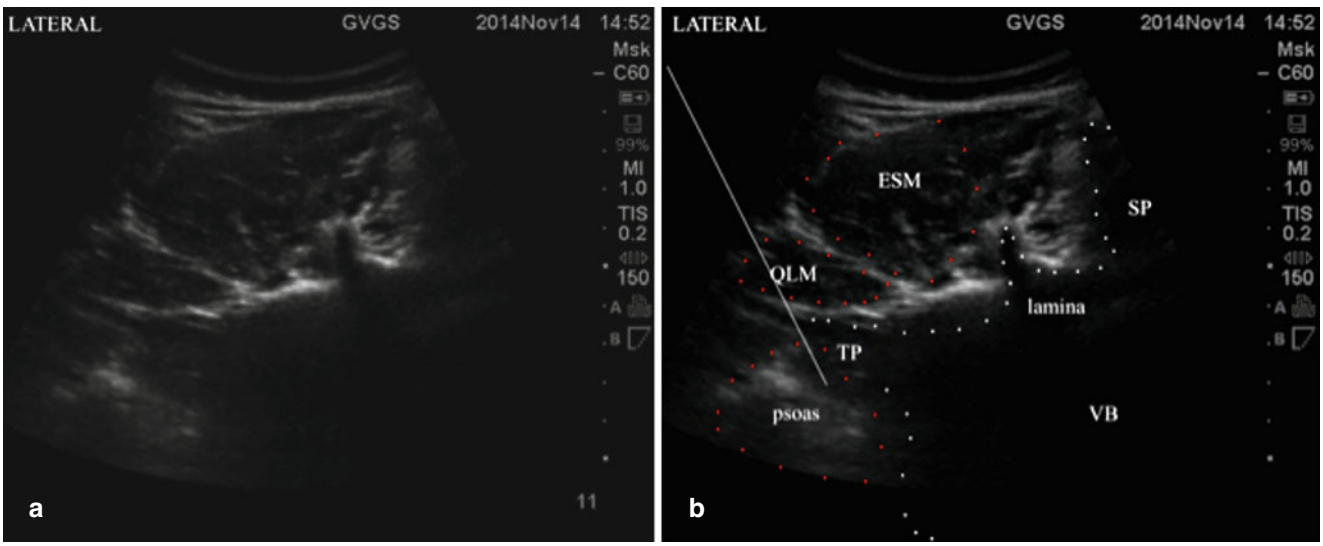

Fig. 49.18 Horizontal lateral view of the lumbar plexus (**a**), with labels and simulated needle (**b**). *SP* spinous process, *VB* vertebral body, *TP* transverse process, *ESM* erector spinous muscle, *QLM* quadratus lumborum muscle (Image courtesy of Agnes Stogicza, MD)

Fluoroscopy-Guided Technique

The patient is positioned in the prone position, and the transverse processes at L3 or L4 are identified. The psoas shadow should be visible (Fig. 49.19), and the injection site should correlate with the lateral or superior aspect of the transverse process to avoid the nerve roots and the epidural space (Fig. 49.20). Insert a 22-gauge, 5-in., B-bevel needle using the "gun-barrel" technique until the needle is approximately at the anterior one third of the vertebral body in the lateral view. Then, inject 1 cc of nonionic contrast, which should show the oblique flow of contrast cephalad and caudad (Fig. 49.21). In the lateral view, the psoas major muscle spreads vertically over the anterior one third of the lumbar vertebral body when the contrast is injected (Fig. 49.22). Note that it is always anterior to the foramina. After the correct needle placement is confirmed, 5–10 mL of a local anesthetic (e.g., 0.25 % bupivacaine or 0.2 % ropivacaine) is

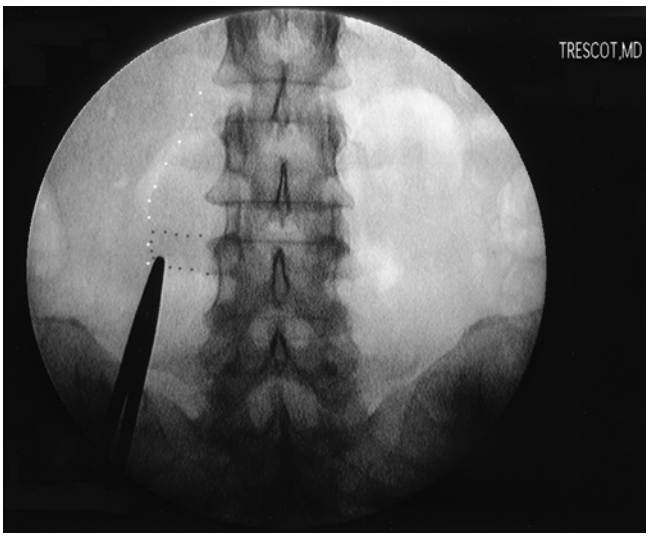

Fig. 49.19 Fluoroscopic image of the lumbar spine. Marker is on the transverse process of L4; note the distortion of the psoas shadow due to cancer mass (*white dotted line*) (Image courtesy of Andrea Trescot, MD)

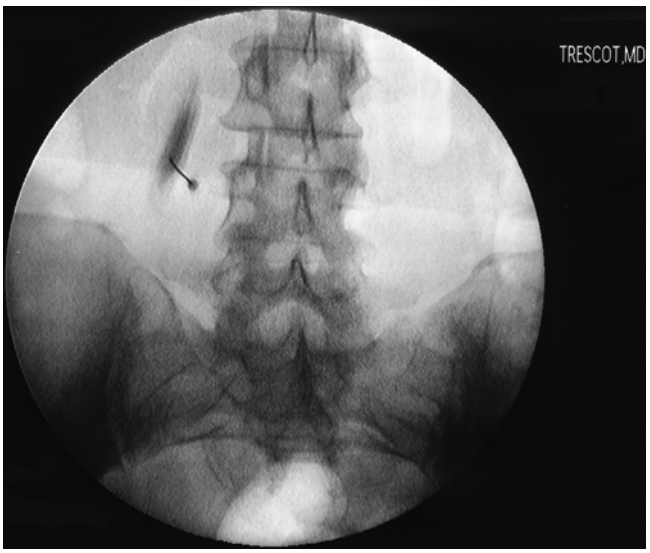

Fig. 49.21 Fluoroscopic psoas injection, contrast pattern (Image courtesy of Andrea Trescot, MD)

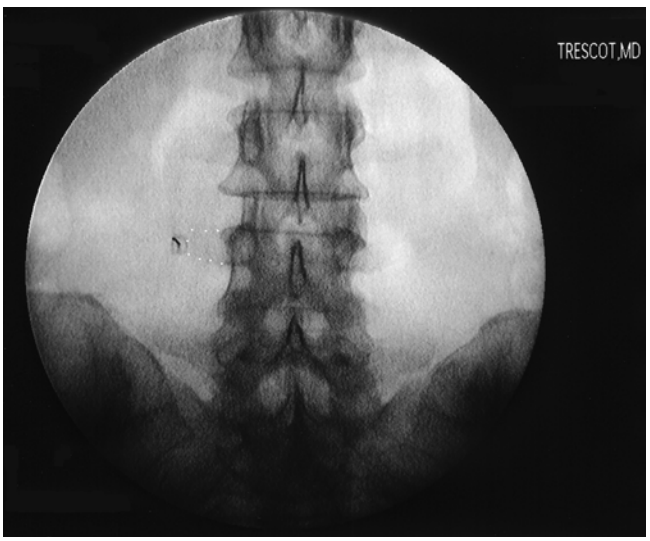

Fig. 49.20 Fluoroscopic psoas injection, needle on the transverse process of L4 (outlined) (Image courtesy of Andrea Trescot, MD)

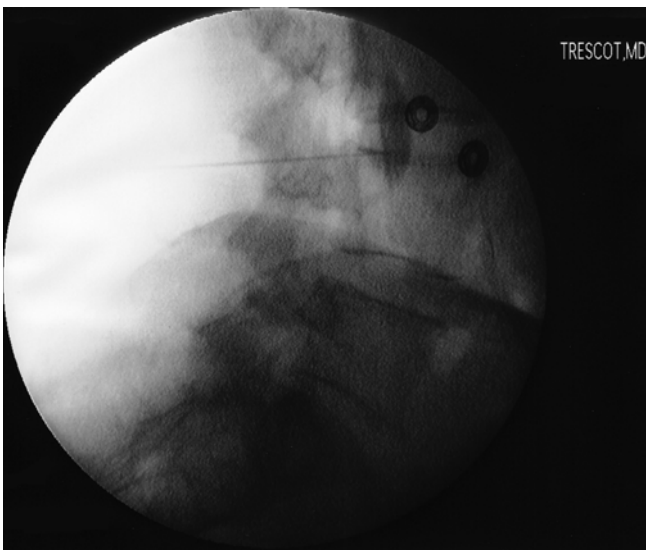

Fig. 49.22 Fluoroscopic psoas injection, lateral view (Image courtesy of Andrea Trescot, MD)

injected into the psoas muscle on one side. Pain relief should occur approximately 30 min after injection of the local anesthetic. On examination, pain should be gone on flexion and extension of the hip [38].

Inguinal Perivascular Injection or Compartment Block (Three-in-One Block)

The *inguinal perivascular block* is based on the concept of injecting local anesthetic near the femoral nerve in an amount sufficient to track proximally along fascial planes to anesthetize the lumbar plexus [39]. The three principal nerves of the lumbar plexus pass from the pelvis anteriorly: the lateral femoral cutaneous, the femoral, and the obturator nerves. The theory behind this block presumes that the local anesthetic will track in the fascial plane between the iliacus and psoas muscles to reach the region of the lumbar plexus roots, so that the only anatomy one needs to visualize is the extension of sheathlike fascial planes that surround the femoral nerve. The patient should be placed supine on the operating table with the anesthesiologist standing at the patient's side in a position to palpate the ipsilateral femoral artery. After local anesthetic infiltration, a short-beveled, 22-gauge

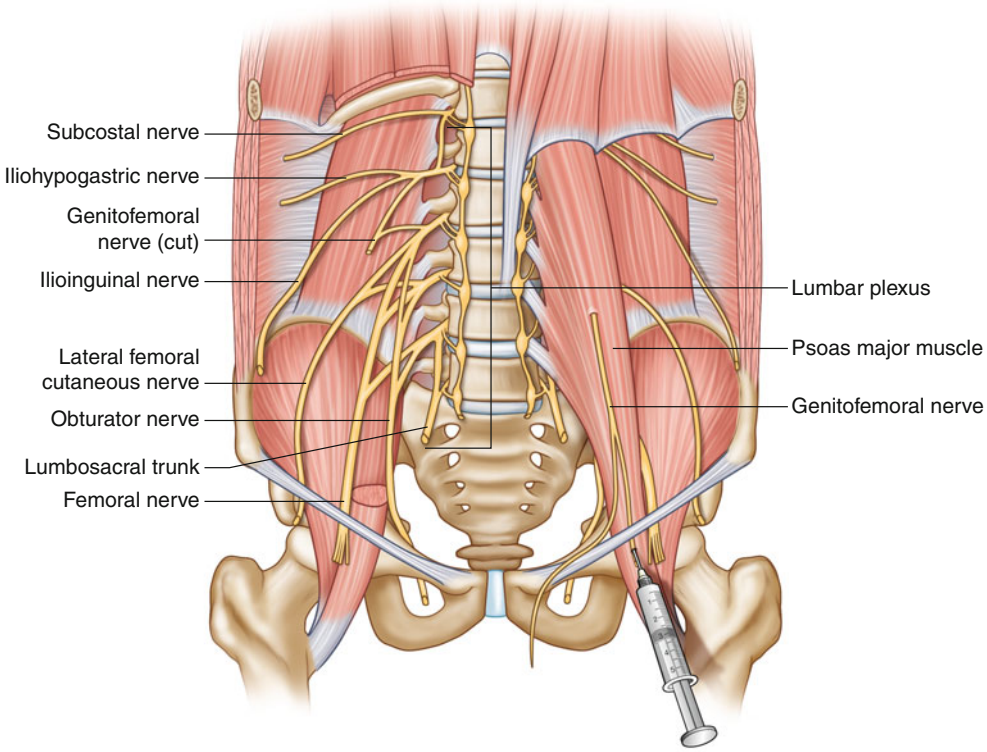

Fig. 49.23 Needle location for inguinal perivascular injection (Image courtesy of Springer)

5-cm needle is inserted immediately lateral to the femoral artery, caudad to the inguinal ligament (Fig. 49.23), and advanced with cephalic angulation until a femoral paresthesia is obtained. At this point, the needle is firmly fixed, and, while the distal femoral sheath is digitally compressed, the entire volume of local anesthetic is injected.

Neurolytic/Surgical Technique

Cryoneuroablation

Because the plexus is a large structure, it is not amenable to a precise technique like cryoneuroablation.

Radiofrequency Lesioning (RF)

There is no literature on this technique, but the same issues as above are involved. The lumbar plexus is not amenable to a precise technique like radiofrequency.

Alcohol/Phenol

To provide longer-term relief, injection of neurolytic substances (6 % aqueous phenol or absolute alcohol) into the psoas sheath was described by Calava et al. [40] for a *malignant psoas syndrome* (metastatic lipoma) patient for whom a prior psoas compartment anesthetic block failed [40]. Depending on life expectancy, a lumbar plexus catheter could be considered for placement [41].

Surgery

Surgical dorsal rhizotomy may provide pain relief from lumbar plexopathy, especially in tumor patients where other conservative measures failed [42]. Surgical nerve repair and nerve grafting may lead to partial recovery of plexopathy from trauma or fractures [43].

Complications

The most serious complications associated with lumbar plexus block are related to the close proximity to the spinal cord and exiting nerve roots, as the needle can cause trauma to the exiting lumbar nerve roots [44]. If the needle is directed too dorsally and medially, there may be inadvertent subarachnoid, subdural, and/or epidural injection. While inadvertent dural puncture is rare, unintentional dural or subdural injection can result in immediate total spinal anesthesia with associated loss of consciousness, hypotension, and apnea [45]. This must be recognized immediately. Intravascular injections can lead to local anesthetic toxicity with loss of consciousness, hypotension, apnea, or cardiac arrest. This risk can be decreased by using digital subtraction

(fluoroscopy) or color mode for Doppler (ultrasound). There can also be delayed hematomas after these injections [46].

Summary

Lumbar plexus entrapment can present in a myriad of ways, including pelvic pain, which makes diagnosis difficult if there is not a high index of suspicion. A careful history and physical exam along with a diagnostic injection can help to elucidate the cause.

References

1. Yeo PT, Grice AC. Lumbosacral nerve plexus compression by ovarian-fallopian cysts. Clin Exp Neurol. 1986;22:139–43.
2. Batchelor TJ, Vowden P. A persistent sciatic artery aneurysm with an associated internal iliac artery aneurysm. Eur J Vasc Endovasc Surg: Off J Eur Soc Vasc Surg. 2000;20(4):400–2.
3. Vinnicombe SJ, Norman AR, Nicolson V, Husband JE. Normal pelvic lymph nodes: evaluation with CT after bipedal lymphangiography. Radiology. 1995;194(2):349–55.
4. Griffiths EA, Kalbassi MR, Cunliffe WJ, Browell DA. Psoas abscess as a secondary complication of gastrointestinal disease: report of five cases. Case Rep Clin Pract Rev. 2003;4(2):73–6.
5. Chevalier X, Larget-Piet B. Femoral neuropathy due to psoas hematoma revisited. Report of three cases with serious outcomes. Spine (Phila Pa 1976). 1992;17(6):724–6.
6. Ozkan OF, Guner A, Cekic AB, Turan T, Kaya U, Reis E. Iliopsoas haematoma: a rare complication of warfarin therapy. J Coll Physicians Surg Pak: JCPSP. 2012;22(10):673–4.
7. Paley M, Sidhu PS, Evans RA, Karani JB. Retroperitoneal collections – aetiology and radiological implications. Clin Radiol. 1997;52(4):290–4.
8. Jaeckle KA, Young DF, Foley KM. The natural history of lumbosacral plexopathy in cancer. Neurology. 1985;35(1):8–15.
9. Stevens MJ, Gonet YM. Malignant psoas syndrome: recognition of an oncologic entity. Australas Radiol. 1990;34(2):150–4.
10. Gibbons KJ, Soloniuk DS, Razack N. Neurological injury and patterns of sacral fractures. J Neurosurg. 1990;72(6):889–93.
11. Irvin W, Andersen W, Taylor P, Rice L. Minimizing the risk of neurologic injury in gynecologic surgery. Obstet Gynecol. 2004; 103(2):374–82.
12. Hughes MS, Burd TA, Allen WC. Post-traumatic catamenial sciatica. Orthopedics. 2008;31(4):400.
13. Feasby TE, Burton SR, Hahn AF. Obstetrical lumbosacral plexus injury. Muscle Nerve. 1992;15(8):937–40.
14. Rancont CM. Chronic psoas syndrome caused by the inappropriate use of a heel lift. J Am Osteopath Assoc. 2007;107(9):415–8.
15. Soldatos T, Andreisek G, Thawait GK, Guggenberger R, Williams EH, Carrino JA, et al. High-resolution 3-T MR neurography of the lumbosacral plexus. Radiographics. 2013;33(4):967–87.
16. Whiteside JL, Barber MD, Walters MD, Falcone T. Anatomy of ilioinguinal and iliohypogastric nerves in relation to trocar placement and low transverse incisions. Am J Obstet Gynecol. 2003; 189(6):1574–8.
17. Petchprapa CN, Rosenberg ZS, Sconfienza LM, Cavalcanti CF, Vieira RL, Zember JS. MR imaging of entrapment neuropathies of the lower extremity. Part 1. The pelvis and hip. Radiographics. 2010;30(4):983–1000.
18. Hacker HS, King JC. Plexopathy: lumbosacral. In: Frontera WR, Silver SK, Rizzo Jr TD, editors. Essentials of physical medicine and rehabilitation: musculoskeletal disorders, pain, and rehabilitation. Philadelphia: Sanders Elsevier; 2008. p. 779–86.
19. Jaeckle KA. Nerve plexus metastases. Neurol Clin. 1991;9(4): 857–66.
20. Taylor BV, Kimmel DW, Krecke KN, Cascino TL. Magnetic resonance imaging in cancer-related lumbosacral plexopathy. Mayo Clin Proc. 1997;72(9):823–9.
21. Tonsgard JH, Kwak SM, Short MP, Dachman AH. CT imaging in adults with neurofibromatosis-1: frequent asymptomatic plexiform lesions. Neurology. 1998;50(6):1755–60.
22. Ozcakar L, Sivri A, Aydinli M, Tavil Y. Lumbosacral plexopathy as the harbinger of a silent retroperitoneal hematoma. South Med J. 2003;96(1):109–10.
23. Cottier JP, Descamps P, Sonier CB, Rosset P. Sciatic endometriosis: MR evaluation. AJNR Am J Neuroradiol. 1995;16(7):1399–401.
24. Whitaker R, Borley N. Chapter 6. Instant anatomy. 1st ed. Oxford: Blackwell Scientific; 1994. p. 114–23.
25. Planner AC, Donaghy M, Moore NR. Causes of lumbosacral plexopathy. Clin Radiol. 2006;61(12):987–95.
26. Thomas JE, Cascino TL, Earle JD. Differential diagnosis between radiation and tumor plexopathy of the pelvis. Neurology. 1985;35(1):1–7.
27. Donaghy M. Lumbosacral plexus lesions. In: Dyk PJ, Thomas PK, editors. Peripheral neuropathy, vol. 2. 4th ed. Philadelphia: Elsevier Saunders; 2005. p. 1375–90.
28. Laughlin RS, Dyck PJ. Electrodiagnostic testing in lumbosacral plexopathies. Phys Med Rehabil Clin N Am. 2013;24(1):93–105.
29. Boon AJ, Gertken JT, Watson JC, Laughlin RS, Strommen JA, Mauermann ML, et al. Hematoma risk after needle electromyography. Muscle Nerve. 2012;45(1):9–12.
30. Hammersted JP. Plexopathy syndromes. In: Goetz CG, editor. Textbook of clinical neurology. 2nd ed. Philadelphia: Saunders; 2003. p. 271–2.
31. Waldman SD. Lumbar plexus block: psoas compartment technique. In: Waldman SD, editor. Atlas of interventional pain management. 2nd ed. Philadelphia: Saunders; 2004. p. 443–8.
32. Waldman SD. Ultrasound-guided lumbar plexus nerve block. In: Waldman SD, editor. Comprehensive atlas of ultrasound-guided pain management injection techniques. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2014. p. 719–26.
33. David L. Brown. Lumbar plexus block. In: Brown DL, editor. Atlas of regional anesthesia. Philadelphia: W. B. Saunders; 1992. p. 76–7.
34. Capdevila X, Macaire P, Dadure C, Choquet O, Biboulet P, Ryckwaert Y, et al. Continuous psoas compartment block for postoperative analgesia after total hip arthroplasty: new landmarks, technical guidelines, and clinical evaluation. Anesth Analg. 2002;94(6):1606–13. table of contents.
35. Hadzic A, Vioka JD. Lumar plexus block. In: Hadzic A, editor. Hadzic's peripheral nerve blocks and anatomy for ultrasound-guided regional anesthesia. 2nd ed. New York: McGraw-Hill; 2012. p. 217–28.
36. Enneking FK, Chan V, Greger J, Hadzic A, Lang SA, Horlocker TT. Lower-extremity peripheral nerve blockade: essentials of our current understanding. Reg Anesth Pain Med. 2005;30(1): 4–35.
37. Mulchandani H, Awad I, McCaetney C. Ultrasound-guided nerve blocks of the lower limb. In: Narouze SM, editor. Atlas of ultrasound-guided procedures in interventional pain management. New York: Springer; 2010. p. 249–50.
38. Paradise LA, Raj PP, Fryer JR. Lumbar myofascial blocks. In: Raj PP, Lou L, Erdine S, Staats PS, Waldman SD, Racz GB, et al., editors. Interventional pain management image-guided procedures. 2nd ed. Philadelphia: Saunders; 2008. p. 382–90.

39. Lang SA, Yip RW, Chang PC, Gerard MA. The femoral 3-in-1 block revisited. J Clin Anesth. 1993;5(4):292–6.
40. Calava JM, Patt RB, Reddy S, Varma DG, Chiang J. Psoas sheath chemical neurolysis for management of intractable leg pain from metastatic liposarcoma. Clin J Pain. 1996;12(1):69–75.
41. Douglas I, Bush D. The use of patient-controlled boluses of local anaesthetic via a psoas sheath catheter in the management of malignant pain. Pain. 1999;82(1):105–7.
42. Lang EM, Borges J, Carlstedt T. Surgical treatment of lumbosacral plexus injuries. J Neurosurg Spine. 2004;1(1):64–71.
43. Kutsy RL, Robinson LR, Routt Jr ML. Lumbosacral plexopathy in pelvic trauma. Muscle Nerve. 2000;23(11):1757–60.
44. Gungor I, Zinnuroglu M, Tas A, Tezer T, Beyazova M. Femoral nerve injury following a lumbar plexus blockade. Balk Med J. 2014;31(2):184–6.
45. Dogan Z, Bakan M, Idin K, Esen A, Uslu FB, Ozturk E. Total spinal block after lumbar plexus block: a case report. Braz J Anesthesiol (Elsevier). 2014;64(2):121–3.
46. Aveline C, Bonnet F. Delayed retroperitoneal haematoma after failed lumbar plexus block. Br J Anaesth. 2004;93(4):589–91.

Inferior Cluneal Nerve Entrapment: Pelvic

50

Terri Dallas-Prunskis

Introduction

Neuropathic perineal pain is often associated with a pudendal neuropathy. However, some patients present with pain located more laterally on the anal margin and on areas including the scrotum or the labia majora, the caudal and medial parts of the buttock, and the upper part of the posterior thigh. These pains extend beyond the territory of the pudendal nerve. The inferior cluneal nerve (ICN), which emerges from the posterior femoral cutaneous nerve, has some branches innervating the perineum. These areas of overlapping symptoms must be clearly defined in order to determine the etiology of the pain.

Clinical Presentation (Table 50.1)

Patients with ICN entrapment complain of a burning, tingling, or numbing sensation along the inferior and medial aspect of the buttocks (Fig. 50.1) and/or along the dorsal and proximal thigh, as well as the lateral anal margin and the skin of the scrotum or labia majora (Fig. 50.2). Pain will increase with sitting on hard surfaces, such as chairs or bicycle seats. Like the pudendal nerve (see Chap. 47), patients may complain of dyspareunia.

Anatomy (Table 50.2)

The cluneal nerves are divided into three groups: the *superior cluneal nerves* (see Chap. 51), the *middle cluneal nerves (lateral branches of the sacral nerves)*, and the *inferior or lateral cluneal nerves* (Fig. 50.3). The ICN arises from the inferior portion of the *posterior femoral cutaneous nerve of the thigh* (PFCN) (see Chap. 46). This nerve is made up of the sensory branches of S1, S2, and S3, traveling parallel with the sciatic nerve and the pudendal nerve through the sciatic notch. After reaching the subgluteal area, the PFCN gives rise to the *inferior cluneal branch* and the *perineal branch* (Fig. 50.4). The nerves then go to the infe-

Table 50.1 Occupation/exercise/trauma history relevant to inferior cluneal entrapment

Trauma	Fall onto buttocks; hamstring injury; intramuscular injection into gluteal muscle; piriformis trauma or injury
Direct compression of nerve	Sitting on a hard seat, bicycle-riding
Myofascial compression	Piriformis spasm; gluteal muscle spasm

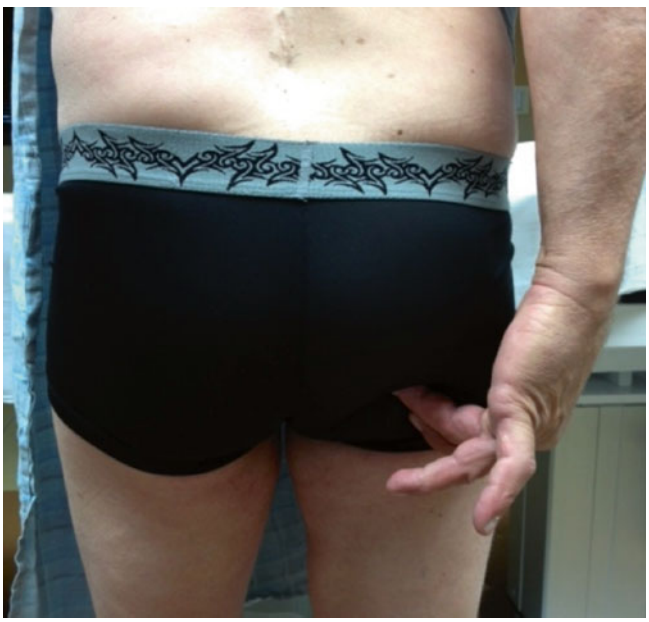

Fig. 50.1 Pain location from inferior cluneal neuralgia (Image courtesy of Terri Dallas-Prunskis, MD)

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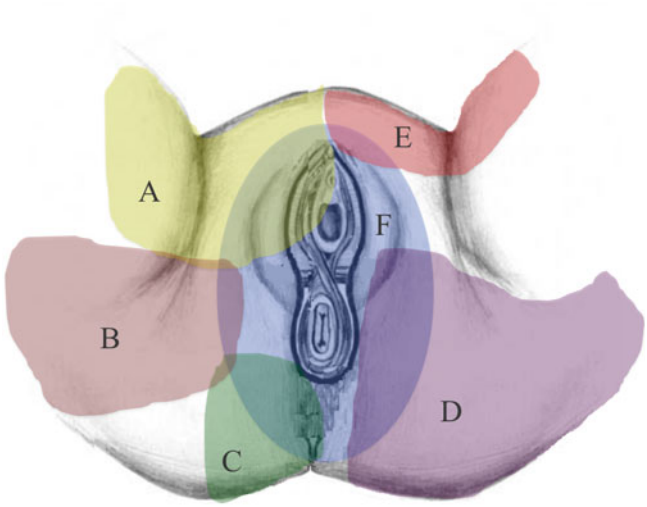

Fig. 50.2 Innervation of perineum: (*A*) genitofemoral nerve; (*B*) obturator nerve; (*C*) inferior cluneal nerve; (*D*) peroneal branch of the posterior femoral cutaneous nerve, (*E*) ilioinguinal nerve, and (*F*) pudendal nerve (Image inspired by Hibner et al. [8], courtesy of Andrea Trescot, MD)

Table 50.2 Inferior cluneal nerve anatomy

Origin	The nerves arise from the inferior portion of the posterior femoral cutaneous nerve of the thigh
General route	The posterior femoral cutaneous nerve is made up of the sensory branches of S1, S2, and S3. It travels through the sciatic notch and, after reaching the subgluteal area, gives rise to the inferior cluneal branch and the perineal branches. The nerves then go to the inferior edge of the gluteus maximus, travel anterior to it, and then circumvent and have a recurrent course behind the muscle, innervating its target areas
Sensory distribution	Provides cutaneous innervation to the inferior part of the buttock, lateral anus region (but not the anus), and the lateral region of the labium majorum
Motor innervation	None
Anatomic variability	Only a few variations are noted in the literature, giving a description of the inferior cluneal nerves going through the gluteus maximus muscle; other sources indicate that the nerves reach the caudal edge of the gluteus maximus and then circumvent it at various levels

rior edge of the gluteus maximus muscle, course in front of it, and then circumvent and have a recurrent course behind the muscle. The inferior cluneal nerves provide cutaneous innervation to the inferior part of the buttocks and lateral anus region, but not the anus. It also innervates the lateral region of the labium majorum, but not the labia minora or the vagina. It does not innervate the penis or clitoris (Fig. 50.2) [1].

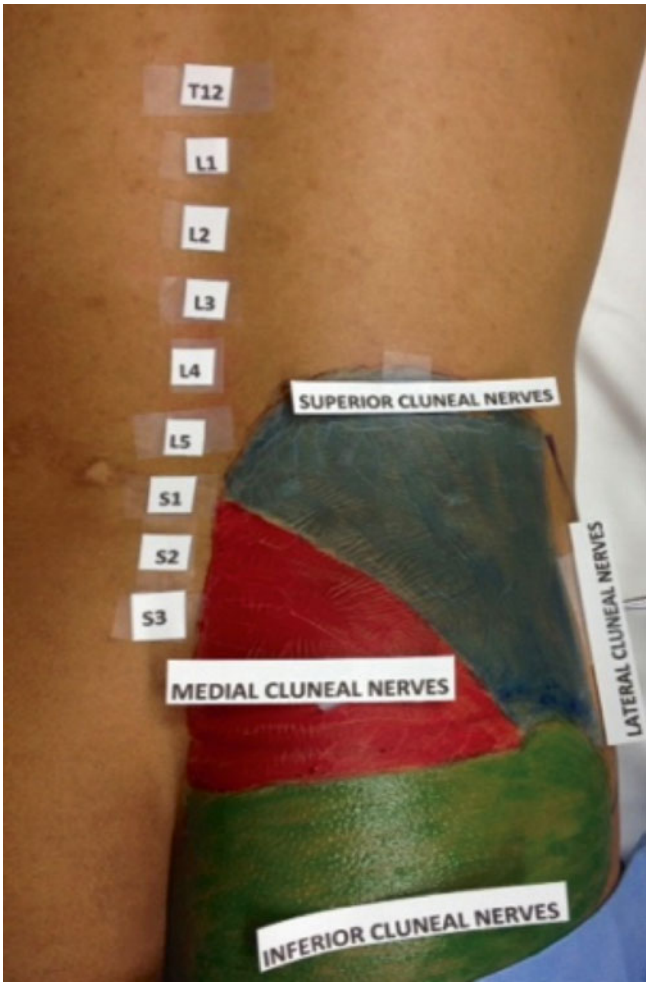

Fig. 50.3 Cutaneous distribution of the cluneal nerves (Image courtesy of Terri Dallas-Prunskis, MD)

Entrapment

There are two common areas where entrapment may occur. The first one would extend from the passage of the perineal ramus under the ischium to the perineum (Site A on Fig. 50.5). This entrapment may be due to nerve compression by the ischium on the gluteus maximus and the hamstring muscles in a sitting position and stretching of the perineal ramus with internal rotation of the thigh.

The second site of entrapment is more proximal, at the level of the sciatic spine and the piriformis. At this point, the roots of the posterior femoral cutaneous nerve, which gives rise to the inferior cluneal nerve, may be encircled by the piriformis against the sciatic notch (Site B on Fig. 50.5). However, whatever the etiology, it is the sitting position that triggers the entrapment, giving the inferior cluneal entrapment syndrome the same general appearance as a pudendal syndrome [2].

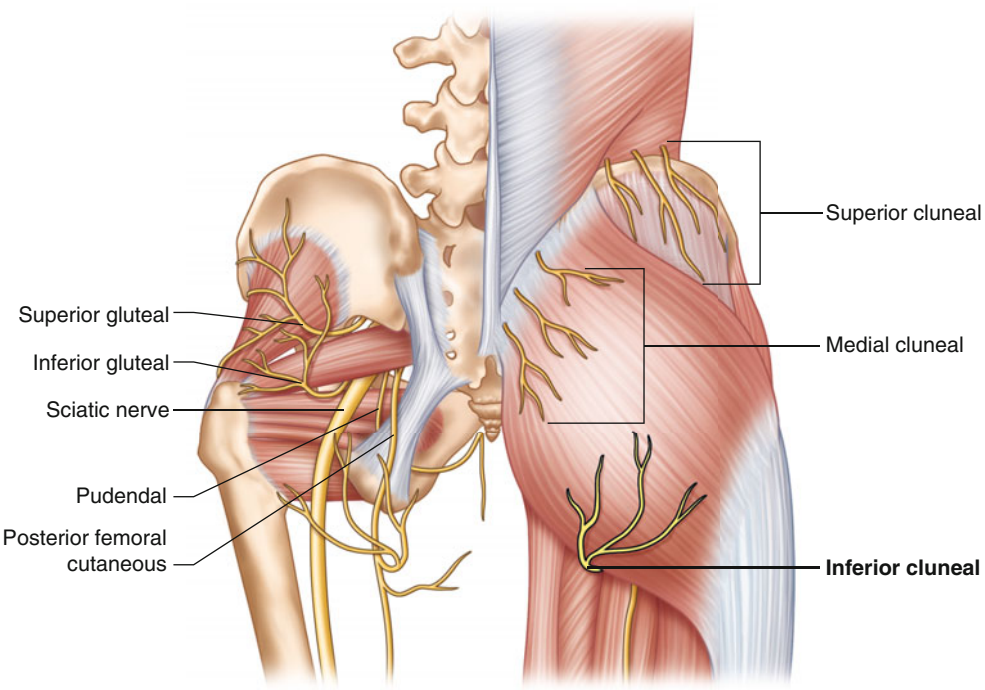

Fig. 50.4 Anatomy of the buttocks nerves (Image by Springer)

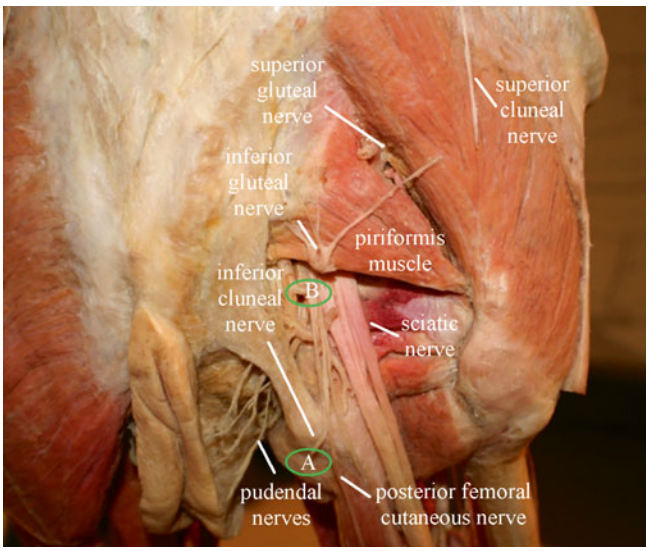

Fig. 50.5 Gluteal muscle dissection modified from an image from *Bodies*, *The Exhibition*, with permission. *A* distal entrapment, *B* proximal entrapment (Image courtesy of Andrea Trescot, MD)

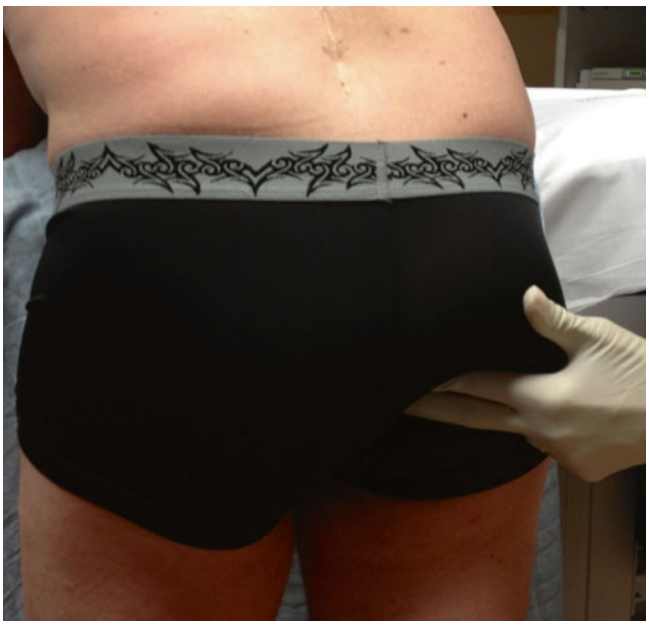

Fig. 50.6 Physical exam of the inferior cluneal nerve; palpation over the inferior cluneal nerve near the sciatic notch (Image courtesy of Terri Dallas-Prunskis, MD)

Physical Examination

The examination should begin by evaluating the entire back to rule out other causes for the pain. Palpation should illicit non-radiating pain increasing with deep pressure over the sciatic notch. Palpate the inferior edge of the gluteus maximus between the ischium and the greater trochanter, where the inferior cluneal nerves circumvent the muscle at various points (Fig. 50.6). Hyperesthesia to pin scratch and decreased sensation to touch over the inferior buttocks corresponding to the distribution of the inferior cluneal nerve will also be noted; no evidence of motor involvement is appreciated. Pain may be induced on digital rectal examination of the ischium more superficial than that associated with pudendal canal syndrome (at the pelvic head of the obturator internus) [3].

Differential Diagnosis (Table 50.3)

Inferior cluneal nerve entrapment must be differentiated from other pelvic pain disorders, including *posterior femoral cutaneous nerve entrapment* (see Chap. 46), pudendal neuralgia (see Chap. 47), or *obturator nerve entrapment* (see Chap. 48) caused by reflex muscle reactions of the piriformis and obturator internus muscles, inducing "irritation" of these nerve trunks. This entrapment is also misdiagnosed as *pudendalgia* (*pudendal canal syndrome*) [4] (Table 50.4).

Contributing Factors

The inferior cluneal nerves may be injured either by a fall onto the buttocks or by a hamstring injury, and sometimes it is not clear what caused the cluneal nerves to be symptomatic. Intramuscular injection into the medial inferior quadrant of the buttock leading to muscle spasm, myositis, and nerve entrapment of nerves within the muscle has been reported. Mechanical damage may result to the inferior cluneal nerves during their course through the piriformis [5].

Sitting on a hard seat will increase the compression of the nerves in the buttocks or underneath the ischium (possibility of subischial tunnel syndrome in contact with the insertion of the hamstring muscles) [2]. Piriformis trauma or injury may also be causative.

Table 50.3 Differential diagnosis of buttock and pelvic pain

	Potential distinguishing features
Posterior femoral cutaneous neuritis	Innervates the lateral and lower portions of the gluteus maximus muscle and the posterior parts of the leg and thigh; the skin of the perineum
Obturator neuritis	Medial thigh or groin pain, weakness with leg adduction, and sensory loss in the medial thigh
Piriformis syndrome	Pain, tingling, and numbness in the buttocks and along the path of the sciatic nerve, descending down the lower thigh and into the leg; hypertonicity of the piriformis muscle
Obturator internus muscle spasm	Difficulty with lateral rotation of the femur with hip extension and abduction of the femur with hip flexion; instability of the femoral head in the acetabulum; hypertonicity of the obturator internus muscle
Pudendalgia (Pudendal canal syndrome)	Pain is positional; worsened by sitting, relieved by standing, absent when recumbent or sitting on the toilet; genital numbness, fecal incontinence, and urinary incontinence may occur

Injection Technique

Landmark-Guided Technique

The procedure may be difficult to perform blindly and will depend on the ability to adequately palpate the patient's anatomy. The patient should be placed in a prone position or standing leaning securely over a cart/bed. Use an aseptic technique to prep the buttocks. Palpate the inferior aspect of the ischium, and mark the site. Next, localize the gluteus maximus muscle and the lateral edge of the hamstring muscle insertion. After local infiltration to the skin and subcutaneous tissue, insert a 22-gauge 3.5 in. block needle through the gluteus maximus onto the lateral edge of the hamstring muscles insertion (the lateral and inferior edges of the ischium) (Fig. 50.7). Following negative aspiration, inject 2–3 cc of a local anesthetic/steroid solution. Using peripheral nerve stimulation would be appropriate for this procedure.

Fluoroscopy-Guided Techniques

With the patient in a prone position, utilizing the fluoroscopic image in an AP view, the lateral and inferior edges of the ischium are identified. Using an aseptic technique, local infiltration with 1 % lidocaine is given to the skin and

Table 50.4 Diagnostic tests for inferior cluneal neuralgia

	Potential distinguishing features
Physical exam	Palpation should illicit non-radiating pain increasing with deep pressure over the sciatic notch; hyperesthesia to pin scratch and decreased sensation to touch over the inferior buttocks corresponding to the nerve distribution. Pain may be induced on digital rectal examination of the ischium more superficial than that associated with pudendal canal syndrome
Diagnostic injection	Utilizing landmark or fluoroscopic technique, local anesthetic/steroid solution may be injected through the gluteus maximus, directed toward the lateral and inferior edge of the ischium
Ultrasound	The nerves are located by identifying the inferior border of the ischium, the gluteus maximus muscle and the lateral edge of the hamstring muscle insertion; the block needle is placed on the lateral and inferior edges of the ischium
MRI	Not useful
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies	Not useful

subcutaneous tissue over the targeted point. Next, a 22-gauge 3.5 in. needle is inserted 1 cm laterally from the caudal edge of the ischium, which is under the gluteus maximus and on the lateral edge of the hamstring muscle insertion (Fig. 50.8). Following a negative aspiration, inject 2–3 cc of a local anesthetic/steroid solution. A peripheral nerve stimulator may also be used to confirm proper needle placement.

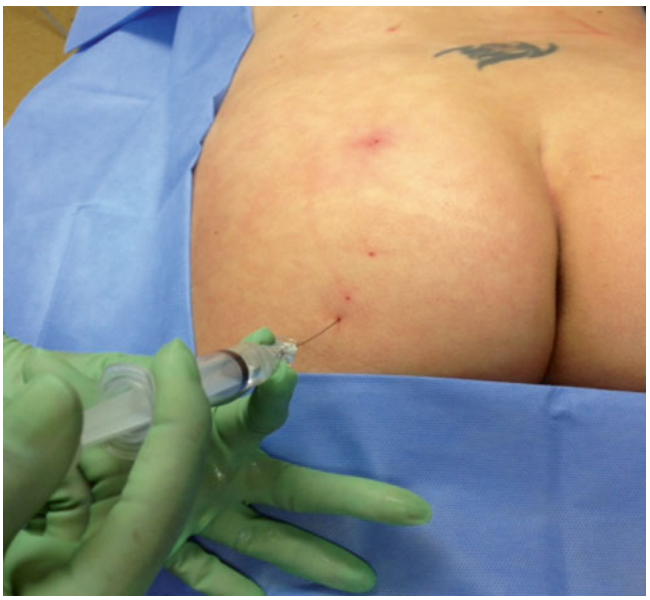

Fig. 50.7 Landmark-guided injection of the inferior cluneal nerve; through the gluteus maximus, and lateral and inferior of the ischium (Image courtesy of Terri Dallas-Prunskis, MD)

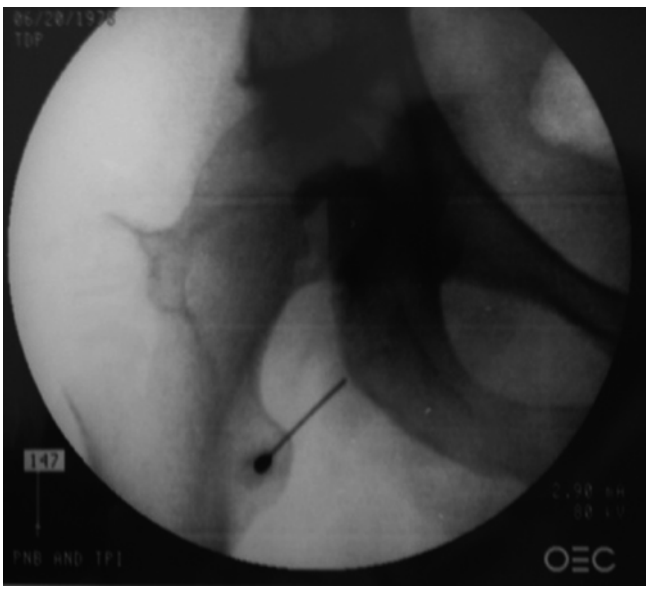

Fig. 50.8 Fluoroscopic injection of the inferior cluneal nerve (Image courtesy of Terri Dallas-Prunskis, MD)

Ultrasound-Guided Techniques

The patient is placed in a prone position. Noninvasive monitors are applied and intravenous access obtained, if the patient requires it. Palpate the inferior edge of the ischium. Skin disinfection is then performed over the gluteal area. A high-frequency (7–12 MHz) linear array probe is appropriate for this block. An in-plane or an out-of-plane approach may be used. Locate the inferior border of the ischium (the ischium casts a bony shadow on the US image), the gluteus maximus muscle, and the lateral edge of the hamstring muscle insertion (Fig. 50.9). The skin is infiltrated with lidocaine, and the block needle is inserted through the gluteus maximus on the lateral and inferior edges of the ischium. Using US guidance, the ICN can be blocked with 2–3 cc of a local anesthetic/steroid solution. Peripheral nerve stimulation can confirm the proper needle placement.

Neurolytic/Surgical Techniques

Following successful infiltrations, if there is temporary relief of pain, neurolytic or surgical techniques may be considered. All of the neurolytic techniques should be performed using a method of imaging.

Cryoneuroablation

Cryoneuroablation may be performed at the lateral and inferior edges of the ischium, with the patient in a prone position. Utilizing an aseptic technique, a small amount of local anesthetic is infiltrated subcutaneously using a 25-gauge 1.5 in. needle. A small incision is made into the skin. An IV introducer (size 12 or 14 gauge, depending on the probe size) is advanced to the target area. The stylet is removed, and the cryoprobe is then advanced through the catheter. The tip of the probe should be exposed by withdrawing the catheter back into the subcutaneous tissues. The probe placement should be confirmed with maximal sensory stimulation and devoid of motor stimulation. This should be followed by a series of three 2-min freezes, with 30 s defrosting between each cycle. The patient may experience burning pain initially during the first freeze cycle, which often replicates the pain and should resolve within approximately 30 s [6].

Radiofrequency Lesioning

Radiofrequency lesioning has also been utilized for extended pain relief of inferior cluneal neuropathies following successful infiltration. The patient is placed in a prone position,

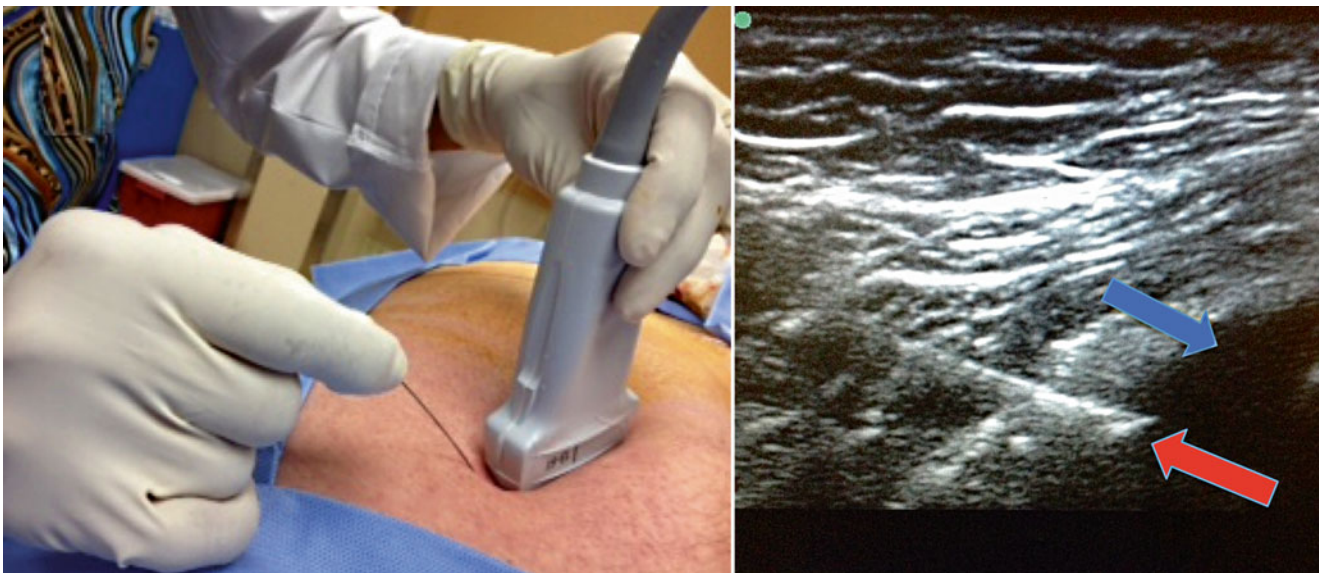

Fig. 50.9 Ultrasound image of the inferior cluneal nerve. *Blue arrow* = ischium; *red arrow* = needle injection (Image courtesy of Terri Dallas-Prunskis, MD)

and utilizing imaging in an AP view, the lateral and inferior edges of the ischium are identified. Using aseptic technique, the skin is anesthetized subcutaneously, and the radiofrequency cannula is advanced to the target site at the ischium. After the radiofrequency probe is advanced through the cannula appropriately, maximal sensory and devoid motor stimulation is used to confirm that the tip of the probe is placed adequately. Either pulsed or non-pulsed lesioning may be used [7].

Surgery

Surgery may be considered after the infiltration provides improvement or temporary pain relief. Two surgical approaches have been discussed in the literature. For instance, a transgluteal approach for decompression and transposition is described when the clunealgia is associated to a pudendalgia, caused by a piriformis syndrome [4]. The second preferred approach is when there is an isolated clunealgia with an under-and-into ischiatic entrapment. The surgical approach is on the dorsal and cranial parts of the thigh [2].

Complications

General complications may occur, based on the location of the needle placement, neural trauma, or hematoma formation. Infectious complications include abscess and side effects related to the administration of local anesthetic and/or steroids and other drugs. Caution must be exercised when performing the procedures blindly. The bone should be contacted so as not to place the needle too deeply.

In performing cryoneuroablation, risk of depigmentation or hyperpigmentation at the cryolesion site and neuritis has been reported [6].

The most common complications of radiofrequency include those related to the placement of the needle and to the neurolysis. The majority of problems are short-lived and self-limited, and they include local swelling and pain at the site of the needle insertion, as well as somatic pain from the site of insertion. Other reported complications of radiofrequency thermoneurolysis include a worsening of the usual pain, burning or dysesthesias, decreased sensation, and allodynia over the skin [7].

Summary

The inferior cluneal nerve is a cause of both low back pain and pelvic pain. It is rarely diagnosed and even more rarely taught. Understanding the clinical presentation and the physical exam will perhaps increase the diagnosis and therefore treatment of this potentially debilitating problem.

References

1. Netter FH. Atlas of human anatomy: ICON learning systems. 1997. p. 468–509.
2. Darnis B, Robert R, Labat JJ, Riant T, Gaudin C, Hamel A, et al. Perineal pain and inferior cluneal nerves: anatomy and surgery. Surg Radiol Anat. 2008;30(3):177–83.

3. Labat JJ. Pudendal neuralgia: clinical signs and diagnosis. In: Baranowski AP, Abrams P, Fall M, editors. Urogenital pain in clinical practice. London: Informa Healthcare Publishing; 2007. p. 361–72.
4. Robert R, Prat-Pradal D, Labat JJ, Bensignor M, Raoul S, Rebai R, et al. Anatomic basis of chronic perineal pain: role of the pudendal nerve. Surg Radiol Anat. 1998;20(2):93–8.
5. Hanson D. Intramuscular injection injuries and complications. Gen Pract. 1963;27:109–15.
6. Trescot AM. Cryoneuroablation. In: Manchikanti L, Singh V, editors. Interventional techniques in chronic non-spinal pain. Paducah: ASIPP Publishing; 2009. p. 69–86.
7. Trescot A, Hansen H. Radiofrequency neurolysis. In: Manchikanti L, Singh V, editors. Interventional techniques in chronic non-spinal pain. Paducah: ASIPP Publishing; 2009. p. 59–68.
8. Hibner M, Desai N, Robertson LJ, Nour M. Pudendal neuralgia. J Minim Invasive Gynecol. 2010;17(2):148–53.

Part VIII

Low Back Pain

Terri Dallas-Prunskis

Introduction

Low back pain remains the most common health complaint in the United States and around the world. Americans spend between $38 and $50 billion each year on low back pain, which is also the most common cause of job-related disability and a leading contributor to missed work. Over a lifetime, 80 % of people have lower back pain, with the difficulty most often beginning between 20 and 40 years old and in women. Although the pain is due in part to the aging process, a sedentary life style with too little exercise and cigarette smoking also play a large part. Back pain is the leading cause of disability in Americans under 45 years old, and over 26 million Americans between the ages of 20 and 64 expeiernce frequent back pain [1].

The back is an intricate structure of ligaments, fascia, nerves, muscles, bones, and other tissues that form the posterior part of the body's trunk from the neck to the pelvis. Several structures have been incriminated as possible sources of chronic low back pain. The majority of low back pain is referred to as *nonspecific low back pain* and does not have a definitive cause [2]. It is believed to stem from benign musculoskeletal problems such as muscle or soft tissue sprain or strains [3]. Over 99 % of back pain instances fall into this category. Many other less common conditions may include mechanical (i.e., degenerative disc, disc herniation, sacroiliac joint dysfunction, spondylolisthesis, and other congenital abnormalities); neoplastic (i.e., bone tumors, intradural spinal tumors); metabolic (i.e., osteoporotic fractures, osteomalacia); psychosomatic (i.e., tension myositis syndrome); Paget's disease; referred pain (i.e., pelvic/abdominal disease, prostate cancer, posture); oxygen deprivation; and inflammatory diseases (i.e., rheumatoid arthritis, seronegative spondyloarthritis, infections, sacroiliitis) [4].

Peripheral nerve entrapment in low back pain is frequently an under-recognized diagnosis. Symptoms may include tingling, numbness, and cutaneous pain. The symptoms may affect just one particular part of the body, depending on which nerve is affected. The neuropathy can be diagnosed confidently on the basis of the signs and symptoms alone, if they are recognized appropriately.

In this section, we will review some of the peripheral nerve entrapments that can cause low back pain, including superior cluneal, inferior cluneal, superior gluteal, inferior gluteal, sacral, and sciatic nerves.

References

1. Dionne CE. Low back pain. In: Crombie IK, Linton S, Von Korff M, LeResche L, editors. Epidemiology of Pain. Seattle: IASP Press; 1999.
2. Casazza BA. Diagnosis and treatment of acute low back pain. Am Fam Physician. 2012;85(4):343–50.
3. Lower back pain fact sheet. nih.gov. http://www.ninds.nih.gov/disorders/bckpain/detail_backpain.htm. Retrieved 16 June 2008.
4. Low back pain. Wikipedia. 2013. http://en.wikipedia.org/wiki/Low_back_pain.

Superior Cluneal Nerve Entrapment

51

Terri Dallas-Prunskis

Introduction

Low back pain remains the most common health complaint in the United States and around the world. The back is an intricate structure of ligaments, fascia, nerves, muscles, bones, and other tissues that form the posterior part of the body's trunk from the neck to the pelvis. Several structures have been incriminated as possible sources of chronic low back pain. However, even though diagnostic radiologic imaging and anesthetic block techniques are used, no apparent cause can be found in approximately 50 % of cases [1].

Iatrogenic injury to the superior cluneal nerves (SCN) was once a common complication associated with *autologous bone graft harvesting* from the posterior iliac crest used in reconstructive orthopedic surgery. A common complaint was postoperative pain at the donor site [2]. In one study of almost 100 patients who underwent lumbar disc surgery with fusion using iliac crest grafts, 37 % complained of persistent graft site pain during more than 10 years of follow-up [3]. With the use of alternative techniques such as allograft (cadaveric bone) or synthetic bone substitutes, the incidence of postoperative SCN entrapments has lessened.

Trescot stated, however, that cluneal neuralgia is more commonly the result of an entrapped nerve than a nerve injury following iliac crest harvest [4]. Talu and colleagues found that the SCN is prone to entrapment where it passes through the fascia near the iliac crest [5]. Whatever the cause, cluneal entrapment neuropathies may be underdiagnosed and should be considered as a potential cause of chronic low back pain. This condition has also been called *posterior rami syndrome*, *thoracolumbar junction syndrome*, *Maigne syndrome*, and *dorsal ramus syndrome*.

Electronic supplementary material The online version of this chapter (doi:10.1007/978-3-319-27482-9_51) contains supplementary material, which is available to authorized users.

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Clinical Presentation (Table 51.1)

Strong and Davila [8] first described superior cluneal neuropathy in 1957. Patients with SCN pathology may present with severe low back pain that radiates to the gluteal region (Fig. 51.1) [9]. Several different sensations at the donor site have been described, such as chronic pain or dysesthesia localized to the sensory distribution of the involved nerve. Others describe the pain as "intense" or "dull." It begins in the medial region of the iliac crest and radiates to the ipsilateral buttock and posterior thigh [10].

There should not be any other pathology detected by lumbosacral radiography, computerized tomography (CT), or magnetic resonance imaging (MRI) [11].

Anatomy (Table 51.2)

The cluneal nerves are divided into three groups: the *superior cluneal nerve* (SCN), the *middle cluneal nerve* (lateral sacral nerve), and the *inferior or lateral cluneal nerve* (see Chap. 52) (Figs. 51.2 and 51.3). The superior cluneal nerves are formed by the lateral cutaneous branches of the dorsal rami of T12 to L3; they are further divided into three branches: the *medial branches*, the *intermediate branches*, and the *lateral branches* (Fig. 51.3). Table 51.3 shows the distribution of the origins of the superior cluneal nerve.

The *medial superior cluneal (mSCN) nerves* emerge from the deep fascia and are found to cross the posterior

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Table 51.1 Occupation/exercise/trauma history relevant to superior cluneal nerve entrapment

Surgical trauma	Iliac crest graft [2]
	Decubitus surgery [6]
Myofascial compression	Quadratus lumborum spasm
Maigne syndrome	T12/L1 pathology
Injection trauma	Infragluteal injection [7]

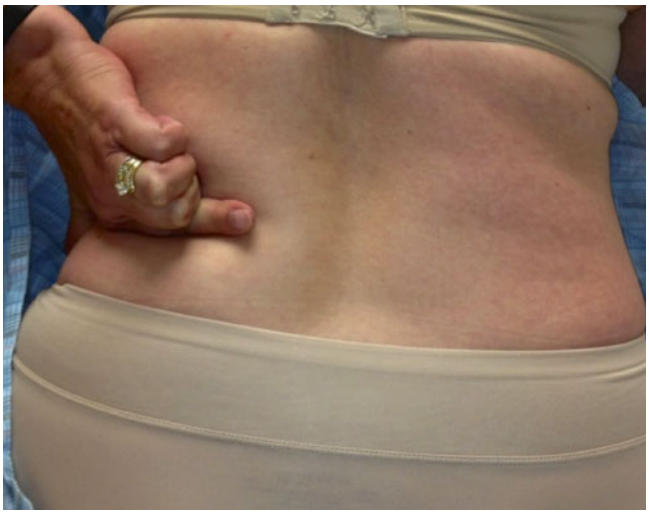

Fig. 51.1 Pain location from superior cluneal neuralgia (Image courtesy of Terri Dallas-Prunskis, MD)

iliac crest at a distance of approximately 7 cm from the midline. At that point, they become superficial, passing through a rigid osseoaponeurotic space formed by the thoracolumbar fascia and the posterior superior iliac crest (PSIS), first described by Lu et al. in 1998 [13]. Entrapment of the nerves is thought to occur mostly at this point [14–16]. Kuniya and colleagues [17] dissected 109 specimens; 61 (56 %) had at least one branch running through an osteofibrous tunnel. The SCN innervates the superior two-thirds of the skin of the gluteal region as far as the greater trochanter (Fig. 51.2) [18].

Entrapment

Nerve compression lesions usually result from the combination of several types of trauma on the nerve including traction, friction, and repetitive compression that lead to local edema in the surrounding tissue and interference with the normal sliding movement of the nerve. In SCN entrapment neuropathy, the anatomic and functional bases for the development of the lesion are a rigid fascial edge, combined with the stretch of the gluteus maximus and skin over a large range by flexion of the hip joint, especially during sports activities. The nerve becomes subjected to stretching forces that cause tissue irritation, edema, inflammatory cell infiltration, and scarring, leading to the subsequent entrapment [19].

The area where the SCN becomes entrapped is located on the iliac crest, approximately 7 cm lateral from the midline as it pierces the quadratus lumborum muscle (Fig. 51.4). Nerve entrapment may occur during spasm or hypertonicity

Table 51.2 Superior cluneal nerve anatomy

Origin	Lateral cutaneous branches of dorsal rami T12, L1, L2, and L3
General route	Nerve emerges from the deep fascia and crosses the posterior iliac crest at a distance of ~6–7 cm from the midline; it then becomes superficial by passing through a space formed by the thoracolumbar fascia and the iliac crest
Sensory distribution	Superior two-thirds of the skin of the gluteal region as far as the greater trochanter
Motor innervation	None
Anatomic variability	Several variations of the running patterns of the SCN branches exist; more commonly, the medial branch of the SCN passes through an osteofibrous tunnel. Some studies have noted that all the branches of the SCN pierce the thoracolumbar fascia above the iliac crest

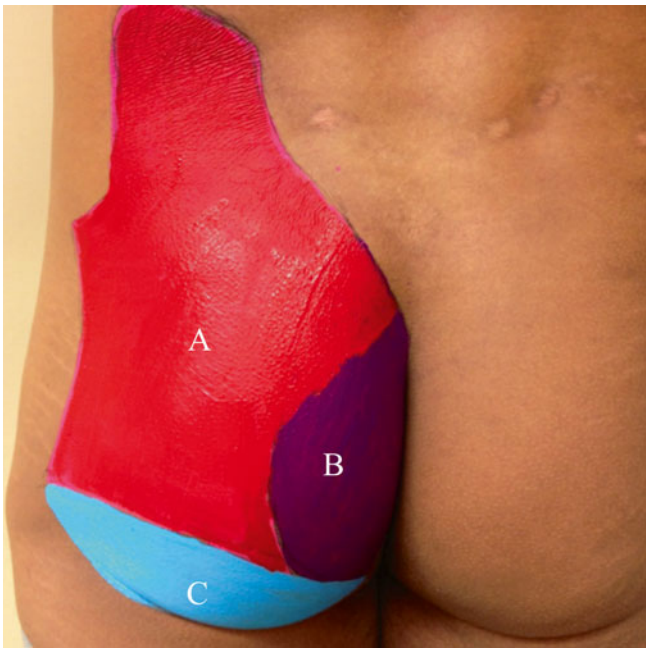

Fig. 51.2 Cutaneous distribution of the cluneal nerves: *A* superior cluneal nerve, *B* medial cluneal nerve, *C* inferior cluneal nerve (Image courtesy of Terri Dallas-Prunskis, MD)

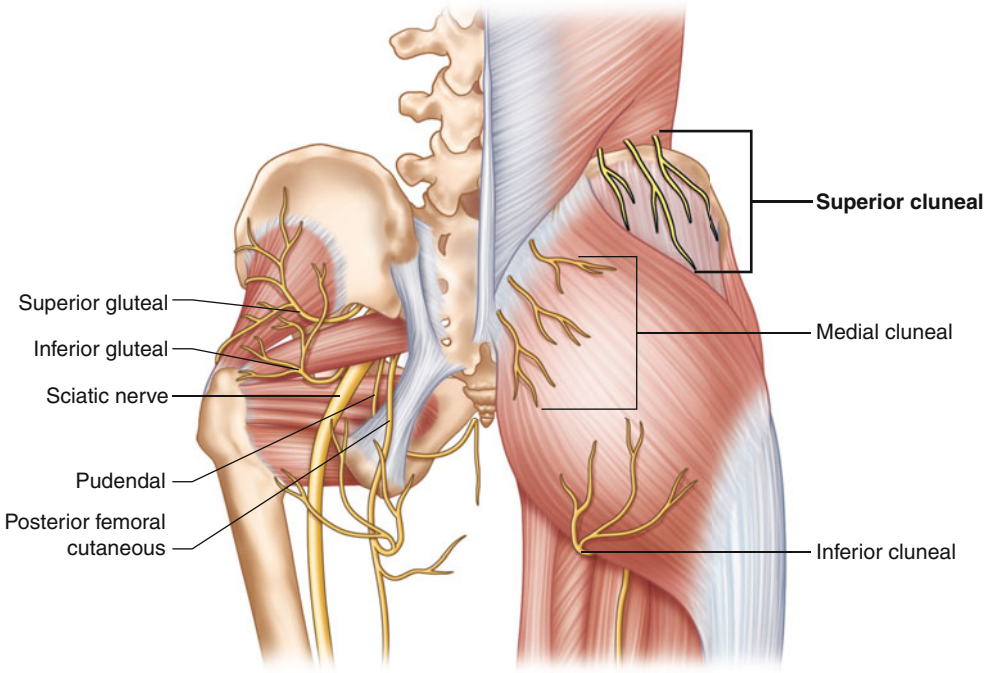

Fig. 51.3 Cluneal anatomy (Image by Springer)

Table 51.3 Distribution of the origin of the superior cluneal nerve in 37 dissections

Origin	Cases (%)
T12 and L1	22 (60 %)
T12, L1, L2	10 (27 %)
T12, L1, L2, L3	5 (13 %)

Data from Maigne et al. [12]

of the quadratus lumborum muscle, causing referred pain to the buttock, leg, and potentially all the way to the foot, clinically mimicking a radiculopathy ("pseudosciatica") (Fig. 51.5).

Physical Examination

The physical examination should begin by examining the entire back to rule out other causes of pain, since this pain may be confused with lumbar spine disorders such as facet syndrome, or with pathology of the sacroiliac joint. The symptoms of entrapment can be reproduced by palpation of the iliac crest approximately 6–7 cm lateral of the midline (Video 51.1) (Fig. 51.6) [20]. This tender point may elicit pain radiating from the low back to the posterior thigh, consistent with the distribution of the SCN. Pain may also be elicited with hyperextension, lateral bending toward the side opposite the complaint, and rotation of the lumbar spine, all of which can cause traction of the quadratus lumborum muscle at the attachment of the iliac spine over the SCN [21]. Aly et al. [9] proposed simultaneous full flexion of the ipsilateral hip and knee joints as a provocative test that reproduced symptoms.

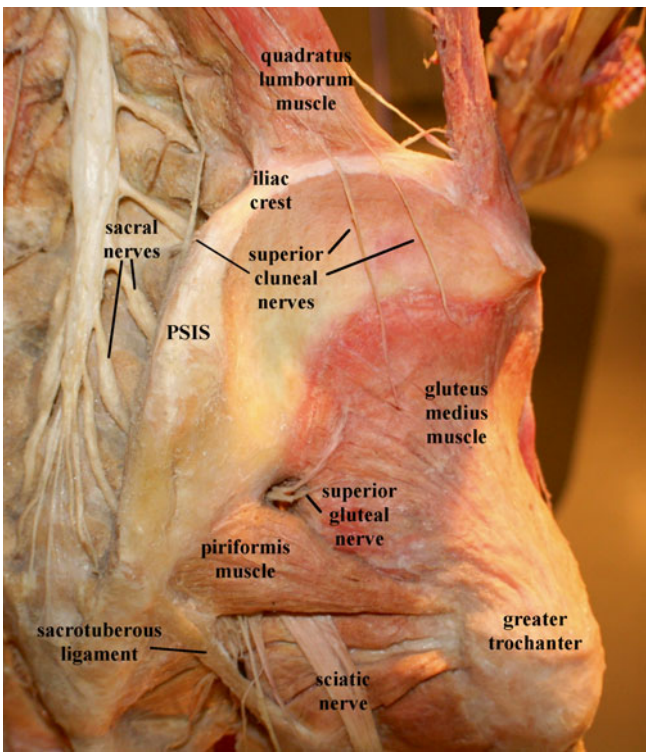

Fig. 51.4 Iliac fossa dissection modified from an image from *Bodies, The Exhibition*, with permission (Image courtesy of Andrea Trescot, MD)

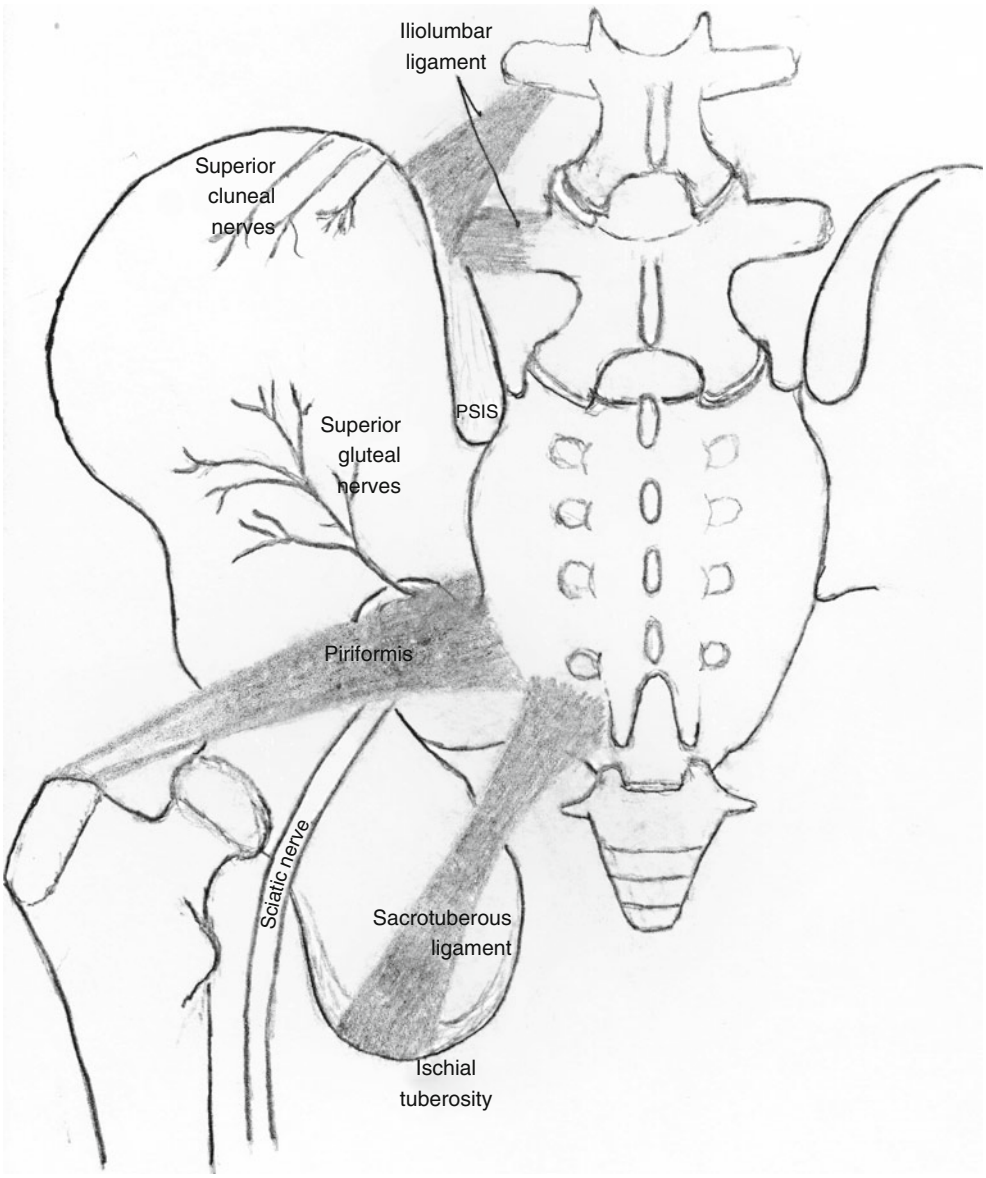

Fig. 51.5 Sites of several causes of "pseudosciatica" (Image courtesy of Luis N. Hernandez, MD)

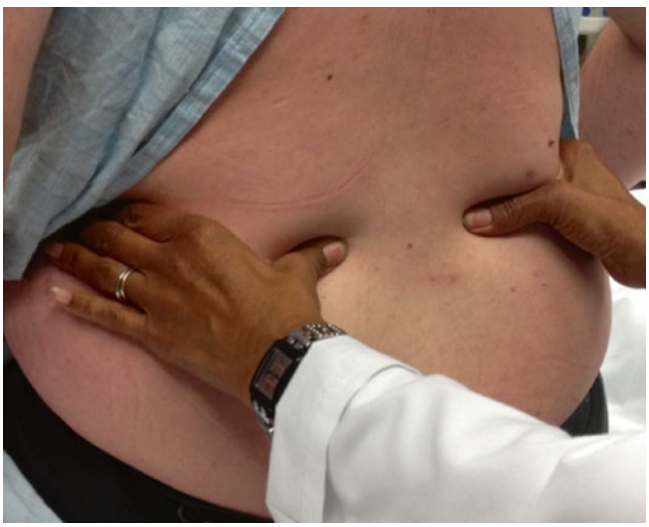

Fig. 51.6 Physical exam of the cluneal nerve (Image courtesy of Terri Dallas-Prunskis, MD)

Differential Diagnosis (Table 51.4)

The possibility of entrapment neuropathy should be considered only when other causes of pain have been reasonably ruled out. The differential diagnosis should include other causes of low back pain specific to the area of complaint (Table 51.4). This includes lumbar spine disorders such as facet syndrome, or with pathology of the sacroiliac joints, lower lumbar disc problems, iliolumbar syndrome, and myofascial pain to the quadratus lumborum muscle [19].

Posterior rami syndrome, also referred to as *thoracolumbar junction syndrome*, *Maigne syndrome*, and *dorsal ramus syndrome*, is a T12/L1 facet pathology causing unilateral low back pain referred to the iliac crest and buttock via the cluneal nerves. There is a tender point over the posterior iliac crest; however, pain is relieved by injection of local anesthetic into the correct facet joint [14, 15]. Foraminal

Table 51.4 Differential diagnosis of buttock pain

	Potential distinguishing features
Lumbar spine disorders	Physical exam and MRI scan should confirm the diagnosis
Sacroiliac joint disorders	Pain may be present in the lower back, back of hips, groin, and thighs; physical exam, X-rays, CT, and MRI scans can help identify pathology; relief of pain from a SI joint injection will confirm the diagnosis
Lower lumbar disc problems	Local pain in the affected area; abnormal X-ray tests or MRI scans
Iliolumbar syndrome/iliac crest pain syndrome	Acute attacks of pain secondary to inflammation or tear of the iliolumbar ligament
Myofascial pain syndromes	Visible hypertonicity or knots in the muscle may be seen; trigger point injections into the muscle will alleviate the symptoms
Posterior rami syndrome, thoracolumbar junction syndrome, Maigne syndrome, and dorsal ramus syndrome	Pain is relieved by injection of local anesthetic into the T12/L1 facet joint

stenosis or a herniated disc at T12, L1, or L2 can cause radicular pain to the iliac crest as well as proximal entrapment of the SCN.

Diagnostic Tests (Table 51.5)

The diagnosis of SCN entrapment neuropathy is made primarily on the basis of the history and physical examination, confirmed by a diagnostic injection.

Maigne et al. [21] and Kunlya et al. [17] suggested diagnostic criteria for an entrapment neuropathy of the SCN:

- Unilateral LBP referring to iliac crest and buttock
- A tender point over the posterior iliac crest located about 7 cm from the midline
- Replication of back and leg pain with palpation of that area
- Relief of symptoms by injection

Identification and Treatment of Contributing Factors

SCN injury is a frequent cause of chronic pain complicating bone graft harvesting from the posterior iliac crest, as previously discussed. However, apart from this surgical iatrogenic lesion, other factors may contribute to entrapment [5, 21]. It was hypothesized that during sports activities such as tennis and volleyball, where the player positions the hip throughout the range of flexion, the nerve is subjected to stretching forces that lead to irritation and edema. Left untreated, it can progress to scarring and

Table 51.5 Diagnostic tests for superior cluneal neuralgia

	Potential distinguishing features
Physical exam	Symptoms of entrapment can be reproduced by palpation of the iliac crest ~ 6–7 cm lateral of the midline; hyperextension, lateral bending toward the side opposite the complaint, rotation of the lumbar spine due to traction of the quadratus lumborum muscle at the attachment of the iliac crest over the SCN
Diagnostic injection	A landmark-guided or fluoroscopic technique using a local anesthetic/steroid solution ~6–7 cm lateral of the midline
Ultrasound	The nerves appear as a small hypoechoic structure found just inferior to the border of the iliac crest
MRI	Not useful
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies	Not useful

inflammatory cell infiltration with consequential entrapment [12]. Idiopathic (spontaneous) mechanical irritation of the nerve secondary to severe myofascial pain syndrome to the quadratus lumborum muscle may also be a contributing factor [12].

Myofascial pain involving the quadratus lumborum muscle may perpetuate the irritation of the SCN and will need to be treated by trigger point injections and/or myofascial release during physical therapy.

Injection Technique

Landmark-Guided Technique

With the patient in a prone position, palpate the posterior wings of the iliac crest approximately 6–7 cm lateral from the midline (Fig. 51.6), and mark the area (Fig. 51.7). Using aseptic technique, a 1.5–2-inch 25- or 22-gauge needle is advanced perpendicularly to the marked area until the bone is contacted (Video 51.2). Following negative aspiration, inject 1 mL of a local anesthetic/steroid solution [21]. A peripheral nerve stimulator may also be used to assist in the proper site of injection.

Herring et al. [22] described using a landmark-guided superior cluneal nerve block to provide analgesia for a buttock abscess drainage.

Fluoroscopy-Guided Technique

Under fluoroscopy guidance, the point of maximum tenderness is usually seen at the medial iliac crest, and a traction spur of the attachment of the quadratus lumborum may be

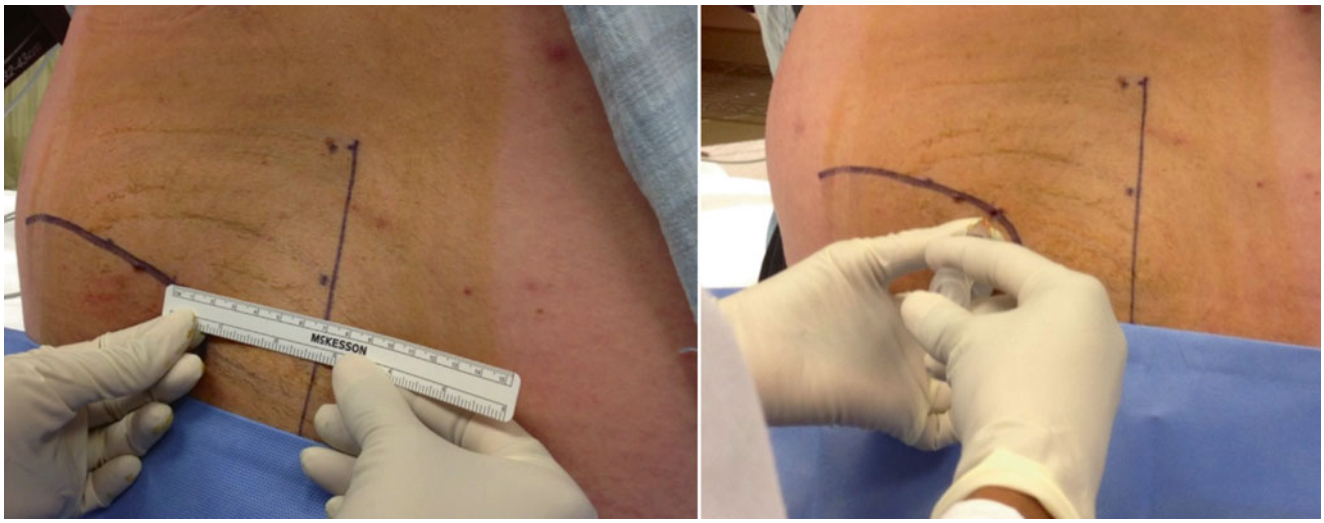

Fig. 51.7 Landmark-guided injection of the superior cluneal nerve (Image courtesy of Terri Dallas-Prunskis, MD)

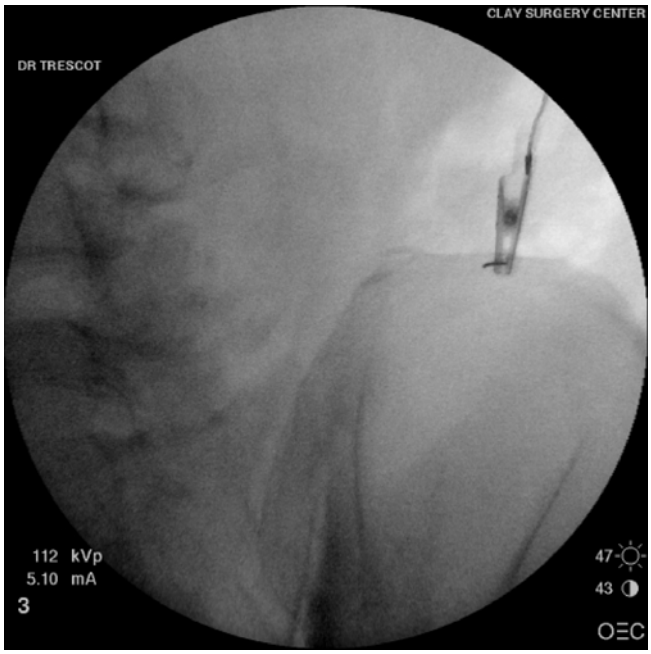

Fig. 51.8 Fluoroscopic injection of the superior cluneal nerve with peripheral nerve stimulator (Image courtesy of Andrea Trescot, MD)

visualized. Palpate the area to confirm the target. Using aseptic technique, 1 mL of a local/steroid solution can be injected at the site. Peripheral nerve stimulation can confirm the proper needle placement (Fig. 51.8) [23].

Ultrasound-Guided Technique (US)

The patient is placed in a prone position; noninvasive monitors are applied and intravenous access obtained if the patient requires it. Palpate the posterior wing of the iliac crest approximately 6–7 cm lateral to the midline to determine the point of maximum tenderness; mark the area. Skin disinfection is then performed over the iliac crest area.

For this superficial technique, a 7–12 MHz high-frequency linear array probe is placed at the marked site on the iliac crest in a transverse view. The iliac crest casts a bony shadow on the US image. The US probe is moved superior and posterior from this point. An in-plane or out-of-plane approach may be used. The nerves are identified as small hypoechoic structures found just inferior of the border of the iliac crest (Fig. 51.9). As it is a superficial structure, an in-plane needle approach may be easiest, with a shallow angle of approach. The skin is infiltrated with lidocaine and the injection needle is inserted to reach the desired site immediately inferior to the iliac crest. Using US guidance, the SCN can be blocked with 1 mL of a local anesthetic/steroid solution, and a peripheral nerve stimulator may assist in confirmation of the appropriate nerve.

Lieba-Samal et al. [24] used high-resolution US to identify the medial SCN (mSCN) on 14 cadavers, which they confirmed after injection of dye under US followed by dissection. They described starting the US scan above the gluteus maximus muscle and then moving the linear probe proximally until the insertion of the gluteus medius muscle and the posterior edge of the iliac crest become visible, which will be the site of the most medial branch of the SCN. They then would trace the nerve proximally until it entered the *autochthone* muscles of the back. At this point, they noted that the mSCN was long and "wavy," which they attributed to the need for slack in the nerve due to range of motion of the lumbar spine (Fig. 51.10). Using the same technique, they identified and injected under US guidance nine patients with mSCN entrap-

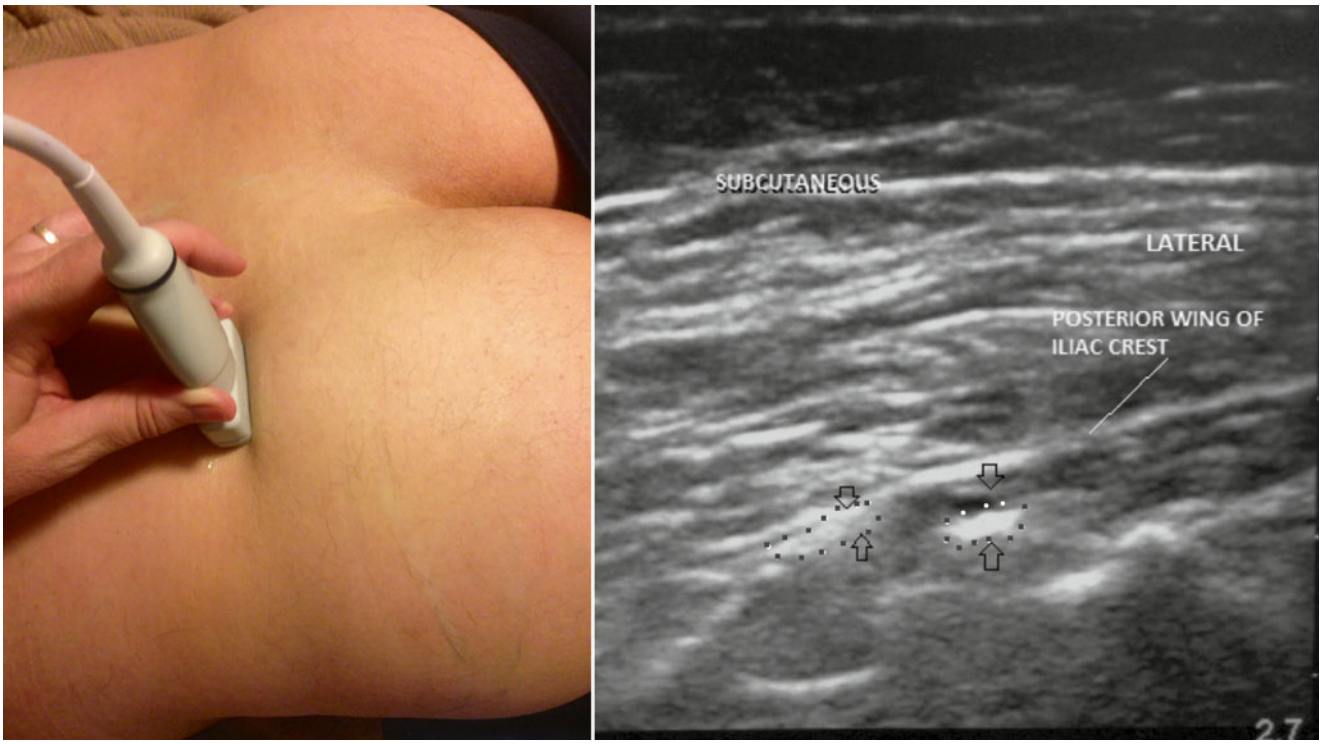

Fig. 51.9 Ultrasound image of the superior cluneal nerve. *Arrows and outline* = superior cluneal nerves (Image courtesy of Terri Dallas-Prunskis, MD)

ment; all the patients had numbness and temporary pain relief after the injection. Interestingly, two of these patients had been originally diagnosed with "sacroiliitis," but had failed SI joint and facet joint injections.

Acupuncture

Electroacupuncture is a common technique in traditional Chinese medicine. Place an acupuncture needle at the apex of the iliac crest on the affected side and a second needle at an auxiliary point (GB 34) on the affected side. After the needling sensation is elicited, the handles of the needles are connected to a stimulator apparatus. The stimulation should be a continuous wave at a frequency of 80 cycles/min with a pulse intensity tolerable to the patient. Treatment should be given once every other day, 30 min each time, with ten sessions constituting one course of treatment [25].

A study for superior cluneal nerve injury was performed utilizing triple acupuncture needling with massage applied alternately once every other day, with eight sessions constituting one course of treatment. Of the 67 cases in this study, 56 (83.58 %) of patients reported that the cluneal pain disappeared completely, and in 9 cases (13.43 %), the local pain and radiating pain disappeared, except for some dull pain on movement. The total effective rate was 97.1 % [16].

Neurolytic Techniques

If pain relief is complete but only temporary, longer-lasting modalities may include cryoneuroablation, radiofrequency lesioning, and chemical neurolysis with a neurolytic agent and/or surgery.

Cryoneuroablation

Cryoneuroablation may be performed at the quadratus lumborum attachment on the iliac crest at the site that was previously injected. The procedure may be performed with or without fluoroscopy or ultrasound.

With the patient in a prone position, utilizing aseptic technique, a small amount of local anesthetic with epinephrine 1:200,000 is infiltrated subcutaneously using a 25-gauge 1.5-in. needle. A small incision is made into the skin, and an I.V. introducer (size 12 or 14 gauge, depending on the probe size) is advanced to the target area. The stylet is removed, and the cryoprobe is advanced through the catheter (Fig. 51.11). Expose the tip of the probe by withdrawing the catheter back into the subcutaneous tissues. Confirm probe placement with maximal sensory stimulation and lack of motor stimulation. This should be followed by a series of three 2-min freezes, with 30-s defrosting between each cycle. There may be burning pain which replicates the initial pain on initiation of the

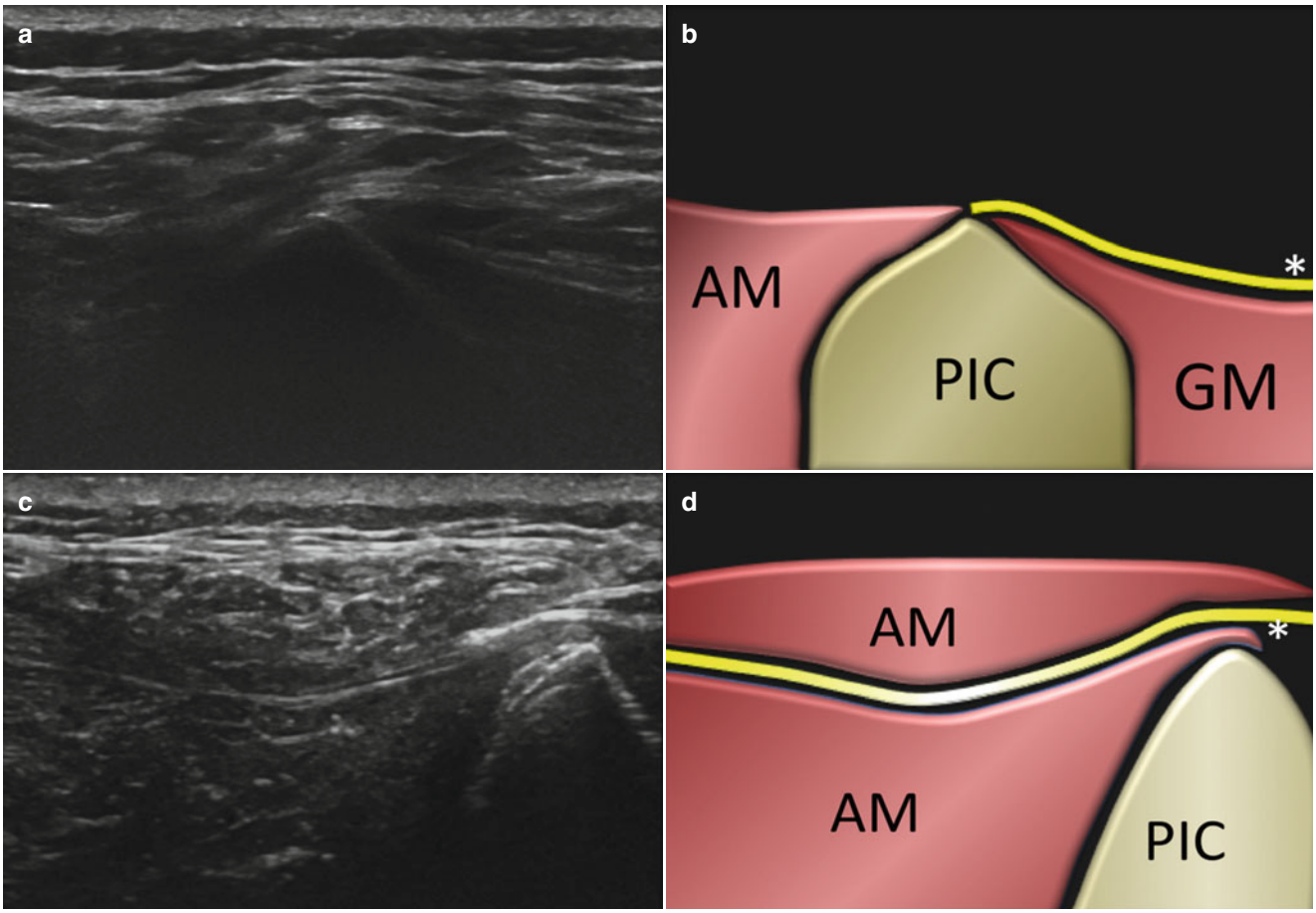

Fig. 51.10 Composite of transverse ultrasound images of the superior cluneal nerve below and above the iliac crest. *A* ultrasound image below the iliac crest, *B* schematic of the ultrasound image below the iliac crest, *C* ultrasound image above the iliac crest, *D* schematic of the ultrasound image above the iliac crest. *AM* autochthome muscles, *GM* gluteus medius muscle, *PIC* posterior superior iliac crest, * = medial superior cluneal nerve (From Lieba-Samal et al. [24]. Reprinted with permission from American Society of Interventional Pain Physicians)

first freeze cycle, but this should resolve within approximately 30 s [4].

Radiofrequency Lesioning

Radiofrequency lesioning (RF) has been utilized for extended relief of superior cluneal neuropathy following a successful diagnostic injection. The target is identified and the site sterilely prepped appropriately. After the skin is anesthetized subcutaneously, the radiofrequency cannula is advanced to the target site at the iliac crest using fluoroscopy or anatomical landmarks. After the radiofrequency probe is advanced through the cannula appropriately, maximal sensory stimulation and lack of motor stimulation are used to confirm that the tip of the probe is placed adequately. Either pulsed or conventional radiofrequency lesioning may be used [26], though the use of conventional RF for large, myelinated nerves such as the SCN would be likely to cause neuromas (see Chap. 8).

Chemical Neurolysis with Neurolytic Agents

Mahli et al. [2] experimented with the effectiveness of alcohol neurolysis in the treatment of persistent pain caused by the entrapment of superior cluneal neuropathy. At the beginning of the neurolysis procedure, 1 mL of 0.5 % lidocaine was injected into the injured branch of the SCN. If and when resolution of pain was observed, 1 mL of absolute alcohol (100 %) was injected via the same cannula. If after 1 week the pain was not resolved, the procedure was repeated. The study patients were observed up to 4 years; no one reported any problems or recurrent pain complaints.

Surgery

Surgery may be considered when less invasive procedures fail to control the patient's symptoms. Berthelot et al. [14] reported dramatic pain relief in a patient with a superior cluneal entrapment after surgical release of a large dorsal ramus

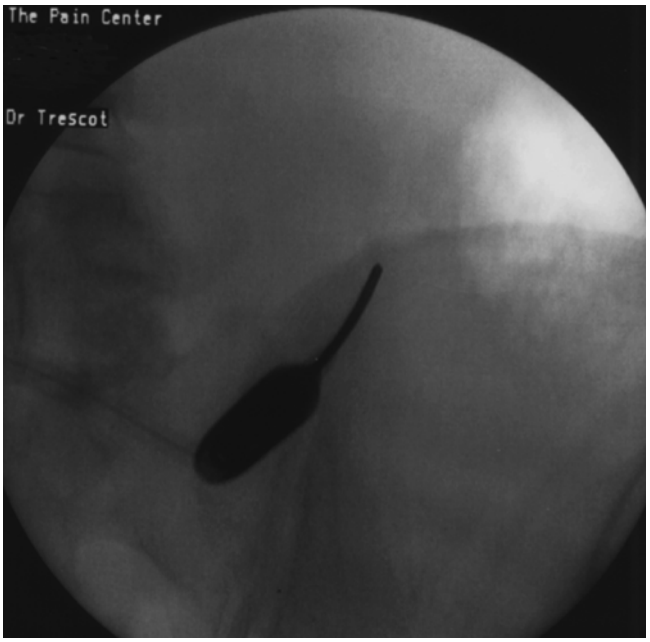

Fig. 51.11 Cryoneuroablation of the superior cluneal nerve (Image courtesy of Andrea Trescot, MD)

entrapped by the rigid opening between the upper rim of the iliac crest and the thoracolumbar fascia. Another group of authors described the surgical release of the SCN under local anesthetic; in all 34 patients, the SCN penetrated the orifice of the thoracolumbar fascia and could be released by dissection of the fascia [27].

Complications

Precautions related to anticoagulant therapy and proximity to vascular structures and other cavities should be taken. General complications are based on the location of the needle placement, neural trauma, and hematoma formation. Infectious complications include abscess and side effects related to the administration of local anesthetic and/or steroids and other drugs. Caution must be exercised when performing this procedure blindly. The needle should come into contact with the bone so as not to place the needle too deeply.

When performing steroid injections the risk of depigmentation or hyperpigmentation at the injection site, alopecia, and neuritis has been reported [28], though not for this specific nerve. Cryoneuroablation, in general, could be associated with depigmentation or hyperpigmentation as well as skin necrosis in this superficial location..

The most common complications of radiofrequency include those related to the placement of the needle and those related to the neurolysis. The majority of problems are short-lived and self-limited. These include local swelling and pain at the site of the needle insertion. Other reported complications of radiofrequency thermoneurolysis include a worsening of the usual pain, burning or dysesthesias, decreased sensation, and allodynia over the skin [26].

Injectable chemoneurolytic agents are fluids and can spread to unintended locations, leading to potentially devastating consequences. Because these medications are designed to destroy tissue, it is not surprising that tissue sloughing and infarcts are potential complications. Clearing the needle prior to withdrawal may decrease the risk of superficial tissue damage. Local pain at the injection site is to be expected but should resolve after a few days; prolonged pain at the injection site may represent partial neurolysis or neuritis.

The use of fluoroscopy or US or peripheral nerve stimulator is strongly encouraged for accurate needle placement.

Summary

Although considered a relatively rare cause of low back pain, superior cluneal nerve entrapment is likely under-recognized and therefore undertreated. Having the patient point to the site of pain can help to diagnose this treatable cause of low back pain.

References

1. Frymoyer JW, Howe J, Kuhlmann D. The long-term effects of spinal fusion on the sacroiliac joints and ilium. Clin Orthop Relat Res. 1978;134:196–201.
2. Mahli A, Coskun D, Altun NS, Simsek A, Ocal E, Kostekci M. Alcohol neurolysis for persistent pain caused by superior cluneal nerves injury after iliac crest bone graft harvesting in orthopedic surgery: report of four cases and review of the literature. Spine. 2002;27(22):E478–81.
3. Delawi D, Dhert WJ, Castelein RM, Verbout AJ, Oner FC. The incidence of donor site pain after bone graft harvesting from the posterior iliac crest may be overestimated: a study on spine fracture patients. Spine. 2007;32(17):1865–8.
4. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
5. Talu GK, Ozyalcin S, Talu U. Superior cluneal nerve entrapment. Reg Anesth Pain Med. 2000;25(6):648–50.
6. Akbas M, Yegin A, Karsli B. Superior cluneal nerve entrapment eight years after decubitus surgery. Pain Pract. 2005;5(4):364–6.
7. Massey EW. Cluneal neuropathy. Anesth Analg. 1978;57(3):381–2.
8. Strong EK, Davila JC. The cluneal nerve syndrome; a distinct type of low back pain. Ind Med Surg. 1957;26(9):417–29.
9. Aly TA, Tanaka Y, Aizawa T, Ozawa H, Kokubun S. Medial superior cluneal nerve entrapment neuropathy in teenagers: a report of two cases. Tohoku J Exp Med. 2002;197(4):229–31.
10. Ermis MN, Yildirim D, Durakbasa MO, Tamam C, Ermis OE. Medial superior cluneal nerve entrapment neuropathy in military personnel; diagnosis and etiologic factors. J Back Musculoskelet Rehabil. 2011;24(3):137–44.
11. Woodburne RT. Essentials of human anatomy. Oxford: Oxford University Press; 1957.

12. Maigne JY, Lazareth JP, Guerin Surville H, Maigne R. The lateral cutaneous branches of the dorsal rami of the thoraco-lumbar junction. An anatomical study on 37 dissections. Surg Radiol Anat. 1989;11(4):289–93.
13. Lu J, Ebraheim NA, Huntoon M, Heck BE, Yeasting RA. Anatomic considerations of superior cluneal nerve at posterior iliac crest region. Clin Orthop Relat Res. 1998;347:224–8.
14. Berthelot JM, Delecrin J, Maugars Y, Caillon F, Prost A. A potentially underrecognized and treatable cause of chronic back pain: entrapment neuropathy of the cluneal nerves. J Rheumatol. 1996;23(12):2179–81.
15. Maigne R. Low back pain of thoracolumbar origin (T11-T12-L1). In: Maigne R, editor. Diagnosis and treatment of pain of vertebral origin. 2nd ed. Boca Raton: Tayloe and Francis Group; 2006. p. 289–98.
16. Wanshun S, Jing L. Treatment for injury of superior cluneal nerves by triple puncture needling with massage. J Tradit Chin Med. 2002;22(1):24–5.
17. Kuniya H, Aota Y, Saito T, Kamiya Y, Funakoshi K, Terayama H, et al. Anatomical study of superior cluneal nerve entrapment. J Neurosurg Spine. 2013;19(1):76–80.
18. McKain CW, Urban BJ. Pain and cluneal neuropathy following intragluteal injection. Anesth Analg. 1978;57(1):138–41.
19. Kaufman MS, Domroese ME. Peripheral nerve injuries of the proximal lower limb in athletes. In: Akuthota V, Herring SA, editors. Nerve and vascular injuries in sports medicine. New York: Springer; 2009. p. 161–70.
20. Lu J, Ebraheim NA, Huntoon M, et al. Anatomic considerations of superior cluneal nerve at posterior iliac crest region. Clin Ortho Rel Res. 1998;347:224–8.
21. Maigne JY, Doursounian L. Entrapment neuropathy of the medial superior cluneal nerve. Nineteen cases surgically treated, with a minimum of 2 years' follow-up. Spine (Phila Pa 1976). 1997;22(10):1156–9.
22. Herring A, Price DD, Nagdev A, Simon B. Superior cluneal nerve block for treatment of buttock abscesses in the emergency department. J Emerg Med. 2010;39(1):83–5.
23. Trescot A. Pseudosciatica. Pain Clin. 2001;3(6):21–5.
24. Lieba-Samal D, Bodner G, Platzgummer H, Meng S, Brugger PC, Gruber G. Successful identification and treatable cause of chronic back pain: entrapment neuropathy assessment of the cluneal nerves. J Rheumatol. 1996;23(12):2179–81.
25. Yuzhen T. Eighty cases of injury of the superior cluneal nerve treated by electroacupuncture. J Tradit Chin Med = Chung i tsa chih ying wen pan/sponsored by All-China Assoc Trad Chin Med Acad Tradit Chin Med. 2000;2(20):132–3.
26. Bogduk N. Pulsed radiofrequency. Pain Med. 2006;7(5):396–407.
27. Morimoto D, Isu T, Kim K, Imai T, Yamazaki K, Matsumoto R, et al. Surgical treatment of superior cluneal nerve entrapment neuropathy. J Neurosurg Spine. 2013;19(1):71–5.
28. Shields KG, Levy MJ, Goadsby PJ. Alopecia and cutaneous atrophy after greater occipital nerve infiltration with corticosteroid. Neurology. 2004;63(11):2193–4.

Inferior Cluneal Nerve Entrapment: Low Back

52

Terri Dallas-Prunskis

Introduction

Neuropathic ischial pain is often associated with a sciatic neuropathy. However, some patients present with pain located on the caudal and medial parts of the buttocks, and the upper part of the posterior thigh, including the scrotum or the labia majora. These pains extend beyond the territory of the sciatic nerve. The inferior cluneal nerve, which emerges from the posterior femoral cutaneous nerve, has some branches innervating the ischium as well as the perineum. These areas of overlapping symptoms must be clearly defined in order to determine the etiology of the pain.

Clinical Presentation (Table 52.1)

Patients with inferior cluneal nerve (ICN) entrapment complain of a burning, tingling, or numbness sensation along the inferior and medial aspect of the buttocks (Fig. 52.1) and/or along the dorsal and proximal thigh, as well as the lateral anal margin and the skin of the scrotum or labia majora (Fig. 52.2). Pain will increase with sitting on hard surfaces, such as chairs or bicycle seats.

Anatomy (Table 52.2)

The cluneal nerves are divided into three groups: the *superior cluneal nerve* (see Chap. 51), the *middle cluneal nerve (lateral branches of the sacral nerves)*, and the *inferior or lateral cluneal nerves* (Fig. 52.3). The ICN arises from the inferior portion of the *posterior femoral cutaneous nerve of the thigh* (PFCN) (see Chap. 56). This nerve is made up of sensory branches of S1, S2, and S3, traveling parallel with the sciatic nerve and the pudendal nerve through the sciatic notch. After reaching the subgluteal area, the PFCN gives rise to the *inferior cluneal branch* and the *perineal branch* (Fig. 52.4). The nerves then go to the inferior edge of the gluteus maximus muscle, course in front of it, then circumvent, and have a recurrent course behind the muscle. The

Table 52.1 Occupation/exercise/trauma history relevant to inferior cluneal entrapment

Trauma	Fall onto buttocks; hamstring injury; intramuscular injection into gluteal muscle; piriformis trauma or injury
Direct compression of nerve	Sitting on a hard seat or bicycle riding
Myofascial compression	Piriformis spasm; gluteal muscle spasm

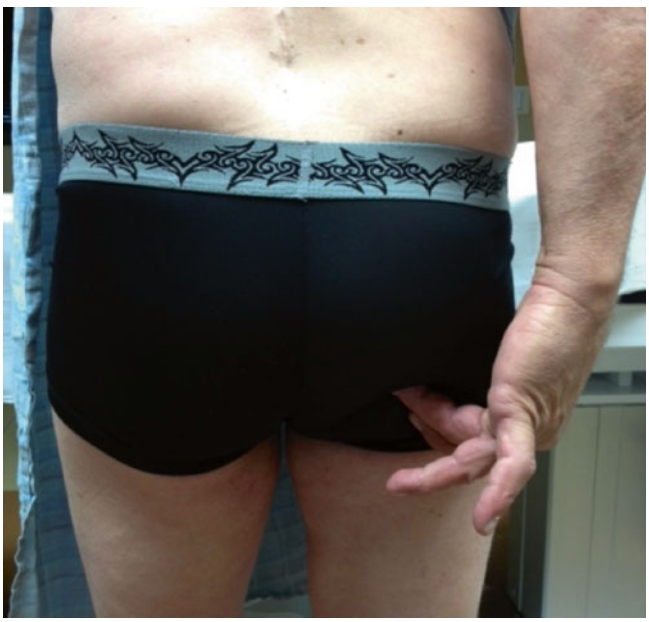

Fig. 52.1 Pain location from inferior cluneal neuralgia (Image courtesy of Terri Dallas-Prunskis, MD)

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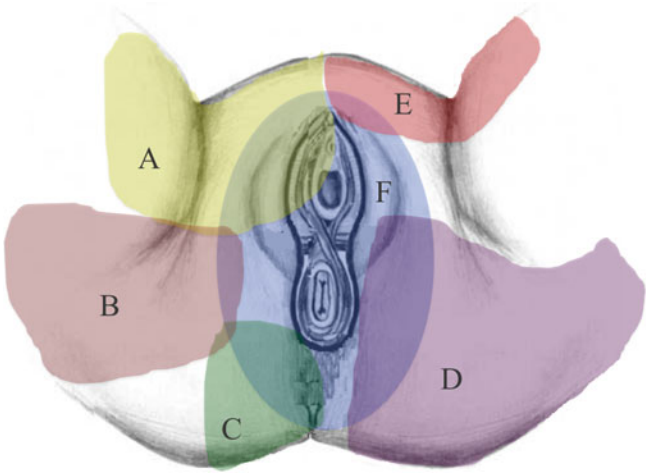

Fig. 52.2 Innervation of perineum: (*A*) genitofemoral nerve; (*B*) obturator nerve; (*C*) inferior cluneal nerve; (*D*) perineal branch of the posterior femoral cutaneous nerve, (*E*) ilioinguinal nerve, and (*F*) pudendal nerve (Image inspired by Hibner et al. [7], courtesy of Andrea Trescot, MD)

Table 52.2 Inferior cluneal nerve anatomy

Origin	The nerves arise from the inferior portion of the posterior femoral cutaneous nerve of the thigh
General route	The posterior femoral cutaneous nerve is made up of the sensory branches of S1, S2, and S3. It travels through the sciatic notch and after reaching the subgluteal area gives rise to the inferior cluneal branch and the perineal branches. The nerves then go to the inferior edge of the gluteus maximus, anterior to it, then circumvent, and have a recurrent course behind the muscle, innervating its target areas
Sensory distribution	Provides cutaneous innervation to the inferior part of the buttocks, lateral anus region (but not the anus), and the lateral region of the labium majorum
Motor innervation	None
Anatomic variability	Only a few variations are noted in the literature, giving a description of the inferior cluneal nerves going through the gluteus maximus muscle; other sources indicate that the nerves reach the caudal edge of the gluteus maximus, then circumvents it at various levels

inferior cluneal nerves provide cutaneous innervation to the inferior part of the buttocks, lateral anus region (but not the *anus* itself), and the lateral region of the *labium majorum*, but not the *labia minora*, nor the vagina. It also does not innervate the *penis* or *clitoris* (Fig. 52.2) [1].

Entrapment

There are two common areas where entrapment may occur. The first one occurs at the site of the passage of the perineal ramus under the ischium to the perineum (Site A on Fig. 52.5). This entrapment may be due to nerve compression by the ischium on the gluteus maximus and the hamstring muscles in a sitting position and stretching of the perineal ramus in internal rotation of the thigh.

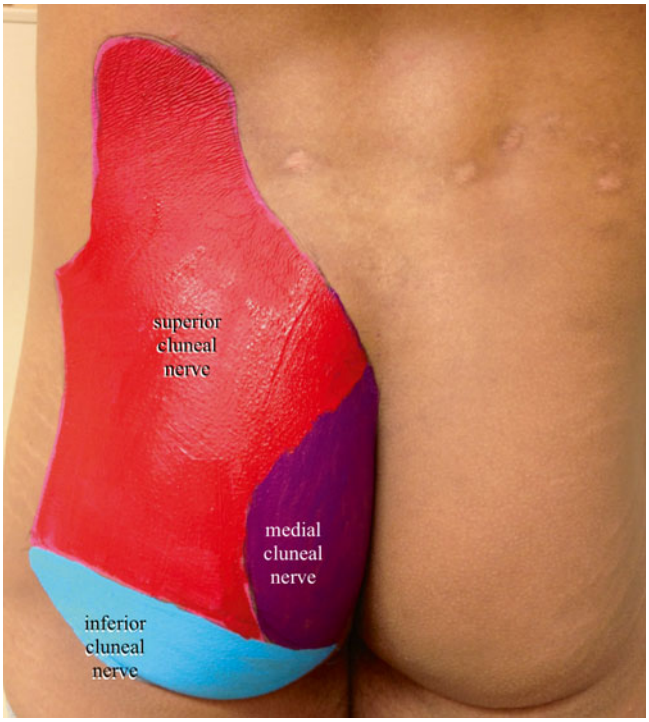

Fig. 52.3 Cutaneous distribution of the cluneal nerves (Image courtesy of Terri Dallas-Prunskis, MD)

The second site of entrapment is more proximal, at the level of the sciatic spine and the piriformis. At this point, the roots of the posterior femoral cutaneous nerve, which gives rise to the inferior cluneal nerve, may be compressed by the piriformis against the sciatic spine (Site B on Fig. 52.5). However, whatever the etiology, it is the sitting position that triggers the entrapment, giving inferior cluneal entrapment syndrome the same general appearance as a pudendal syndrome or ischial bursitis [2].

Physical Examination

The examination should begin by evaluating the entire back to rule out other causes for the pain. Palpation should elicit non-radiating pain, increasing with deep pressure over the sciatic notch. Palpate the inferior edge of the gluteus maximus between the ischium and the greater trochanter (Fig. 52.6). Hyperesthesia to pin scratch and decreased sensation to touch over the inferior buttocks corresponding to the distribution of the inferior cluneal nerve will also be noted. There is no motor involvement. Tenderness will be slightly lateral to the most inferior portion of the ischium.

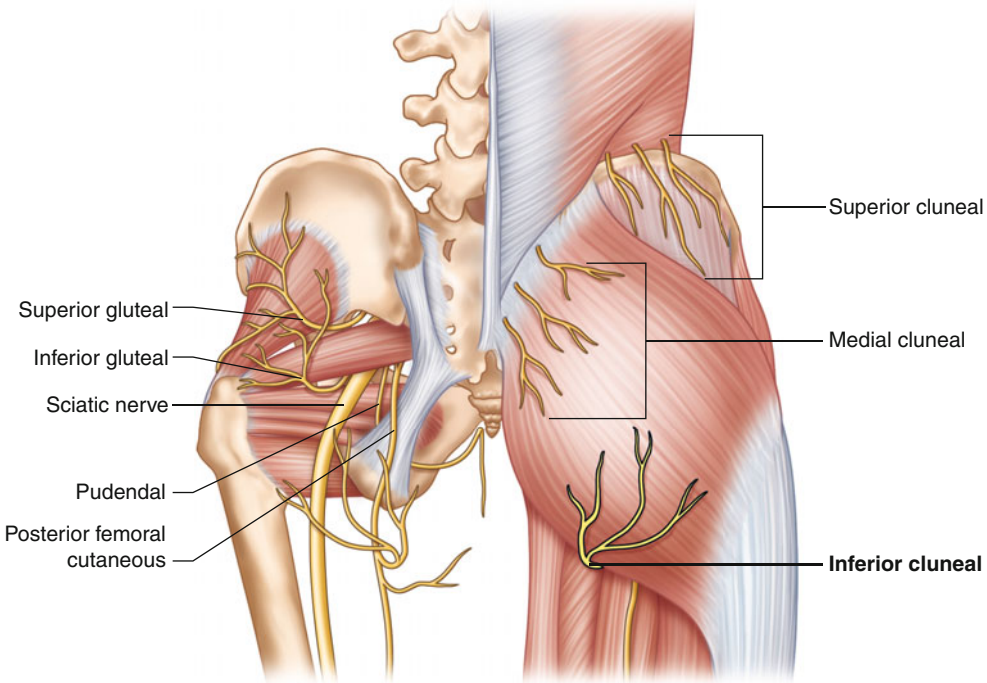

Fig. 52.4 Anatomy of the buttock nerves (Image courtesy of Springer)

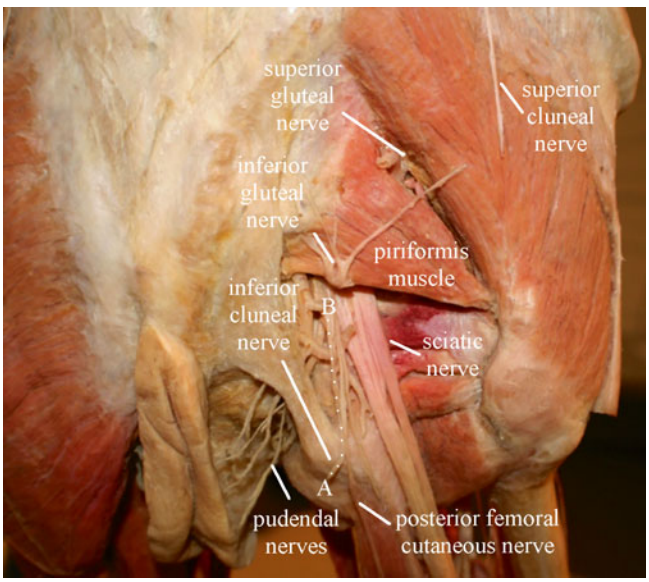

Fig. 52.5 Gluteal muscle dissection modified from an image from *Bodies*, *The Exhibition*, with permission. Inferior cluneal nerve (*white dots*) entrapment sites: *A* distal entrapment, *B* proximal entrapment (Image courtesy of Andrea Trescot, MD)

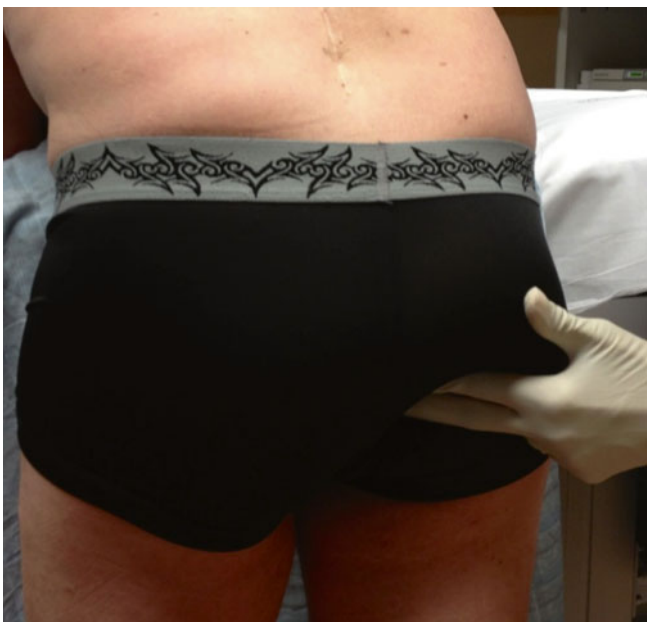

Fig. 52.6 Physical exam of the inferior cluneal nerve; palpation over the inferior cluneal nerve near the sciatic notch (Image courtesy of Terri Dallas-Prunskis, MD)

Differential Diagnosis (Table 52.3)

Inferior cluneal nerve entrapment must be differentiated from other lower limb pain disorders of the *sciatic nerve*, *posterior femoral cutaneous nerve*, or *obturator nerve* (*see* Chaps. 62, 64, and 65) caused by reflex muscle contraction of the piriformis and obturator internus muscles, inducing "irritation" of these nerve trunks. This entrapment is also misdiagnosed as *pudendalgia* (*pudendal canal syndrome*) [3] Table 52.4.

Table 52.3 Differential diagnosis of buttock pain

	Potential distinguishing features
Lower limb pain disorders	
Sciatica	Weakness, numbness, or difficulty moving the leg or foot
Posterior femoral cutaneous neuritis	Innervates the lateral and lower portions of the gluteus maximus muscle and the posterior parts of the leg and thigh; the skin of the perineum
Obturator neuritis	Medial thigh or groin pain, weakness with leg adduction, and sensory loss in the medial thigh
Piriformis syndrome	Pain, tingling, and numbness in the buttocks and along the path of the sciatic nerve, descending down the lower thigh and into the leg; hypertonicity of the piriformis muscle
Obturator internus muscle spasm	Difficulty with lateral rotation of the femur with hip extension and abduction of the femur with hip flexion; instability of the femoral head in the acetabulum; hypertonicity of the obturator internus muscle

Table 52.4 Diagnostic tests for inferior cluneal neuralgia

	Potential distinguishing features
Physical exam	Palpation should elicit non-radiating pain, increasing with deep pressure over the sciatic notch; hyperesthesia to pin scratch and decreased sensation to touch over the inferior buttocks corresponding to the nerve distribution; tenderness will be slightly lateral to the most inferior portion of the ischium
Diagnostic injection	Utilizing landmark or fluoroscopic technique, local anesthetic/steroid solution may be injected through the gluteus maximus directed toward the lateral and inferior edge of the ischium
Ultrasound	The nerves are located by identifying the inferior border of the ischium, the gluteus maximus muscle, and the lateral edge of the hamstring muscle insertion; the block needle is placed on the lateral and inferior edges of the ischium
MRI	Not useful
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies	Not useful

Treatment of Contributing/Perpetuating Factors

The ICN may be injured either by a fall onto the buttocks, by a hamstring injury, and sometimes it is not clear what caused the cluneal nerves to be symptomatic. Intramuscular injection into the medial inferior quadrant of the buttocks leading to muscle spasm, myositis, and nerve entrapment of nerves within the muscle has been reported. Mechanical damage may result to the inferior cluneal nerves during their course through the piriformis [4].

Sitting on a hard seat will increase the compression of the nerves in the buttocks or underneath the ischium [2]. Piriformis trauma or injury may also be causative.

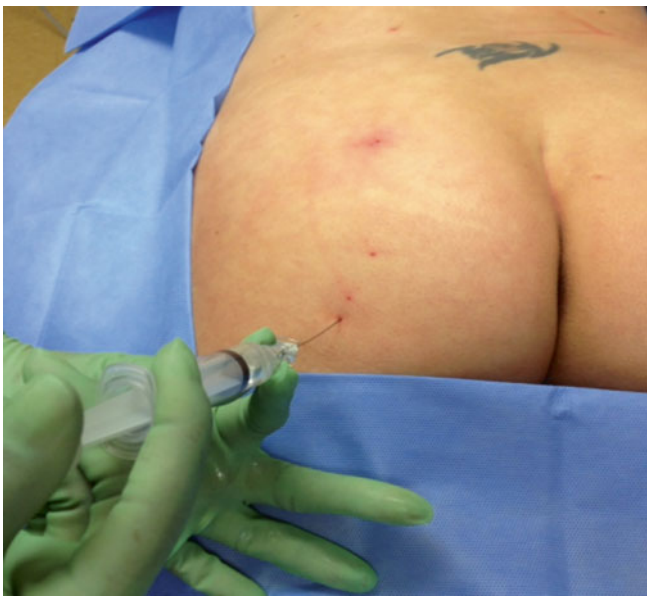

Fig. 52.7 Landmark-guided injection of the inferior cluneal nerve; needle is directed through the gluteus maximus, lateral, and inferior to the ischium (Image courtesy of Terri Dallas-Prunskis, MD)

Injection Technique

Landmark-Guided Technique

The procedure may be difficult to perform blindly and will depend on the ability to adequately palpate the patient's anatomy. The patient should be placed in a prone position or standing flexed securely over a cart/bed. Utilizing aseptic technique, prep the buttocks. Palpate the inferior aspect of the ischium, and mark the site. Next, localize the gluteus maximus muscle and the lateral edge of the hamstring muscle insertion. After local infiltration to the skin and subcutaneous tissue, insert a 22-gauge 3.5-in. block needle through the gluteus maximus on to the lateral edge of the hamstring muscles insertion (the lateral and inferior edges of the ischium) (Fig. 52.7). Following a negative aspiration, inject 2–3 mL of a local anesthetic/steroid solution. Utilizing peripheral nerve stimulation would be appropriate for this procedure.

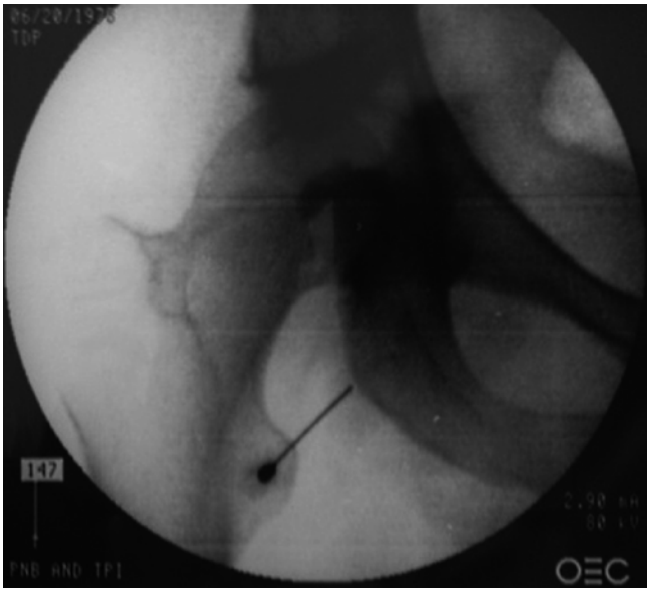

Fig. 52.8 Fluoroscopic injection of the inferior cluneal nerve (Image courtesy of Terri Dallas-Prunskis, MD)

Fluoroscopy-Guided Technique

With the patient in a prone position, utilizing the fluoroscopic image in an AP view, the lateral and inferior edges of the ischium are identified. Using aseptic technique, local infiltration with 1 % lidocaine is injected into the skin and subcutaneous tissue over the targeted point. Next, a 22-gauge 3.5-in. needle is inserted 1 cm laterally from the caudal edge of the ischium, which is under the gluteus maximus and on the lateral edge of the hamstring muscle insertion (Fig. 52.8). Following a negative aspiration, inject 2–3 mL of a local anesthetic/steroid solution. A peripheral nerve stimulator may also be used to confirm proper needle placement.

Ultrasound-Guided Technique

The patient is placed in a prone position. Noninvasive monitors are applied and intravenous access obtained, if the patient requires it. Palpate the inferior edge of the ischium. Skin disinfection is then performed over the gluteal area. A high-frequency (7–12 MHz) linear array probe is appropriate for this block. Either an in-plane or an out-of-plane approach may be used. Locate the inferior border of the ischium (the ischium casts a bony shadow on the US image), the gluteus maximus muscle, and the lateral edge of the hamstring muscle insertion (Fig. 52.9). The skin is infiltrated with lidocaine, and the block needle is inserted through the gluteus maximus on the lateral and inferior edges of the ischium. Using US guidance, the ICN can be blocked with 2–3 mL of a local anesthetic/steroid solution. Peripheral nerve stimulation can confirm the proper needle placement.

Neurolytic/Surgical Techniques

If there is temporary relief of pain after successful infiltrations, neurolytic or surgical techniques may be considered. Any neurolytic technique should be performed using a choice of imaging.

Cryoneuroablation

Cryoneuroablation may be performed at the lateral and inferior edges of the ischium, with the patient in a prone position. Utilizing an aseptic technique, a small amount of local anesthetic is infiltrated subcutaneously with a 25-gauge 1.5-in. needle. A small incision is made into the skin, and an IV introducer (size 12- or 14-gauge, depending on the probe size) is advanced to the target area. The stylet is removed, and the cryoprobe is then advanced through the catheter. Expose the tip of the probe by withdrawing the catheter back into the subcutaneous tissues. Probe placement should be confirmed with maximal sensory stimulation and the absence of motor stimulation. This should be followed by a series of three 2-min freezes, with 30 s of defrosting between each cycle. At the beginning of the first freeze cycle, the patient may experience burning pain which often replicates their original pain; this should resolve within approximately 30 s [5].

Radiofrequency Lesioning

Radiofrequency lesioning has also been utilized for extended pain relief of inferior cluneal neuropathies following successful infiltration. The patient is placed in a prone position, and utilizing imaging in an AP view, the lateral and inferior edges of the ischium are identified. Using aseptic technique, the skin is anesthetized subcutaneously, and the radiofrequency cannula advanced to the target site at the ischium. When the probe is thought to be at the correct site, sensory stimulation and the absence of motor response are used for confirmation. Either pulsed or conventional lesioning may be used [6].

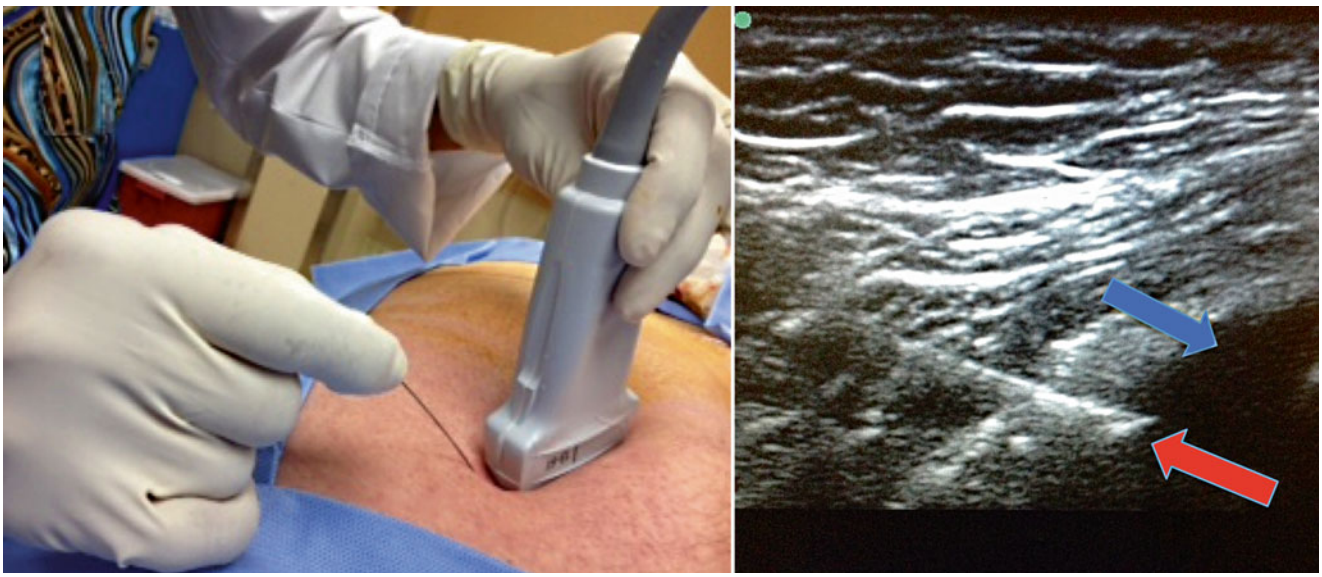

Fig. 52.9 Ultrasound image of the inferior cluneal nerve. *Blue arrow* = ischium; *red arrow* = needle injection (Image courtesy of Terri Dallas-Prunskis, MD)

Surgery

Surgery may be considered after infiltration provides improvement or temporary pain relief. Two surgical approaches have been discussed in the literature. A transgluteal approach for decompression and transposition has been described for *clunealgia* associated with *pudendalgia*, caused by a piriformis syndrome [3]. The second approach on the dorsal and cranial parts of the thigh is recommended for isolated clunealgia with an under-and-into ischiatic entrapment [2].

Complications

General complications may occur based on the location of the needle placement, neural trauma, and hematoma formation. Infectious complications include abscess and side effects related to the administration of local anesthetic and/or steroids and other drugs. Caution must be exercised when performing the procedures blindly. The bone should be contacted so as not to place the needle too deeply.

Neuritis and depigmentation or hyperpigmentation at the cryolesion site have been reported after cryoneuroablation [5].

The most common complications of radiofrequency include those related to the placement of the needle and to the neurolysis. Most of these problems are short-lived and self-limited and include local swelling and pain at the site of the needle insertion, as well as somatic pain from the site of insertion. Other reported complications of radiofrequency thermoneurolysis include a worsening of the usual pain, burning or dysesthesias, decreased sensation, and allodynia over the skin [6].

Summary

The inferior cluneal nerve is a cause of both a low back pain and pelvic pain. It is rarely diagnosed and even more rarely taught. Understanding the clinical presentation and the physical exam perhaps will increase awareness of the diagnosis and therefore increase treatment of this potentially debilitating problem.

References

1. Netter FH. Atlas of Human Anatomy. Bsael, Switzerland: Novartis Medical Education; 1997. p. 468–509.
2. Darnis B, Robert R, Labat JJ, Riant T, Gaudin C, Hamel A, et al. Perineal pain and inferior cluneal nerves: anatomy and surgery. Surg Radiol Anat. 2008;30(3):177–83.
3. Robert R, Prat-Pradal D, Labat JJ, Bensignor M, Raoul S, Rebai R, et al. Anatomic basis of chronic perineal pain: role of the pudendal nerve. Surg Radiol Anat. 1998;20(2):93–8.
4. Hanson D. Intramuscular injection injuries and complications. Gen Pract. 1963;27:109–15.
5. Trescot AM. Cryoneuroablation. In: Manchikanti L, Singh V, editors. Interventional techniques in chronic non-spinal pain. Paducah: ASIPP Publishing; 2009. p. 69–86.
6. Trescot A, Hansen H. Radiofrequency neurolysis. In: Manchikanti L, Singh V, editors. Interventional techniques in chronic non-spinal pain. Paducah: ASIPP Publishing; 2009. p. 59–68.
7. Hibner M, Desai N, Robertson LJ, Nour M. Pudendal neuralgia. J Minim Invasive Gynecol. 2010;17(2):148–53.

Superior Gluteal Nerve Entrapment

53

Andrea M. Trescot

Introduction

The superior gluteal nerve (SGN) entrapment is an often-missed cause of "pseudo-sciatica," mimicking a herniated disc. The SGN is a branch of the lumbar plexus. When piriformis and/or gluteus medius spasm injures it, the result can be pain going down the leg to the foot.

Clinical Presentation (Table 53.1)

The patient with superior gluteal neuralgia has often suffered a bending, lifting, or twisting injury; a fall on the buttock or a poorly placed IM injection can also injure the SGN. Other injuries might include forced internal rotation of the leg and extension of the hip under mechanical load (such as seen with the knee extended during a motor vehicle accident (MVA)).

These patients have pain in the lower back and buttock (Fig. 53.1), with vaguer pain down the back of the leg to the popliteal fossa and occasionally down to the foot (Fig. 53.2). These symptoms can be misdiagnosed as a lumbar herniated disc, "sciatica," and is sometimes termed "pseudo-sciatica" [9].

Patients generally experience pain from the buttocks to the foot with prolonged sitting, leaning forward, or twisting to the contralateral side. The pain increases with standing and walking and decreases with sitting. They have difficulty getting out of a chair or climbing stairs, and weak abduction

Table 53.1 Occupation/exercise/trauma history relevant to superior gluteal nerve entrapment

Trauma	Fall on the buttocks
	Twisting, bending injury
	Forced extension of the hip, during MVA
	Poorly placed IM injection [1]
	Iliac artery aneurysm [1]
	Piriformis hypertrophy [1]
	Pelvic pathology [2]
	Lumbar spondylolisthesis [3]
	Hip fracture [4]
Surgery	Injury during direct lateral hip surgery, with branches injured during anterior hip surgery [5]
	Injury during minimally invasive anterolateral approaches [6]
	Total hip replacement [7]
Entrapment	Piriformis hypertrophy [8]
	Vascular dilation

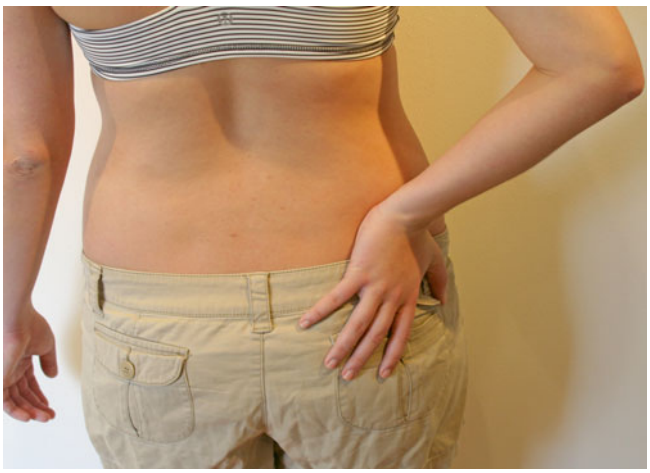

Fig. 53.1 Patient localization of superior gluteal nerve pain (Image courtesy of Andrea Trescot, MD)

Electronic supplementary material The online version of this chapter (doi:10.1007/978-3-319-27482-9_53) contains supplementary material, which is available to authorized users.

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of the affected hip causes a "waddling" gait. They sit with their weight on the contralateral buttock or cross their legs so as to minimize pressure on the involved side. Other reported symptoms include the inability to sit more than 20–30 min, limping, disturbed or loss of sensation in the affected extremity, "lumbago," and pain at night getting better during the day.

Fig. 53.2 Pain pattern associated with superior gluteal nerve entrapment (Image courtesy of Andrea Trescot, MD)

Anatomy (Table 53.2)

The SGN is formed from the posterior branches of the L4, L5, and S1 anterior rami, passes inferiorly through the *sciatic notch* above the *piriformis muscle* (Fig. 53.3), and turns superiorly to pass between the piriformis and *gluteus medius muscles* (Fig. 53.4). It lies deep to the *gluteus maximus muscle* and accompanies the *superior gluteal artery* and the large *superior gluteal vein* over the surface of the gluteus minimus muscle (Fig. 53.5). The upper branch of the SGN innervates the gluteus medius muscle, while the larger inferior branch innervates the gluteus medius and minimus muscles, as well as the *tensor fascia lata muscle* [10], which accompanies the lower branch of the deep division of the superior gluteal artery. The SGN also innervates the superior portion of the femoral neck. The SGN has no superficial sensory fibers.

In 1989, Jacobs and Buxton [11] dissected ten cadavers, and the SGN and its branches were identified bilaterally. They used the midpoint of the superior border of the *greater trochanter* as a reference point for measurements and described two patterns of neural branching. All the branches ended in an arcuate pattern along the middle one-third of the deep surface of the gluteus medius muscle. They described a "safe area" of the gluteus medius muscle about 5 cm medial to the greater trochanter: they thought that an intramuscular (IM) injection less than 5 cm from the trochanter would pose a minimal risk of injury to the superior gluteal nerve and its branches.

However, an anatomic study by Ince et al. [6] in 2007 found the branch of the superior gluteal nerve leading to the gluteal minimus muscle was 33 mm (range 20–50 mm) from the tip of the greater trochanter, within a deeper layer. The nearest point of the superior gluteal nerve branches from the tip of the greater trochanter in the posterior region was 19 mm (range 10–30 mm), 20 mm (range

Table 53.2 Superior gluteal nerve anatomy

Origin	Sacral plexus – posterior branches of L4, L5, and S1 nerves
General route	The SGN passes out from the pelvis through the greater sciatic notch above the piriformis, travels laterally between the gluteus minimus and gluteus medius muscles, and divides into superior and inferior branches
Sensory distribution	No superficial sensory fibers; sensory to superficial femoral neck
Motor innervation	Superior branch of SGN – gluteus medius muscle
	Inferior branch of SGN – gluteus medius, gluteus minimus, tensor fascia lata muscles
Anatomic variability	Multiple variations in branching pattern (see section "Anatomy")
Other relevant structures	SGN is accompanied by the superior gluteal artery and the very large superior gluteal vein

20–30 mm) in the middle region, and 20 mm (range 10–35 mm) in the anterior region. In half of the cases, they found a distal intermuscular branch between gluteal medius and tensor fascia lata muscle, 27 mm (range 10–40 mm) caudal and 38 mm (range 25–60 mm) ventral to the tip of the greater trochanter. This distal branch is considered to create a loop with upper branches of the SGN within the tensor fascia lata muscle, so they concluded that the "safe area" is smaller than had been previously thought.

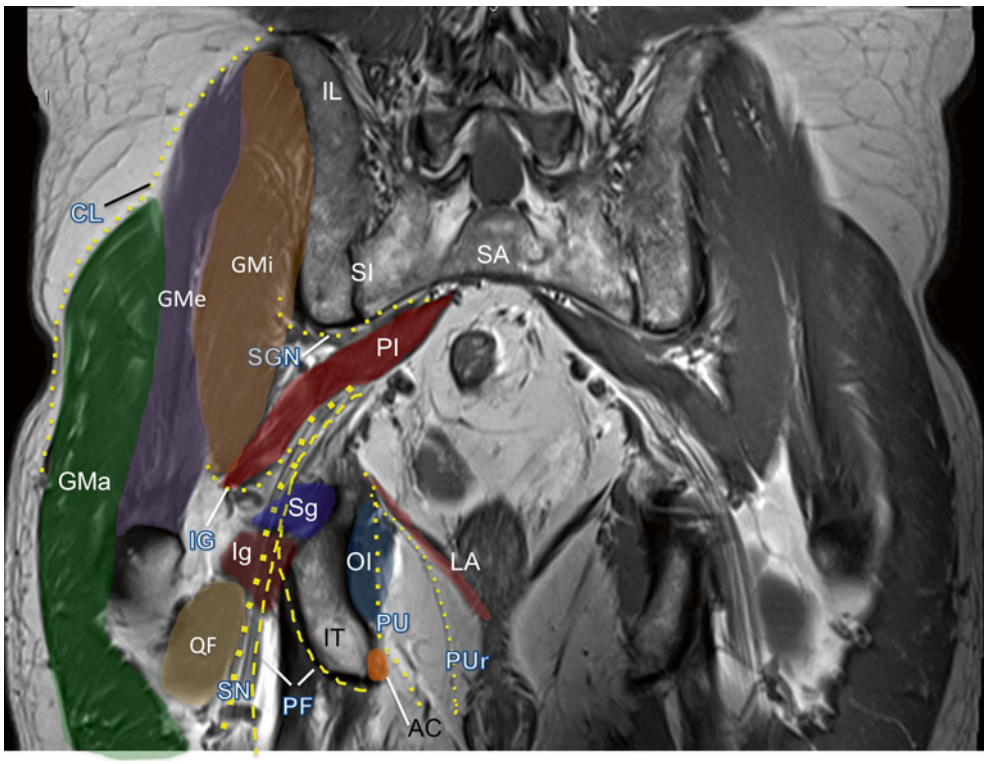

Fig. 53.3 MRI coronal images showing gluteal muscles and nerves. *AC* Alcock's canal, *CL* superior cluneal nerve, *IL* iliac crest, *IG* inferior gluteal nerve, *Ig* inferior gemellus muscle, *IT* ischial tuberosity, *Pi* piriformis, *Pu* pudendal, *Gmi* gluteus minimus, *Gme* gluteus medius, *Gma* gluteus maximus, *GT* greater trochanter, *LA* levator ani muscle, *OI* obturator internus muscle, *PF* posterior femoral cutaneous nerve, *PU* pudendal nerve, *PUr* pudendal nerve (rectal branch), *QF* quadratus femoris muscle, *SA* sacrum, *SGN* superior gluteal nerve, *Sg* superior gemellus muscle, *SI* sacroiliac joint, *SN* sciatic nerve (Image courtesy of Andrea Trescot, MD)

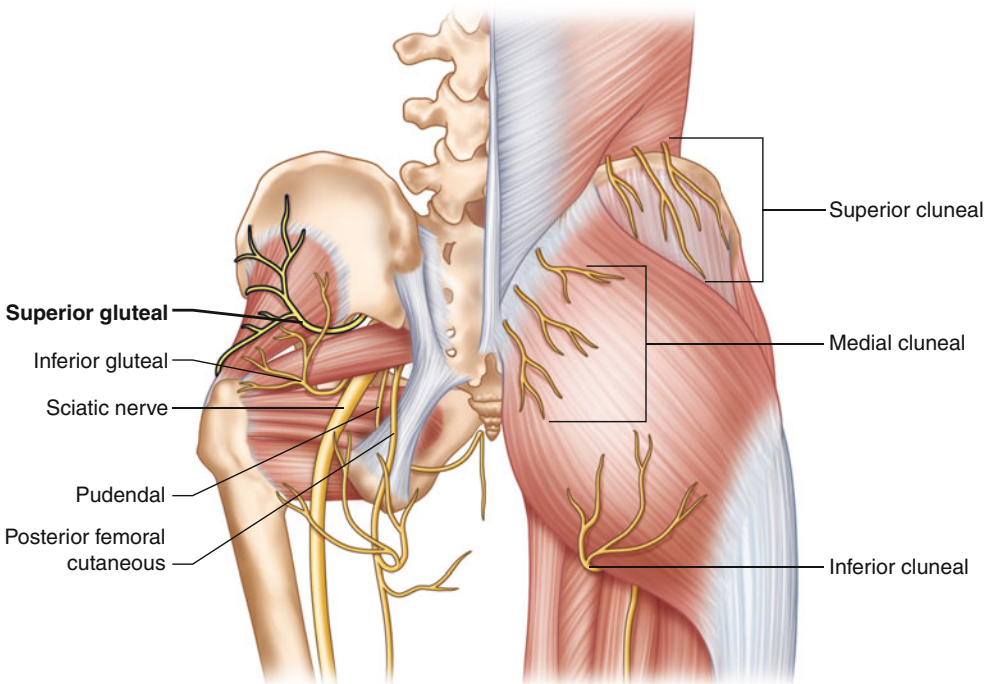

Fig. 53.4 Nerves of the buttocks (Image by Springer)

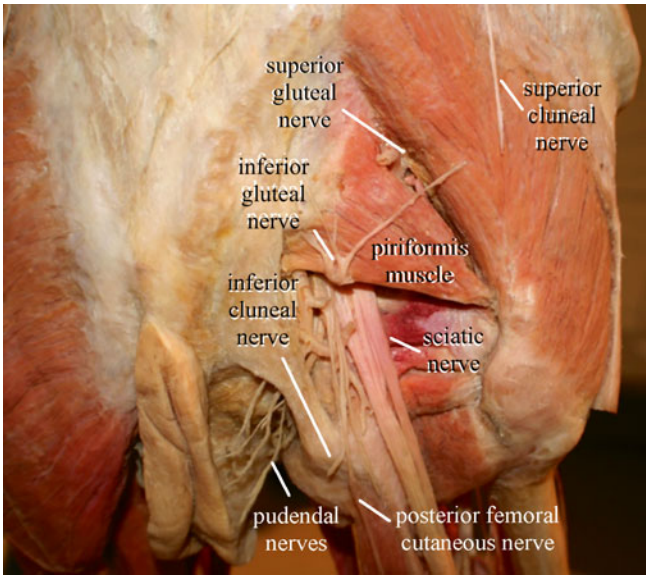

Fig. 53.5 Buttocks dissection modified from an image from *Bodies, The Exhibition*, with permission (Image courtesy of Andrea Trescot, MD)

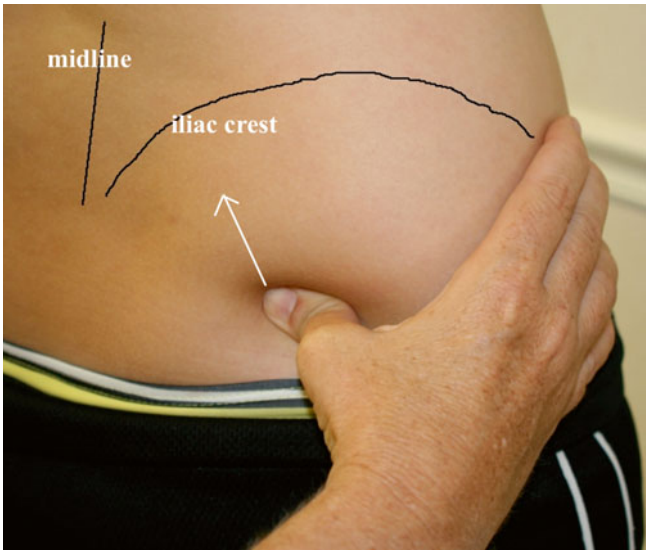

Fig. 53.6 Physical exam of the superior gluteal nerve (Image courtesy of Andrea Trescot, MD)

Entrapment/Injury

The SGN is injured by direct trauma, such as a fall on the buttock. It is also entrapped as it passes in the fascial plane between the gluteus medius and gluteus minimus musculature, as a result of shearing between the gluteal muscles with forced external rotation of the leg. It can also be injured with extension of the hip under mechanical load, as might occur in a head-on automobile collision, where the foot is pressed against the automobile floorboards with the knee in extension, as the patient anticipates impact. Lumbar lordosis and internal rotation of the hip put tension on the piriformis muscle, which then traps the SGN between the piriformis, gluteus minimus, and ilium, creating a viscious cycle, with edema of the nerve from the entrapment creating more pressure injury [2]. Entrapment can also occur at the superior edge of the piriformis muscle.

Rask [3] described a patient who had a SGN entrapment after a fall on the ice; she described an aching pain in the buttocks and a paresis of the hip abductors. She then had a second fall (felt to be due to the abductor weakness) and fractured her hip. Surgical exploration showed entrapment of the SGN in a tunnel formed by the anterior-superior fibers of the piriformis, the inferior border of the gluteus minimus, and the ilium.

The nerve can also be injured by IM injections. According to dissections done by Jacobs and Buxton [11], the so-called "safe area" of the gluteus medius muscle was found to be up to 5 cm medial to the greater trochanter. According to the authors, if this distance is not exceeded by the IM injection, the risk to the superior gluteal nerve and its branches will be minimal [11]. Direct trauma from a slip and fall or an inappropriate injection site can also trigger spasm of the piriformis and therefore cause entrapment.

Surgery is probably the most common source of injury to the SGN. The inferior branches are most often injured during the direct lateral hip surgery approach, while the branches to the tensor fascia lata are injured during anterolateral or anterior hip surgery approaches [5]. According to Ramesh et al. [7], regardless of the approach, 77 % of patients after total hip replacement have at least subclinical superior and inferior gluteal nerve (Chap. 54) EMG findings. The SGN also may be injured during percutaneous placement of iliosacral screws.

Physical Exam

With the patient standing, flexed at the hips, the examiner's hands are placed on the top of the iliac crest; the examiner's thumb drops down to the posterior superior iliac spine (PSIS, the site of tenderness for sacroiliac (SI) pathology) and then laterally to the iliac fossa (Video 53.1) (Fig. 53.6). Pushing medially and superiorly between the piriformis and the gluteus medius at the sciatic notch will entrap the SGN and replicate the pain. A patient with SGN entrapment may point to the iliac fossa as the site of their pain. When there is weakness of the gluteus medius muscle, the uninjured hip will drop when the foot on the uninjured side is lifted off the ground (*Trendelenburg's sign*) (Fig. 53.7). Because the abductors (gluteus medius, gluteus minimus,

Fig. 53.7 Trendelenburg's sign (Image courtesy of David Trescot)

and tensor fascia lata) are weak when the SGN is injured, they are not able to hold the pelvis level (see https://www.youtube.com/watch?v=HE0lk5MVFEg for a video describing the gait, courtesy of Dr. Nabil Ebraheim, with permission).

Differential Diagnosis (Table 53.3)

The clinical presentation of SGN entrapment can be similar to SI pathology, but the exam will show tenderness from the SI to be medial to the posterior iliac crest (PSIS), while the SGN is more lateral, in the iliac fossa. It is easy to confuse this nerve entrapment with myofascial pain of a piriformis or gluteus medius muscle itself, especially since myofascial spasms of either muscle can create the SGN entrapment. However, careful palpation should identify that the tenderness of SGN entrapment is primarily located medially in the groove between the two muscles.

Rask [3] described "the triad of superior gluteal nerve entrapment" – buttocks pain, weakness of the hip adductors, and tenderness just lateral to the sciatic notch. The diagnostic tests for SGN entrapment are described in Table 53.4.

Table 53.3 Differential diagnosis of buttock pain

	Potential distinguishing features
Lumbar spine disorders	Physical exam and MRI scan should confirm the diagnosis
Sacroiliac pathology	Tenderness over PSIS
Piriformis or gluteus medius spasm	Tenderness and spasm of the muscles. Stretching the muscle increases pain; trigger point injections into the muscle alleviate the symptoms
Sciatic entrapment	US shows compression of the nerve (see Chap. 54)
DJD hip	X-rays show degenerative changes
Maigne syndrome	Pain is relieved by injection of local anesthetic into the T12/L1 facet joint
Posterior femoral cutaneous nerve	Pain radiates down the leg

Table 53.4 Diagnostic tests for superior gluteal nerve entrapment

	Potential distinguishing features
Physical exam	Buttocks pain, hip adductor weakness, buttocks tenderness (SGN triad)
Diagnostic injection	Landmark, fluoroscopic, or US guidance
Ultrasound	May show piriformis spasm
MRI	SGN can sometimes be seen on coronal and sagittal images as it exits the pelvis in the suprapiriformis foramen [5]
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies	EMG abnormalities in 77 % of patients after total hip replacements [7]

Identification and Treatment of Contributing Factors

Minimally invasive hip surgery places trocars very close to the path of the SGN. Orthopedic surgeons have been taught that there is a "safe triangle" region for insertion of trocars and pins so as to avoid the SGN. However, Eksioglu et al. [12] showed that the boundaries of that triangle change with the height of the patient and found that 78 % of the time the SGN was found in the "safe area." They concluded that the "safe area" is "not so safe." Ozsoy and colleagues [13] noted that the risk of injury to both the superior gluteal nerve and the gluteus medius muscle is higher with limited degrees of hip flexion and adduction (for instance, on a fracture table) than with greater degrees of hip flexion and adduction, which are possible in the lateral position on a fracture table or in the so-called sloppy lateral position on an ordinary OR table.

Piriformis hypertrophy and spasm can entrap the SGN [8] and/or the sciatic nerve and result in similar presentations. Poor IM injection technique may also traumatize the SGN.

Knowing the location of the SGN can prevent surgical and injection trauma. Myofascial release, dry needling, or local anesthetic injection of the piriformis muscle can relieve the entrapment.

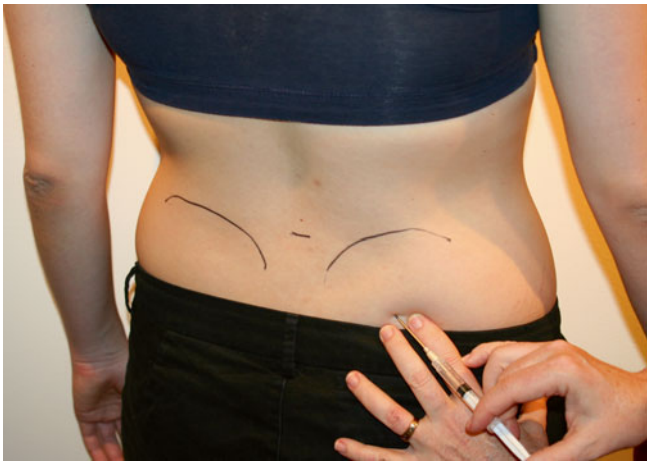

Fig. 53.8 Landmark-guided superior gluteal injection (Image courtesy of Andrea Trescot, MD)

Injection Technique

Landmark-Guided Technique

For injections without fluoroscopy, the patient is positioned standing, flexed at the hips, and holding onto the exam table, in the same position as the physical exam. Place the index and middle fingers of the non-injecting hand so they straddle the injection site. Using a 25-gauge 2-in. needle, direct the injection caudal to cephalad, lateral to medial, and advance to the periosteum (Video 53.2) (Fig. 53.8).

Fluoroscopy-Guided Technique

For injections under fluoroscopy, the patient is positioned prone on the fluoroscopy table. Place the C-arm in a straight A-P position and identify the sciatic notch. After the patient is prepped and draped as usual, administer subcutaneous local anesthetic approximately 1 cm above the sciatic notch. Radiographic landmarks include the greater sciatic foramen and the junction of the ilium and the sacrum at the lower pole of the SI joint (Fig. 53.9). Using fluoroscopic control, guide a 22-gauge SAB needle toward an area approximately 1 cm cephalad and 1 cm medial to the sciatic notch and advance to the bone (Fig. 53.10). Only a small volume, 1–2 cc, of therapeutic injectate is required. A larger volume will result in a loss of specificity, with inadvertent treatment of the piriformis muscle and possibly sciatic nerve. Additionally, a peripheral nerve stimulator can be used for precise guidance (Fig. 53.11).

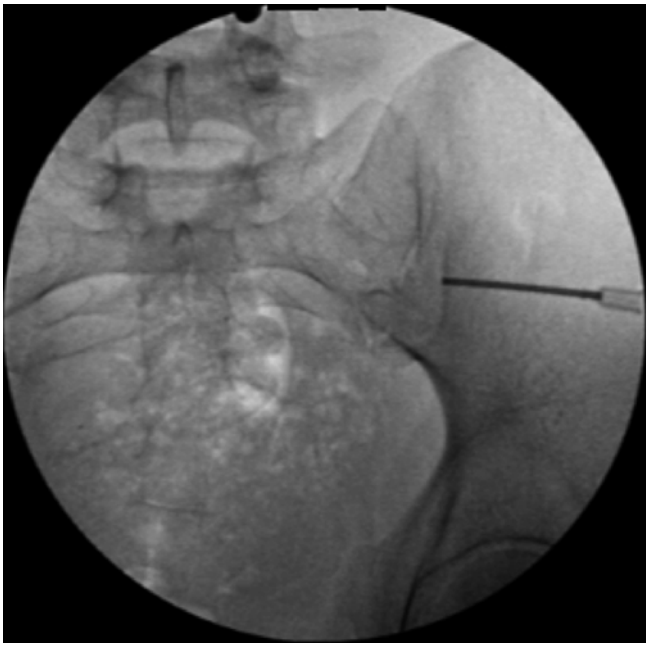

Fig. 53.9 Fluoroscopic location of superior gluteal nerve injection (Image courtesy of Andrea Trescot, MD)

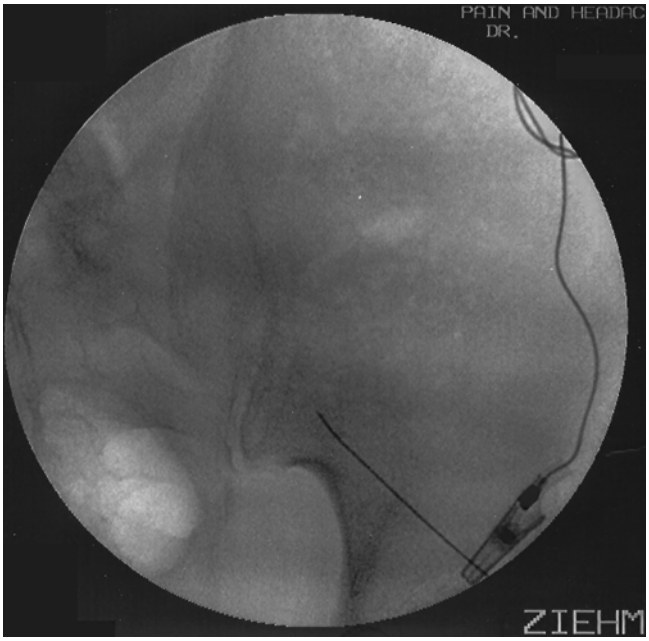

Fig. 53.11 Superior gluteal nerve injection under fluoroscopic guidance with peripheral nerve stimulator (Image courtesy of Andrea Trescot, MD)

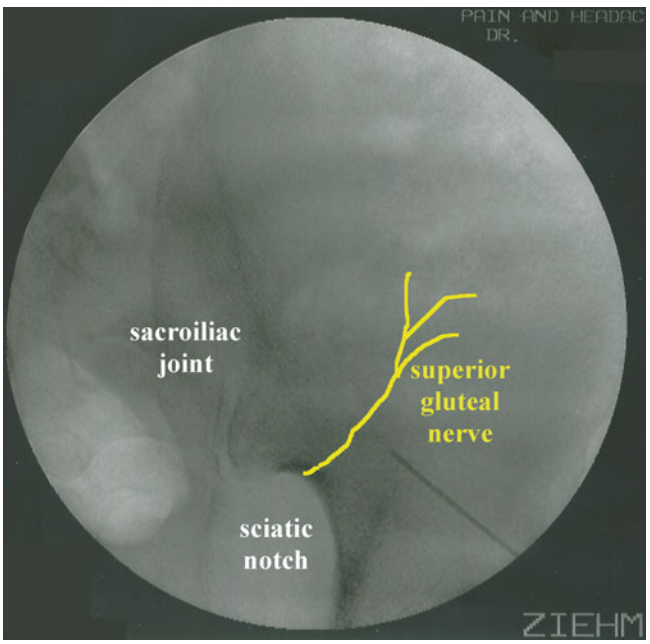

Fig. 53.10 Superior gluteal nerve injection under fluoroscopic guidance (Image courtesy of Andrea Trescot, MD)

Ultrasound-Guided Technique

Although there are no reported descriptions of US injections of the SGN, the superior gluteal artery and the piriformis muscle can be seen with US, using the curvilinear, low-frequency probe (Fig. 53.12).

Neurolytic/Surgical Technique

Cryoneuroablation

The SGN can be lesioned with a cryo-probe as described by Trescot [14]. The probe is placed in a similar manner as the diagnostic injection (Fig. 53.13), and the built-in stimulator is used to identify the nerve, with or without fluoroscopic or US guidance.

Radiofrequency Lesioning

There are no descriptions of radiofrequency lesioning of the SGN.

Surgery

Rask [3] described surgical exploration of the SGN; he found the nerve to be entrapped in a tunnel formed by the anterior-superior tendinous fibers of the piriformis, the inferior border of the gluteus minimus muscle, and the iliac crest.

Complications

The risk of injections always includes bleeding or infection, and SGN injections are no different. Misdirection of the injection can result in sciatic or piriformis injections. Paralysis of the SGN was the presumed cause of a femur fracture in one patient [2].

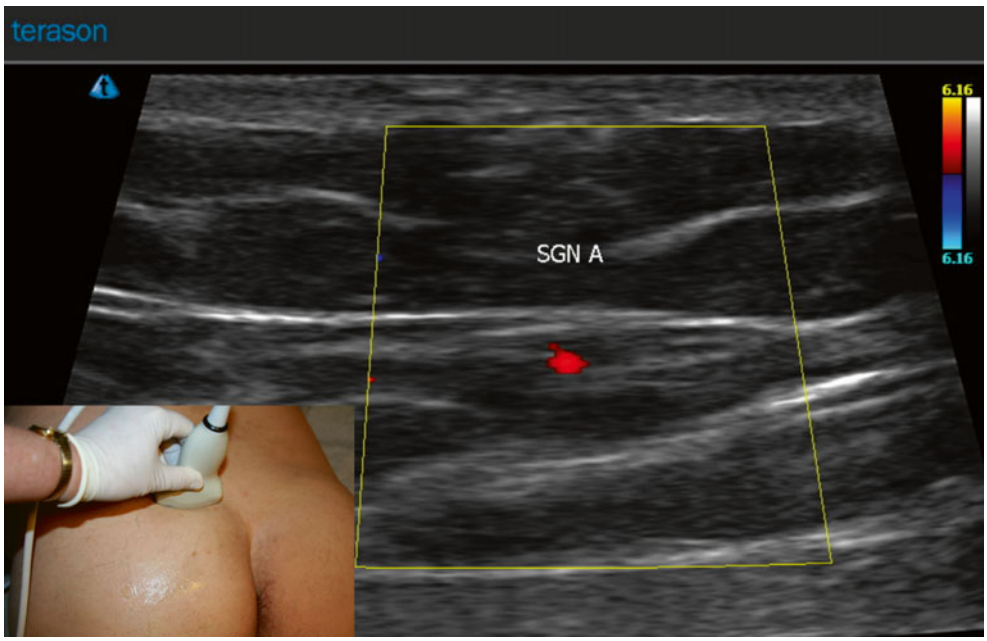

Fig. 53.12 Ultrasound image of the superior gluteal nerve. *SGN A* superior gluteal artery (Image courtesy of Andrea Trescot, MD)

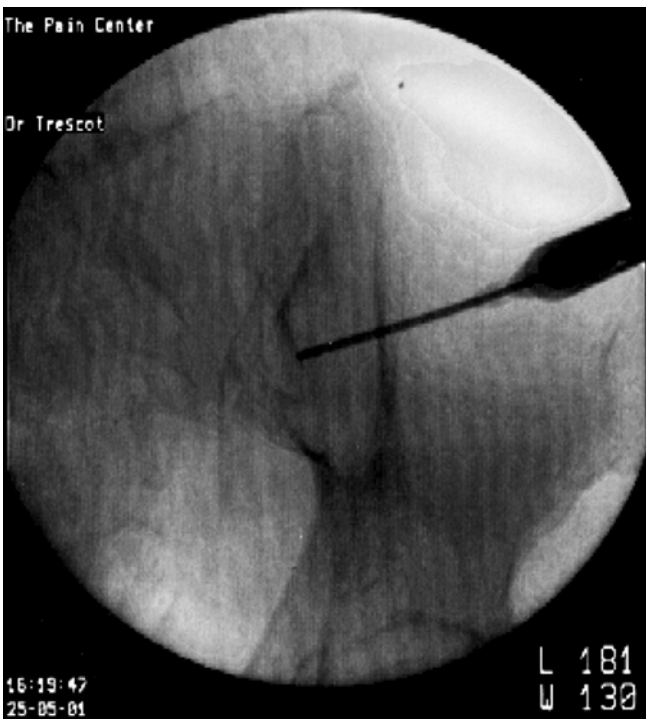

Fig. 53.13 Fluoroscopic image of cryoneuroablation of the superior gluteal nerve (Image courtesy of Andrea Trescot, MD)

Summary

The piriformis and gluteus medius muscles can entrap the SGN, causing pain down the leg ("pseudo-sciatica") that can lead to misdiagnosis and inappropriate surgeries. In addition, the nerve can be injured by injection and surgery. Recognition of the entrapment is critical for appropriate treatment.

References

1. Mondelli M, Martelli G, Greco G, Ferrari F. Mononeuropathies of inferior and superior gluteal nerves due to hypertrophy of piriformis muscle in a basketball player. Muscle Nerve. 2008;38(6):1660–2.
2. de Jong PJ, van Weerden TW. Inferior and superior gluteal nerve paresis and femur neck fracture after spondylolisthesis and lysis: a case report. J Neurol. 1983;230(4):267–70.
3. Rask MR. Superior gluteal nerve entrapment syndrome. Muscle Nerve. 2004;3(4):304–7.
4. Willick SE, Margherita AJ, Carter GT. Isolated superior gluteal nerve injury: two case reports. Muscle Nerve. 1998;21(7):951–3.
5. Petchprapa CN, Rosenberg ZS, Sconfienza LM, Cavalcanti CF, Vieira RL, Zember JS. MR imaging of entrapment neuropathies of the lower extremity. Part 1. The pelvis and hip. Radiographics. 2010;30(4):983–1000.
6. Ince A, Kemper M, Waschke J, Hendrich C. Minimally invasive anterolateral approach to the hip: risk to the superior gluteal nerve. Acta Orthop. 2007;78(1):86–9.
7. Ramesh M, O'Byrne JM, McCarthy N, Jarvis A, Mahalingham K, Cashman WF. Damage to the superior gluteal nerve after the Hardinge approach to the hip. J Bone Joint Surg Br. 1996;78(6):903–6.
8. Diop M, Parratte B, Tatu L, Vuillier F, Faure A, Monnier G. Anatomical bases of superior gluteal nerve entrapment syndrome in the suprapiriformis foramen. Surg Radiol Anat. 2002;24(3–4):155–9.

9. Trescot AM, Klein DS, Edwards LW. Superior gluteal nerve pathology as a cause of sciatica (poster). American Academy of Pain Management annual meeting; Dallas; 1995.
10. Ray B, D'Souza AS, Saxena A, Nayak D, Sushma RK, Shetty P, et al. Morphology of the superior gluteal nerve: a study in adult human cadavers. Bratisl Lek Listy. 2013;114(7):409–12.
11. Jacobs LG, Buxton RA. The course of the superior gluteal nerve in the lateral approach to the hip. J Bone Joint Surg Am. 1989; 71(8):1239–43.
12. Eksioglu F, Uslu M, Gudemez E, Atik OS, Tekdemir I. Reliability of the safe area for the superior gluteal nerve. Clin Orthop Relat Res. 2003;412:111–6.
13. Ozsoy MH, Basarir K, Bayramoglu A, Erdemli B, Tuccar E, Eksioglu MF. Risk of superior gluteal nerve and gluteus medius muscle injury during femoral nail insertion. J Bone Joint Surg Am. 2007;89(4):829–34.
14. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.

Inferior Gluteal Nerve Entrapment

54

Andrea M. Trescot

Introduction

Entrapment of the *inferior gluteal nerve* (IGN) causes buttock pain and weakness of the gluteal muscle and usually occurs in combination with *superior gluteal nerve* (SGN) (Chap. 53), *posterior femoral cutaneous nerve* (PFCN) (see Chap. 56), and *sciatic nerve* (Chap. 55) entrapments. It is a rare (or under-recognized) pathology occurring after buttock procedures (such as hip surgery) and trauma.

Clinical Presentation (Table 54.1)

The patient with IGN entrapment (IGNE) will present with pain, weakness, and numbness of the buttocks (Fig. 54.1). Because of its location, the nerve is often entrapped along with the PFCN and the *sciatic nerve* and thus may refer pain down the posterior thigh (Fig. 54.2).

The IGN is the sole innervation of the gluteus maximus (GM). The GM acts to extend the trunk at the hip (such as when extending the trunk from the stooped position) and to extend the hip when rising from sitting or climbing stairs. Patients therefore complain of weakness getting out of chairs or climbing stairs.

Gluteal augmentation (to increase the size and improve the contour of the buttocks) can traumatize the IGN along its course within the GM muscle, under which the gluteal implants are placed. In the same way, posterior and posterolateral surgical approaches to the hip may injure the IGN.

Table 54.1 Occupation/exercise/trauma history relevant to inferior gluteal nerve entrapment

Compression	Pelvic pathology
	Piriformis entrapment [1]
	Direct compression by sciatic lesions
	Compression during coma or general anesthesia [1]
	Colorectal cancer [2]
Trauma	Intramuscular injections
Surgery	Augmentation gluteoplasty [3]
	Posterior and posterolateral hip surgeries [4]

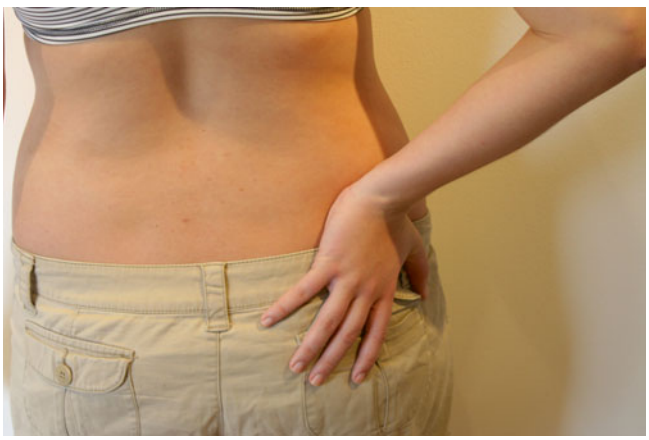

Fig. 54.1 Patient localization of inferior gluteal nerve pain (Image courtesy of Andrea Trescot, MD)

Anatomy (Table 54.2)

The IGN arises from the sacral plexus (the L5, S1, and S2 dorsal rami), just one nerve root down from the SGN (see Chap. 53). The sacral plexus gives rise to the *sciatic nerve*, the superior and inferior gluteal nerves, the *pudendal nerve* (see Chap. 47), and the *posterior femoral cutaneous nerve* (see Chap. 61). The IGN leaves the pelvis with the sciatic nerve through the *sciatic notch* below the piriformis (the

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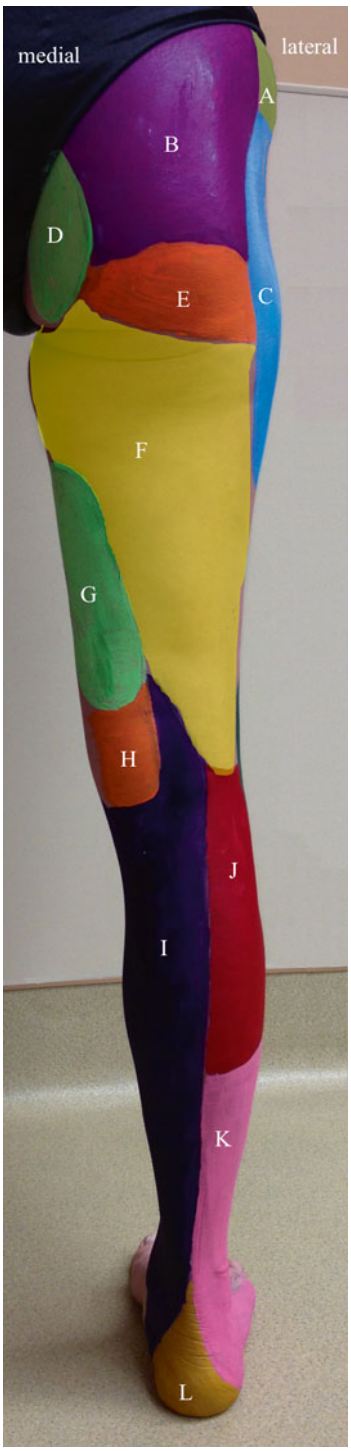

Fig. 54.2 Pain pattern from nerve entrapments of the posterior leg. *A* lateral branch iliohypogastric nerve, *B* superior cluneal nerve, *C* lateral femoral cutaneous nerve, *D* middle cluneal/sacral nerve, *E* inferior cluneal nerve, *F* posterior femoral cutaneous nerve, *G* obturator nerve, *H* femoral nerve, *I* saphenous nerve, *J* lateral sural cutaneous nerve, *K* superficial peroneal nerve, *L* medial calcaneal nerve (Image courtesy of Terri Dallas- Prunskis, MD)

Table 54.2 Inferior gluteal nerve anatomy

Origin	Dorsal rami of L5, S1, and S2
General route	Lies medial to sciatic nerve and passes out of the pelvis inferior to the piriformis through the greater sciatic foramen (the infra-piriformis foramen). At the lower border of the piriformis, it divides into superior-heading and inferior-heading branches
Sensory distribution	None
Motor innervation	Sole motor innervation of the gluteus maximus [5]
Anatomic variability	Like the sciatic nerve, the IGN can congenitally pass *through* instead of *under* the piriformis muscle
Other relevant structures	Piriformis muscle, sciatic nerve, posterior femoral cutaneous nerve, and gluteus minimus

infra-piriformis foramen) (Fig. 54.3) and bends retrograde (cephalad) over the piriformis and under the GM. It then divides into upward and downward branches and supplies motor innervation to the superior GM muscle (Figs. 54.4, 54.5, and 54.6).

There may also be a branch connecting to the *PFCN* [5]. Sforsini et al. [6] actually describes the IGN as a branch of the "posterior cutaneous nerve of the thigh" rather than having an independent origin. According to this group, the IGN (or a branch of the PFCN) has a cutaneous branch that innervates the skin of the lower border of the gluteal region and a perineal branch that innervates the skin of the perineum and scrotum/labia majora (which is called the *inferior cluneal nerve* by other authors – see Chap. 52).

Skalak and colleagues [7] developed an implantable gluteal stimulation device to prevent pressure decubitus ulcers and needed to find a reliable surface landmark for the "motor point" of the IGN. Therefore, they dissected nine cadavers and identified that the IGN could be reliably found at the junction of a line connecting "the most prominent lateral borders of the greater trochanters" horizontally with a perpendicular vertical line centered at the ischial tuberosity (Fig. 54.7a).

Because the IGN is vulnerable during posterior approaches to the hip, Apaydin and colleagues [4] dissected 36 gluteal regions, looking for surgical landmarks for the IGN. They were able to define a triangular region that contained the IGN: the apex is the posterior inferior iliac spine (PIIS); the other two legs connect the greater trochanter (GT) and the ischial tuberosity (IT) (Fig. 54.7b). This triangle could be further divided into two regions, with the superior region being the "danger zone." In all of

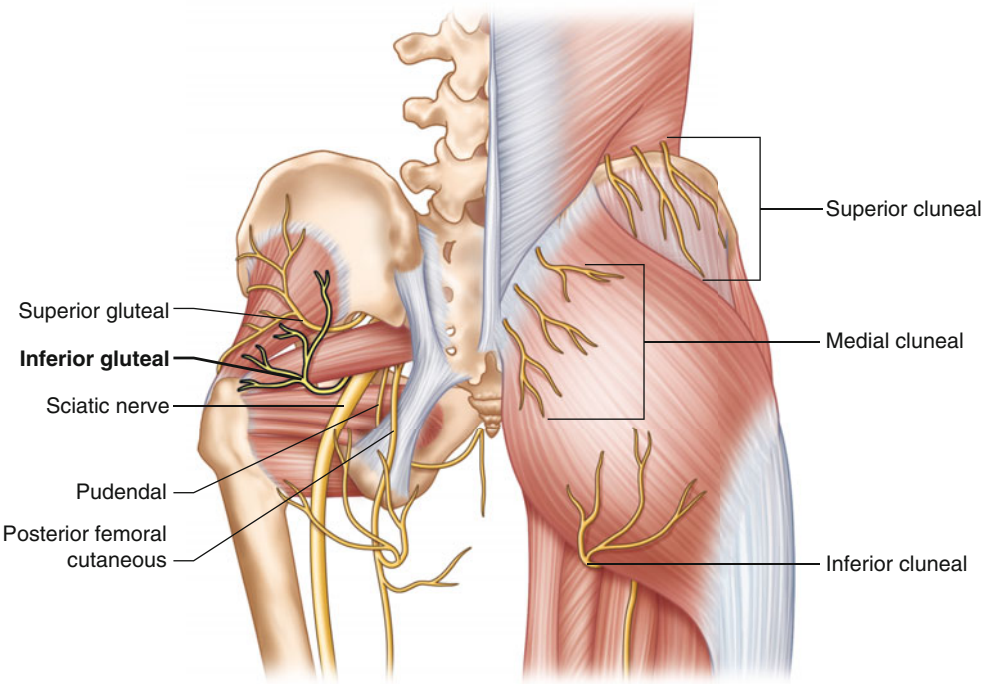

Fig. 54.3 Anatomy of the buttock region (Image by Springer)

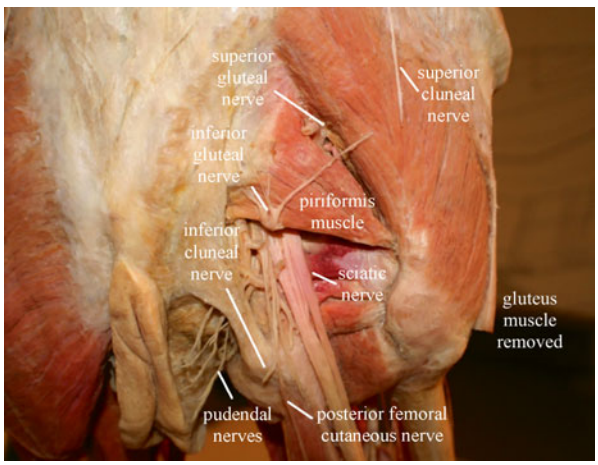

Fig. 54.4 Gluteal dissection modified from an image from *Bodies, The Exhibition,* with permission (Image courtesy of Andrea Trescot, MD)

their specimens, the IGN entered the deep surface of the GM approximately 5.4 cm from the apex of the GT.

Hwang and colleagues [3] dissected 10 adult Korean cadavers (20 buttocks) from the *infra-piriformis foramen* to the deep surface of the GM in order to measure the relative depth of the IGN based on the thickness of the GM. Except for the region below the coccyx-greater trochanter line, the IGN traveled relatively superficially in the medial portion of the GM (at a depth less than 70 % of the muscle thickness), but relatively deep in the lateral portion of the muscle (more than 70 % of the thickness).

Like most of the nerve entrapments discussed in this book, the IGN has several variations in its path, which perhaps contribute to its entrapment. Tillmann [8] evaluated 112 cadavers; in 17 of them, the IGN left the pelvis through the piriformis, similar to what is seen occasionally with the *sciatic nerve* (Chap. 55). In three cases, this variation was seen bilaterally, more often in females than males. In all the cases, the peroneal division of the *sciatic nerve* also passed through the piriformis muscle. The PFCN was seen to often travel with the IGN through the *infra-piriformis foramen*.

Entrapment

Pathology of the IGN comes primarily from pelvic pathology and intramuscular (IM) injection-related injury of the SGN and IGN. However, because of the medial intrapelvic fixation of the IGN at its origin from the sciatic nerve, lumbar lordosis and internal rotation of the hip put tension on the piriformis muscle. This then entraps the IGN between the piriformis, gluteus minimus, and dorsal rim of the sciatic notch above and the inferior gluteal blood vessels and lymph nodes below, creating a vicious cycle, as edema of the entrapped nerve creates more entrapment. As noted in section "Anatomy", Tillmann [8] evaluated 112 subjects; in 17

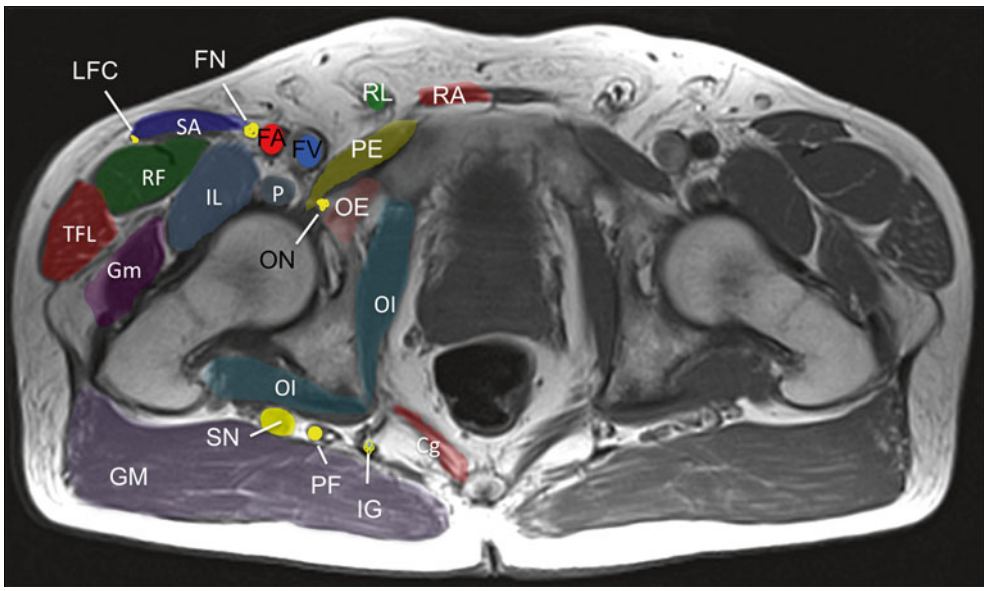

Fig. 54.5 MRI axial image of pelvis. *Cg* coccygeus muscle, *FA* femoral artery, *FN* femoral nerve, *FV* femoral vein, *GM* gluteus maximus muscle, *GME* gluteus medius, *IG* inferior gluteal nerve, *IL* iliopsoas muscle, *LFC* lateral femoral cutaneous nerve, *OI* obturator internis muscle, *P* psoas muscle, *PE* pectineus muscle, *PF* posterior femoral cutaneous nerve, *QF* quadratus femoris muscle, *RA* rectus abdominis muscle, *RF* rectus femoris muscle, *RL* round ligament, *SA* sartorius muscle, *SN* sciatic nerve, *TFN* tensor fascia lata muscle (Image courtesy of Andrea Trescot, MD)

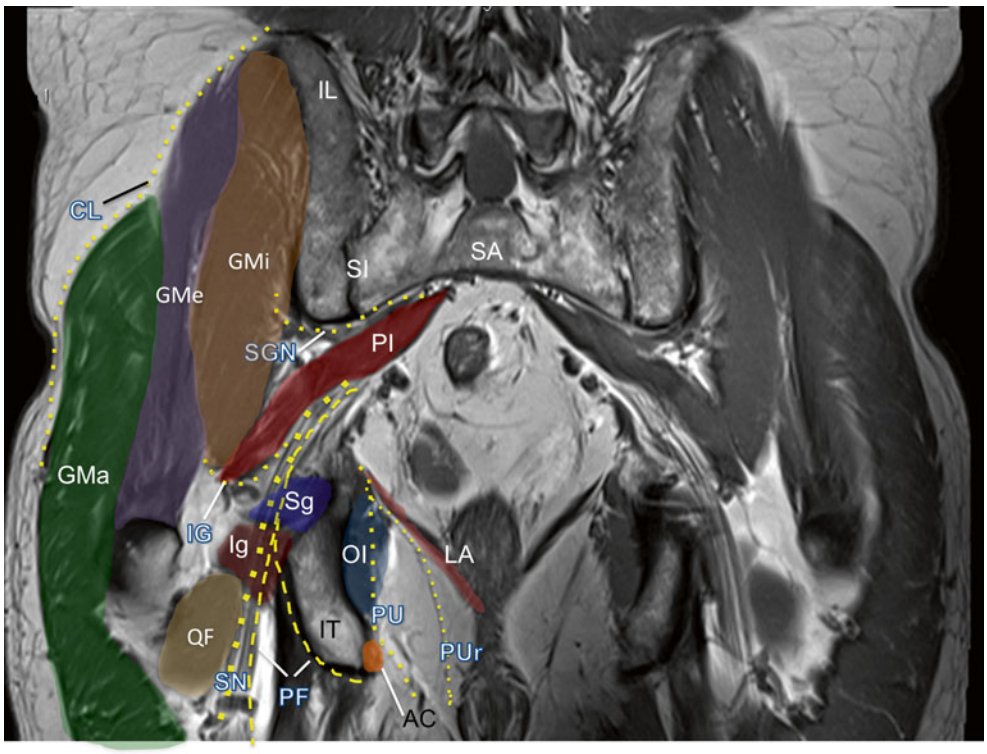

Fig. 54.6 MRI coronal images showing gluteal muscles and nerves. *AC* Alcock's canal, *CL* superior cluneal nerve, *IL* iliac crest, *IG* inferior gluteal nerve, *Ig* inferior gemellus muscle, *IT* ischial tuberosity, *Pi* piriformis, *Pu* pudendal, *Gmi* gluteus minimus, *Gme* gluteus medius, *Gma* gluteus maximus, *GT* greater trochanter, *LA* levator ani muscle, *OI* obturator internus muscle, *PF* posterior femoral cutaneous nerve, *PU* pudendal nerve, *PUr* pudendal nerve (rectal branch), *QF* quadratus femoris muscle, *SA* sacrum, *SGN* superior gluteal nerve, *Sg* superior gemellus muscle, *SI* sacroiliac joint, *SN* sciatic nerve (Image courtesy of Andrea Trescot, MD)

of them, the IGN left the pelvis through the piriformis, similar to that seen occasionally with the sciatic nerve. Subclinical electromyography (EMG) changes of the SGN and IGN have been seen in up to 77 % of patients after total hip replacement, regardless of approach. The IGN can also be injured during intramuscular augmentation gluteoplasty.

Interestingly, LaBan et al. [2] noted lumbosacral or buttock pain, cutaneous anesthesia in the PFCN, and EMG evidence of IGN entrapment in five patients diagnosed with recurrent colon cancer. They concluded that, due to the medial intrapelvic origin of the IGN and the "crowding effect" of the piriformis muscle above, the dorsal rim

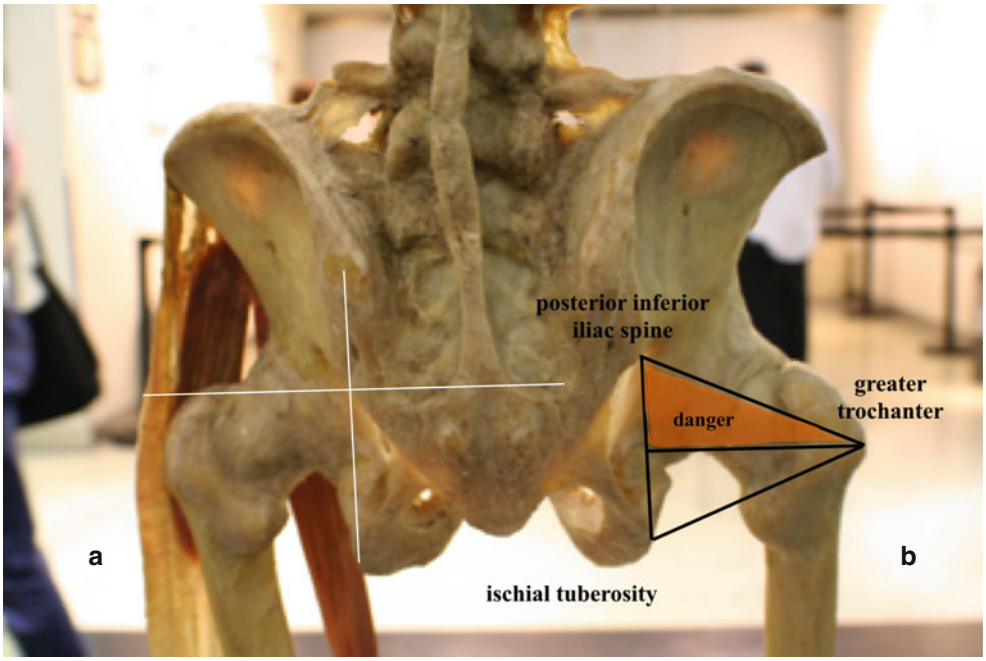

Fig. 54.7 (**a**) Location of the IGN using the landmarks described by Skalal et al. [7]. (**b**) "Danger" triangle by Apaydin et al. [4] identifying the location of the inferior gluteal nerve (Image courtesy of Andrea Trescot, MD)

of the sciatic notch behind, and the inferior gluteal vessels and nodes below, the IGN was particularly susceptible to entrapment by colorectal cancer. IGN entrapment should be considered in patients with hypoesthesia over the inferior lateral buttocks and a history of colorectal malignancy.

Physical Exam

Patients with IGN entrapment may have a GM "lurch" – when the GM is weak, the trunk hyperextends with heel strike to compensate for weak hip extension (see https://www.youtube.com/watch?v=bTQ5ID7Tpa4 for a video demonstration, courtesy of Dr. Nabil Ebraheim, with permission). Palpation of the IGN is similar to that of the sciatic nerve or PFCN. With the patient standing and flexed at the hips, identify the piriformis muscle; the IGN is at the inferior border of the piriformis, superior to the ischial tuberosity (Fig. 54.8).

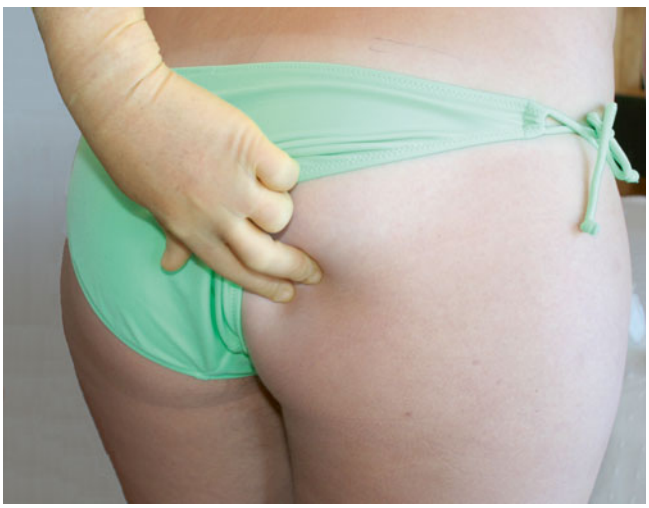

Fig. 54.8 Physical exam of the inferior gluteal nerve (Image courtesy of Andrea Trescot, MD)

Differential Diagnosis (Table 54.3)

It can be difficult to differentiate IGN dysfunction from a variety of gluteal pathologies, including sciatic nerve entrapment (Chap. 55), piriformis syndrome, PFCN entrapment (Chap. 56), and pathology of the sacroiliac or L5 facet joints or superior cluneal (Chap. 51) or inferior cluneal nerves (Chap. 52). It is not uncommon to have several concurrent entrapments from piriformis spasm (IGN, sciatic, and PFCN), since they travel together. Since the treatment is similar, there may be no clinical need to differentiate, at least early on. The history and mechanism of injury may provide the most useful information. Table 54.4 lists the diagnostic tests for IGN entrapments.

Table 54.3 Differential diagnosis of buttock pain

	Potential distinguishing features
Lumbar spine disorders	Physical exam and MRI scan should confirm the diagnosis
Sacroiliac joint disorders	Pain may be present in the lower back, back of hips, groin, and thighs. Physical exam, X-rays, CT, and MRI scans can help identify pathology. Relief of pain from a SI joint injection will confirm the diagnosis
Piriformis or gluteus medius spasm	Tenderness and spasm of the muscles, stretching the muscle will increase the pain; trigger point injections into the muscle will alleviate the symptoms
Posterior rami syndrome, thoracolumbar junction syndrome (Maigne syndrome), and dorsal ramus syndrome	Pain is relieved by injection of local anesthetic into the T12/L1 facet joint
Sciatic entrapment	US shows compression of the nerve (see Chap. 55)
DJD hip	X-rays show degenerative changes
Posterior femoral cutaneous nerve	Pain radiates down the leg

Table 54.4 Diagnostic tests for inferior gluteal nerve entrapment

	Potential distinguishing features
Physical exam	Tenderness inferior to the piriformis, medial to sciatic nerve
Diagnostic injection	Landmark or US injection inferior to piriformis muscle
Ultrasound	Not described, but the location of the sciatic nerve will identify the direction to the IGN
MRI	The IGN can be seen on coronal images, exiting the pelvis adjacent to the sciatic nerve. With IGN injury, there may be signal abnormalities in the gluteus maximus [5]
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies	May show denervation pattern of the gluteus maximus [2]; EMG abnormalities in 77 % of patients after total hip replacements [9]

Identification and Treatment of Contributing Factors

Piriformis muscle spasm is the major factor contributing to entrapment and perhaps a "double crush" situation (see Chap. 1). Piriformis stretches and injections can be very useful, and botulinum toxin may be appropriate for recalcitrant cases. De Jong and van Weerden [10] described a patient with substantial lordosis from wearing high heels that led to piriformis entrapment of the SGN and IGN. The resulting lack of gluteal muscle function resulted in a hip fracture.

Fig. 54.9 Landmark-guided injection of the inferior gluteal nerve (Image courtesy of Andrea Trescot, MD)

Knowing the location of the IGN should decrease the risk of iatrogenic injury during IM injections and hip surgery. Apaydin et al. [4] stated that the posterior approach to the hip, while the most common, was more likely to result in damage to the IGN. Ling and Kumar [11] recommended a muscle-splitting incision not more than 5 cm medial to the greater trochanter to avoid the IGN. Hwang et al. [3] (see section "Anatomy") concluded that intramuscular augmentation gluteoplasty could be performed without injuring the IGN, as long as the dissection was not extended too deeply.

Injection Technique

Since IGN entrapment is not well recognized, there is relatively little literature on the injection techniques for this nerve. Most of the techniques described here are modifications of techniques used to treat other nerves in the area.

Landmark-Guided Technique

For the landmark-guided injection, the patient is positioned standing, flexed at the hips, which tightens the gluteal tissues and makes palpation of the structures easier. Alternatively, especially if a peripheral nerve stimulator is to be used, the patient can be placed prone. The inferior border of the piriformis muscle is palpated, and the maximal tenderness identified, medial to the midbody of the muscle (Fig. 54.9). A 25-gauge 2-in. needle is usually all that is necessary, though

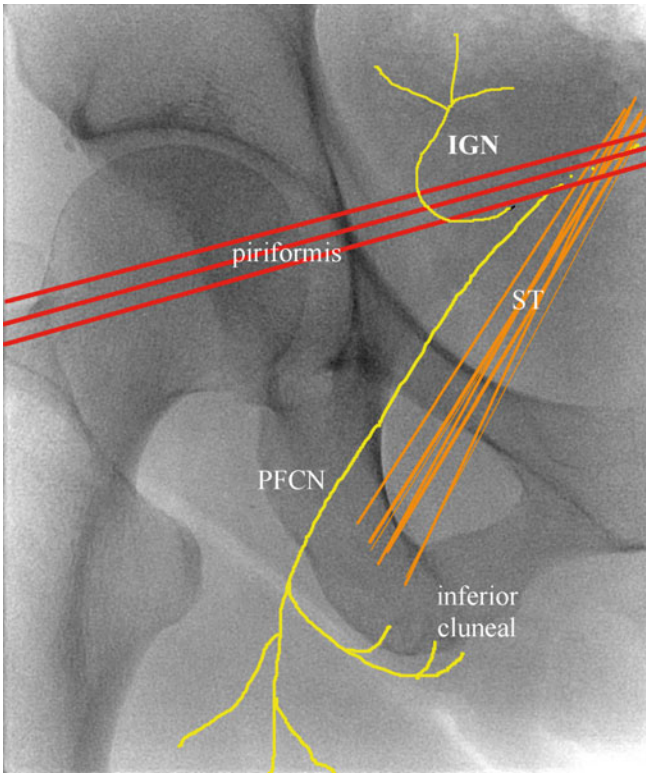

Fig. 54.10 Fluoroscopic landmarks of the inferior gluteal nerve (Image courtesy of Andrea Trescot, MD)

for the morbidly obese, a 22-gauge 3-in. Quincke needle may be needed. As described above, a peripheral nerve stimulator may be useful, seeking stimulation of the GM and not the posterior leg (which would be the sciatic nerve).

Fluoroscopy-Guided Technique

There are no specific fluoroscopic landmarks for the IGN, but, since it travels with the PFCN, a similar technique could be used (see Chap. 56) (Fig. 54.10).

Ultrasound-Guided Technique

There are no published descriptions of US-guided IGN injections. However, when using an US-guided approach to the sciatic nerve at the level of the piriformis, the IGN should be medial to the sciatic nerve.

Neurolytic/Surgical Technique

There are no published cryoneuroablation or radiofrequency techniques described.

Complications

Because of the proximity of the sciatic nerve, there is a risk of sciatic nerve anesthesia (from spillover of the local anesthetic) or sciatic nerve damage from the needle.

Summary

IGN entrapment is not well recognized, which probably contributes to the limited available literature. This nerve is in the same area as the sciatic and posterior femoral cutaneous nerves, both of which can cause severe pain in the buttocks. Inferior gluteal entrapment should be considered in the patient with buttock pain.

References

1. Mondelli M, Martelli G, Greco G, Ferrari F. Mononeuropathies of inferior and superior gluteal nerves due to hypertrophy of piriformis muscle in a basketball player. Muscle Nerve. 2008;38(6):1660–2.
2. LaBan MM, Meerschaert JR, Taylor RS. Electromyographic evidence of inferior gluteal nerve compromise: an early representation of recurrent colorectal carcinoma. Arch Phys Med Rehabil. 1982;63(1):33–5.
3. Hwang K, Nam YS, Han SH, Hwang SW. The intramuscular course of the inferior gluteal nerve in the gluteus maximus muscle and augmentation gluteoplasty. Ann Plast Surg. 2009;63(4):361–5.
4. Apaydin N, Bozkurt M, Loukas M, Tubbs RS, Esmer AF. The course of the inferior gluteal nerve and surgical landmarks for its localization during posterior approaches to hip. Surg Radiol Anat. 2009;31(6):415–8.
5. Petchprapa CN, Rosenberg ZS, Sconfienza LM, Cavalcanti CF, Vieira RL, Zember JS. MR imaging of entrapment neuropathies of the lower extremity. Part 1. The pelvis and hip. Radiographics. 2010;30(4):983–1000.
6. Sforsini C, Wikinski JA. Anatomical review of the lumbosacral plexus of the lower extremity. Tech Reg Anesth Pain Manag. 2006;10:138–44.
7. Skalak AF, McGee MF, Wu G, Bogie K. Relationship of inferior gluteal nerves and vessels: target for application of stimulation devices for the prevention of pressure ulcers in spinal cord injury. Surg Radiol Anat. 2008;30(1):41–5.
8. Tillmann B. Variations in the pathway of the inferior gluteal nerve (author's transl). Anat Anz. 1979;145(3):293–302.
9. Ramesh M, O'Byrne JM, McCarthy N, Jarvis A, Mahalingham K, Cashman WF. Damage to the superior gluteal nerve after the hardinge approach to the hip. J Bone Joint Surg Br. 1996;78(6):903–6.
10. De Jong PJ, van Weerden TW. Inferior and superior gluteal nerve paresis and femur neck fracture after spondylolisthesis and lysis: a case report. J Neurol. 1983;230(4):267–70.
11. Ling ZX, Kumar VP. The course of the inferior gluteal nerve in the posterior approach to the hip. J Bone Joint Surg Br. 2006;88(12):1580–3.

Proximal Sciatic Nerve Entrapment: Low Back

55

Annemarie E. Gallagher, Amitabh Gulati, and Terri Dallas-Prunskis

Introduction

Low back and buttocks pain may be caused by entrapment of the sciatic nerve, an etiology that is often underdiagnosed. There are many potential areas of entrapment of this nerve. The focus of this chapter will be primarily on *proximal* sciatic nerve entrapment causing low back pain. *Proximal sciatic entrapment* can also cause lower extremity pain, which will be discussed in Chap. 65.

Clinical Presentation (Table 55.1)

The *sciatic nerve* (SN), the largest nerve in the body, can be entrapped anywhere along its path from the sacrum to the foot. There are multiple causes of low back and buttocks pain, as well as leg pain related to proximal sciatic entrapment (Table 55.1). Proximally, the sciatic nerve may become entrapped in the region of the hip and, less commonly, in the thigh. Sciatic neuropathy may be secondary to acetabular fracture, trauma from hip arthroplasty, muscular entrapment by the hamstrings, posterior thigh compartment syndrome, tumor, vascular compression of the sacral plexus, or heterotopic ossification. Sciatic neuropathy may also occur from piriformis syndrome or from scarring due to trauma or radiation [28].

Table 55.1 Occupation/exercise/trauma history relevant to proximal sciatic entrapment

Endopelvic pathology [1, 2]	Pregnancy [3]
	Infection [4]
	Vascular causes [5, 6]
Hip etiology	Hip surgery [7, 8]
	Hematoma after hip surgery [9]
	Hip fracture/heterotopic ossification [10, 11]
Spinal pathology	Lumbar spinal stenosis/disc herniation/ spondylolisthesis [12]
Extrapelvic pathology	Piriformis syndrome [13]
	Sacroiliitis [14]
	Prolonged squatting or sitting position [15–18]
	Intraneural tumor/cyst [19]
	Intramuscular gluteal injection [20]
	Acute external compression [21, 22]
	Lipoma [5, 23]
	Penetrating injuries [24]
	Poor IM injection technique [25, 26]
	Endovascular vein ablation [27]

The patient with proximal sciatic nerve entrapment will initially present with one of three clinical stages. During *Stage I*, the patient will complain of resting low back, buttocks, and leg pain and paresthesias or dysesthesias in the sciatic distribution, which is often worse at night. The patient in *Stage II* will present with leg weakness and numbness in the sciatic distribution (Fig. 55.1). *Stage III* symptoms consist of complaints of constant pain, with muscle atrophy and sensory loss that will be apparent on physical examination [29]. The patient may also complain of sharp low back pain, aching buttock pain, and occasionally dull pain from the popliteal fossa to the foot [30] (Fig. 55.2). Since each lumbar and sacral root innervates a different part of the foot, the patient who complains of pain or numbness of the "whole foot" (minus the medial part of the foot innervated by the saphenous nerve) is likely to have a sciatic entrapment rather than a radiculopathy.

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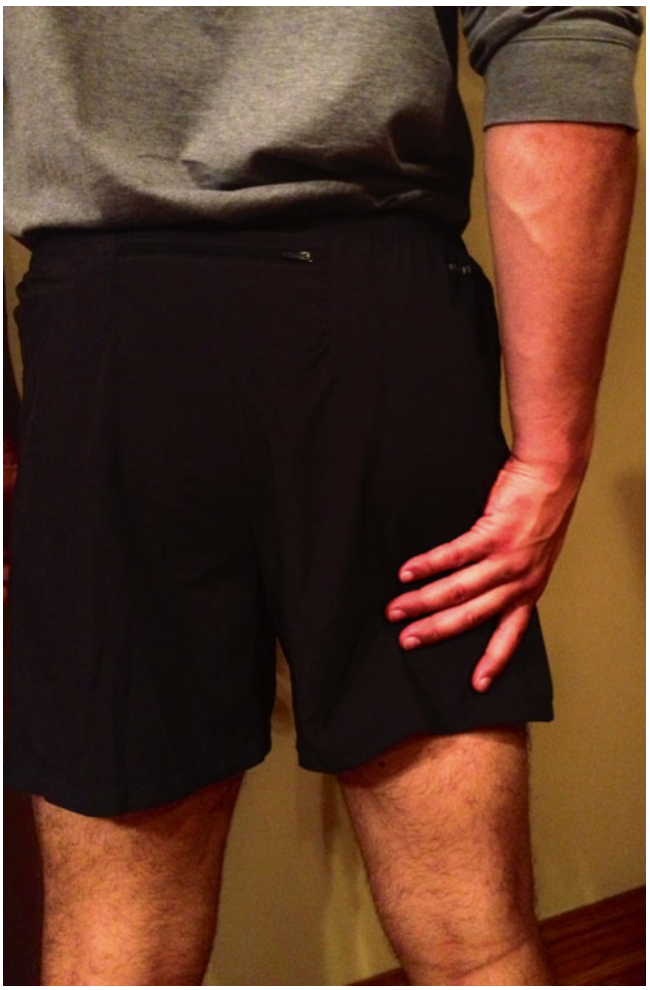

Fig. 55.1 Patient's pain complaints from the proximal sciatic nerve (Image courtesy of Terri Dallas-Prunskis MD)

Other common symptoms include foot drop and pain with extended periods of sitting or twisting toward the unaffected side. Patients will often weight shift when sitting to minimize pressure on the painful side [30].

Anatomy (Table 55.2)

The sciatic nerve is the largest branch of the *sacral plexus* and is the largest nerve in the human body. Originating from the ventral divisions of L4-S3 of the sacral plexus, the sciatic nerve courses through the pelvis, enters the gluteal region through the *greater sciatic foramen* (Fig. 55.3), and then exits the greater sciatic foramen at the inferior border of the piriformis muscle in 79 % of patients [11]. Anatomic variants include having the sciatic nerve pass through the piriformis muscle itself (either the tibial or peroneal divisions or both) or superior to it [12].

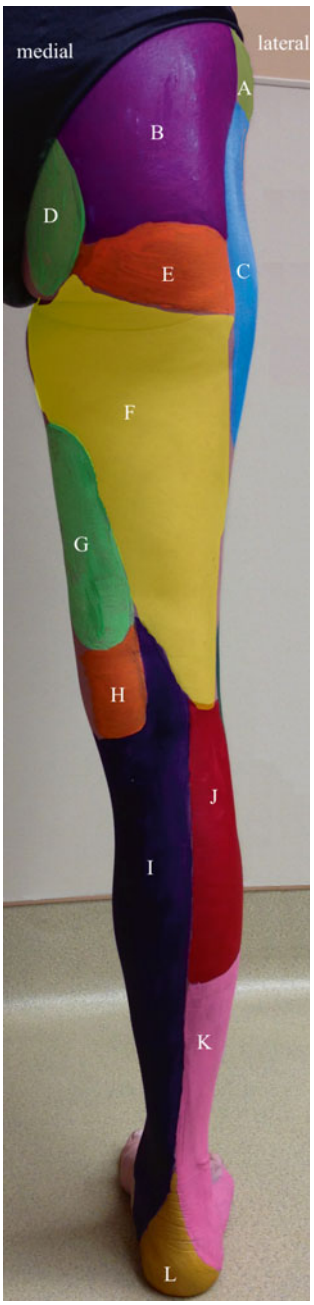

Fig. 55.2 Pain pattern from nerves of the posterior leg. *A* lateral branch iliohypogastric nerve, *B* superior cluneal nerve, *C* lateral femoral cutaneous nerve, *D* middle cluneal/sacral nerve, *E* inferior cluneal nerve, *F* posterior femoral cutaneous nerve, *G* obturator nerve, *H* femoral nerve, *I* saphenous nerve, *J* lateral sural cutaneous nerve, *K* superficial peroneal nerve, *L* medial calcaneal nerve (Image courtesy of Terri Dallas-Prunskis, MD)

The sciatic nerve travels between the *greater trochanter* and the *ischial tuberosity*, passing deep to the *gluteus maximus muscle* and posteriorly to the *obturator internus* and *adductor magnus muscles* (Fig. 55.4). It then curves around the ischial spine and descends laterally past the origin of the hamstrings. In the proximal thigh, the sciatic nerve runs

Table 55.2 Sciatic nerve anatomy

Origin	Ventral divisions of L4-S3 of the sacral plexus
General route	The sciatic nerve comprises the lateral division, which forms the *peroneal nerve*, eventually forming the *common peroneal (fibular) nerve* (see Chap. 67) and the medial division, which forms the *tibial nerve* (see Chap. 73), each separately encased from the outset [31]. The sciatic nerve courses through the pelvis, enters the gluteal region through the *greater sciatic foramen*, then exits at the inferior border of the *piriformis muscle*; the nerve is covered by the gluteus maximus muscle and soft tissue. The nerve then travels halfway between the bony landmarks (greater trochanter laterally and ischial tuberosity medially) and then descends into the subgluteal area. It then runs posteriorly in the midthigh, remaining dorsal to the adductor magnus and ventral to the long head of the biceps femoris
Sensory distribution	Back of the leg, back and lateral side of the calf, and most of the foot
Motor innervation	Tibial division: hamstring muscles (*semimembranosus*, *semitendinosus*, long head of the *biceps femoris*); adductor *magnus in the thigh*
	Common peroneal (fibular) division: short head of the biceps femoris
Anatomic variability	The sciatic nerve usually travels under the piriformis muscle, except in 10–30 % cases, where the nerve passes through the piriformis muscle or above it [31]
Other relevant structures	Sciatic foramen, piriformis muscle

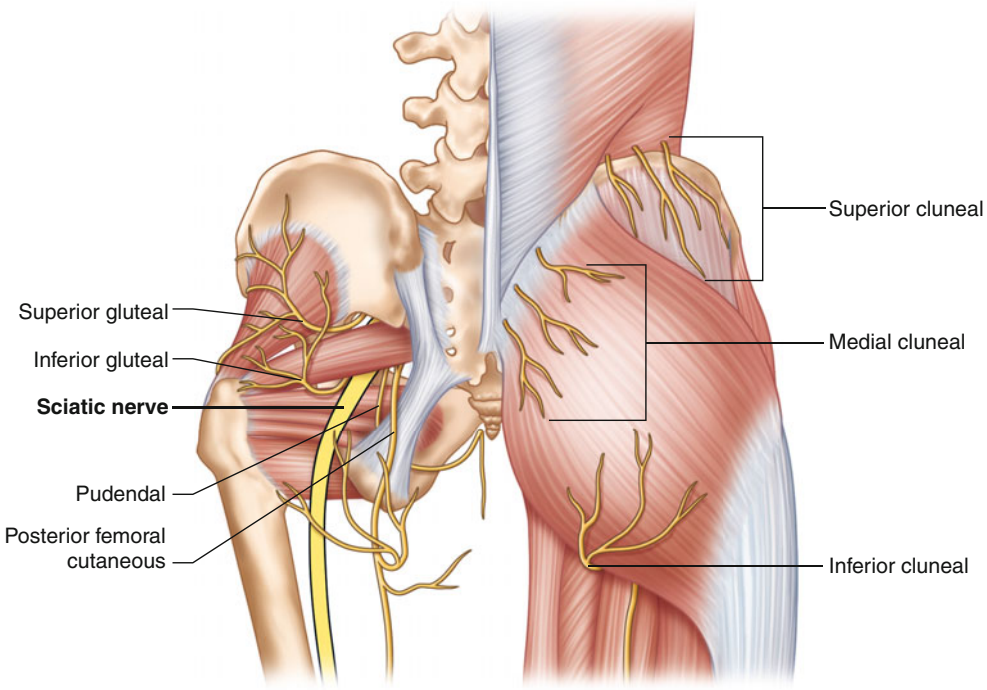

Fig. 55.3 Sciatic anatomy (Image courtesy of Springer)

posteriorly to the hamstrings and anteriorly to the adductor magnus. A branch of the sciatic nerve, the *superior gluteal nerve* (see Chap. 53), may also be entrapped (Fig. 55.5), contributing to the patient's symptoms. This branch of the sciatic nerve exits the sciatic notch and travels between the fascial planes of the gluteus medius and the piriformis muscles (Fig. 55.6), where it may undergo shearing forces resulting in injury [30].

Distally, the sciatic nerve divides into the *common peroneal* and *tibial nerves*, just proximal to the knee. The sciatic nerve innervates most of the muscles of the posterior compartment of the thigh (*semitendinosus*, *semimembranosus*, and the *biceps femoris*) and is responsible for most sensorimotor functions below the knee (Table 55.2) [32].

Although this is the classic description of the sciatic nerve, Ogeng'o et al. [33] dissected 164 sciatic nerves and found variations of this classic anatomy in more than 30 % of the dissections. For instance, in 20 % of the cases that they examined, the sciatic nerve division occurred within the pelvis instead of proximal to the knee, which was described above; in those cases, the tibial nerve was always below the piriformis, while the common peroneal nerve pierced the piriformis muscle in 13 cases traveled below

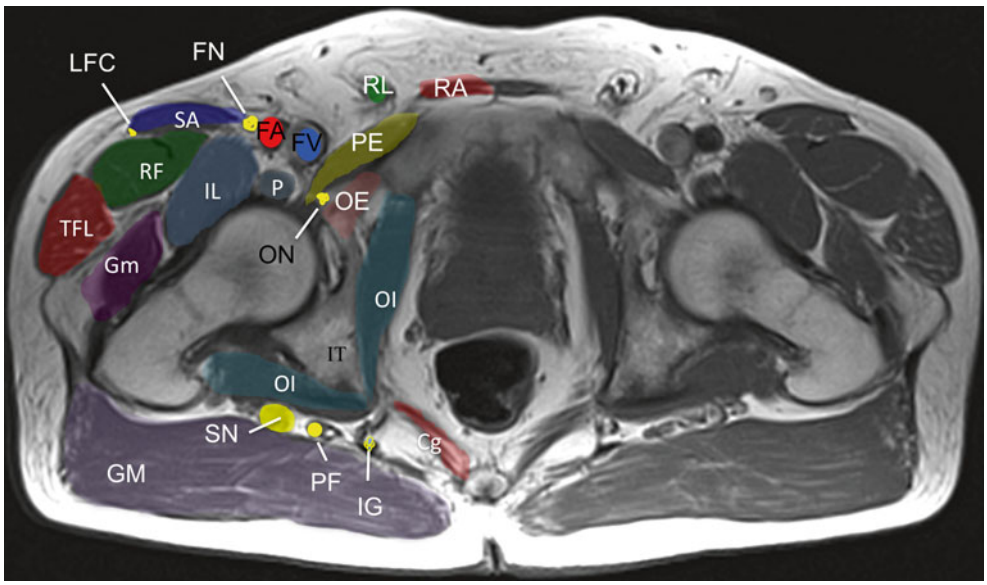

Fig. 55.4 MRI axial image of the pelvis. *Cg* coccygeus muscle, *FA* femoral artery, *FN* femoral nerve, *FV* femoral vein, *GM* gluteus maximus muscle, *Gm* gluteus medius, *IG* inferior gluteal nerve, *IL* iliopsoas muscle, *IT* ischial tuberosity, *LFC* lateral femoral cutaneous nerve, *OI* obturator internus muscle, *P* psoas muscle, *PE* pectineus muscle, *PF* posterior femoral cutaneous nerve, *QF* quadratus femoris muscle, *RA* rectus abdominis muscle, *RF* rectus femoris muscle, *RL* round ligament, *SA* sartorius muscle, *SN* sciatic nerve, *TFN* tensor fascia lata muscle (Image courtesy of Andrea Trescot, MD)

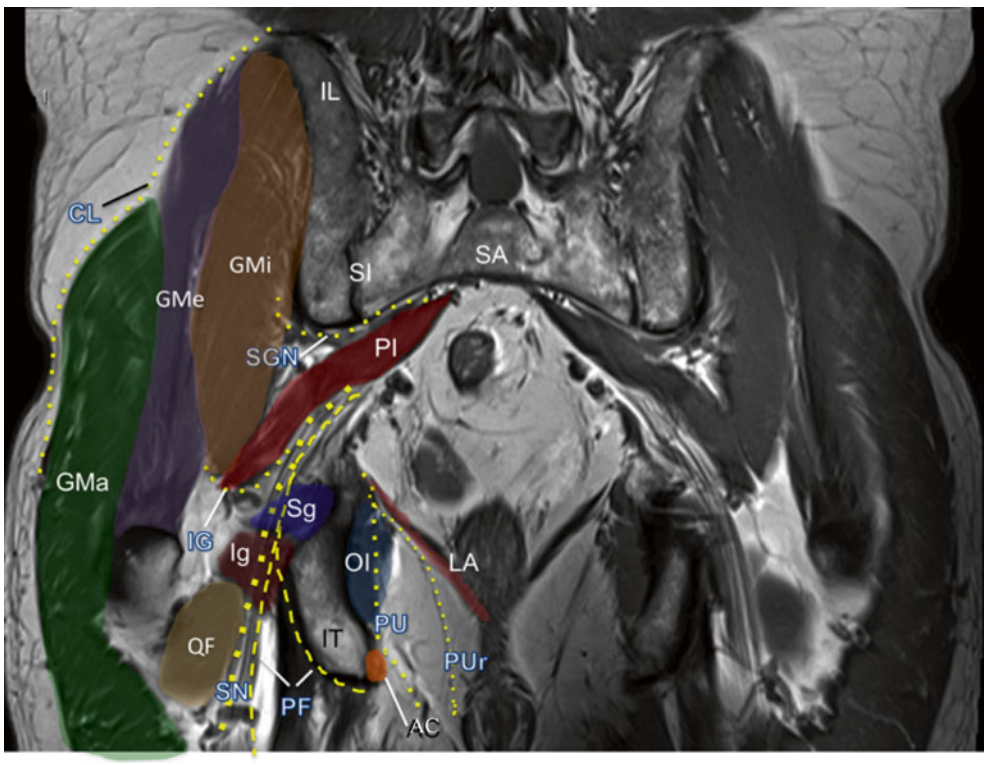

Fig. 55.5 MRI coronal images showing gluteal muscles and nerves. *AC* Alcock's canal, *CL* superior cluneal nerve, *IL* iliac crest, *IG* inferior gluteal nerve, *Ig* inferior gemellus muscle, *IT* ischial tuberosity, *Pi* piriformis, *Pu* pudendal, *GMi* gluteus minimus, *GMe* gluteus medius, *GMa* gluteus maximus, *GT* greater trochanter, *LA* levator ani muscle, *OI* obturator internus muscle, *PF* posterior femoral cutaneous nerve, *PU* pudendal nerve, *PUr* pudendal nerve (rectal branch), *QF* quadratus femoris muscle, *SA* sacrum, *SGN* superior gluteal nerve, *Sg* superior gemellus muscle, *SI* sacroiliac joint, *SN* sciatic nerve (Image courtesy of Andrea Trescot, MD)

the muscle in 16 cases and above the muscle in 4 cases. Of the 131 specimens where the sciatic nerve division was outside the pelvis, the division occurred at the popliteal fossa (as classically described) in 110 cases, but 17 occurred at the middle of the thigh, and 4 were found to divide in the gluteal region.

Entrapment

The most common SN entrapment is at the level of the piriformis muscle (Fig. 55.7). The peroneal division of the sciatic nerve is more frequently injured because its fibers are more superficial, have less supporting connective tissue, and

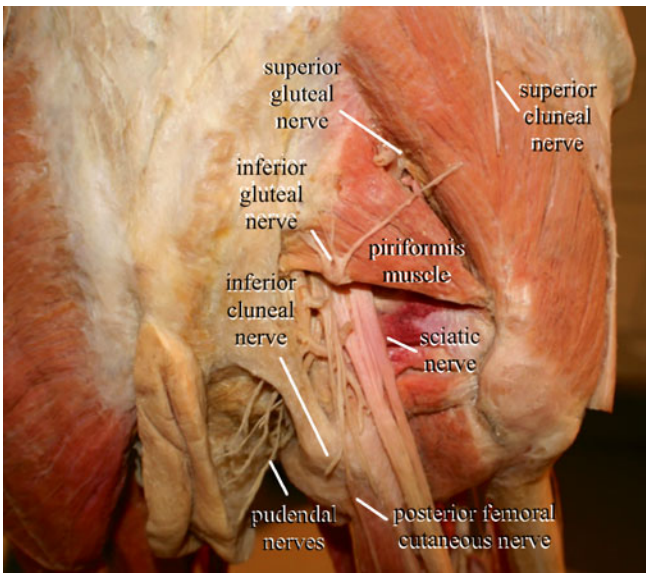

Fig. 55.6 Buttocks dissection, modified from an image from the *Bodies*, *The Exhibition*, with permission (Image courtesy of Andrea Trescot, MD)

are fixed at two points (the sciatic foramen and the fibular head), whereas the tibial division is fixed only at the sciatic foramen [24]. The piriformis may become hypertrophied secondary to extreme lumbar lordosis or hip flexion deformities, resulting in gait abnormalities. The hypertrophied piriformis may cause constriction at the greater sciatic foramen with resultant sciatic nerve compression [32]. *Piriformis syndrome* is considered a clinical diagnosis of exclusion when other potential causes of a patient's symptoms, including discogenic disease, have been ruled out.

There are various potential causes for proximal sciatic nerve entrapment that include traumatic, compressive, ischemic, neoplastic, and iatrogenic causes (Table 55.1). When examining a patient with possible sciatic nerve compression, it is important to keep in mind the possibility of an *acetabular fracture* and/or dislocation [11]. In fact, a study by Letournel and Judet reported that the highest incidence of sciatic palsy was due to posterior fracture/dislocation of the hip [34].

Probably the most common iatrogenic cause of proximal sciatic nerve entrapment occurs in the retroacetabular region secondary to total hip arthroplasty and may be attributed to a posterior surgical approach, limb lengthening, compression from a postoperative hematoma, laceration from a screw used for acetabular cup fixation (Fig. 55.8), or hardware migration [11, 32]. Other iatrogenic causes include injury from intramuscular injections; Obach et al. [35] discussed 131 cases of sciatic paralysis after injections.

Heterotopic ossification around the sciatic nerve is also a common cause of sciatic nerve entrapment in the postoperative period. Heterotopic ossification is an extra-skeletal bone formation, most often around the hip, and occurs commonly following hip arthroplasty, but it may also occur in the setting of fracture, traumatic brain injury, spinal cord injury, and burns [11, 22]. Cases have been reported where mature heterotopic bone formed around the hip and extended around the sciatic nerve, resulting in pain and weakness in the sciatic distribution [11, 36].

It is important to keep in mind other diagnoses that can result in proximal sciatic nerve entrapment, including endopelvic pathology (e.g., infiltrating *endometriosis*), metastatic tumors, or scarring from radiation therapy in those with a known cancer diagnosis [37, 38]. Infiltration of the sciatic nerve by tumors can occur from nearby pelvic bones, including the sacrum and the periacetabular region [32]. Vascular compression of the sacral plexus may also occur from local tumor infiltration.

Physical Exam

The physical examination of a possible sciatic nerve entrapment should start with inspection and palpation of the hip, buttock, and posterior thigh area, as well as passive stretch and resistance testing of the hamstring. A complete examination of the hip and lumbar spine should also take place, in conjunction with a neurological exam of the lower limb. In examining the hip and lumbar spine, range of motion testing, palpation of the sacroiliac joint, and dural tensions signs should be tested, in order to rule out other etiologies, including *disc herniation*, *facet arthropathy*, *sacroiliac joint dysfunction*, or *piriformis syndrome*. One of the diagnostic tests is the *straight leg raise* (SLR) to produce *Lasegue's sign*, which is considered positive if pain in the distribution of the sciatic nerve is reproduced with between 30 and 70° passive flexion of the straight leg (Fig. 55.9) [39]. While this test is positive in about 90 % of people with sciatic entrapment, approximately 75 % of people with a positive test do not have sciatica (resulting in a high false-positive rate) [12]. This test can also be confused with the positive SLR of radiculopathy.

If SI joint pathology is present, tenderness will be at the *posterior superior iliac spine (*PSIS) (Fig. 55.10). Other provocative maneuvers include *Freiberg's maneuver*, where the affected leg is forcefully internally rotated while the patient is in the supine position, in an attempt to stretch the piriformis and exacerbate a sciatic nerve compression. The *Beatty test* is another maneuver, where the patient lies on the

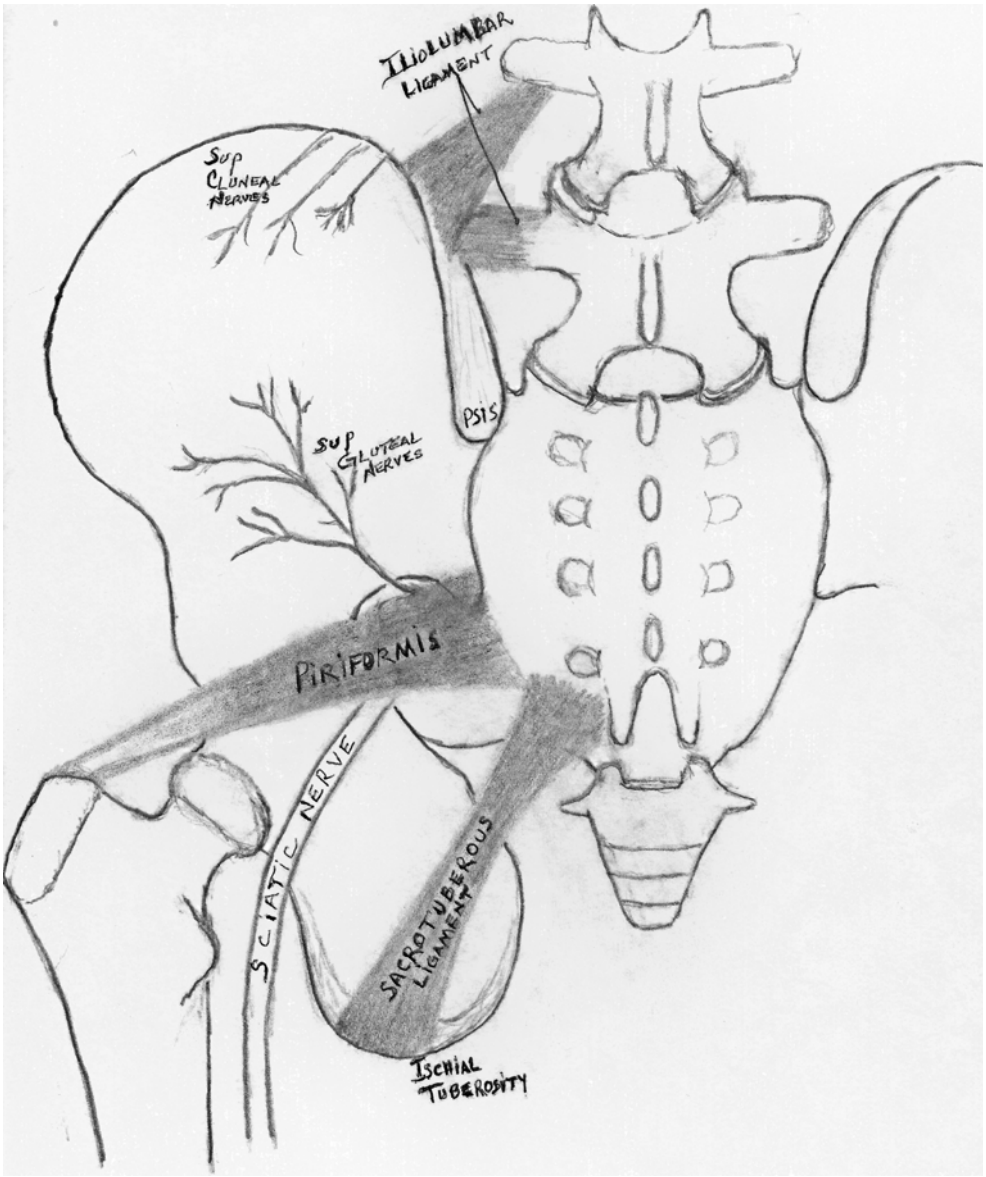

Fig. 55.7 Sites of several causes of "pseudosciatica," including piriformis entrapment of the sciatic nerve (Image drawn by Luis N. Hernandez, MD, with permission)

unaffected side with the hip and knee flexed and attempts to abduct the affected thigh. Deep buttock pain will be elicited in those with likely piriformis syndrome [40].

If there is *superior gluteal nerve* involvement, the patient will experience pain more laterally (Fig. 55.11) [30]. Piriformis tenderness [30] and spasm (Fig. 55.12) can also be a diagnostic clue. Gait should also be observed for any abnormalities consistent with weakness in the particular nerve distribution [39].

Differential Diagnosis (Table 55.3)

The history and physical examination will narrow the list of differential diagnoses (Table 55.3). If a patient is complaining of leg pain, weakness, and/or sensory disturbances, particularly in a radicular distribution, then lumbar pathology must be ruled out. Hip joint pathology, as well as hamstring tendinopathy, should also be among the differential diagnoses.

If sciatic nerve entrapment remains at the top of the differential diagnoses, it is important to localize the site of entrapment, as both proximal and distal nerve compressions can present with similar findings, including foot drop. Electrodiagnostic studies can help to differentiate between an entrapment proximally along the sciatic nerve itself or more distally along the peroneal or tibial divisions [32]. If the diagnostic tests show that proximal sciatic nerve entrapment is the cause of the patient's symptoms, then the etiology of the entrapment itself must be further explored, including traumatic, compressive, ischemic, neoplastic, and iatrogenic reasons, as described above. Both electrodiagnostic and magnetic resonance imaging can be useful in assessing the cause of symptoms (Table 55.4) [11, 32].

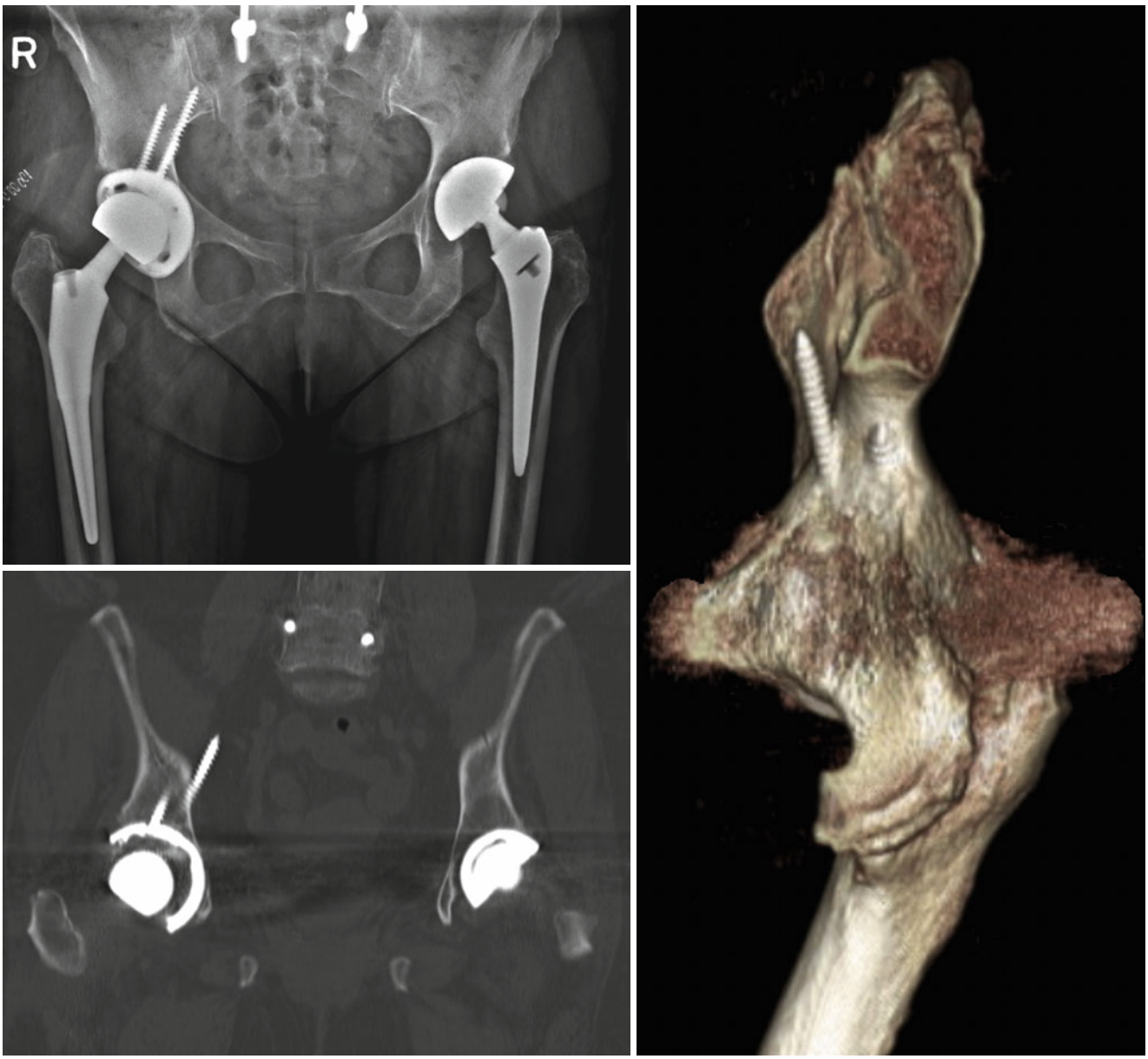

Fig. 55.8 Composite of X-ray, CT, and 3D reconstruction images of an acetabular screw (Image courtesy of Andrea Trescot, MD)

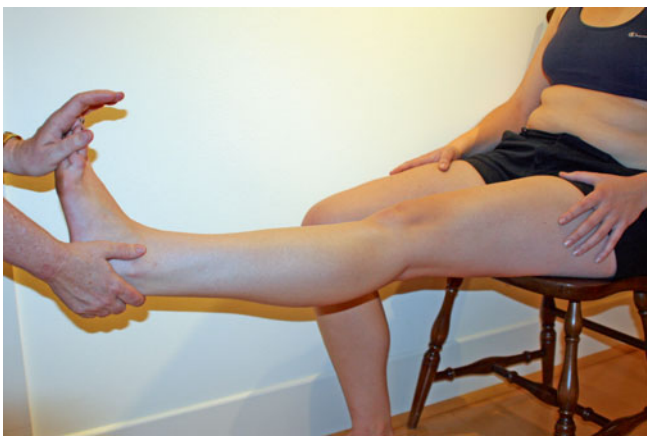

Fig. 55.9 Lasegue's sign (*straight leg raise*) (Image courtesy of Terri Dallas-Prunskis, MD)

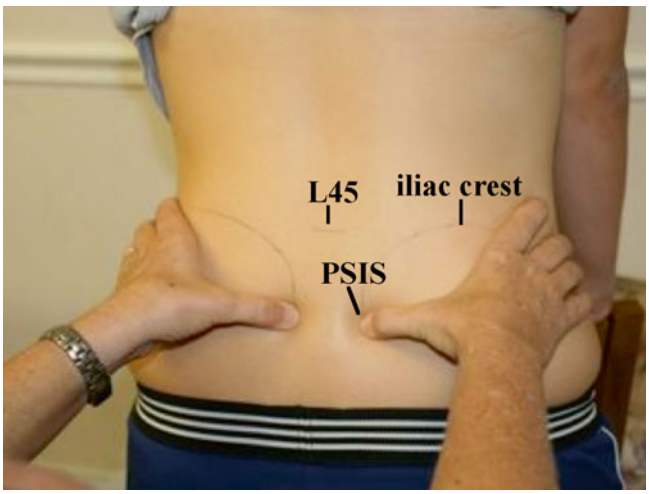

Fig. 55.10 Physical exam of the sacroiliac joint (Image courtesy of Andrea Trescot, MD)

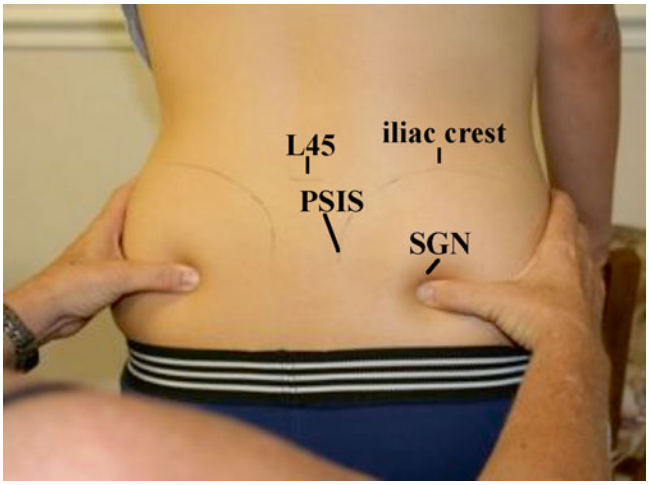

Fig. 55.11 Physical exam of the superior gluteal nerve (Image courtesy of Andrea Trescot, MD)

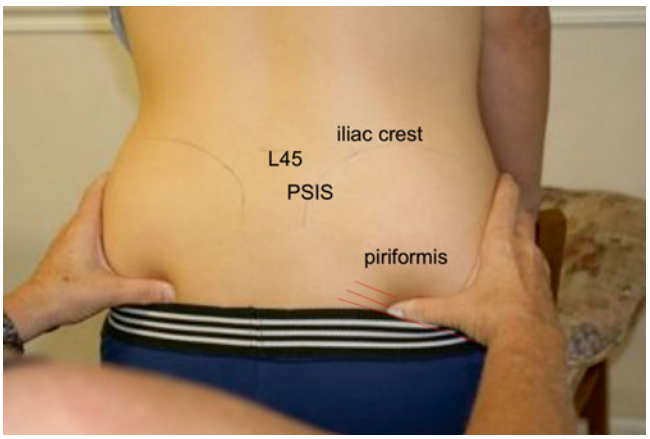

Fig. 55.12 Physical exam of the piriformis muscle (Image courtesy of Andrea Trescot, MD)

Identification and Treatment of Contributing Factors

A recognized entity known as "*wallet sciatica*" or "*fat wallet syndrome*" can cause significant sciatic nerve irritation, mimicking an actual nerve entrapment. This should prompt the clinician to ask about the patient's other habits, including frequently sitting on a hard surface [40].

High-risk occupations or jobs that require awkward positions or cause whole body vibration, such as long-distance truck driving, place workers at particular risk. Other risk factors may include *lumbar hyperlordosis* and *lumbar vertebral fractures* [51].

If a patient's sciatic nerve entrapment initially required surgical intervention, the entrapment symptoms can be perpetuated by postoperative complications such as hematoma or postoperative scarring. Sciatic nerve compression due to postoperative hematoma can result in continued symptoms [32]. While time can alleviate the hematoma-induced nerve entrapment, sometimes the hematoma must be surgically evacuated for symptom relief. Other times, the sciatic nerve can become entrapped from acetabular cup fixation or from

Table 55.3 Differential diagnosis of posterior leg pain

	Potential distinguishing features
Lumbosacral plexopathy (Chap. 66) [41]	Greater degree of weakness in the gluteal muscle, ankle dorsi, and plantar flexion
L5 radiculopathy [42]	Low back pain → posterolateral thigh
S1 radiculopathy [42]	Low back pain → posterior thigh
Common fibular neuropathy [42]	Sensory loss in the distal two third of lateral leg
Hip joint pathology [43]	Stiffness, limited range of motion; X-ray and MRI show abnormality
Hamstring tendinopathy [44]	The sciatic nerve is under compression only with certain activities; EMG is inconclusive
Piriformis muscle syndrome [40, 45]	Increased pain with hip internal rotation; taut piriformis muscle
Inferior cluneal entrapment (Chap. 63) [46]	Innervates the lateral anus and lateral region of the labium majorum
Posterior femoral cutaneous entrapment (Chap. 62) [47]	Innervates the skin of the perineum and the back surface of the thigh and leg

Table 55.4 Diagnostic tests for sciatic entrapment

	Potential distinguishing features
Physical exam	Weakness in hamstring, with foot inversion, toe flexion, possible knee flexion; decrease sensory to upper one third of the lateral leg; + SLR
Diagnostic injection	Local anesthetic is suitable to confirm a diagnosis
Ultrasound	Limited ability in diagnosis
MRI	Depending on the severity of nerve injury, a high-intensity signal in the nerve fibers or increased nerve dimension, deformation, or total loss of the nerve integrity may be seen [48]. Affected muscles may show fatty infiltration, edema, and atrophy [49]
CT Scan	Reveals lesions that impact the sciatic nerve involving bone (e.g., sacrum fractures) and vessel abnormalities (e.g., aneurysm) or hematoma [50]
Arteriography	Not helpful
X-ray	To rule out other causes
Electrodiagnostic studies	Needle EMG consistent with denervation depending on the lesion site: short and long heads of the biceps femoris, semimembranosus, and semitendinosus [42]

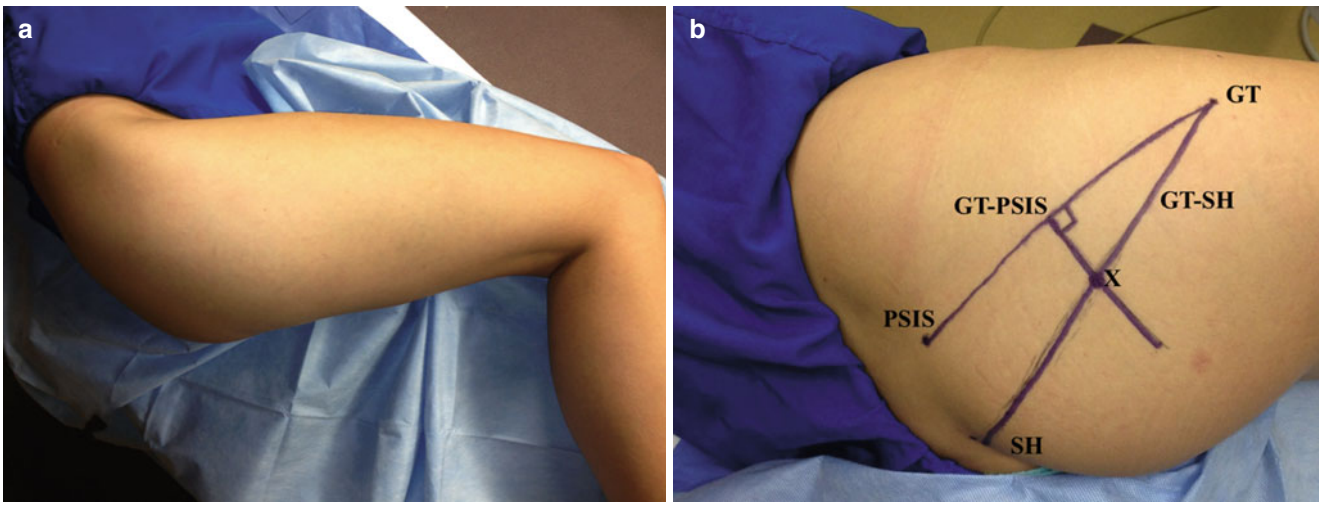

Fig. 55.13 Landmark-guided sciatic nerve injection – classic/posterior/transgluteal approach. (**a**) positioning; (**b**) injection site. *GT* greater trochanter, *PSIS* posterior superior iliac spine, *SH* sacral hiatus (Image courtesy of Terri Dallas-Prunskis, MD)

hardware migration, even years after the initial hip arthroplasty [32]. Hip revision surgery is required if the hardware is the perpetrator. Following surgical intervention, particularly at the hip joint, patients can also develop heterotopic ossification that can result in sciatic nerve entrapment. Surgical removal of the mature heterotopic bone is required to alleviate this etiology of nerve entrapment [11].

Surgical intervention is also the reported treatment for sciatic entrapment by the hamstring tendons and entrapment by a proximal sciatic nerve intraneural ganglion cyst [19]. In the cases of vascular malformations, Van Gompel et al. [52] described external neurolysis and limited microvascular dissection of the sciatic nerve and its terminal branches.

Entrapment of the sciatic nerve by the piriformis muscle will cause persistent pathology. Kirschner et al. described the use of myofascial release, trigger point massage, and possible trigger point injections [40]. Botulinum toxin has been used to alleviate the piriformis entrapment syndrome [53].

Injection Technique

Landmark-Guided Injection

For both diagnostic and anesthetic purposes, a proximal sciatic nerve block may be performed blindly in the gluteal region. Landmarks for the injection should be determined first.

In the *classic or posterior approach (Labat)*, the patient is in the lateral position with the extremity to be blocked flexed maximally at the hip and slightly at the knee. The gluteal region, the greater trochanter (GT) of the affected side, the posterior superior iliac spine (PSIS), the sacral hiatus, and the ischial tuberosity (IT) are identified by palpation (Fig. 55.13). Utilizing an aseptic technique, a line is drawn between the greater trochanter and the PSIS (from the GT to the PSIS). A second line is drawn between the greater trochanter and the sacral hiatus (from the GT to the SH). Then, a perpendicular line is drawn that bisects the first line and should extend thru the second line, which is the injection site (X). The needle should be inserted at the marked point, perpendicular to the skin in all planes, and advanced until paresthesia are noted. If a nerve stimulator is utilized, search for movement in the leg and foot [54].

The *subgluteal or subtrochanteric approach* does not require painful repositioning and can be used, for example, in patients who may have experienced trauma or fracture to the limb. The patient is placed lateral or prone, and the leg is in a neutral position or rotated slightly inward. Palpate and mark the greater trochanter and ischial tuberosity (Fig. 55.14). A line is drawn joining the greater trochanter and the ischial tuberosity. From the midpoint of this line, a perpendicular line is extended caudally about 4 cm. At this level, a skin depression is palpated, representing the groove between the biceps femoris and the semitendinosus muscles. This point marks the site of the introduction of a 22-gauge 100 mm needle. Nerve stimulation should elicit plantar or dorsiflexion of the foot at less than 0.5 mV [55].

With the *parasacral approach*, a block is performed in the sacral plexus after its emergence from the greater sciatic foramen. This technique, however, will block the sciatic nerve as well as the *superior gluteal nerve* (see Chap. 53), *the inferior gluteal nerve* (see Chap. 54), *the posterior cutaneous nerve of the thigh* (see Chap. 56), and the pudendal nerve (Chap. 47). The patient is positioned in the lateral or prone position. The anatomic landmarks are the posterior superior iliac spine and the ischial tuberosity (Fig. 55.15). A point is marked 7 cm distal from the posterior iliac spine and

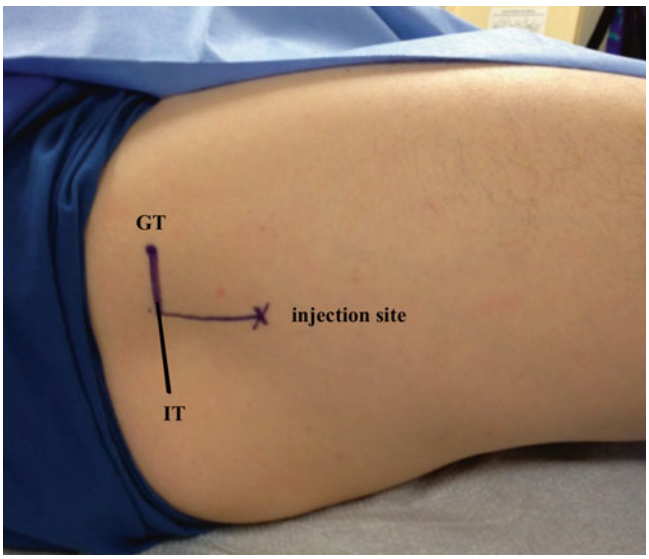

Fig. 55.14 Landmark-guided sciatic nerve injection – subgluteal/subtrochanteric approach. *GT* greater trochanter, *IT* ischial tuberosity (Image courtesy of Terri Dallas-Prunskis, MD)

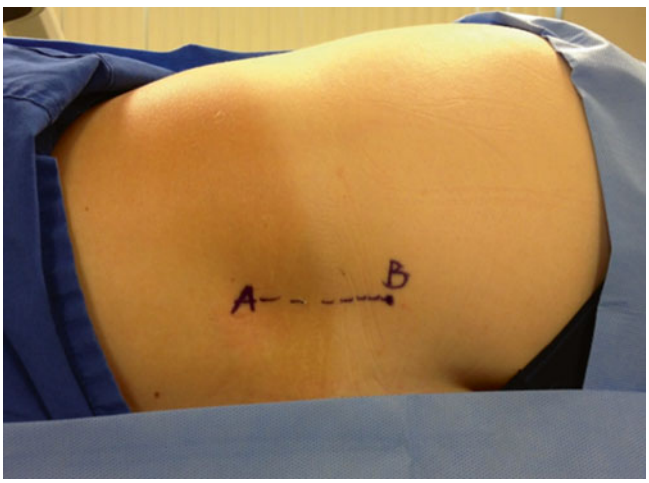

Fig. 55.15 Landmark-guided sciatic nerve injection – parasacral approach. *A* PSIS (posterior superior iliac spine), *B* ischial tuberosity (Image courtesy of Terri Dallas-Prunskis, MD)

a 22-gauge 100 mm needle is introduced perpendicular to the skin and advanced 6–8 cm. If the bone is contacted, the needle is reintroduced along a line more caudally until it misses the bone [55].

Ripart et al. [56] prospectively evaluated 400 patients undergoing sciatic nerve blocks from a parasacral approach; the mean time to perform the procedure was 7 ± 5 min, with a 96 % success rate.

Another option is the *anterior approach (Beck)* (Fig. 55.16). With the patient in the supine position, a line (A) is drawn between the lower border of the anterior superior iliac spine (ASIS) and the pubic tubercle (PT),

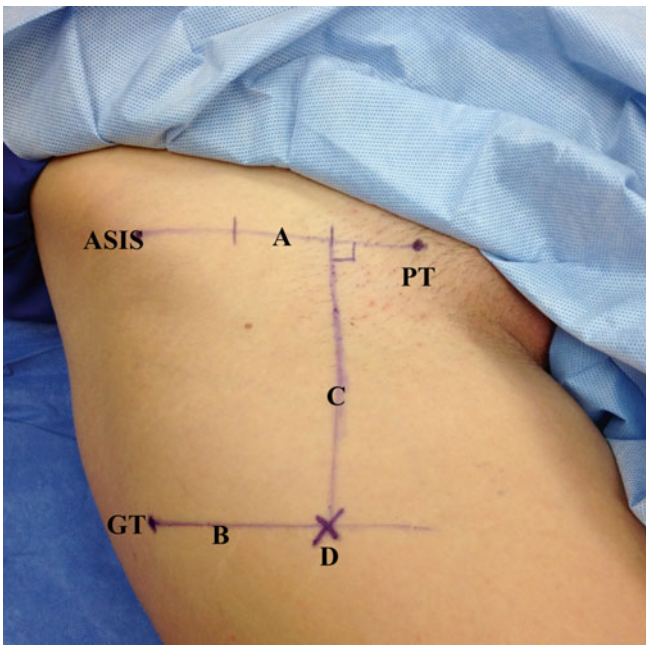

Fig. 55.16 Landmark-guided sciatic nerve injection – anterior approach. *ASIS* anterior superior iliac spine, *GT* greater trochanter, *PT* pubic tubercle (Image courtesy of Terri Dallas-Prunskis, MD)

representing the inguinal ligament. This line is then divided into three thirds. A second line (B) is drawn through the proximal pole of the greater trochanter (GT). A third line (C), perpendicular to both (A) and (B) and crossing (A) at the junction between the medial third and lateral two thirds, is drawn. The intersection between (B) and (C), point (D), is the needle insertion point. Utilizing a 12- or 15-cm insulated needle connected to a nerve stimulator, advance the needle perpendicular to the floor, through aseptic skin and the quadriceps muscle. Typically, the lesser trochanter will be encountered at a depth around 7–8 cm in an average-sized person. The needle should be withdrawn 1–2 cm and walked medially until bone contact is lost. The sciatic nerve will be encountered 2–3 cm deeper than the bone depth, i.e., between 9 and 12 cm from the skin. Sometimes the nerve is stimulated without having contacted the bone. A response to stimulation distal to the knee, either in the tibial or peroneal innervation territory, is acceptable [57].

Fluoroscopic-Guided Injection

Under fluoroscopic guidance, a sciatic nerve block can be performed at the sciatic notch. The patient is placed in a prone position, utilizing the same sterile precautions described above, and a 22-gauge 3.5 in. spinal needle is advanced toward the sciatic notch, where the sciatic nerve crosses the pelvic rim, parallel to the fluoroscopy beam (Fig. 55.17). As the needle tip comes into close proximity

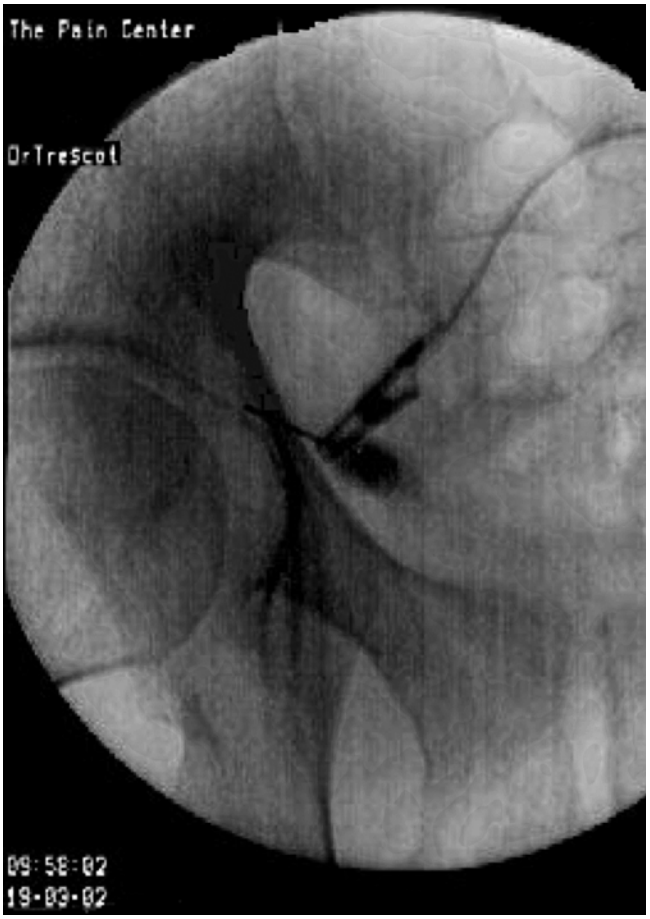

Fig. 55.17 Fluoroscopic sciatic nerve injection (Image courtesy of Andrea Trescot, MD)

with the sciatic nerve, a paresthesia may be elicited or the procedure can be performed with peripheral nerve stimulation for confirmation of needle placement. Radiopaque contrast dye may be injected for the purpose of outlining the sciatic nerve [58].

Ultrasound-Guided Injection

Using ultrasound guidance, the sciatic nerve may be blocked from a gluteal approach or from a proximal thigh approach. For the gluteal approach, the technique is the same as when the injection is performed using landmarks (as above), with the added benefit of being able to visualize the path of the needle and the injectate in real time. The sciatic nerve is found medial to the greater trochanter and lateral to the ischium, usually deep to the gluteus maximus and piriformis muscles (Fig. 55.18). Using an in-plane approach, an 8 cm 25-gauge or 22-gauge insulated block needle is inserted adjacent to the lateral aspect of the ultrasound transducer, approximately 3 cm below the midpoint of the greater trochanter and the PSIS. After local anesthetic infiltration of the skin and under sterile precautions, the needle is then advanced along the long axis of the transducer until the needle reaches the area around the sciatic nerve, usually deep to the piriformis muscle. After negative aspiration for blood, the local anesthetic spread can then be observed [59].

Regardless of the approach, visualizing the sciatic nerve with ultrasound guidance can be challenging. The sciatic nerve can also be targeted using an in-plane approach and the anterior thigh technique discussed above. The thigh should be externally rotated in order to laterally displace the femoral neurovasculature. An 8–12 cm 22-gauge insulated block needle is utilized, depending on the thickness and depth of the patient's thigh muscles. Under sterile precautions and after local skin anesthetic, the needle is inserted adjacent to the medial aspect of the ultrasound transducer, medial to the femoral vessels. The needle is then advanced along the transducer beam and moved posteriorly from medial to lateral. Again, needle placement can be confirmed with the use of peripheral nerve stimulation [60].

If an anterior sciatic nerve block approach is performed, utilizing the out-of-plane technique, the sciatic nerve should be aligned at the midpoint of the transducer, and the block needle should be inserted at that same location. Needle placement may be confirmed with peripheral nerve stimulation and visualization of local anesthetic around the targeted nerve [59].

Neurolytic Technique

The sciatic nerve is predominately a motor nerve, with branches of sensory innervation. Direct ablation of the sciatic nerve would cause significant loss of motor function; thus, ablation of the sciatic nerve directly would only be used for very rare situations where the motor function is not needed or could be sacrificed, such as for lower extremity phantom limb pain. Branches of the sciatic nerve, such as the superior gluteal nerve, have both sensory and motor innervation, but since the muscular innervation is not as significant clinically, these branches can be targeted for pain relief.

Cryoneuroablation

Trescot described the technique for cryoneurolysis of the superior gluteal branch of the sciatic nerve. The patient is placed in a prone position during the procedure, and the medial border of the ilium is palpated. A point approximately 5 cm inferior and lateral to the attachment site of the gluteus medius is identified. Under sterile precautions, local skin anesthesia is given. A 12-gauge intravenous catheter is used as an introducer for a 2.0 mm cryoprobe. The probe is

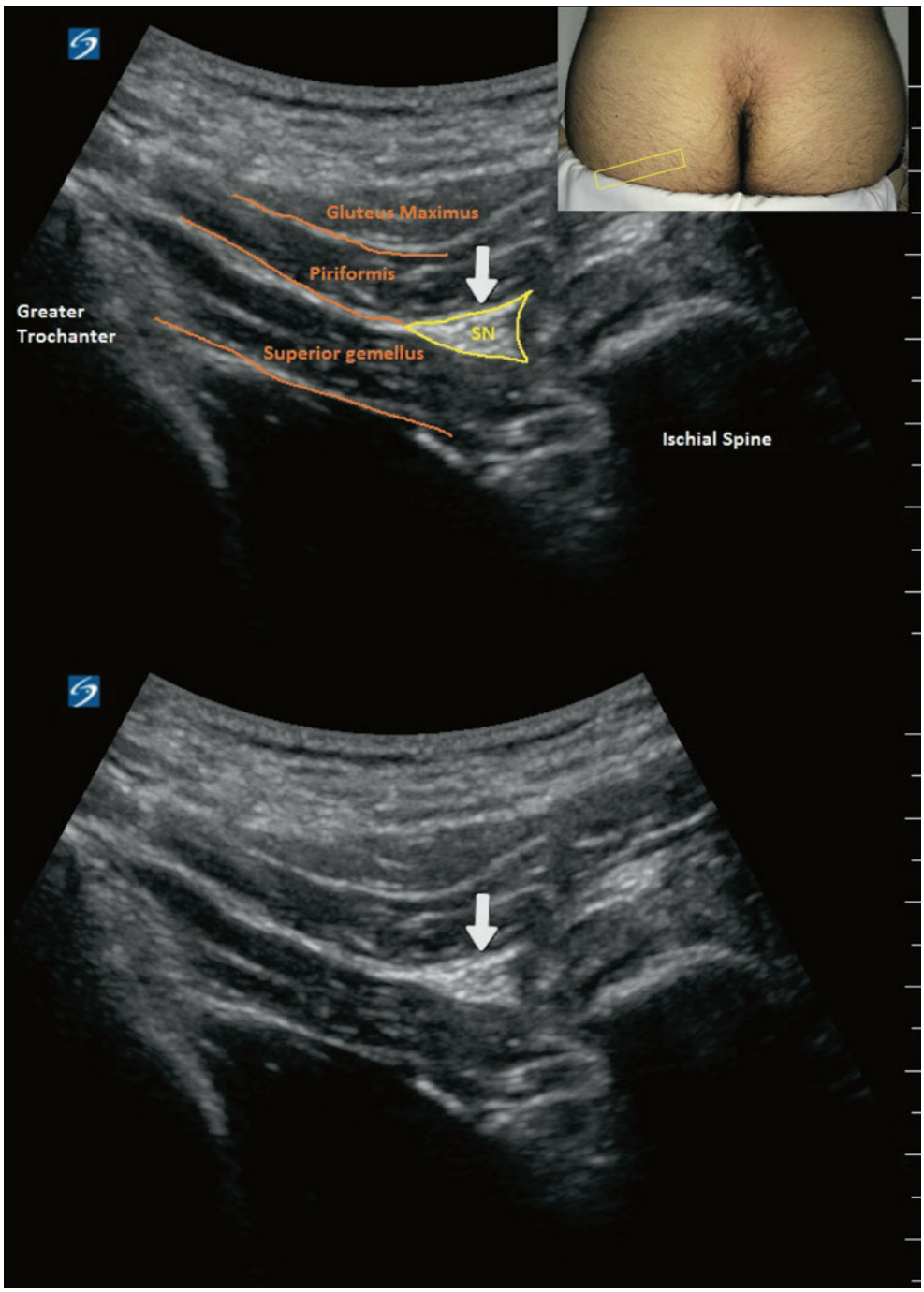

Fig. 55.18 Ultrasound localization of the sciatic nerve (Image courtesy of Agnes Stogicza, MD)

advanced in an inferomedial (Fig. 55.19) or superolateral direction. Peripheral nerve stimulation is used to confirm placement of the needle tip and to avoid the motor nerves (e.g., sciatic nerve) [30].

Radiofrequency (RF) Lesioning

Conventional radiofrequency lesioning of large myelinated nerves such as the sciatic nerve cannot be recommended. However, pulsed RF has been used for denervation of the sciatic nerve to treat chronic knee pain [61]. The target is identified and the site sterilely prepped. After the skin is anesthetized subcutaneously, the radiofrequency cannula is advanced to the target site at the iliac crest using fluoroscopy, US, or anatomical landmarks. After the radiofrequency probe is advanced through the cannula appropriately, maximal sensory and devoid motor stimulation is used to confirm that the tip of the probe is placed adequately.

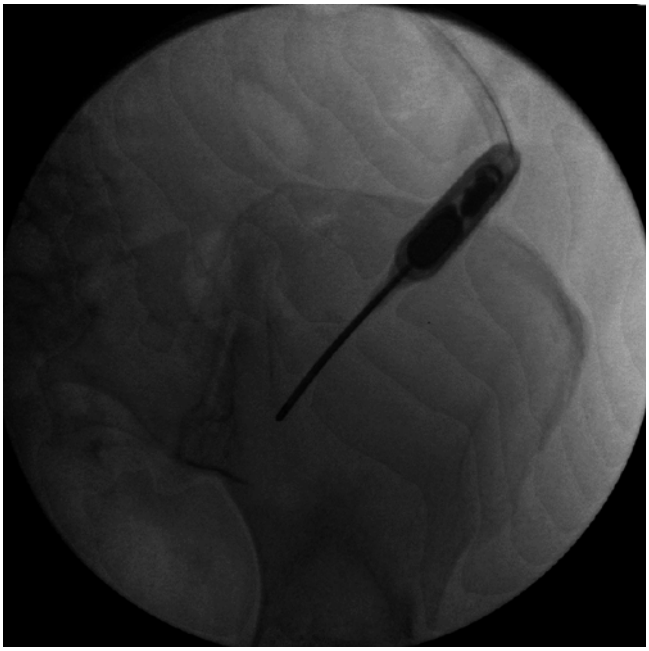

Fig. 55.19 Cryoneuroablation of the superior gluteal nerve (Image courtesy of Andrea Trescot, MD)

Surgical Technique

Depending on the etiology, sometimes a surgical intervention is required for the alleviation of proximal sciatic nerve entrapment. If the cause is an acetabular fracture, then surgical fixation can decompress the nerve [32]. If posterior thigh compartment syndrome is the etiology, fasciotomy is necessary [32, 62]. In endopelvic pathology, laparoscopic therapy for infiltrating endometriosis may be performed to alleviate vascular entrapment of the sacral plexus [37]. Pelvic tumor excision may need to be performed if the tumor or metastasis is infiltrating the nerve. With any hip surgery, the possibility of the patient developing heterotopic ossification should be kept in mind. If this occurs, surgical excision of heterotopic bone is performed, especially if it is causing entrapment of the sciatic nerve [11]. If piriformis syndrome is diagnosed and if conservative therapy, including physical therapy, anti-inflammatory medications, and piriformis injection, has failed to alleviate symptoms, the patient may be a candidate for surgical release of the sciatic nerve proximally [63]. It is important to note that the very reason for the surgical intervention may result in further sciatic nerve entrapment.

Complications

Any therapeutic injection of the sciatic nerve can result in nerve injury, due to direct trauma, nerve stretching, or ischemic compression [64]. MRI with contrast of the injured region may show the extent of the nerve injury [65]. There is a significant risk of life-threatening arterial puncture, hematoma formation, and localized bleeding because of the close proximity of the inferior gluteal artery to the sciatic nerve when using the transgluteal approach [66]. With the anterior approach, vessel puncture of the femoral artery and profunda femoris arteries, as well as neural injury to the femoral nerve or its branches, may occur [67].

Summary

Etiologies of proximal sciatic neuropathy outside of the neural foramina are uncommon. A review of the anatomy is essential for the understanding of the biological basis of its various injuries.

References

1. Vilos GA, Vilos AW, Haebe JJ. Laparoscopic findings, management, histopathology, and outcome of 25 women with cyclic leg pain. J Am Assoc Gynecol Laparosc. 2002;9(2):145–51.
2. Bodack MP, Cole JC, Nagler W. Sciatic neuropathy secondary to a uterine fibroid: a case report. Am J Phys Med Rehabil. 1999;78(2):157–9.
3. Silva M, Mallinson C, Reynolds F. Sciatic nerve palsy following childbirth. Anaesthesia. 1996;51(12):1144–8.
4. Goh KJ, Tan CB, Tjia HT. Sciatic neuropathies – a retrospective review of electrodiagnostic features in 29 patients. Ann Acad Med Singap. 1996;25(4):566–9.
5. Chatillon CE, Guiot MC, Jacques L. Lipomatous, vascular, and chondromatous benign tumors of the peripheral nerves: representative cases and review of the literature. Neurosurg Focus. 2007;22(6):E18.
6. Ricci S, Georgiev M, Jawien A, Zamboni P. Sciatic nerve varices. Eur J Vasc Endovasc Surg: Off J Eur Soc Vasc Surg. 2005; 29(1):83–7.
7. Fischer SR, Christ DJ, Roehr BA. Sciatic neuropathy secondary to total hip arthroplasty wear debris. J Arthroplasty. 1999;14(6):771–4.
8. Schmalzried TP, Amstutz HC, Dorey FJ. Nerve palsy associated with total hip replacement. Risk factors and prognosis. J Bone Joint Surg Am. 1991;73(7):1074–80.
9. Weil Y, Mattan Y, Goldman V, Liebergall M. Sciatic nerve palsy due to hematoma after thrombolysis therapy for acute pulmonary embolism after total hip arthroplasty. J Arthroplasty. 2006;21(3): 456–9.
10. Kemler MA, de Vries M, van der Tol A. Duration of preoperative traction associated with sciatic neuropathy after hip fracture surgery. Clin Orthop Relat Res. 2006;445:230–2.
11. Issack PS, Helfet DL. Sciatic nerve injury associated with acetabular fractures. HSS J: Musculoskelet J Hosp Spec Surg. 2009;5(1): 12–8.
12. Valat JP, Genevay S, Marty M, Rozenberg S, Koes B. Sciatica. Best practice & research. Clin Rheumatol. 2010;24(2):241–52.
13. Robinson DR. Pyriformis syndrome in relation to sciatic pain. Am J Surg. 1947;73(3):355–8.
14. Wong M, Vijayanathan S, Kirkham B. Sacroiliitis presenting as sciatica. Rheumatol (Oxf Engl). 2005;44(10):1323–4.
15. Holland NR, Schwartz-Williams L, Blotzer JW. "Toilet seat" sciatic neuropathy. Arch Neurol. 1999;56(1):116.

16. Scherer K, Skeen MB, Strine SA, Juel VC. Hanging leg syndrome: combined bilateral femoral and sciatic neuropathies. Neurology. 2006;66(7):1124–5.
17. Gozal Y, Pomeranz S. Sciatic nerve palsy as a complication after acoustic neurinoma resection in the sitting position. J Neurosurg Anesthesiol. 1994;6(1):40–2.
18. Wilbourn AJ, Mitsumoto H. Proximal sciatic neuropathies caused by prolonged sitting. Neurology. 1988;38:400.
19. Swartz KR, Wilson D, Boland M, Fee DB. Proximal sciatic nerve intraneural ganglion cyst. Case reports in medicine. 2009:on line.
20. Ramtahal J, Ramlakhan S, Singh K. Sciatic nerve injury following intramuscular injection: a case report and review of the literature. J Neurosci Nurs: J Am Assoc Neurosci Nurs. 2006;38(4):238–40.
21. Shields Jr RW, Root Jr KE, Wilbourn AJ. Compartment syndromes and compression neuropathies in coma. Neurology. 1986; 36(10):1370–4.
22. Safaz I, Alaca R, Bozlar U, Yasar E. Bilateral sciatic nerve entrapment due to heterotopic ossification in a traumatic brain-injured patient. Am J Phys Med Rehabil. 2008;87(1):65–7.
23. Botwin KP, Shah CP, Zak PJ. Sciatic neuropathy secondary to infiltrating intermuscular lipoma of the thigh. Am J Phys Med Rehabil. 2001;80(10):754–8.
24. Yuen EC, So YT. Sciatic neuropathy. Neurol Clin. 1999;17(3):617–31, viii.
25. Mishra P, Stringer MD. Sciatic nerve injury from intramuscular injection: a persistent and global problem. Int J Clin Pract. 2010;64(11):1573–9.
26. Jung Kim H, Hyun PS. Sciatic nerve injection injury. J Int Med Res. 2014;42(4):887–97.
27. Shahid KR, Dellon AL, Amrami KK, Spinner RJ. Sciatic and peroneal nerve injuries after endovascular ablation of lower extremity varicosities: case reports and review of the literature. Ann Plast Surg. 2015;74(1):64–8.
28. Dong Q, Jacobson JA, Jamadar DA, Gandikota G, Brandon C, Morag Y, et al. Entrapment neuropathies in the upper and lower limbs: Anatomy and MRI features. Radiol Res Pract. 2012:on line.
29. Hirose CB, McGarvey WC. Peripheral nerve entrapments. Foot Ankle Clin. 2004;9(2):255–69.
30. Trescot AM. Cryoanalgesia in interventional pain management. Pain Phys. 2003;6(3):345–60.
31. Sunderland S. Nerves and nerve injuries. 2nd ed. London: Churchill Livingstone; 1978.
32. Martinoli C, Miguel-Perez M, Padua L, Gandolfo N, Zicca A, Tagliafico A. Imaging of neuropathies about the hip. Eur J Radiol. 2011;82(1):17–26.
33. Ogeng'o JA, El-Busaidy H, Mwika PM, Khanbhai MM, Munguti J. Variant anatomy of sciatic nerve in a black Kenyan population. Folia Morphol (Warsz). 2011;70(3):175–9.
34. Letournel E, Judet R. Early complications of operative treatment within three weeks of injury. In: Elso RA, editor. Fractures of the acetabulum. 2nd ed. Berlin: Springer; 1993. p. 535–40.
35. Obach J, Aragones JM, Ruano D. The infrapiriformis foramen syndrome resulting from intragluteal injection. J Neurol Sci. 1983;58(1):135–42.
36. Thakkar DH, Porter RW. Heterotopic ossification enveloping the sciatic nerve following posterior fracture-dislocation of the hip: a case report. Injury. 1981;13(3):207–9.
37. Possover M, Schneider T, Henle KP. Laparoscopic therapy for endometriosis and vascular entrapment of sacral plexus. Fertil Steril. 2011;95(2):756–8.
38. Pradat PF, Bouche P, Delanian S. Sciatic nerve mononeuropathy: an unusual late effect of radiotherapy. Muscle Nerve. 2009;40(5):872–4.
39. Speed C. Low back pain. BMJ. 2004;328(7448):1119–21.
40. Kirschner JS, Ben-Meir RS, Diamond J. Piriformis syndrome. In: Wyss JF, Patel AD, editors. Therapeutic programs for musculoskeletal disorders. 1st ed. New York: Demos; 2013. p. 363–9.
41. Katirji B. Electromyography in clinical practice – a case study approach. St. Louis: Mosby; 1998.
42. Distad BJ, Weiss MD. Clinical and electrodiagnostic features of sciatic neuropathies. Phys Med Rehabil Clin N Am. 2013; 24(1):107–20.
43. Nguyen T, Sheikh K. Sciatic neuropathy: Pathogenesis and pathophysiology 2011 [10/14/14]. Available from: http://www.medmerits.com/index.php/article/sciatic_neuropathy.
44. Saikku K, Vasenius J, Saar P. Entrapment of the proximal sciatic nerve by the hamstring tendons. Acta Orthop Belg. 2010;76(3):321–4.
45. Vallejo MC, Mariano DJ, Kaul B, Sah N, Ramanathan S. Piriformis syndrome in a patient after cesarean section under spinal anesthesia. Reg Anesth Pain Med. 2004;29(4):364–7.
46. Darnis B, Robert R, Labat JJ, Riant T, Gaudin C, Hamel A, et al. Perineal pain and inferior cluneal nerves: anatomy and surgery. Surg Radiol Anat. 2008;30(3):177–83.
47. Chutkow JG. Posterior femoral cutaneous neuralgia. Muscle Nerve. 1988;11:1146–8.
48. Maravilla KR, Bowen BC. Imaging of the peripheral nervous system: evaluation of peripheral neuropathy and plexopathy. AJNR Am J Neuroradiol. 1998;19(6):1011–23.
49. Chhabra A, Chalian M, Soldatos T, Andreisek G, Faridian-Aragh N, Williams E, et al. 3-T high-resolution MR neurography of sciatic neuropathy. AJR Am J Roentgenol. 2012;198(4):W357–64.
50. Ergun T, Lakadamyali H. CT and MRI in the evaluation of extraspinal sciatica. Br J Radiol. 2010;83(993):791–803.
51. Curlee PM. Other disorders of the spine. In: Canale ST, Beatty JH, editors. Campbell's operative orthopaedics. Philadelphia: Mosby Elsevier; 2007.
52. Van Gompel JJ, Griessenauer CJ, Scheithauer BW, Amrami KK, Spinner RJ. Vascular malformations, rare causes of sciatic neuropathy: a case series. Neurosurgery. 2010;67(4):1133–42; discussion 42.
53. Fishman LM, Anderson C, Rosner B. BOTOX and physical therapy in the treatment of piriformis syndrome. Am J Phys Med Rehabil. 2002;81(12):936–42.
54. Stein BE, Srikumaran U, Tan EW, Freehill MT, Wilckens JH. Lower-extremity peripheral nerve blocks in the perioperative pain management of orthopaedic patients: AAOS exhibit selection. J Bone Joint Surg Am. 2012;94(22):e167.
55. Taboada M, Rodriguez J, Valino C, Carceller J, Bascuas B, Oliveira J, et al. What is the minimum effective volume of local anesthetic required for sciatic nerve blockade? A prospective, randomized comparison between a popliteal and a subgluteal approach. Anesth Analg. 2006;102(2):593–7.
56. Ripart J, Cuvillon P, Nouvellon E, Gaertner E, Eledjam JJ. Parasacral approach to block the sciatic nerve: a 400-case survey. Reg Anesth Pain Med. 2005;30(2):193–7.
57. Atchabahian A. Sciatic nerve block: anterior approach using neurostimulator without ultrasound guidance. JNYSORA. 2009;12:24–7.
58. Thota RS, Aravind A, Mahapatro SB, Anantsingh R, Gvalani S, Dewoolkar LV. The anterior approach to sciatic nerve block using fluoroscopic guidance and radio-opaque dye. Anesth Analg. 2007;104(2):461–2.
59. Sites BD, Antonakakis JG. Ultrasound guidance in regional anesthesia: state of the art review through challenging clinical scenarios. Local Reg Anesth. 2009;2:1–14.
60. Barrington MJ, Lai SL, Briggs CA, Ivanusic JJ, Gledhill SR. Ultrasound-guided midthigh sciatic nerve block-a clinical and anatomical study. Reg Anesth Pain Med. 2008;33(4):369–76.

61. Fucci RED, Pascual-Ramirez J, Martinez-Marcos A, Mantecon JM. Ultrasound-guided sciatic nerve pulsed radiofrequency for chronic knee pain treatment: a novel approach. J Anesth. 2013;27(6):935–8.
62. Mallo GC, Stanat SJ, Al-Humadi M, Divaris N. Posterior thigh compartment syndrome as a result of a basketball injury. Orthopedics. 2009;32(12):923.
63. Kobbe P, Zelle BA, Gruen GS. Case report : recurrent piriformis syndrome after surgical release. Clin Orthop Relat Res. 2008;466(7):1745–8.
64. Borgeat A, Blumenthal S. Nerve injury and regional anaesthesia. Curr Opin Anaesthesiol. 2004;17(5):417–21.
65. Pham M, Wessig C, Brinkhoff J, Reiners K, Stoll G, Bendszus M. MR neurography of sciatic nerve injection injury. J Neurol. 2011;258(6):1120–5.
66. Charous MT, Mariano ER. Proximal sciatic nerve blocks. In: Chu LF, Fuller AJ, editors. Manual of clinical anesthesiology. 1st ed. Philadelphia: Lippincott Williams & Wilkins; 2012.
67. Shah S, Hadzic A, Vloka JD, Cafferty MS, Moucha CS, Santos AC. Neurologic complication after anterior sciatic nerve block. Anesth Analg. 2005;100(5):1515–7, table of contents.

Posterior Femoral Cutaneous Nerve Entrapment: Low Back

56

Natalia Murinova, Daniel Krashin, and Andrea M. Trescot

Introduction

Posterior femoral cutaneous nerve (PFCN) neuropathy is an uncommon, potentially under-recognized cause of low back pain, as well as posterior and lateral thigh pain and numbness. The PFCN provides sensation to the posterior thigh and perineal region, mimicking sciatic and pudendal nerve pathologies. An isolated PFCN neuropathy is rare. In the literature, the most commonly reported causes of PFCN injury have been intramuscular injections or pressure injuries, as from a thigh pressure cuff during knee surgery. The alternate names for this nerve and syndrome include *lesser sciatic nerve* or *posterior cutaneous nerve of the thigh*. The perineal branch of the PFCN has been called the *inferior pudendal nerve*, *pudendal longus inferior*, *long pudendal nerve*, or *nerve of Soemmering*. Consider PFCN entrapment neuropathy in a patient presenting with posterior and medial thigh pain.

Clinical Presentation (Table 56.1)

A patient with PFCN entrapment has posterior buttocks and thigh pain, from the gluteal fold to the back of the knee, and includes a variable amount of the posterior calf (Fig. 56.1). There can be sensory abnormalities in the lower buttock and posterior thigh (Fig. 56.2). Through its perineal branch, the PFSN also innervates the perineal region (Fig. 56.3). PFCN injury has been reported after intramuscular gluteal injections [1]. This nerve is also traumatized by prolonged bicycle rides, pelvic tumors, venous malformation, and pressure on the nerve at the inferior margin of the gluteus maximus, due to sitting on hard surfaces (Table 56.1) [2–4]. It can also present as perineal pain in the rectum, scrotum/labia majora, and/or penis/clitoris. The pain down the back of the leg after intraoperative use of a thigh cuff (such as for knee surgery) may represent PFCN pathology. Isolated lesions of the PFCN are described in a small number of individual case studies (Table 56.2).

Table 56.1 Clinical presentation

Branches of the PFCN	Site of sensory innervation and skin pain
Major branch of PFCN	Back and medial side of the thigh
	Popliteal fossa
	Upper part of the back of the leg
Inferior cluneal nerve of PFCN	Inferior buttocks
Perineal branch of PFCN	Lateral perineum
	Upper and medial thigh
	Posterolateral scrotum
	Labium majora
	Part of the penis/clitoris
Gluteal branches of PFCN	Lower and lateral part of gluteus maximus
Other name: inferior cluneal nerves	
Collateral branches from the anterior divisions	Quadratus femoris
	Inferior gemellus muscles (L4, L5, S1)
	Internal obturator
	Superior gemellus muscles (L5, S1, S2)

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Fig. 56.1 Patient pain complaint from posterior femoral cutaneous nerve entrapment (Image courtesy of Andrea Trescot, MD)

Anatomy (Table 56.3)

The PFCN is a pure sensory nerve. It originates from the anterior and posterior rami of the first three sacral nerves [9], as well as from the anterior divisions of S2 and S3, and the posterior rami of S1–S3 (Table 56.3). The anatomy of PFCN is very variable, and it may involve branches as high as L4 or as low as S4 [10]. The PFCN exits the pelvis beneath the gluteus maximus (Figs. 56.4 and 56.5), accompanied by the inferior gluteal artery, and passes through the sciatic foramen, below the piriformis muscle, and down the buttock and thigh on the medial aspect of the sciatic nerve (Fig. 56.6). It is sometimes referred to as the "*lesser sciatic nerve*."

Deep to the gluteus maximus muscle, the PFCN gives off the *inferior cluneal nerve* (Chap. 63) (Fig. 56.7) and the *perineal branch of the PFCN* (PBPFCN). The inferior cluneal nerve provides the cutaneous innervation of the inferior buttocks, while the perineal branch innervates the lateral perineum, proximal medial thigh, posterolateral scrotum/labium majora, and part of the penis/clitoris (Fig. 56.3) [11]. The perineal branch courses medially, staying about 4 cm inferior to the attachment of the sacrotuberous ligament onto the ischial tuberosity, parallel to the ischial ramus [11]. However, Bergman et al. [12] described the perineal branch piercing the sacrotuberous ligament, which would place it close to the *pudendal nerve* in *Alcock's canal* (Chap. 47).

The rest of the nerve continues inferiorly from the lower edge of the gluteus maximus in a muscular groove formed by the medial and lateral hamstring muscles, with branches providing the sensation to the posterior thigh. It becomes superficial near the popliteal fossa, joining with the *lateral sural cutaneous nerve* (Chap. 72) and the distal *saphenous nerve* (Chap. 59) to innervate the posterior calf. The PFCN extends a variable distance into the calf, occasionally all the way to the calcaneal region [13].

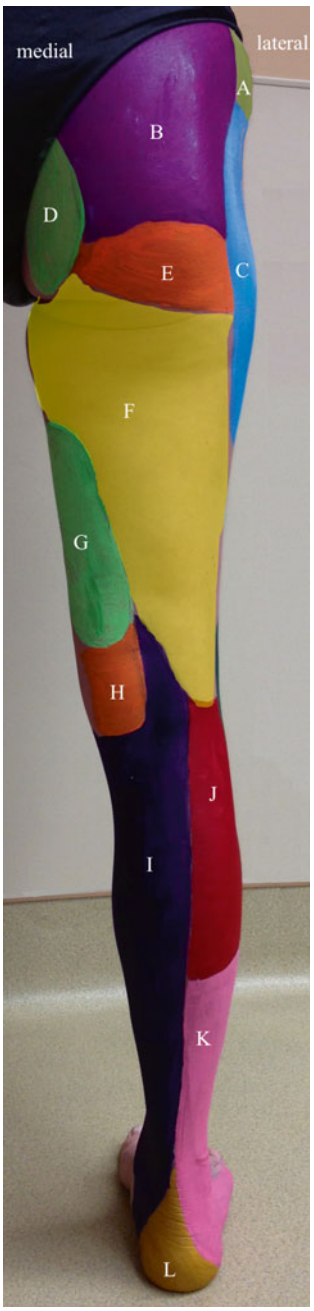

Fig. 56.2 Pain pattern from nerve entrapments of the posterior leg. *A* lateral branch iliohypogastric nerve, *B* superior cluneal nerve, *C* lateral femoral cutaneous nerve, *D* middle cluneal/sacral nerve, *E* inferior cluneal nerve, *F* posterior femoral cutaneous nerve, *G* obturator nerve, *H* femoral nerve, *I* saphenous nerve, *J* lateral sural cutaneous nerve, *K* superficial peroneal nerve, *L* medial calcaneal nerve (Image courtesy of Terry Dallas- Prunskis, MD)

The *main trunk of the PFCN* to the back of the thigh and leg consists of numerous filaments derived from both sides

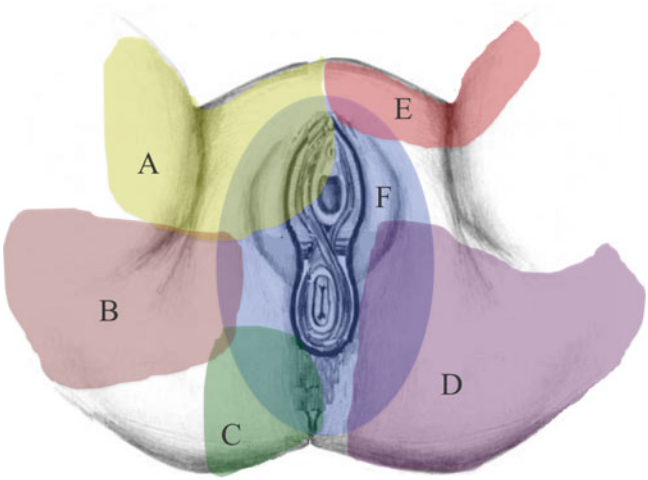

Fig. 56.3 Innervation of perineum: (*A*) genitofemoral nerve, (*B*) obturator nerve, (*C*) inferior cluneal nerve, (*D*) perineal branch of the posterior femoral cutaneous nerve, (*E*) ilioinguinal nerve, and (*F*) pudendal nerve (Image inspired by Hibner et al. [21], courtesy of Andrea Trescot, MD)

Table 56.2 Causes of posterior femoral cutaneous neuropathy

Cause of PFCN neuropathy	Presenting symptoms
1. Gluteal injection [5]	54-year-old man with hyperalgesia of posterior thigh and lateral scrotum
2. Gluteal injection describes complete syndrome of the infrapiriformis foramen [6]	137 cases of sciatic nerve injury, including two with PFCN lesions
3. Nerve compression by the ischial tuberosity thought to arise during exercise, sitting, and biking [4]	37-year-old female compressing her PFCN with gymnastics, 47-year-old sedentary male with bilateral hyperpathia, 54 year-old male riding bicycle 30 miles daily with pain in the posterior thigh and paresthesias in lower buttocks and lateral scrotum
4. Unknown etiology [7]	40-year-old woman with decreased sensation in the right posterior thigh 4 years after a left putamen hemorrhage
5. Gluteal intramuscular injections [1]	25-year-old woman with posterior/lateral thigh decreased sensation
6. Gluteal intramuscular injection [8]	22-year-old woman with pain, lack of sensation in posterior thigh and lower half of buttock; she had abnormal nerve conduction study

of the nerve and distributes to the skin covering the back and medial side of the thigh, the popliteal fossa, and the upper part of the back of the leg.

A study by Nakamishi et al. [14] described the dissection of 37 Japanese subjects, which showed that the origin of PFCN was variable. They found that the PFCN nerve may receive root components from the segments S1 through S4 and does not arise from S1 alone.

Table 56.3 Posterior femoral cutaneous nerve anatomy

Origin	Posterior rami of S1–S3 (but may include up to L4 and down to S4)
General route	Exits the pelvis anterior to the piriformis and posterolateral to the sciatic nerve and gives off inferior cluneal nerve and perineal branch of the PFCN. Travels down the posterior thigh between the medial and lateral hamstring muscles, joining the sural nerve, occasionally down to calcaneus
Sensory distribution	Inferior buttocks, lateral perineum, proximal medial thigh, posterolateral scrotum/labia, and part of the penis/clitoris
Motor innervation	None
Anatomic variability	Perineal branch can come from the inferior cluneal nerve
Other relevant structures	Sacrotuberous ligament, piriformis muscle, pudendal nerve

Tubbs et al. [11] dissected 20 cadavers and found that the perineal branch of the PFCN (PBPFCN) arose directly from the PFCN in 55 % of the sides; in 30 %, it arose from the inferior cluneal nerve, and it was absent in 15 % (Table 56.4). The PBPFCN provides two to three branches to the medial thigh and then innervates the scrotum and labia majora. In males, one nerve branch traveled inferior to the corpora cavernosa and anterior to the spermatic cord to cross the midline. Communications between the PBPFCN and the perineal branch of the pudendal nerve are common. There is also a report of a connection between the PFCN and the sciatic nerve at the gluteal region [15].

Entrapment

The posterior femoral cutaneous nerve lies adjacent to the sciatic nerve. Like the sciatic nerve, it may be compressed by the piriformis muscle or injured by prolonged tourniquet. The sciatic foramen is divided into two parts by the piriformis muscle (*foramina suprapiriformis* and *foramina infrapiriformis*). The PFCN reaches the dorsal part of the pelvis in close proximity to the ischial tuberosity as it passes through the infrapiriformis foramen. The compression of the nerve can occur at the ischial tuberosity by prolonged sitting on the edge of a chair [4]. Gluteal intramuscular injections have been reported to injure the PFCN and present as thigh and scrotal/labial pain [5].

The pudendal nerve and the PFCN leave the pelvis together through the greater sciatic foramen after passing through the infrapiriformis canal, so they can be trapped together along that path. They also share innervation of the perineal region. As early as 1900, Cushing [16] suggested that pain after ligation of testicular veins might be due to trauma of the perineal branch of PFCN.

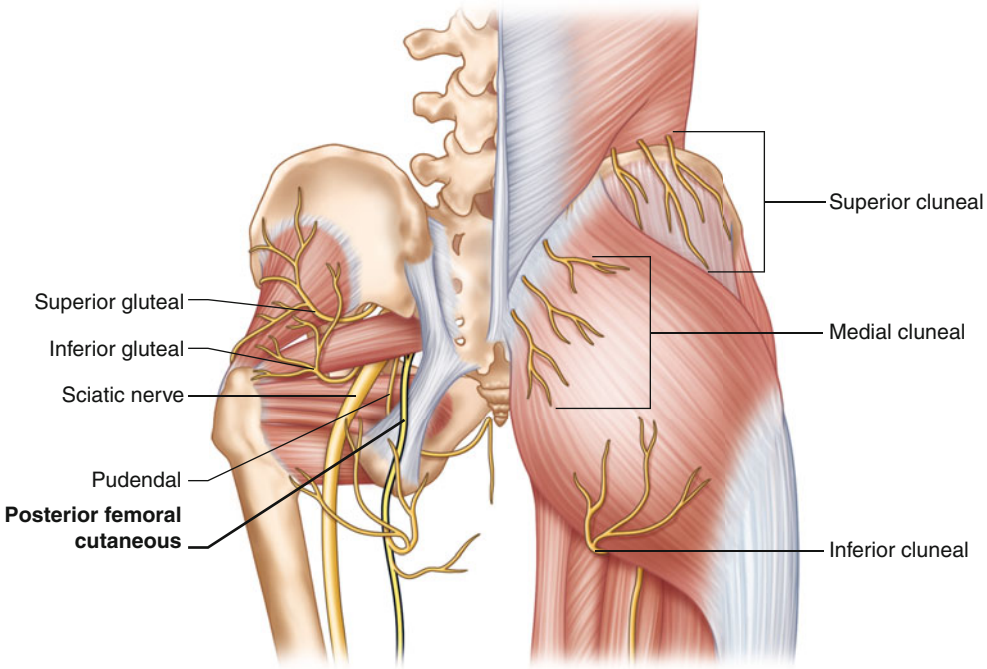

Fig. 56.4 Anatomy of the buttocks and pelvis (Image by Springer)

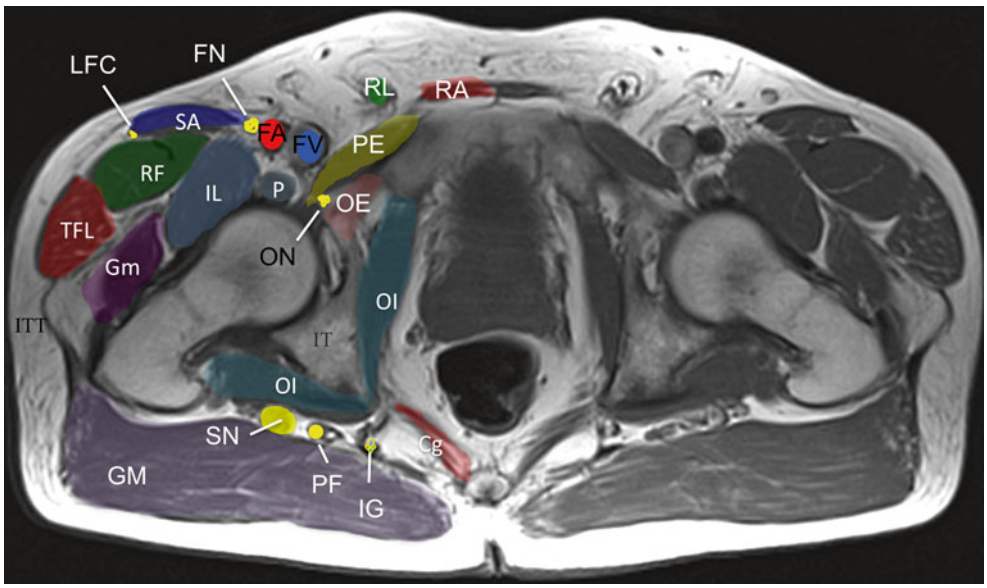

Fig. 56.5 MRI axial image of the pelvis. *Cg* coccygeus muscle, *FA* femoral artery, *FN* femoral nerve, *FV* femoral vein, *GM* gluteus maximus muscle, *Gm* gluteus medius, *IG* inferior gluteal nerve, *IL* iliopsoas muscle, *IT* ischial tuberosity, *LFC* lateral femoral cutaneous nerve, *OI* obturator internus muscle, *P* psoas muscle, *PE* pectineus muscle, *PF* posterior femoral cutaneous nerve, *QF* quadratus femoris muscle, *RA* rectus abdominis muscle, *RF* rectus femoris muscle, *RL* round ligament, *SA* sartorius muscle, *SN* sciatic nerve, *TFN* tensor fascia lata muscle (Image courtesy of Andrea Trescot, MD)

Physical Exam

Because the PFCN lies deep in the thigh, it is relatively hard to examine it by palpation. At the level of the ischium, however, the nerve can be palpated between the heads of the hamstring muscles (Fig. 56.8). Only two case reports mentioned numbness in the region of the PFCN [4, 7], and two patients had decreased sensation in the posterior thigh and lower buttock. This suggests that in a significant number of posterior femoral cutaneous neuropathies, the inferior cluneal nerve is also affected.

On physical examination, look for pain and sensory abnormality to be localized to the area corresponding to the particular branch of PFCN that is injured, such as the lower buttock, the posterior thigh, and the dorsal surface of the upper leg. The rest of the physical examination, including motor strength and reflexes, should be normal.

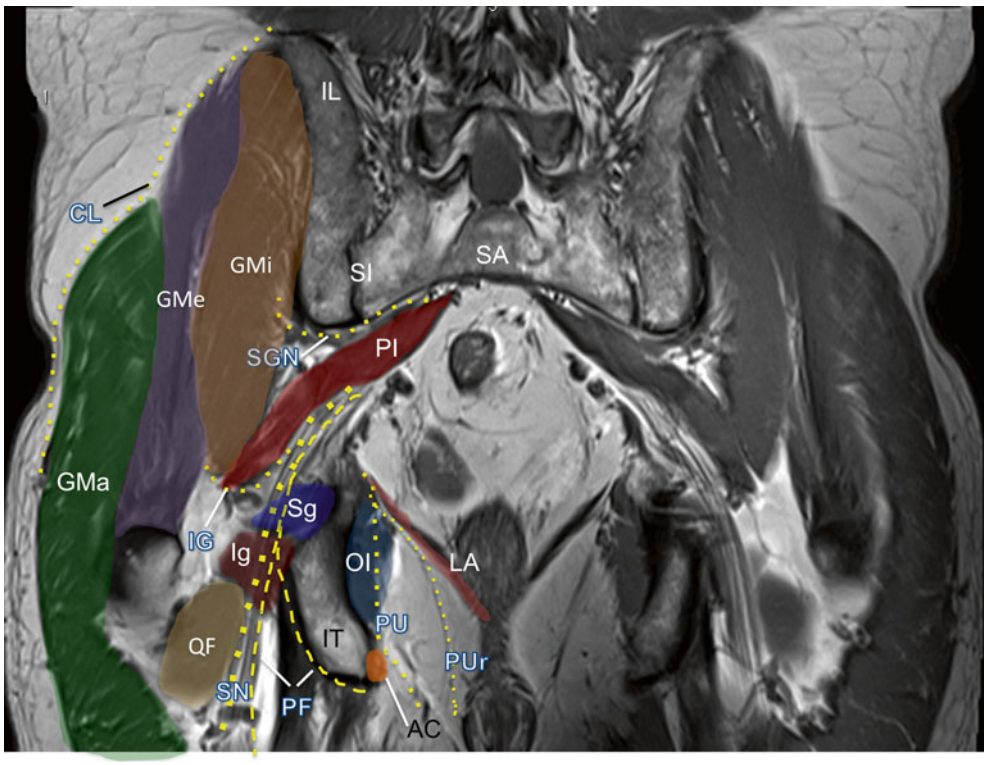

Fig. 56.6 MRI coronal images showing gluteal muscles and nerves. *AC* Alcock's canal, *CL* superior cluneal nerve, *IL* iliac crest, *IG* inferior gluteal nerve, *Ig* inferior gemellus muscle, *IT* ischial tuberosity, *Pi* piriformis, *Pu* pudendal, *Gmi* gluteus minimus, *Gme* gluteus medius, *Gma* gluteus maximus, *GT* greater trochanter, *LA* levator ani muscle, *OI* obturator internus muscle, *PF* posterior femoral cutaneous nerve, *PU* pudendal nerve, *PUr* pudendal nerve (rectal branch), *QF* quadratus femoris muscle, *SA* sacrum, *SGN* superior gluteal nerve, *Sg* superior gemellus muscle, *SI* sacroiliac joint, *SN* sciatic nerve (Image courtesy of Andrea Trescot, MD)

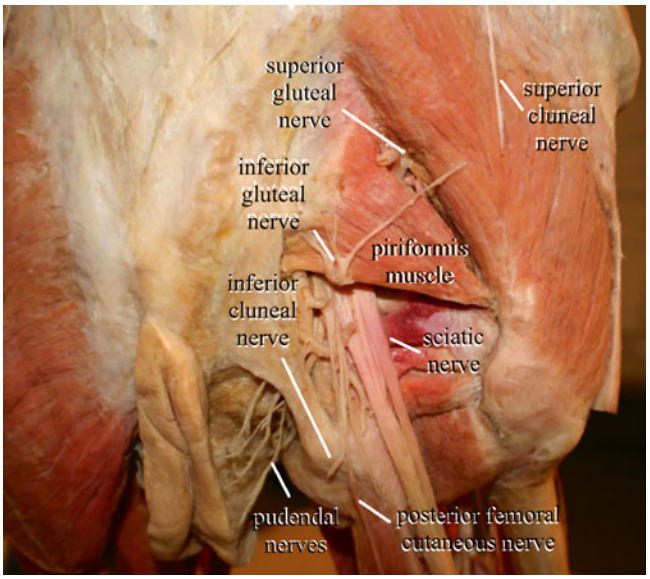

Fig. 56.7 Buttocks dissection, modified from an image from *Bodies, The Exhibition,* with permission (Image courtesy of Andrea Trescot, MD)

Differential Diagnosis (Table 56.5)

The PFCN may be responsible for many symptoms that are attributed to sciatic nerve and "piriformis syndrome" (Table 56.5). Piriformis syndrome presents with pain in the gluteal area that may mimic PFCN neuropathy. However, in piriformis syndrome, the pain can be worsened by internal rotation and flexion at the hip, as well as by pressure over the greater sciatic foramen. There is often tenderness to palpation along the course of the nerve at the thigh. Injury of the *perineal branch of the PFCN* can mimic *pudendal nerve* (Chap. 47) pathology, and it shares much of its innervation. Table 56.5 lists some distinguishing features of the differential diagnosis.

Diagnostic Studies (Table 56.6)

Dumitru and Marquis [7] described somatosensory-evoked potential (SSEP) evaluation of the PFCN. A recording electrode was placed 6 cm proximal to the midpopliteal fossa, and the nerve was stimulated 12 cm proximally on a line between the active electrode and the ischial tuberosity. A ground electrode was placed just proximal to the active recording electrode. The lower extremities of 40 individuals with a mean age of 34 years (20–78 years) were examined. The mean peak latency of the response was 2.8 (2.3–3.4) ms ± 0.2 ms, with a mean amplitude of 6.5 (4.1–12.0) mV ± 1.5 mV.

Identification and Treatment of Contributing Factors

- Iatrogenic trauma with gluteal injections
- Pressure on ischial area from prolonged bicycle riding or prolonged sitting

Table 56.4 Origin of the perineal branch of PFCN (PBPFCN)

From PFCN directly	55 % (22/40)
From inferior cluneal nerve	30 % (12/40)
Absent	15 % (6/40)

Data from Tubbs et al. [11] study of 20 adult cadavers (40 sides)

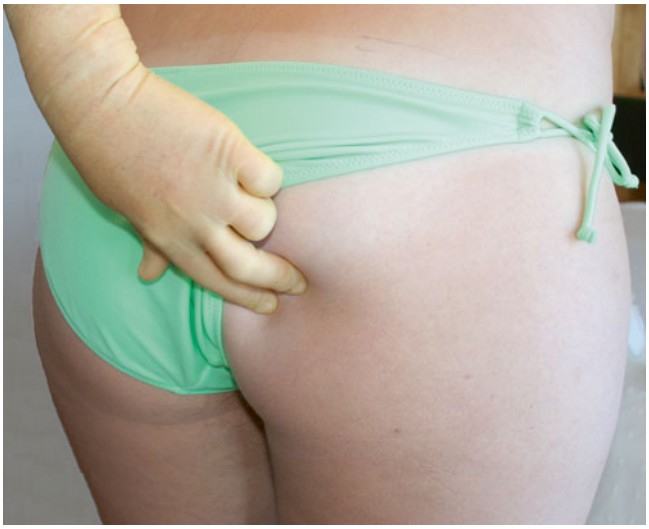

Fig. 56.8 Palpation of the posterior femoral cutaneous nerve at the ischium (Image courtesy of Andrea Trescot, MD)

Table 56.5 Differential diagnosis of low back and perineal pain

S1	Lower back pain
S1 only	Buttock pain
	Pain, numbness, or weakness in various parts of the leg and foot
	Pain may radiate below the knee but not always
Pudendal neuropathy	Pelvic pain
S2, S3, and S4 responsible for perineal innervation	Pain worse with sitting and driving
	Pain reduced by sitting on a toilet seat
	Vaginal pain with intercourse
	Bladder pain during micturition
	Rectal pain during defecation
	Bowel, bladder, and sexual dysfunction
PFCN neuropathy	Back and medial side of the thigh
S1, S2, and S3/4	Popliteal fossa
	Upper part of the back of the leg
	Inferior buttocks
	Normal motor exam
	No problem with urination, defecation, or sexual function
Piriformis syndrome	Pain worse with internal rotation and flexion of the hip

Table 56.6 Diagnostic tests for posterior femoral cutaneous nerve dysfunction

	Potential distinguishing features
Physical exam	Tenderness between the heads of the hamstring muscles at the ischium
Diagnostic injection	At the ischium
Ultrasound	Not described
MRI	May identify extrinsic compression
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies	SSEP

- Anatomic variations
- Smoking
- Diabetes
- Poor circulation
- Cachexia/excessive muscle loss that would function as nerve cushion
- Obesity

Iatrogenic trauma can be prevented with careful intramuscular injections in the gluteal area. Tourniquet times and pressures should be closely monitored to prevent ischemia of the nerve. Prevent pressure into the ischial area from prolonged bicycle riding or prolonged sitting; this is especially significant for people who are at risk for peripheral nerve injuries due to genetic and other medical conditions such as diabetes, poor circulation, and smoking. It is important to educate patients that if they experience recurrent pain in the gluteal area due to exercise, they may be overstretching the PFCN or causing its entrapment. Bikers with gluteal pain should use a cushion to prevent compression. Athletes such as rowers, bikers, and others whose sport leads to compression of the gluteal area are especially prone to this injury and may be misdiagnosed with "sciatica."

Injection Technique

The choice of approach to the PFCN depends on the site of entrapment; that is, entrapment in the sciatic foramen would be targeted differently than pathology at the ischium, perineum (see Chap. 46), or distal thigh (see Chap. 62). Table 56.7 describes the literature regarding PFCN injections.

Landmark-Guided Technique

Only one study describes a landmark-guided injection of the PFCN. Hughes et al. [17] described blocking the nerve where its branches come from below the medial border of the glu-

Table 56.7 Types of posterior femoral cutaneous nerve injections

Nerve block	Study
Landmark guided	Hughes et al. [17] described injection at gluteal fold
Fluoroscopy guided	No literature found
Ultrasound guided	No literature found
CT guided	Block of PFCN in 2 patients [19]; 22-gauge needle was advanced under CT, and the location was documented by injecting 1 cc of 50 % diluted nonionic water-soluble contrast. No improvement in their symptoms
MRI guided	Fritz et al. [18] performed MRI-guided PFCN on 12 patients. A 20-G needle of 10 or 15 cm length was placed in the immediate vicinity of the PFCN and then 4 mL injected around the PFCN, consisting of 1 mL of 1 % preservative-free lidocaine, 1 mL of 0.5 % bupivacaine, and 1 mL of non-particulate dexamethasone. Technically successful injections in 12/12 cases (100 %), with uniform perineural distribution of the injectant
	No comment on improvement of pain

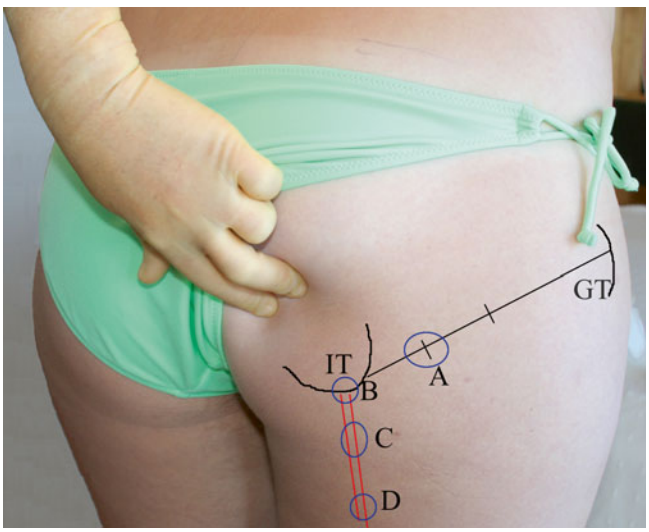

Fig. 56.9 Landmark-guided injections of the posterior femoral cutaneous nerve. *IT* ischial tuberosity, *GT* greater trochanter. *A* site described by Hughes [11], *B* injection at the ischium, *C* site recommended by Fritz [18], 2 cm below the IT, *D* site recommended by Tubbs et al. [11], 4 cm below the IT (Image courtesy of Andrea Trescot, MD)

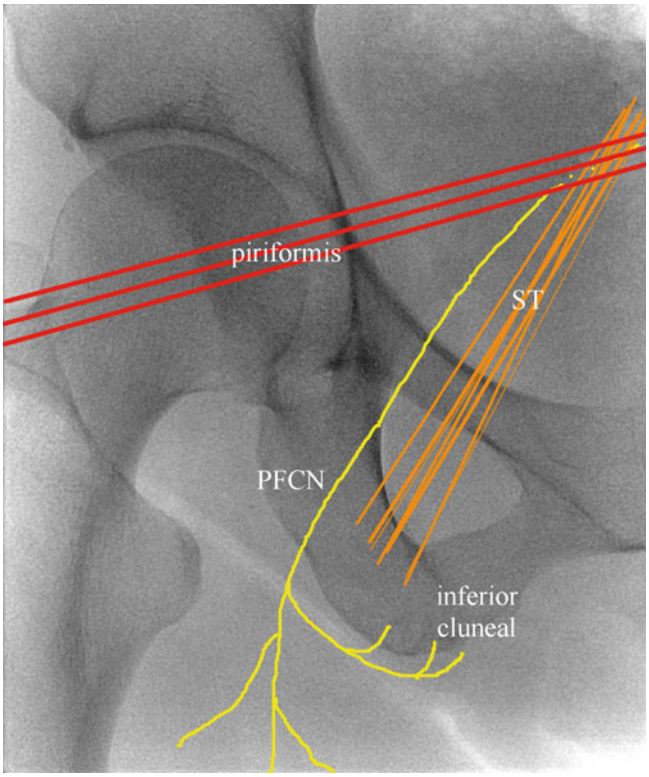

Fig. 56.10 Fluoroscopic landmarks of the posterior femoral cutaneous nerve (Image courtesy of Andrea Trescot, MD)

teus maximus. The site is in the gluteal fold, one quarter of the distance between the ischial tuberosity and the greater trochanter (Fig. 56.9 Site A). The authors suggest trying to feel two distinct losses of resistance as superficial and deep fascia are penetrated with a short-beveled needle. The perineal branch of the PFCN is usually injected at the ischial tuberosity (Fig. 56.9 Site B) [11].

Since the approach to the PFCN depends on the site of entrapment, Fritz et al. [18] recommended injecting the nerve 2 cm below the ischial tuberosity (Fig. 56.9 Site C), proximal to the PBPFCN for patients with pain and paresthesia in the perineal area. In patients with inferior lateral buttock area pain and numbness, target the cluneal nerves under the gluteus maximus muscle. For those with pain in the posterior thigh, the distal portion of the PFCN between the gluteus maximus and the long head of the biceps femoris should be considered for injection [18]. Based on cadaver studies, Tubbs et al. suggested that the PBPFCN is on average located 4 cm inferior to the ischial tuberosity; they recommend injecting this area with anesthetic if this nerve is the source of the pain (Fig. 56.9 Site D) [11].

Fluoroscopy-Guided Technique

There are no good fluoroscopic landmarks, other than the ischium (Fig. 56.10). Although there are no reported fluoroscopic techniques, Trescot (personal communication) describes the injection of the PFCN at the ischium using a peripheral nerve stimulator (PNS).

Ultrasound-Guided Technique

The sciatic nerve is well visualized by ultrasound, but there are no reported US approaches the PFCN.

CT-Guided Technique

Kasper et al. [19] describe CT-guided PFCN block in two patients. They traced the PFCN neurovascular bundle from the inferior margin of the ipsilateral gluteus maximus muscle between the hamstring tendon origin and the sciatic nerve. Under aseptic precautions, a 22-gauge needle was advanced, and the location was documented by injecting 1 cc of 50 % diluted nonionic water-soluble contrast. One patient had decreased sharp sensation in the inferior gluteal region, as well as posterior thigh (the inferior cluneal nerve and PFCN distribution) [19]. Neither patient had improvement of their pain.

MRI-Guided Technique

Fritz et al. [18] performed MRI-guided PFCN injections that were technically successful in 12 out of 12 cases (100 %), with a uniform perineural distribution of the injectant. A total of 4 cc of their solution (1 cc of 1 % preservative-free lidocaine, 1 cc of 0.5 % bupivacaine, and 1 cc of non-particulate dexamethasone (10 mg/mL)) was injected around the PFCN.

Neurolytic Technique

No neurolytic techniques have been described, although cryoneuroablation or radiofrequency lesioning would theoretically be reasonable options.

Surgical Technique

Mobbs et al. [20] described one case of successful surgical release of the PFCN in a 51-year-old healthy male with a 12-month history of shooting pain down the posterolateral aspect of his thigh and buttock down to the knee. Lower limb strength was intact, and preoperative somatosensory-evoked potentials were consistent with PCFN entrapment neuropathy.

Tubbs et al. [11] performed an open exploration of the PFCN. After identification of the sciatic nerve, they found the PFCN, superficial to the proximal sciatic nerve. Although no entrapment site was obvious, release of the fascia over the nerve was successful; their patient had complete resolution of his symptoms 12 months postoperatively.

Complications

No complications of the landmark-guided technique have been reported; since there are multiple nerves in this area, the main concerns are neurological complications. There were no complications using CT-guided or MRI-guided injections of the PFCN during the procedure or during the follow-up [18].

Summary

PFCN is rarely considered as a cause of buttocks pain, which probably contributes to the limited available literature. PFCN entrapment should be considered in the patient with buttocks and posterior thigh pain.

References

1. Tong HC, Haig A. Posterior femoral cutaneous nerve mononeuropathy: a case report. Arch Phys Med Rehabil. 2000;81(8):1117–8.
2. Schuchmann JA. Sural nerve condition: a standardized technique. Arch Phys Med Rehabil. 1977;58:166–8.
3. Chutkow JG. Posterior femoral cutaneous neuralgia. Muscle Nerve. 1988;11:1146–8.
4. Arnoldussen W, Korten J. Pressure neuropathy of the posterior femoral cutaneous nerve. Clin Neurol Neurosurg. 1980;82(1): 57–60.
5. Iyer VG, Shields CB. Isolated injection injury to the posterior femoral cutaneous nerve. Neurosurgery. 1989;25(5):835–8.
6. Obach J, Aragones JM, Ruano D. The infrapiriformis foramen syndrome resulting from intragluteal injection. J Neurol Sci. 1983; 58(1):135–42.
7. Dumitru D, Marquis S. Posterior femoral cutaneous nerve neuropathy and somatosensory evoked potentials. Arch Phys Med Rehabil. 1988;69(1):44–5.
8. Kim JE, Kang JH, Chol Choi J, Seok Lee J, Kang SY. Isolated posterior femoral cutaneous neuropathy following intragluteal injection. Muscle Nerve. 2009;40(5):864–6.
9. Enneking FK, Chan V, Greger J, Hadzic A, Lang SA, Horlocker TT. Lower-extremity peripheral nerve blockade: essentials of our current understanding. Reg Anesth Pain Med. 2005; 30(1):4–35.
10. Hollinshead WH. Anatomy for surgeons: the back and limbs. 3rd ed. Philadelphia: Harper & Row; 1982.
11. Tubbs RS, Miller J, Loukas M, Shoja MM, Shokouhi G, Cohen-Gadol AA. Surgical and anatomical landmarks for the perineal branch of the posterior femoral cutaneous nerve: implications in perineal pain syndromes. Laboratory investigation. J Neurosurg. 2009;111(2):332–5.
12. Bergman RA, Afifi AK, Miyauchi R. Posterior femoral cutaneous nerve. 2014 [Jan 4, 2014]. Available from: http://www.anatomyatlases.org/AnatomicVariants/NervousSystem/Text/PosteriorFemoralCutaneous.shtml.
13. Kosinski C. Course, mutual relations and distribution of the cutaneous nerves of the metazonal regions of leg and foot. J Anat. 1926;60:274–97.
14. Nakanishi T, Kanno Y, Kaneshige. Comparative morphological remarks on the origin of the posterior femoral cutaneous nerve. Anat Anz. 1976;139(1–2):8–23.

15. Tunali S, Cankara N, Albay S. A rare case of communicating branch between the posterior femoral cutaneous and the sciatic nerves. Rom J Morphol Embryol. 2011;52(1):203–5.
16. Cushing HI. The employment of local anaesthesia in the radical cure of certain cases of hernia, with a note upon the nervous anatomy of the inguinal region. Ann Surg. 1900;31(1):1–34.
17. Hughes P, Brown T. An approach to posterior femoral cutaneous nerve block. Anaesth Intensive Care. 1986;14(4):350–1.
18. Fritz J, Bizzell C, Kathuria S, Flammang AJ, Williams EH, Belzberg AJ, et al. High-resolution magnetic resonance-guided posterior femoral cutaneous nerve blocks. Skeletal Radiol. 2013;42(4):579–86.
19. Kasper JM, Wadhwa V, Scott KM, Chhabra A. Clunealgia: CT guided therapeutic posterior femoral cutaneous nerve block. Clin Imaging. 2014;38(4):540–42. http://dx.doi.org/10.1016/s.clininaq.2014.02.017
20. Mobbs R, Szkandera B, Blum P. Posterior femoral cutaneous nerve entrapment neuropathy: operative exposure and technique. Br J Neurosurg. 2002;16(3):309–11.
21. Hibner M, Desai N, Robertson LJ, Nour M. Pudendal neuralgia. J Minim Invasive Gynecol. 2010;17(2):148–53.

Part IX

Lower Extremity

Susan R. Anderson-Jones

Introduction

Peripheral nerve entrapments in the lower extremity can present with a variety of clinical symptoms and can be rather difficult to diagnose. The success in the treatment for peripheral nerve entrapment relies on the correct identification of the specific nerve involved and the exact location of compression [1]. In addition to a detailed history and physical exam during the patient interview, electrodiagnostic and imaging techniques provide more definitive evidence if any neuropathy exists [1]. However, the use of diagnostic injections can significantly improve the clinician's diagnostic armamentarium. The patient presenting with diffuse neuropathic pain in the lower extremity simulating a complex regional pain syndrome (CRPS) condition should be questioned as to whether or not there is any focal region within the painful sensory field that is most intense or occurred first. One may discover this focal region of pain will correlate to a specific known peripheral nerve entrapment that can be diagnosed and treated.

Reference

1. Flanigan RM, DiGiovanni BF. Peripheral nerve entrapments of the lower leg, ankle, and foot. Foot Ankle Clin. 2011;16(2):255–74.

Femoral Nerve Entrapment

57

Christopher J. Burnett

Introduction

The femoral nerve is a mixed motor and sensory nerve that is formed from the L2–L4 nerve roots. It is the largest branch of the lumbar plexus. The femoral nerve can be injured in a variety of different ways and has several areas of potential entrapment.

Clinical Presentation (Table 57.1)

The femoral nerve (FN) is the largest anterior lower extremity nerve, innervating the anterior thigh (Fig. 57.1). Since there are multiple nerves in the femoral nerve complex (including the saphenous nerve – see section "Anatomy"), FN entrapment can cause numbness and paresthesias in the iliac fossa, inguinal region, anterior thigh, medial calf and foot, and great toe (Fig. 57.2). Patients with FN entrapment or neuropathy may have ipsilateral knee weakness or buckling with walking that may result in frequent falls [1]. Severe femoral neuropathy can produce weakness and wasting of the quadriceps muscle. The patient may be able to walk freely on level ground by keeping the knee in a hyperextended position, but they will not be able to use the involved leg to step up when climbing stairs. The affected leg will be kept in the lead position when walking down stairs [1].

FN injections are used in anesthesia for perioperative pain relief for knee surgery, femur fracture, and muscle biopsy. Direct needle trauma can injure the nerve during these procedures.

Femoral neuropathies can be divided into a mixed sensory and motor dysfunction of the proximal FN or a purely sensory neuropathy of the saphenous nerve (see Chaps. 58 and 59). Studies of complications after hip arthroplasties show an incidence of femoral neuropathies of 0.1–2.3 % [1], attributed to pressure from retractors, iliacus hematoma, or postoperative scar tissue, as well as thermal or mechanical damage from the cement used during these procedures. FN injuries during abdominal hysterectomies are not uncommon; two prospective studies showed an incidence of 7.45 % [3] and 11.6 % [12], attributed to self-retaining retractors. The lithotomy position is also associated with unilateral or bilateral FN injuries. These have occurred during vaginal hysterectomies or prolonged labor, presumably because of FN compression under the inguinal ligament with prolonged or extreme hip abduction and external rotation [6]. This may also be the mechanism of FN injuries from laparoscopy [4]. Retroperitoneal renal transplants are associated with (usually transient) FN injuries, again from retractors and hematomas but possibly also from a vascular steal from the graft artery [13]. Inguinal hernia repairs have been associated with FN injuries and entrapment, especially by staples or sutures

Table 57.1 Occupation/exercise/trauma history relevant to femoral nerve entrapment

Surgical procedures	Surgery of hip, abdomen, pelvis, and inguinal region with self-retaining retractors [1]
	Hip arthroplasty [1]
	Pelvic surgery [1]
	Vaginal hysterectomy [2]
	Abdominal hysterectomy [3]
	Laparoscopy [4]
	Renal transplant [5]
Positioning	Lithotomy position [6]
Thermal injury	Heat from hip cement [1]
Anticoagulation (hematoma)	Psoas hematoma [1, 7, 8]
	Aortic aneurysm [9]
	Hemophilia [10]
Infection	Iliacus pyomyositis
Diabetes	Diabetic femoral neuropathy [11]

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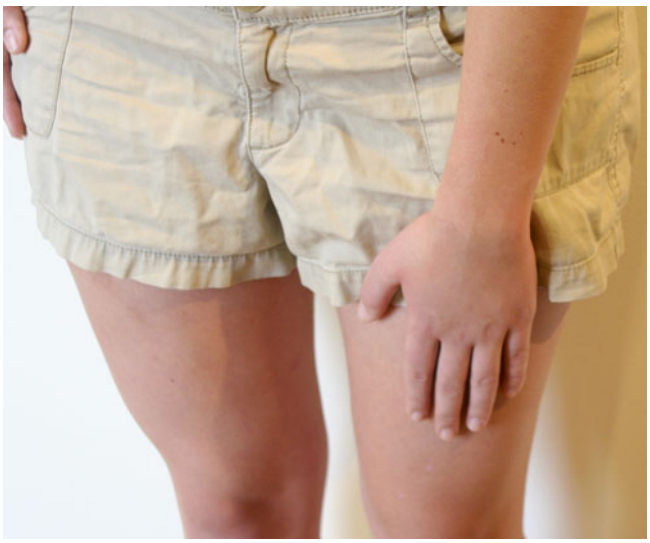

Fig. 57.1 Patient identification of femoral nerve pain (Image courtesy of Andrea Trescot, MD)

from laparoscopic mesh [14] or scar tissue [15]. Injuries to the FN are obviously a potential consequence of femoral artery interventions, such as cardiac catheterization [16] or hemodialysis catheters, and groin procedures such as inguinal node biopsies. Retroperitoneal or groin hematomas, most likely in hemophiliacs or in patients on anticoagulants, can cause FN entrapment [10]. Neuropraxia of the FN has been reported in dancers [17], most likely due to chronic overstretching.

Common signs of FN entrapment include sudden onset of severe groin and thigh pain, flexion contracture of the hip with lateral rotation of the thigh, and loss of patellar reflex. If it has been caused by a hematoma, there may be a palpable mass in the iliac fossa [10].

Anatomy (Table 57.2)

The FN is a mixed motor and sensory nerve that arises from the posterior divisions of the ventral rami of the L2, L3, and L4 spinal nerves within the *psoas muscle* (Fig. 57.3) as part of the *lumbar plexus* (see Chap. 66). It provides motor innervation to all of the anterior muscles of the thigh, with the exception of the *tensor fasciae lata*. It is the largest branch of the lumbar plexus and arises from the lumbar plexus within the *psoas major* muscle. It then travels inferiorly in the pelvis through the *iliacus compartment*, which is bounded by the *psoas* and *iliacus muscles* and is roofed by *the iliacus fascia*. The FN then approaches the *external iliac artery*, which lies anterior and medial to it. As these two structures descend to exit the pelvis, the FN supplies motor branches to both the iliac and psoas muscles (Fig. 57.4) [1, 18–20].

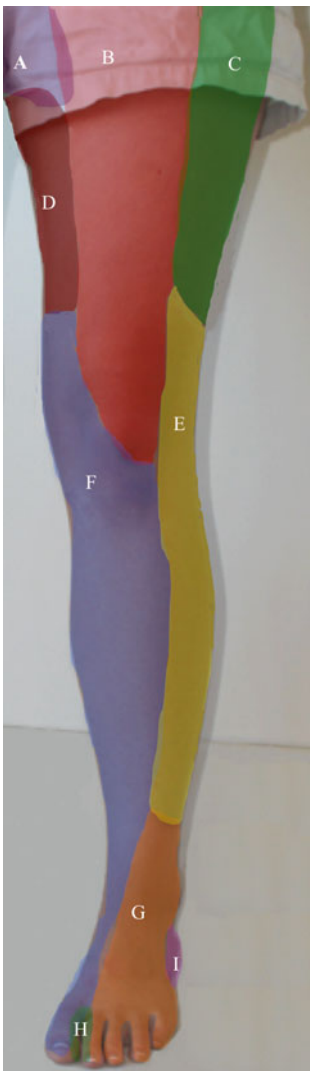

Fig. 57.2 Pattern of pain from anterior lower extremity nerve entrapments. *A* genitofemoral nerve, *B* femoral nerve, *C* lateral femoral cutaneous nerve, *D* obturator, *E* lateral sural cutaneous nerve, *F* saphenous nerve, *G* superficial peroneal nerve, *H* deep peroneal nerve, *I* sural nerve (Image courtesy of Andrea Trescot, MD)

In the proximal thigh, the FN, the psoas and iliacus muscles, and the *iliolumbar vessels* travel in a fibromuscular canal (the *femoral-iliacus compartment*) formed by the *iliac fascia*, with the nerve separated from the vessels by the fascia iliaca. The FN descends under the *inguinal ligament*, giving a branch to the *pectineus muscle*. The FN then continues distally to enter the *femoral triangle*, where it lies lateral to the femoral artery (Fig. 57.5). The common mnemonic is "VAN" (vein, artery, nerve) going from medial to lateral (Fig. 57.6). The FN then divides into an anterior and posterior division, roughly 4 cm below the inguinal ligament. The anterior division innervates the *sartorius muscle* via the *anterior lateral femoral cutaneous nerve* and then generates the *medial and intermediate femoral cutaneous*

nerves, which provide sensory innervation to the anterior thigh above the knee. The posterior division provides motor branches to the *quadriceps muscles* (*rectus femoris*, *vastus lateralis*, *vastus intermedius*, and *vastus medialis*) and terminates as the *saphenous nerve* (see Chaps. 58 and 59). The saphenous nerve receives sensory information from the medial and anterior aspects of the knee joint, the medial leg all the way to the medial arch of the foot and the great toe, and the skin of the medial and anterior surface of the knee [1, 20]. Because innervation of the psoas and iliacus muscles may come from the lumbar plexus or the femoral nerve, FN entrapment may cause weakness of the hip flexors.

Table 57.2 Femoral nerve anatomy

Origin	Posterior divisions of L2, L3, and L4
General route	Within psoas through iliacus compartment, exits pelvis with iliopsoas under inguinal ligament to femoral triangle (lateral to femoral artery); anterior division becomes the medial and intermediate femoral cutaneous nerves; posterior division becomes the saphenous nerve
Sensory distribution	Anterior thigh above the knee and medial lower extremity below the knee to medial arch and great toe
Motor innervation	All of the anterior muscles of the thigh except tensor fascia lata
Anatomic variability	Nerve may be located lateral or posterolateral to the femoral artery and can be oval or triangular in shape; muscular slips from iliacus and psoas may pierce or cover the nerve
Other relevant structures	Iliacus compartment, lacuna musculorum, femoral triangle, and femoral artery and vein

Entrapment

There are several potential sites of entrapment or injury of the FN. The two most common locations are the *iliacus compartment* and at the inguinal ligament (Fig. 57.6). Most FN entrapments occur after injuries to the *iliopsoas compartment*; an iliacus or iliopsoas muscle tear or hematoma can result in compression of the nerve [18]. The FN can also be entrapped at the pelvis as it passes under the inguinal ligament through a rigid tunnel called the *lacuna musculorum*. This channel is bounded by the *iliopectineal arch* and the *inguinal ligament* ventrally and the iliac bone and iliopsoas muscle dorsally [19]. Entrapment has been reported in association with iliopsoas compartment masses, distended *iliopsoas bursa* [18], and aberrant bands of iliacus or psoas muscle covering or even splitting the nerve [21, 22].

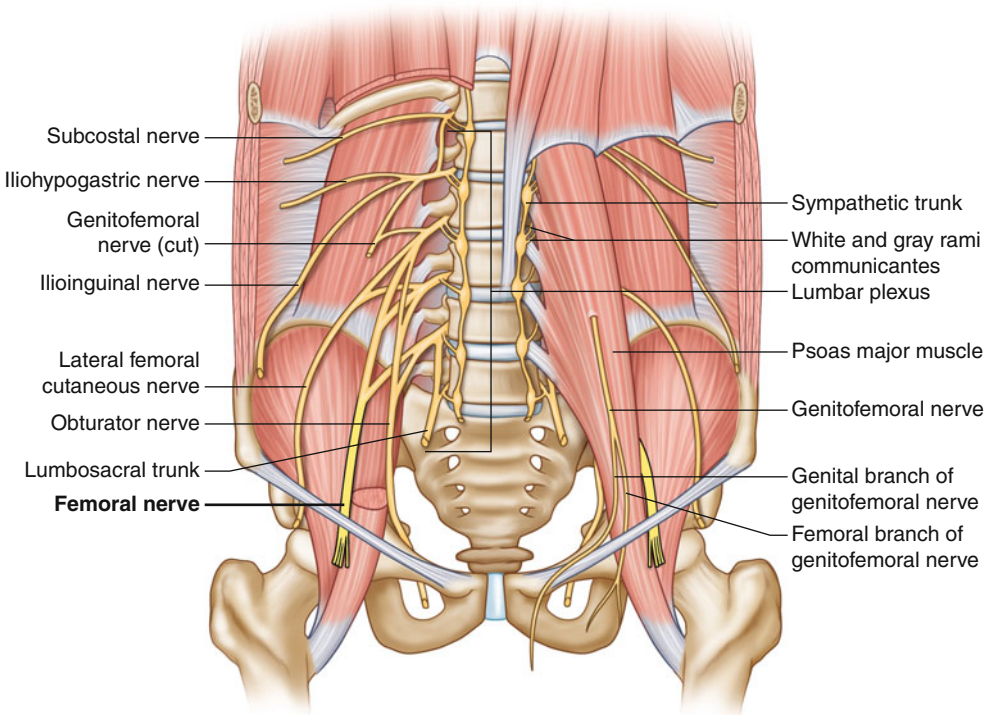

Fig. 57.3 Anatomy of the lumbar plexus (Image by Springer)

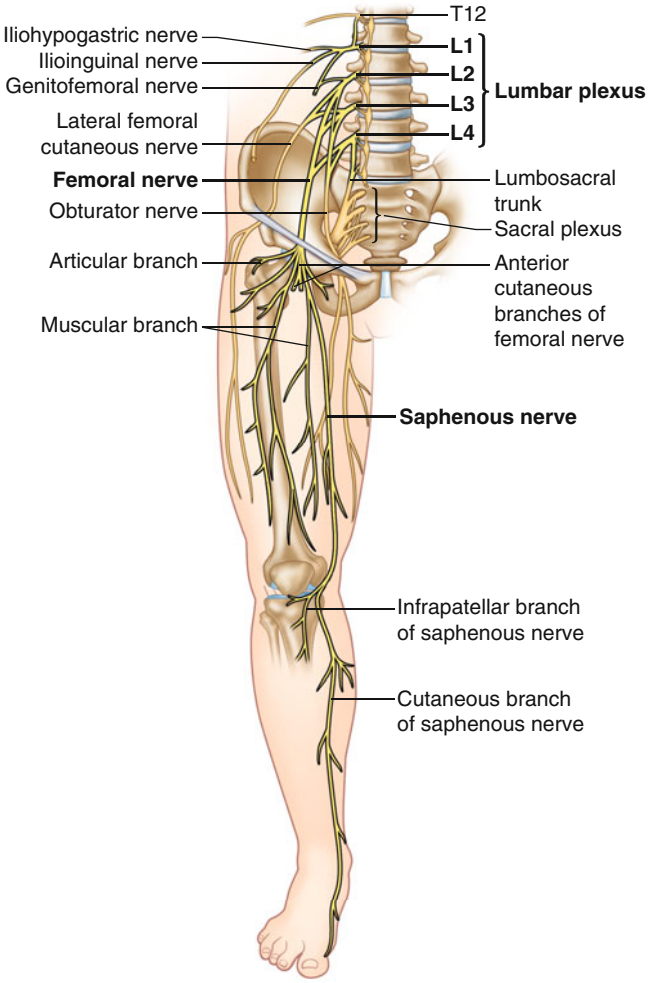

Fig. 57.4 Anatomy of the femoral and saphenous nerve (Image courtesy of Springer)

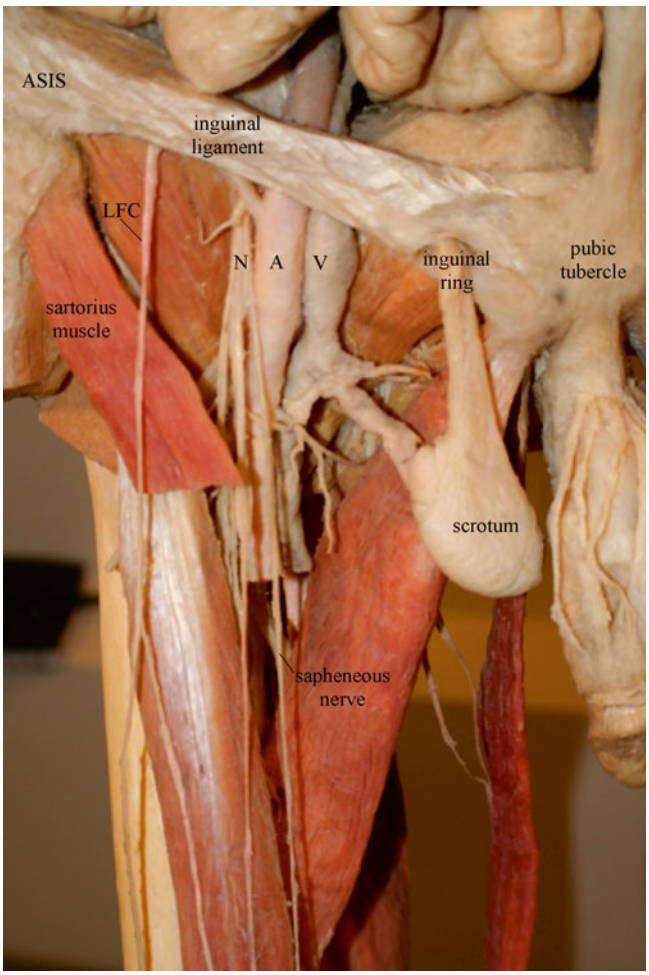

Fig. 57.6 Dissection of the anterior thigh and groin, modified from an image from *Bodies, The Exhibition,* with permission (Image courtesy of Andrea Trescot, MD)

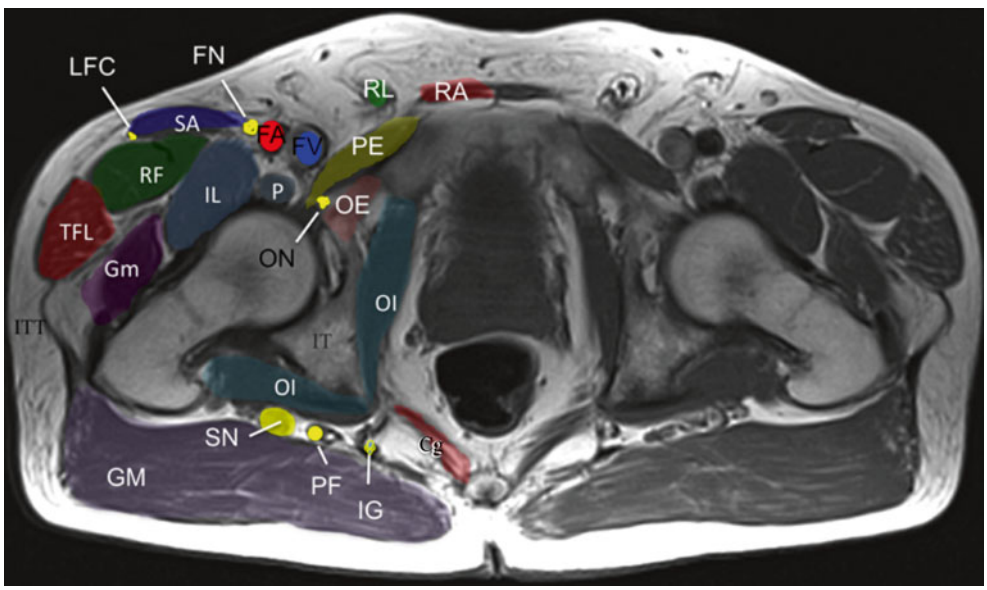

Fig. 57.5 MRI axial image of the pelvis. *Cg* coccygeus muscle, *FA* femoral artery, *FN* femoral nerve, *FV* femoral vein, *GM* gluteus maximus muscle, *Gm* gluteus medius, *IG* inferior gluteal nerve, *IL* iliopsoas muscle, *IT* ischial tuberosity, *ITT* iliotibial tract, *LFC* lateral femoral cutaneous nerve, *OI* obturator internus muscle, *P* psoas muscle, *PE* pectineus muscle, *PF* posterior femoral cutaneous nerve, *QF* quadratus femoris muscle, *RA* rectus abdominis muscle, *RF* rectus femoris muscle, *RL* round ligament, *SA* sartorius muscle, *SN* sciatic nerve, *TFN* tensor fascia lata muscle (Image courtesy of Andrea Trescot, MD)

Physical Exam

Patients with FN entrapment report tenderness at the inguinal ligament (Fig. 57.7), unless the entrapment occurs more proximally. On inspection, an atrophy of the anterior thigh may be noted. Sensory deficits may be present in the anterior thigh, medial calf, medial foot, or great toe. Pain may be reproduced with flexion of the hip (Fig. 57.8) and diminished with external rotation of the hip. The patient may have weak hip flexion, weak knee extension, and impaired or absent quadriceps tendon (patellar) reflex [1, 18, 23].

Differential Diagnosis (Table 57.3)

Diagnosis of FN entrapment is primarily determined from the history and physical examination, and it may be aided by electrodiagnostic studies, magnetic resonance imaging, or high-resolution ultrasonography. The differential diagnosis includes lumbar plexopathy; L4 radiculopathy [19]; endometriosis; diabetic radiculopathy or plexopathy [1]; acute retroperitoneal hematoma [24]; iliacus pyomyositis; metastatic or primary tumor of the femoral nerve, ilium, or iliopsoas muscle [1]; and pseudoaneurysm of the iliac vessels [19]. Needle EMG of the lumbar paraspinals and hip adductors can establish pathology outside of the femoral nerve; EMG abnormalities of the psoas muscle indicate involvement of the FN, lumbar plexus (see Chap. 66), or L2 and L3 nerve roots [1]. The list of diagnostic tests is found in Table 57.4.

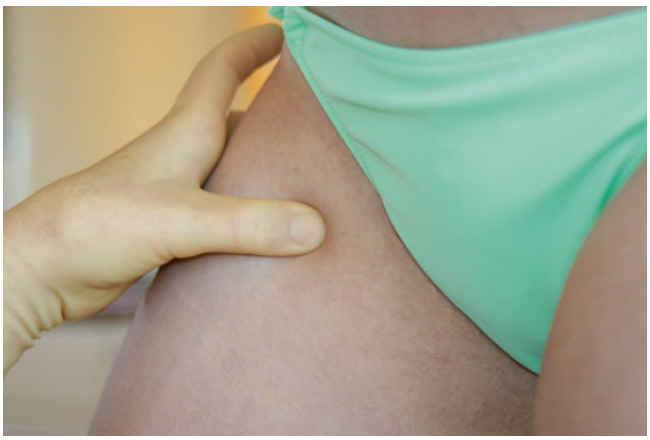

Fig. 57.7 Physical exam of the femoral nerve at the groin (Image courtesy of Andrea Trescot, MD)

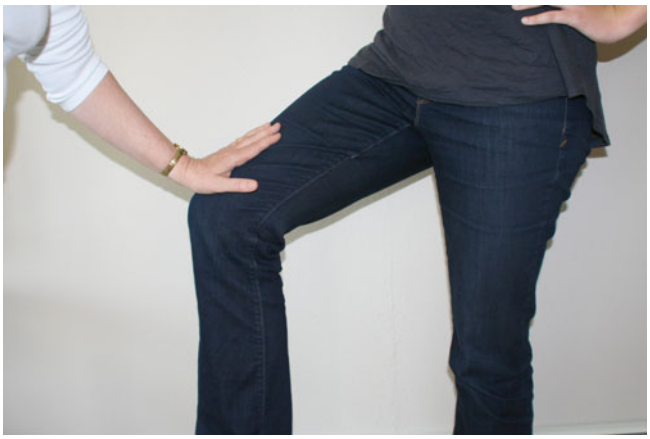

Fig. 57.8 Examination for strength from femoral nerve entrapment (Image courtesy of Andrea Trescot, MD)

Table 57.3 Differential diagnosis of anterior thigh pain

	Potential distinguishing features
Lumbar plexopathy	Multiple nerve entrapments – weakness of the hip adductors as well as hip flexors, abdominal wall pain (ACNE syndrome)
L4 radiculopathy	Medial calf hypesthesia but not anterior thigh numbness, MRI showing proximal pathology, EMG showing paravertebral pathology
Endometriosis	Positive pelvic exam
Diabetic radiculopathy or plexopathy	Multiple nerve entrapments with elevated blood sugar [11]
Acute retroperitoneal hematoma or abscess	Ultrasound showing retroperitoneal fluid collection, with palpable tender mass in the inguinal area and ecchymosis in the inguinal region, flank, or upper thigh [1]
Iliacus pyomyositis	Ultrasound/MRI showing iliac fluid collection, increased WBC
Tumor of the femoral nerve, ilium, or iliopsoas muscle	MRI showing mass
Pseudoaneurysm of the iliac vessels	MRI/arteriogram showing pseudoaneurysm
Vasculitis	Positive ANA, ESR, CRP
Upper motor lesion	Positive Babinski [1]

Table 57.4 Diagnostic tests for femoral nerve entrapment

	Potential distinguishing features
Physical exam	Pain reproduced with hip flexion and decreased by external hip rotation
Diagnostic injection	Confirms nerve etiology, identifies tumors, radiculopathy, or hematoma [1]
Ultrasound	Identifies nerve, identifies potential entrapment
MRI/CT	Confirms site of entrapment
Arteriography	Not diagnostic
X-ray	Not diagnostic
Electrodiagnostic studies	EMG confirms lumbar plexopathy vs. radiculopathy; EMG abnormalities in the iliopsoas muscle indicate femoral nerve, lumbar plexus, or L2/L3 nerve root pathology; proximal lesions (such as L4 radiculopathy) will have normal saphenous sensory nerve action potentials [1]

Identification and Treatment of Contributing Factors

Many factors may contribute to the development of FN entrapment. The nerve is at risk in the groin following inguinal hernia repairs, pelvic surgery, hip surgery, hysterectomy, renal transplantation, cardiac catheterization, intra-aortic balloon pump placement, or arterial bypass procedures. Prolonged labor and delivery with or without sustained lithotomy positioning can compress the femoral nerve against the inguinal ligament. The large self-retaining retractors used during abdominal or pelvic operations can directly compress the nerve, as can femoral artery catheterization, even without subsequent hematoma formation. Injury of the FN can result from hip or pelvic fractures, gunshot wounds, lacerations, or pseudoaneurysm of the iliac vessels [18, 19, 23].

Inadvertent FN palsy can occur after a peripheral field block for inguinal hernia repair. The resulting inability to walk can dramatically limit the early discharge of the patient from the facility. Rosario et al. [25] injected methylene blue in cadavers and showed that the plane between the transversus abdominis muscle and the transversalis fascia connects laterally with the tissue plane deep to the iliacus fascia, the plane containing the FN. Large volumes of local anesthetic in this region can cause FN anesthesia.

Taking care with pelvic retraction can decrease the risk of femoral entrapment. Goldman et al. [3] prospectively followed two groups of pelvic surgery patients over two 5-year periods. When self-retaining retractors were used, there were 282 cases of iatrogenic femoral neuropathy (7.45 %); when those retractors were not used, there were only two cases.

Injection Technique

Landmark-Guided Technique

The patient is placed in the supine position. The ipsilateral leg is abducted 10–20°, and the femoral artery palpated at the level of the femoral crease, below the inguinal ligament. The needle insertion site is approximately 1 cm lateral to the palpated pulse (Fig. 57.9). The needle is advanced at a 45° angle to the thigh in a cephalad direction. The clinician may notice a discreet change in resistance as the needle pierces the fascia lata, and the patient may report a paresthesia as the needle tip approaches the FN. After negative aspiration for blood, the planned injectate can be administered. Many advocate a larger volume of 15–20 mL of local anesthetic for this landmark-guided technique [26]. The same needle approach can be used in conjunction with a nerve stimulator, and optimal needle positioning will result in patellar twitches at 0.2–0.5 mA [20].

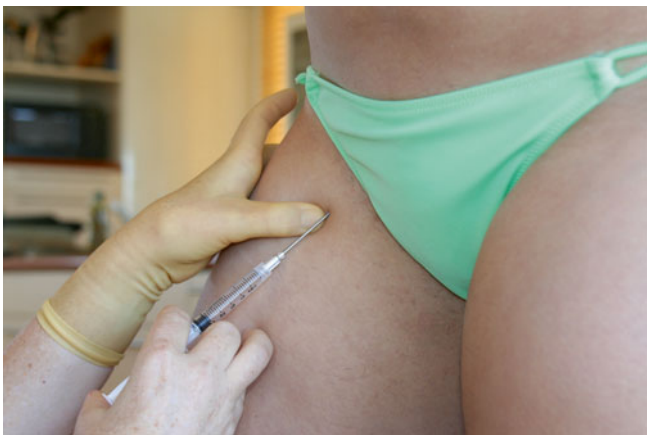

Fig. 57.9 Landmark-guided injection of the femoral nerve (Image courtesy of Andrea Trescot, MD)

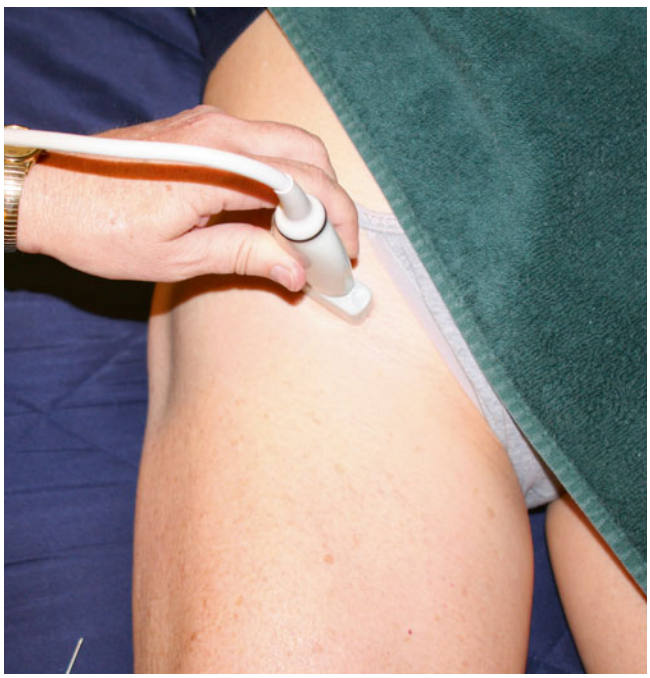

Fig. 57.10 Ultrasound probe location for femoral nerve identification under ultrasound (Image courtesy of Andrea Trescot, MD)

The "3-in-1" injection popularized by Winnie and coworkers [27] in 1973 was used to anesthetize the femoral, obturator (see Chap. 64), and lateral femoral cutaneous nerves (see Chap. 61) with one injection, reflecting the compartmentalization of the fascial layers.

Ultrasound-Guided Technique

The FN can be easily visualized in the inguinal region using ultrasonography with a linear 8–12 MHz transducer. The probe is placed in the inguinal crease on the transverse axial plane (Fig. 57.10). The large femoral artery is

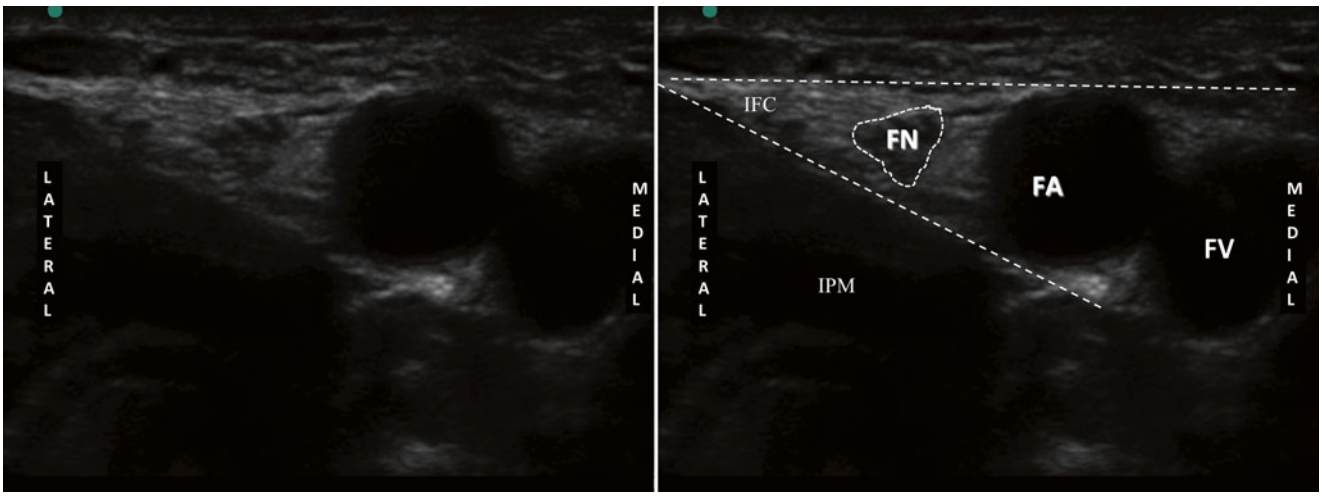

Fig. 57.11 Ultrasound image of the femoral nerve. *FA* femoral artery, *FV* femoral vein, *IPM* iliopsoas muscle, *IFC* iliac fascial compartment (Image courtesy of Thialgo Nouer Frederico, MD)

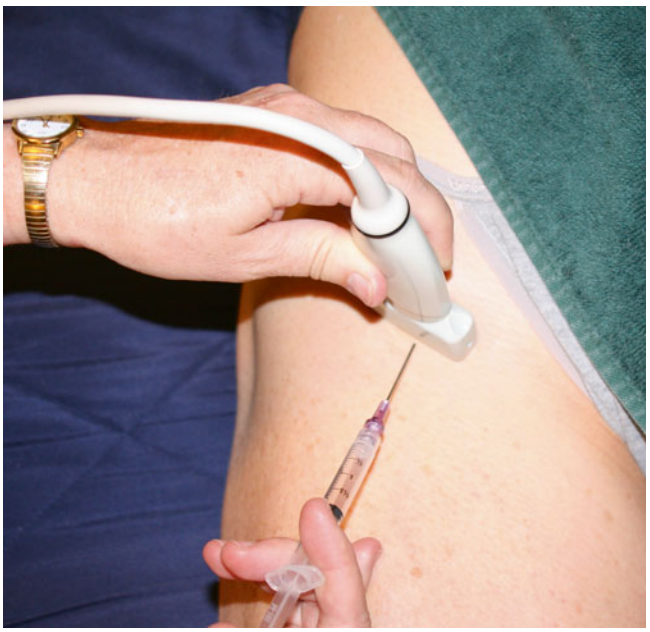

Fig. 57.12 Ultrasound out-of-plane injection of the femoral nerve, showing probe and needle position (Image courtesy of Andrea Trescot, MD)

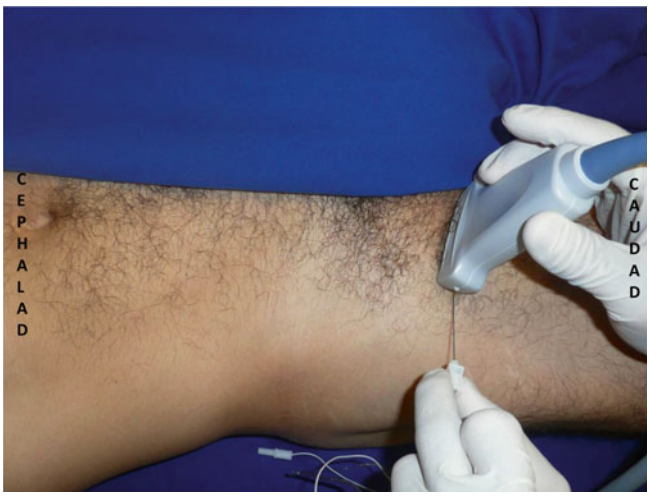

Fig. 57.13 Ultrasound in-plane injection of the femoral nerve, showing probe and needle position (Image courtesy of Thiago Nouer Frederico, MD)

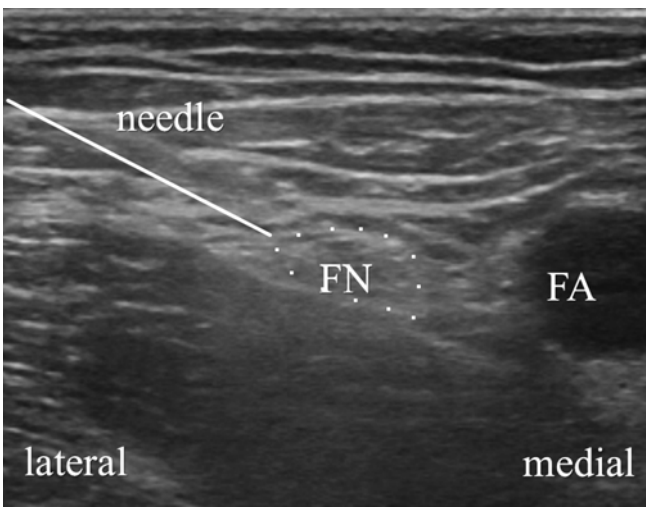

Fig. 57.14 Ultrasound image of simulated needle placement for femoral nerve injection (Image courtesy of Andrea Trescot, MD)

easily seen; the FN is slightly lateral and posterior to the artery (Fig. 57.11). It lies deep to the *iliopectineal arch* and overlies the groove separating the iliac from the psoas muscles and may appear oval or triangular. The needle is placed using either an out-of-plane (Fig. 57.12) or in-plane approach (Fig. 57.13) and advanced until the needle tip is near the nerve (Fig. 57.14). After negative aspiration for blood, watch the local anesthetic spread around the nerve. Smaller volumes may be sufficient with this approach, and a peripheral nerve stimulator may help to identify the nerve.

Fluoroscopy-Guided Technique

The femoral nerve does not have many bony landmarks, so there is little need for fluoroscopically guided injections. One exception is the treatment of hip pain in patients who are not candidates for hip arthroplasty. The hip joint is innervated by sensory branches of the *obturator nerve* (Chap. 64) and the FN. For FN injections of the hip, the patient is placed supine, and the needle is advanced below the anterior superior iliac spine. Using a peripheral nerve stimulator will facilitate the identification of the nerve.

Neurolytic/Surgical Techniques

Neurolysis is not commonly performed due to the extensive motor component of the femoral nerve and the weakness that would result from such a procedure.

Cryoneuroablation

Although neuritis and neuroma formation would not be seen with cryoneuroablation, the damage to motor fibers and resultant weakness of the leg limits the use of this technique.

Radiofrequency Lesioning

Because the sensory branches of the obturator and femoral nerves supply the hip joint, percutaneous RF lesioning of these branches was described in 2001 [28] to denervate painful degenerated hips in patients who were not candidates for hip arthroplasty. Rivera et al. [29] prospectively studied 18 patients with DJD of the hip who were not surgical candidates. Several days after a positive diagnostic injection, each patient underwent RF lesioning of the obturator and femoral sensory branches to the hip. The femoral nerve was identified below the anterior superior iliac spine (ASIS) at the anterolateral margin of the hip joint (Fig. 57.15). Sensory stimulation resulted in groin and thigh pain; after negative motor stimulation, the site was lesioned at 90 °C for 90 s. The obturator sensory branch was also treated (see Chap. 64). Eight patients (44 %) had >50 % pain relief at 6 months. Because of concerns regarding conventional RF and neuroma formation, Wu and Groner [30] proposed using pulsed RF for this technique; they treated two patients, both of whom noted >50 % pain relief at 3 months.

Surgery

In severe cases of FN entrapment, the femoral nerve can be surgically decompressed. Surgical decompression should be reserved for those cases where a clearly defined site or cause of entrapment can be identified [22].

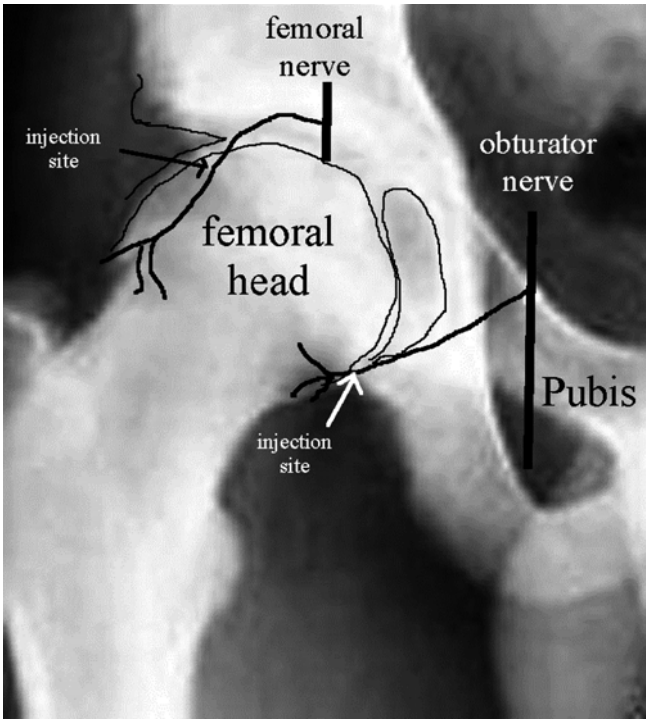

Fig. 57.15 Femoral and obturator sites for injection and denervation of the hip joint (Image courtesy of Andrea Trescot, MD)

Complications

Complications for FN block include infection, direct needle trauma to the neurovascular bundle, intravascular injection, and puncture of the femoral artery resulting in significant blood loss and/or hematoma formation. Transient FN palsy has been described after local anesthetic infiltration field blocks for open inguinal hernia repairs [25].

Summary

Femoral nerve pathology may be proximal (from psoas entrapment) or distal (at or below the inguinal ligament). The most common reason for FN injection is for surgical or post-surgical anesthesia, but several pain conditions can be diagnosed and treated with FN (and FN branch) injections.

References

1. Busis NA. Femoral and obturator neuropathies. Neurol Clin. 1999;17(3):633–53.
2. Hopper CL, Baker JB. Bilateral femoral neuropathy complicating vaginal hysterectomy. Analysis of contributing factors in 3 patients. Obstet Gynecol. 1968;32(4):543–7.
3. Goldman JA, Feldberg D, Dicker D, Samuel N, Dekel A. Femoral neuropathy subsequent to abdominal hysterectomy. A comparative study. Eur J Obstet Gynecol Reprod Biol. 1985;20(6):385–92.
4. Hershlag A, Loy RA, Lavy G, DeCherney AH. Femoral neuropathy after laparoscopy. A case report. J Reprod Med. 1990;35(5):575–6.

5. Meech PR. Femoral neuropathy following renal transplantation. Aust N Z J Surg. 1990;60(2):117–9.
6. Gombar KK, Gombar S, Singh B, Sangwan SS, Siwach RC. Femoral neuropathy: a complication of the lithotomy position. Reg Anesth. 1992;17(5):306–8.
7. Chevalier X, Larget-Piet B. Femoral neuropathy due to psoas hematoma revisited. Report of three cases with serious outcomes. Spine (Phila Pa 1976). 1992;17(6):724–6.
8. Brantigan JW, Owens ML, Moody FG. Femoral neuropathy complicating anticoagulant therapy. Am J Surg. 1976;132(1):108–9.
9. Aune S, Trippestad A. Chronic contained rupture of an abdominal aortic aneurysm complicated by infection and femoral neuropathy. Case report. Eur J Surg. 1995;161(8):613–4.
10. Kashuk K. Proximal peripheral nerve entrapment syndromes in the lower extremity. J Am Podiatry Assoc. 1977;67(8):529–44.
11. Coppack SW, Watkins PJ. The natural history of diabetic femoral neuropathy. Q J Med. 1991;79(288):307–13.
12. Kvist-Poulsen H, Borel J. Iatrogenic femoral neuropathy subsequent to abdominal hysterectomy: incidence and prevention. Obstet Gynecol. 1982;60(4):516–20.
13. Jog MS, Turley JE, Berry H. Femoral neuropathy in renal transplantation. Can J Neurol Sci: J Can Sci Neurol. 1994;21(1): 38–42.
14. Schottland JR. Femoral neuropathy from inadvertent suturing of the femoral nerve. Neurology. 1996;47(3):844–5.
15. Kline DG. Operative management of major nerve lesions of the lower extremity. Surg Clin North Am. 1972;52(5):1247–65.
16. Belli AM, Cumberland DC, Knox AM, Procter AE, Welsh CL. The complication rate of percutaneous peripheral balloon angioplasty. Clin Radiol. 1990;41(6):380–3.
17. Sammarco GJ, Stephens MM. Neurapraxia of the femoral nerve in a modern dancer. Am J Sports Med. 1991;19(4):413–4.
18. Petchprapa CN, Rosenberg ZS, Sconfienza LM, Cavalcanti CF, Vieira RL, Zember JS. MR imaging of entrapment neuropathies of the lower extremity. Part 1. The pelvis and hip. Radiographics. 2010;30(4):983–1000.
19. Beltran LS, Bencardino J, Ghazikhanian V, Beltran J. Entrapment neuropathies III: lower limb. Semin Musculoskelet Radiol. 2010;14(5):501–11.
20. Singelyn FJ. Femoral nerve block. In: Hadzic A, New York School of Regional Anesthesia, editors. Textbook of regional anesthesia and acute pain management. New York: McGraw-Hill, Medical Pub. Division; 2007. p. 499–508.
21. Vazquez MT, Murillo J, Maranillo E, Parkin IG, Sanudo J. Femoral nerve entrapment: a new insight. Clin Anat. 2007;20(2):175–9.
22. Shah A, Busconi B. Hip and pelvis. In: DeLee JC, Drez D, Miller MD, editors. DeLee and Drez's orthopaedic sports medicine. 1. 3rd ed. Philadelphia: Saunders Elsevier; 2010. p. 1451–69.
23. Hollis MH. Nerve entrapment syndromes of the lower extremity 2012. Cited 15 May 2013:[1–16 pp.].
24. Katirji B, Koontz D. Disorders of peripheral nerves. In: Daroff RB, Bradley WG, editors. Bradley's neurology in clinical practice. 6th ed. Philadelphia: Elsevier/Saunders; 2012. p. 1915–2015.
25. Rosario DJ, Jacob S, Luntley J, Skinner PP, Raftery AT. Mechanism of femoral nerve palsy complicating percutaneous ilioinguinal field block. Br J Anaesth. 1997;78(3):314–6.
26. Brown DL. Femoral block. In: Brown DL, editor. Atlas of regional anesthesia. 2nd ed. Philadelphia: Elsevier/Saunders; 1999. p. 105–10.
27. Winnie AP, Ramamurthy S, Durrani Z. The inguinal paravascular technic of lumbar plexus anesthesia: the "3-in-1 block". Anesth Analg. 1973;52(6):989–96.
28. Kawaguchi M, Hashizume K, Iwata T, Furuya H. Percutaneous radiofrequency lesioning of sensory branches of the obturator and femoral nerves for the treatment of hip joint pain. Reg Anesth Pain Med. 2001;26(6):576–81.
29. Rivera F, Mariconda C, Annaratone G. Percutaneous radiofrequency denervation in patients with contraindications for total hip arthroplasty. Orthopedics. 2012;35(3):e302–5.
30. Wu H, Groner J. Pulsed radiofrequency treatment of articular branches of the obturator and femoral nerves for management of hip joint pain. Pain Pract. 2007;7(4):341–4.

Proximal Saphenous Nerve Entrapment: Thigh and Knee

58

Andrea M. Trescot, Helen W. Karl, Michael N. Brown, and Beth S. Pearce

Introduction

The *saphenous nerve (SN)* is a pure sensory nerve. When this nerve is subjected to injury or compressive neuropathy, it can develop not only classic neuropathic pain in the distribution of the nerve but may also result in a cutaneous allodynia and a *complex regional pain syndrome (CRPS)* or CRPS-like picture. With the growing population of elderly patients and the associated increase in total joint arthroplasties [1], *infrapatellar saphenous* (IPS) neuropathies will be seen more often as a complication of procedures involving the knee.

Pain management providers need to understand the specific symptoms and signs that make a SN injury a likely cause of an individual's knee or leg pain. Anatomical understanding is critical to provide effective selective denervation procedures for SN neuropathies.

Clinical Presentation (Tables 58.1 and 58.2)

Proximal saphenous nerve entrapment (SNE) can cause pain in varied places, from the groin, to the medial mid-thigh (Fig. 58.1), to the medial knee, to the great toe (Fig. 58.2). Injuries to the proximal portion of the SN result in three distinct clinical manifestations. First, the SN can become injured by surgery in the groin or proximal thigh such as during femoral surgery or cardiac catheterization; these patients complain of groin and medial upper thigh pain. The second set of patients have the saphenous nerve entrapment in the distal thigh as originally described by Kopell in 1960 [18] and have symptoms in the knee, often extending down the medial calf (Fig. 58.3) and sometimes proximally to the hip or lower abdomen [7, 19, 20]. The third common presentation is caused by entrapment at the first major branch of the SN, the *infrapatellar saphenous nerve* (IPS); this nerve is also called the *infragenicular branch of the saphenous nerve* or the *sartorial nerve*. This is usually the result of iatrogenic or other trauma and results in pain and sensory disturbances more localized to the knee. The specific pattern of IPS entrapment is discussed below, while *distal saphenous* pathology is discussed in Chap. 59.

In 1989, Romanoff and colleagues [21] formulated criteria for SN entrapment in the *adductor canal* (see section "Anatomy" below): pain in the SN distribution, normal motor function, and tenderness over the adductor canal

Table 58.1 History relevant to proximal saphenous nerve entrapment or injury

Compression	Peripheral edema
Trauma	CABG – vein harvest [2]
	Cardiac catheterization
	Femoral vascular surgery [3]
	Varicose vein stripping [4]
	Kneeling ("housemaid's knee) [5]
Sports	Surfing [6]
	Idiopathic [7]

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Table 58.2 History relevant to infrapatellar saphenous nerve injury

Surgery involving the medial knee	Total knee replacement (TKR) [8, 9]
	Patellar tendon harvest [10]
	Hamstring tendon harvest [11]
	Arthroscopy [12, 13]
Trauma	Medial knee trauma [14, 15]
	Knee joint injection [16]
	Idiopathic [17]

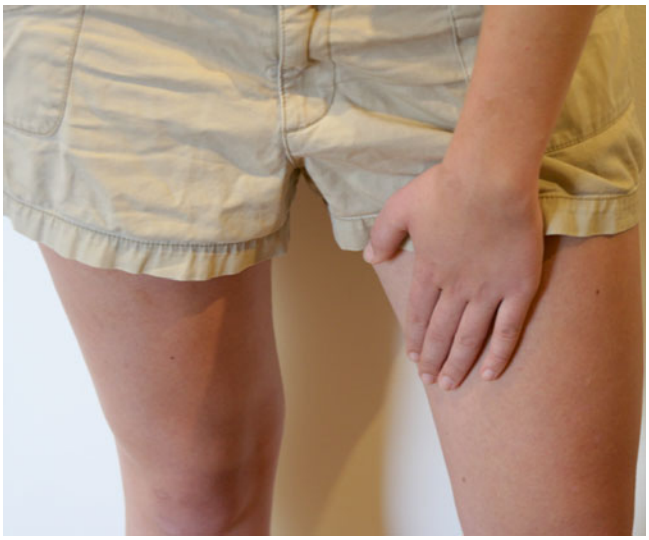

Fig. 58.1 Patient pain complaints from proximal saphenous nerve entrapment (Image courtesy of Andrea Trescot, MD)

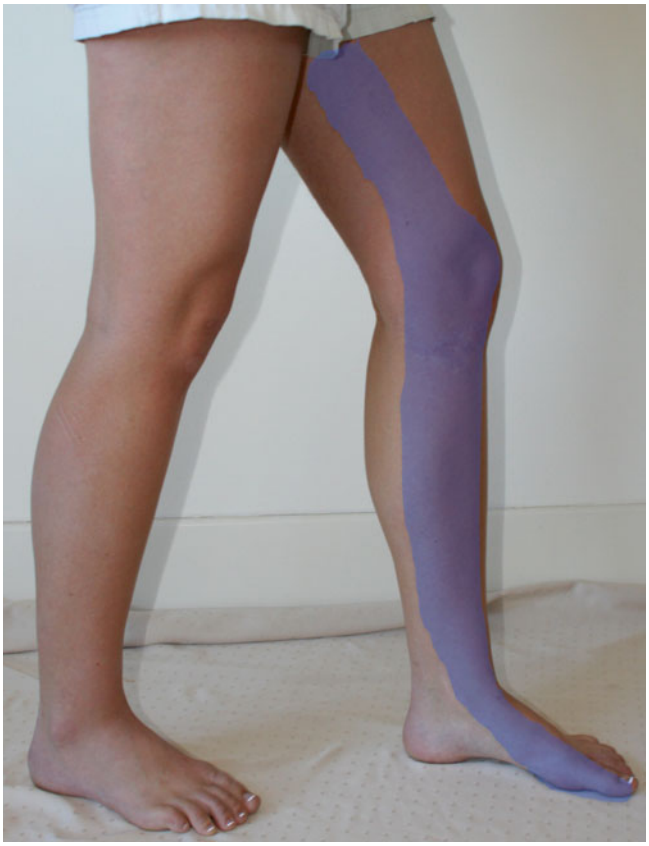

Fig. 58.2 Pattern of pain from proximal saphenous nerve entrapment (Image courtesy of Andrea Trescot, MD)

(Fig. 58.4). Of the 30 patients in their study, 27 (90 %) had knee pain, 2 had thigh pain, and 1 had calf pain [21]. Most investigators report a predominance of women, somewhere between 73 % [21] and 79 % [21] of all patients. The patient's leg may feel tired and heavy [19]; the discomfort is aggravated by walking and standing [22] and sometimes simulates claudication [19]. Almost 70 % of patients in one study had night pain [22], and pain at rest has been described as potentially "severe" [18].

Surfers have been noted to develop medial thigh and knee pain due to proximal saphenous trauma caused by gripping the surfboard between their legs [6]. *Varicose vein stripping* has also been associated with SNE, but is usually less severe. Morrison and Dalsing looked at patients after saphenous vein grafting; small areas of sensory deficits were commonly found on exam (26/45 legs = 58 %), but more than half of those patients were not aware of the numbness. Fewer than 10 % of patients had symptoms of SN injury that negatively impacted their quality of life [4].

The pattern of pain in patients with *IPS entrapment* (IPSE) does not usually help the clinician, since the pain is usually not well localized. Patients describe generalized anterior knee pain, perhaps more medially and possibly radiating to the medial calf. The pain is characteristically neuropathic in nature (see Chap. 1) with complaints of burning, tingling, and sensitivity. They may walk "stiff legged" to avoid flexion of the knee. The joint may be red, hot, and swollen, as though infected, with allodynia and pain on movement. Laboratory measurement of inflammatory markers, however, will usually be normal. Especially postoperatively, patients may present with sharp burning pain, hyperesthesia, and/or allodynia that may mimic CRPS. There may be visible swelling in the *medial tibial fossa*, and palpation of this area usually replicates the pain (see section "Physical exam" below) [23].

Anatomy (Tables 58.3 and 58.4)

The SN, which is composed of sensory fibers from the L3 and L4 nerve roots (Fig. 58.5), branches off of the *femoral nerve* not far below the *inguinal ligament* (Fig. 58.6) and then descends through the anteromedial thigh with the femoral artery and vein to the *adductor (Hunter's) canal*. The femoral vessels travel posteriorly toward the *popliteal fossa*, while the SN and descending *genicular branch of the femoral artery* penetrate the *vastoadductor membrane*, the dense connective tissue between the *adductor magnus* and the *vastus medialis* muscles, about 10 cm above the medial

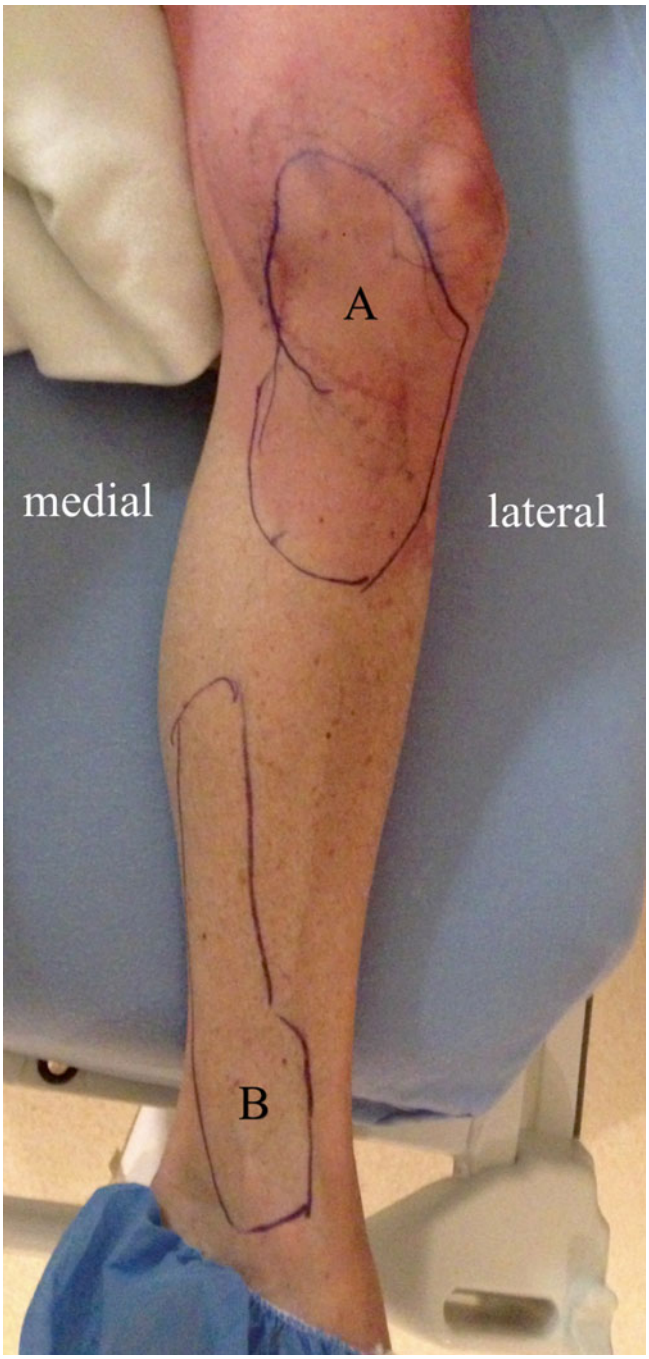

Fig. 58.3 Pattern of pain in a patient with proximal saphenous entrapment (infrapatellar saphenous) (**a**) and distal saphenous nerve entrapment (**b**) (Image courtesy of Agnes Stogicza, MD)

femoral epicondyle (Fig. 58.7). The combination of direction change and a relatively noncompliant corridor is a classic setting for peripheral nerve entrapment (Chap. 1), though the nerve can be injured anywhere along its lengthy course.

Distal to the vastoadductor membrane, the anatomic variability of the SN, and particularly its infrapatellar branch (IPS) (occasionally labeled as the *infragenicular branch of the saphenous nerve* [30]), increases. In most people, two or more branches of the SN leave the adductor canal proximal

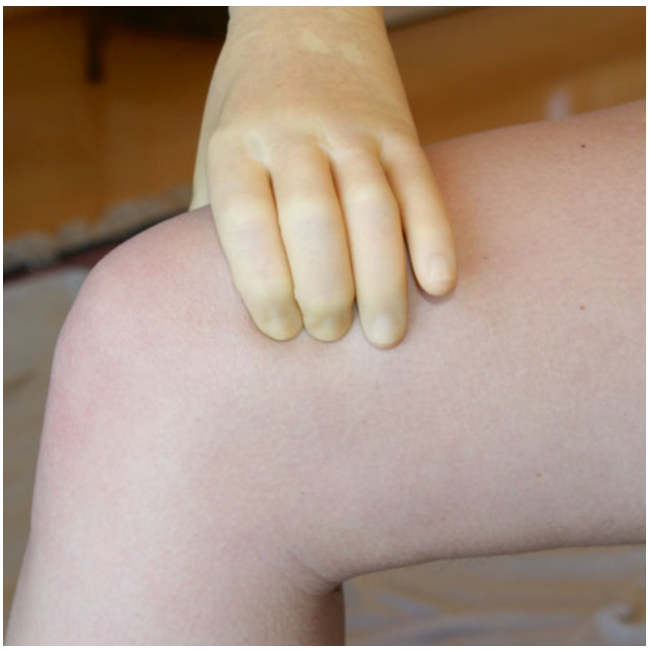

Fig. 58.4 Tenderness over the adductor canal (Image courtesy of Andrea Trescot, MD)

to the joint line, the most anterior of which crosses the knee as the IPS (Fig. 58.8) to innervate the skin below the patella and the anterior inferior knee capsule (Fig. 58.9). The most posterior branch continues as the distal *saphenous (sartorial) nerve*, discussed in Chap. 59.

Arthornthurasook studied the relationship of the IPS to the sartorius muscle [27]. He found that in most (62 %) of the cadaver limbs, the IPS becomes superficial at the posterior border of the sartorius and crosses the surface of that muscle to the front of the knee. In 22 % of his specimens, the nerve penetrated the muscle; in 14 % of knees studied, it ran parallel and posterior to the muscle; and in 3 %, it emerged from the anterior edge of the sartorius. Only half of the knees were identical on both sides.

Mochida et al. examined IPS anatomy with the perspective of an arthroscopist, focusing on the point at which the IPS crosses the proximal edge of the tibia [31]. They found two predominant patterns in the relationship between the patellar ligament and the tibial plateau. More commonly (68 %), the IPS crosses the joint line medial to the medial margin of the patellar ligament; in 32 % of cases, the nerve crosses the patellar ligament proximal to the joint line. Based on the measurement of the distance between the IPS and the medial margin of the patella, they identified a "safe area" for blind puncture to allow passage of an arthroscope.

In one of the most recent anatomic studies of the IPS, Ackmann et al. [29] studied the location of the IPS to determine the site of both the safest surgical incision and the most appropriate location for cryoneuroablation (see "Neurolytic" section below). They dissected 30 knees and identified 4 different patterns (Fig. 58.10): an *anterior type* (the IPS emerges from

Table 58.3 Proximal saphenous nerve anatomy

Origin	The ventral rami of L3–L4 spinal nerves contribute to the femoral nerve. The femoral nerve gives off its long medial sensory branch, the saphenous nerve (SN), about 8 cm below the inguinal ligament
General route	Accompanies the femoral artery part way through the adductor (Hunter's) canal and then passes through the vastoadductor membrane to the knee with the descending genicular branch of the femoral artery. The part of the nerve after the division to the IPS is sometimes known as the *sartorial nerve*
Sensory distribution	Medial knee, calf, and ankle
Motor innervation	None
Anatomic variability	Nerve position may change in obese patients [16]
Other relevant structures	Adductor canal: bounded by the adductor longus, adductor magnus, sartorius, and vastus medialis muscles. It is approximately 15 cm long and extends from the distal femoral triangle to the adductor hiatus about 4 finger breadths above the medial femoral condyle
	Vastoadductor membrane: a thickening of the fascia between the vastus medialis and the adductor magnus [24]
	Sartorius muscle: a landmark for ultrasound guidance [25]

Table 58.4 Infrapatellar saphenous nerve anatomy

Origin	Ventral rami of L3–L4
General route	Branches from the saphenous nerve at a variety of levels and extends toward the patella
Sensory distribution	Skin below the patella
	Anterior knee capsule [26]
Motor innervation	None
Anatomic variability	Branching patterns are highly variable [23, 27, 28]
	Nerve position changes with the position of the knee [29]
Other relevant structures	Sartorius muscle

the anterior border of the sartorius muscle), a *posterior type* (the IPS emerges from the posterior border of the sartorius muscle), a *penetrating type* (the IPS penetrates the sartorius muscle), and a *pes anserinus type* (the IPS penetrates the sartorius muscle close to the pes anserinus). They also found that a medial parapatellar skin incision was associated with the highest risk of damaging the IPS because the incision is over the nerve in 53.3 % of cases. A midline skin incision is the second most likely to damage the IPS, since the IPS fibers crossed or reached the tibial tuberosity in 46.7 % of cases.

Entrapment

Entrapment of the proximal SN may occur at several levels. From proximal to distal, these include the inguinal ligament, the proximal thigh [32], the adductor canal [18], and the IPS [23] (Fig. 58.11). Distal saphenous entrapment sites are discussed in Chap. 59.

The pathophysiology of SN neuropathy may involve acute high-pressure compression or chronic intermittent compression, as well as posttraumatic or postsurgical scarring. For example, the proximal SN can be entrapped by aneurysms of the femoral vessels [32], scar tissue or trauma caused by a femoral catheterization, or a pes anserinus bursa

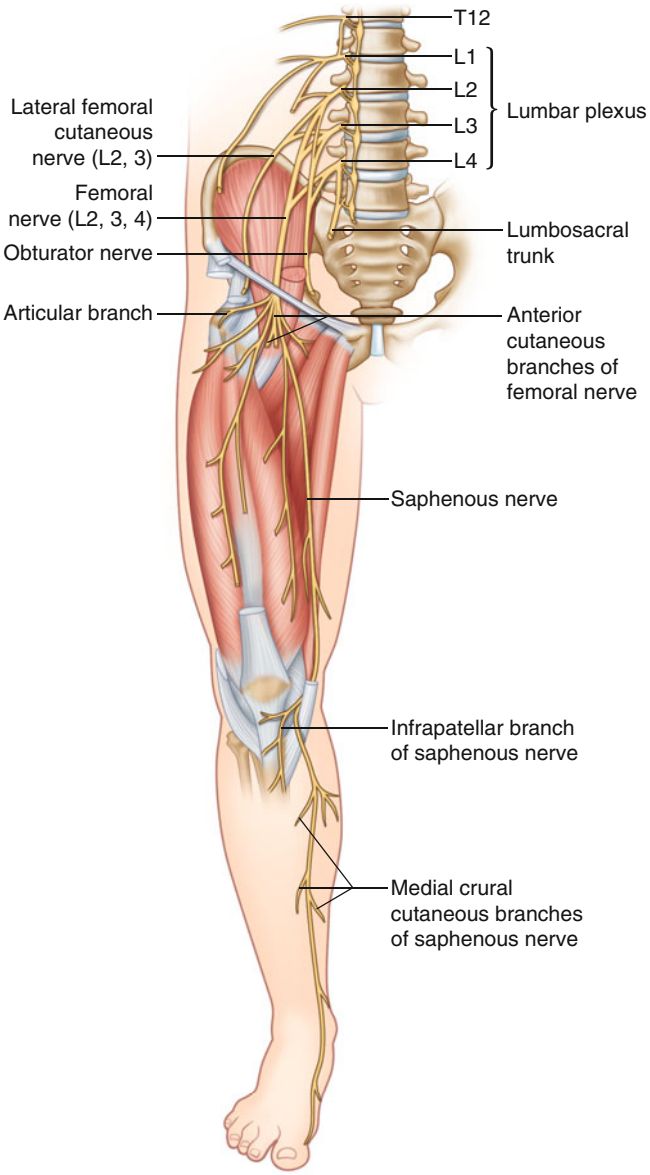

Fig. 58.5 Anatomy of the anterior thigh (Image from Springer)

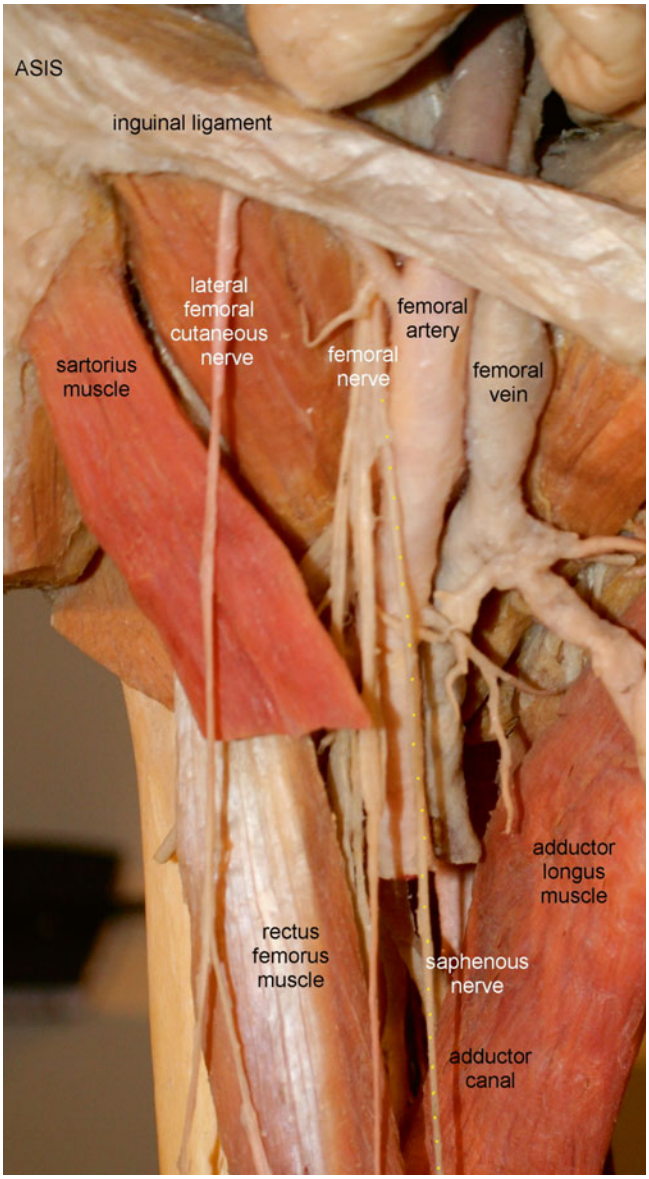

Fig. 58.6 Dissection of the anterior thigh and groin, modified from an image from *Bodies*, *The Exhibition*, with permission (Image courtesy of Andrea Trescot, MD)

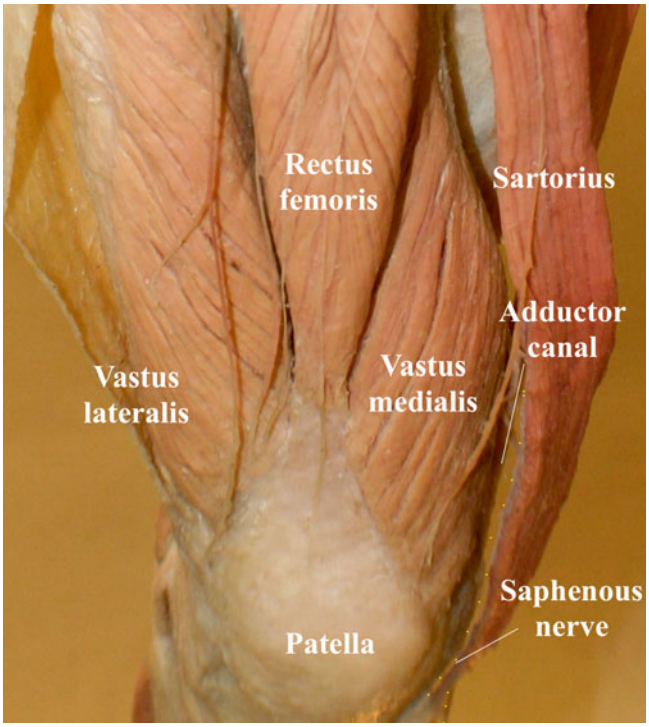

Fig. 58.7 Anterior knee dissection, modified from an image from *Bodies*, *The Exhibition*, with permission (Image courtesy of Andrea Trescot, MD)

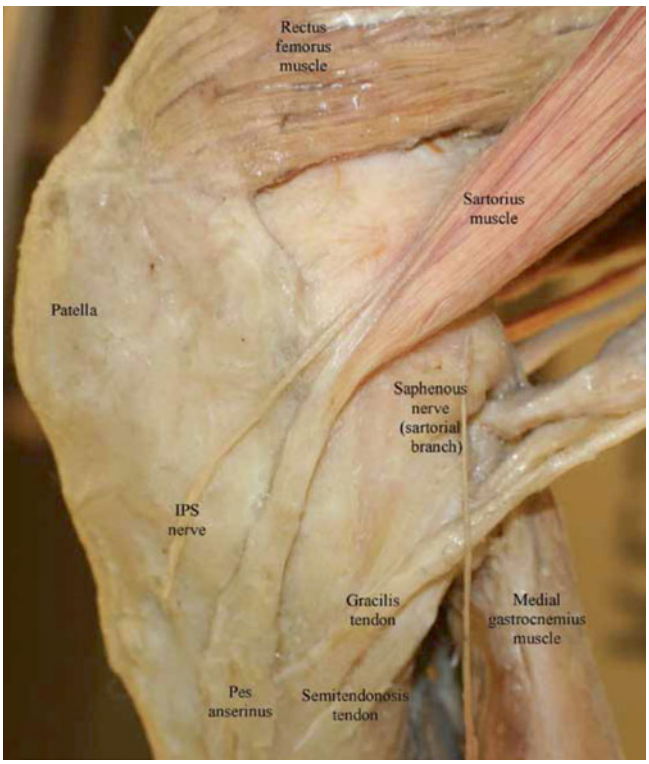

Fig. 58.8 Dissection of the medial knee, modified from an image from *Bodies*, *The Exhibition*, with permission (Image courtesy of Andrea Trescot, MD)

[33]. Entrapment by scar tissue or direct trauma after knee surgery is the most common cause of IPS pain [23].

Physical Exam

Physical examination and diagnostic injections are the cornerstones of clinical diagnosis of SN and IPS neuropathies. Palpation along the route of the nerve may show tenderness; local pressure may aggravate sensory symptoms (*Hoffman-Tinel's sign*) and indicate the site of the lesion. Pressure at the site where the SN leaves the adductor canal, approximately 4 finger breadths above the medial femoral condyle, often

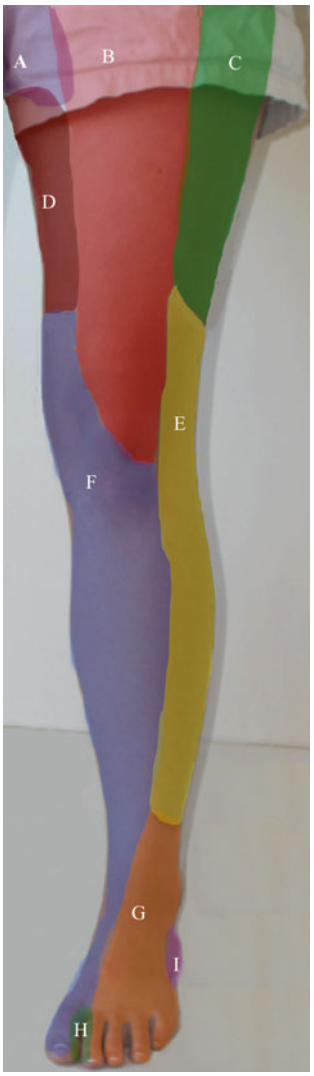

Fig. 58.9 Pattern of pain from anterior lower extremity nerve entrapments. *A* genitofemoral nerve, *B* femoral nerve, *C* lateral femoral cutaneous nerve, *D* obturator, *E* lateral sural cutaneous nerve, *F* saphenous nerve, *G* superficial peroneal nerve, *H* deep peroneal nerve, *I* sural nerve (Image courtesy of Andrea Trescot, MD)

causes pain which radiates proximally and distally [18, 19] (Video 58.1, Fig. 58.12). If the IPS is injured or entrapped, pressure at the depression just distal to the medial tibial flare is likely to reproduce symptoms [23] (Video 58.2, Fig. 58.13).

Symptoms may be increased with hyperextension and external rotation of the hip (*the femoral stretch test*) [20]. Reflexes and other motor function testing are normal. The presence of normal motor function is a key distinction between the SN symptoms and lumbar radiculopathy.

Differential Diagnosis (Table 58.5)

SN neuropathy can mimic diverse musculoskeletal, vascular, and other nervous system problems. As described in Table 58.5, there is often confusion between saphenous neuralgia and a variety of other knee pathologies, including medial collateral ligament, pes anserinus, and sartorius pathologies. Because of medial groin pain, there may be confusion regarding hip pathologies, and the claudication-like heaviness with ambulation from saphenous entrapment can mimic vascular pathologies.

Diagnostic Tests (Tables 58.6 and 58.7)

Sensory nerve conduction studies, although technically challenging, have been described for the SN [35] and for the IPS [37]. Lesions proximal to the dorsal root ganglion (DRG), such as L4 radiculopathy, will have normal saphenous sensory nerve action potential (SNAP) amplitudes, while lesions distal to the DRG classically have reduced SNAPs [5]. Electrodiagnostic testing in the elderly may be less valuable, since the superficial location of the nerve allows repeated microtrauma, which, on testing, may appear as pathology. As a result, abnormalities in conduction velocity are common, and test results should always be compared to the findings on the contralateral asymptomatic side. In general, the authors have found that diagnostic injections may be equally, if not more, useful than electrodiagnostic testing in the diagnosis of SN pathologies.

Ultrasound has helped to identify the site of entrapment as well as guide the diagnostic injections (see "Injection" section below) [25, 34]. MRI findings of the proximal saphenous nerve are not well described, but the IPS can be seen on sagittal knee images (Fig. 58.14).

Identification and Treatment of Contributing Factors

Simple approaches should be tried first, including weight loss, reduction of chronic edema, and adjustment of tight clothing that might be leading to local irritation of the nerve. Decreasing mechanical stress forces by treating *genu varum* and the accompanying internal tibial torsion will also reduce stress on the nerve.

Neural mobilization aims to reduce neuropathic pain by stretching the nerve and freeing it from inflammatory scarring or other attachments [38, 39]. This technique may be particularly helpful when the SN is entrapped at the adductor canal. *Saphenous nerve mobilization* is accomplished by placing the foot of the patient's affected leg against a wall with the other leg in front as for a lunge (Fig. 58.15a). Each time the front leg knee is bent for the lunge, the nerve is stretched. The patient must also stretch the femoral nerve by repeating the lunge while looking up and placing the ipsilateral arm over their head (Fig. 58.15b).

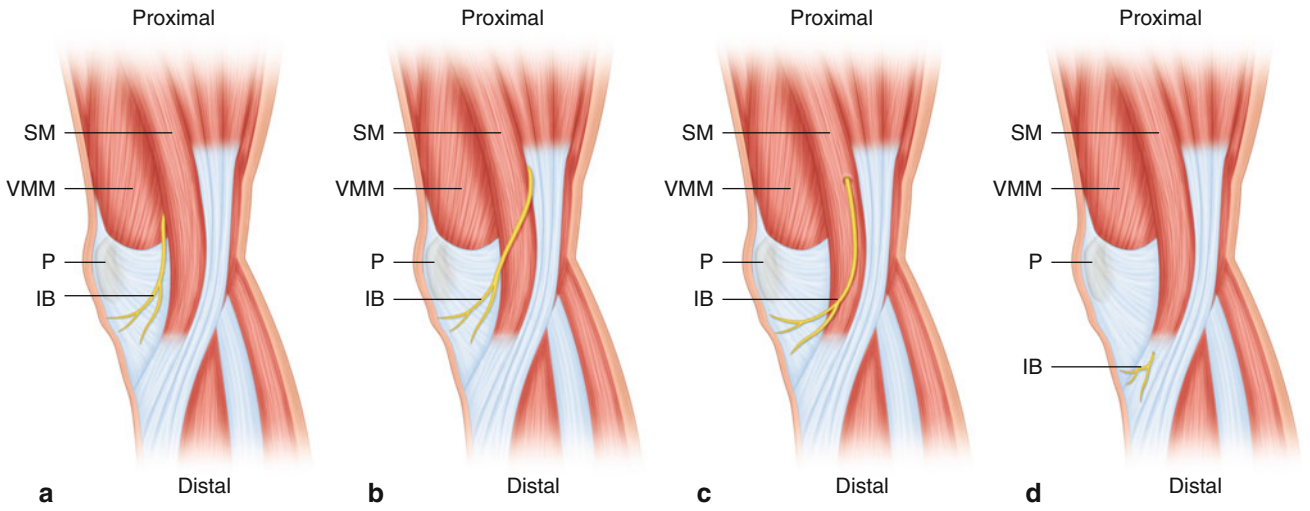

Fig. 58.10 Patterns of infrapatellar saphenous nerve location, seen as medial views of right knee joints. Four types of anatomical variations of the infrapatellar branch (*IB*) in relation to the sartorius muscle (*SM*) are shown. (**a**) Anterior type – the IB emerges from the anterior border of the SM; (**b**) posterior type – the IB emerges from the posterior border of the SM; (**c**) penetrating type – the IB penetrates the belly of the SM; (**d**) the pes anserinus type – the IB penetrates the tendon of the SM close to the superficial pes anserinus; *VMM* vastus medialis muscle, *P* patella, *SM* sartorius muscle (From Ackmann et al. [29]. Reprinted with permission from American Society of Interventional Pain Physicians)

Injection Techniques

Diagnostic injections to confirm SN or IPS neuropathy require low volumes of injectate for specificity. Large quantities of local anesthetics that spread through the mid-thigh or anterior tibial tissues will confound the diagnosis. Careful postinjection assessment of the degree of symptomatic relief should demonstrate significant improvement in order to confirm the diagnosis.

Landmark-Guided Technique

Romanoff et al. first formally described the SN block at the adductor canal in 1989 [19]. The patient is positioned supine or seated with the knee slightly flexed. Their technique is to find the plane between the *vastus medialis* and *sartorius* muscles by palpating the medial mid-thigh from anterior to posterior. Advance a short-beveled needle perpendicular to the skin until the "pop" through the fascia of the vastus medialis is felt. Paresthesias are not needed, and aspiration of blood is uncommon. Repeated injections using this technique led to long-term pain relief in most patients (24/30 = 80 %). Injections at the point of maximum tenderness were somewhat less successful in providing lasting analgesia (12/32 = 38 %) [19]. Because of the nerve's small size and the absence of a motor component, conventional nerve localization techniques, including eliciting paresthesias with successive approximation, have demonstrated inconsistent success [40–42]. Of these, the transsartorial approach was most successful [41].

Alternatively, the needle can be advanced more parallel to the nerve, distal to proximal (Fig. 58.16a), or proximal to distal (Fig. 58.16b, Video 58.3). Dr. Gabor Racz describes the injection of 10 cc of local anesthetic and steroid with proximal compression to perform a hydrodissection (personal communication).

Injection of the IPS is also performed with the knee flexed. The non-injecting hand identifies the point of maximum tenderness, with the index and middle fingers straddling the nerve from below. The needle enters from below and is directed toward the tibial tubercle and advanced to bone (Video 58.4, Fig. 58.17) [23]. Employing a nerve stimulator can be a useful adjunct to landmark-guided SN blockade (Fig. 58.18) [43], although as more practitioners become skilled with ultrasound, landmark-guided techniques will likely be used less frequently (see below).

Fluoroscopy-Guided Injections

Fluoroscopy has been used as an adjunct to pulsed radiofrequency [44] and cryoneuroablation of the IPS (see section "Cryoneuroablation" below), but has very little role in diagnostic injections.

Ultrasound-Guided Technique

Detailed descriptions of the diverse approaches to US-guided SN blockade are beyond the scope of this work. Some authors describe using the descending genicular artery as the central landmark for their technique [45], though this structure may

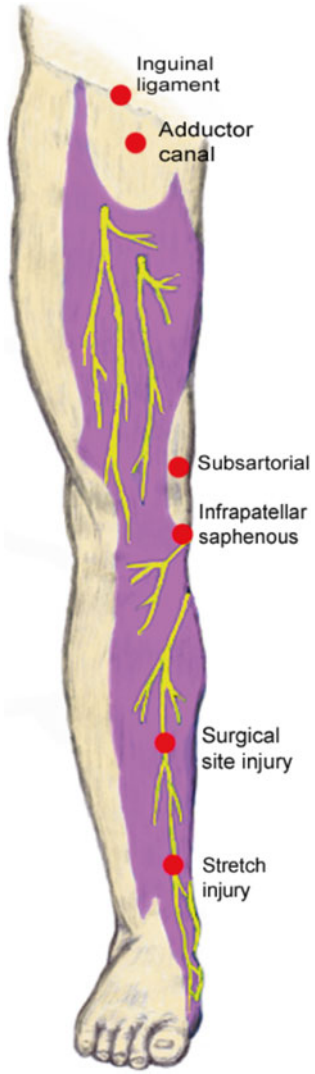

Fig. 58.11 Saphenous entrapment sites (Image courtesy of Michael Brown, MD)

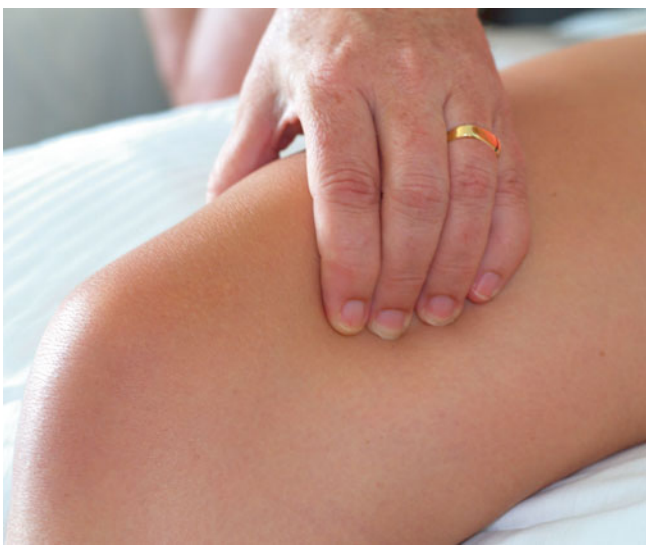

Fig. 58.12 Physical exam of the adductor canal (Image courtesy of Andrea Trescot, MD)

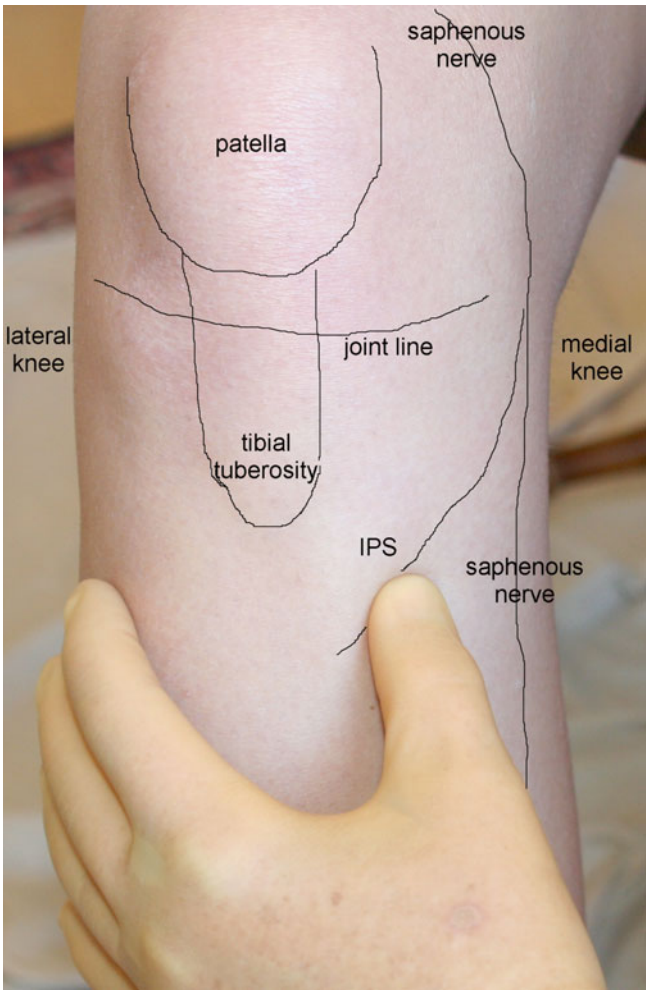

Fig. 58.13 Physical exam of the infrapatellar saphenous nerve (Image courtesy of Andrea Trescot, MD)

Table 58.5 Differential diagnosis of medial knee and/or leg pain

	Potential distinguishing features
Lumbar radiculopathy or plexopathy	Changes in muscles: weakness, atrophy, reflexes
Venous insufficiency or inflammation	Pain is not increased with pressure at the exit of the adductor canal
Medial tibial stress fracture [33]	X-rays and bone scan will show fracture
Arterial insufficiency	Abnormal pedal pulses [19]
Sartorial tendonitis	Tenderness at the sartorius attachment (see Fig. 58.6)
Pes anserinus bursitis	Tenderness more distal over the pes anserinus (see Fig. 58.6)
Medial collateral ligament (MCL) injury	Medial instability, pain with valgus movement
Hip joint pathology [19]	X-rays and MRI will show pathology

be difficult to locate [46]. Others describe using the plane between the sartorius and vastus medialis muscles [47] (Fig. 58.19), although this plane may be difficult to locate in obese patients [46]. The superficial femoral artery in the

Table 58.6 Diagnostic tests for distal thigh SN entrapment

	Potential distinguishing features
Physical exam	SN is tender at the point of exit from the adductor canal [22, 23]
Diagnostic injection	Results in numbness in SN distribution and pain relief [22]
Ultrasound [25, 34]	May improve the accuracy of the injection
MRI	MRI findings of saphenous entrapment neuropathies are not well described
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies [35]	Compare affected NCV with the asymptomatic side; EMG should be normal

Table 58.7 Diagnostic tests for IPS entrapment

	Potential distinguishing features
Physical exam	Tenderness over the medial tibial fossa
Diagnostic injection	1–1.5 mL of local anesthetic and deposteroid
Ultrasound [36]	The nerve is visible in the adductor canal and in the tibial fossa
MRI	Not well described, but IPS is visible on coronal MRI (Fig. 58.13)
Arteriography	Not useful
X-ray	Rule out joint pathology
Electrodiagnostic studies [37]	Compare NCV of affected with the asymptomatic side; EMG should be normal

mid-proximal femur may provide a landmark that is reliable across a range of providers and patients [46, 48]. A prospective, controlled, double-blinded, crossover trial of proximal US SN injections (in the fascial plane between the vastus medialis and sartorius) vs. landmark-guided SN injections showed that the US-guided injections gave 80 % relief, compared to 50 % relief with the landmark-guided injections [46].

US would be expected to improve proximal SN injections for below-the-knee surgeries. A randomized prospective crossover trial of 30 volunteers showed that two US-guided approaches to SN block were substantially more successful (80–100 % loss of sensation to pinprick) than a below-the-knee field block (30 %) [46].

US guidance may also be useful for the IPS block [49]. In a prospective, double-blinded, randomized, placebo-controlled study comparing IPS blocks to placebo for knee arthroscopies, the femoral artery was used as a landmark to find the SN (Fig. 58.20); the nerve was then traced distally to the take off of its infrapatellar branch, which was injected with 10 cc of 0.25 % bupivacaine. This approach provided good anesthesia for the knee arthroscopy, with the advantage of a substantial decrease in the risk of quadriceps weakness when compared to large volume, more proximal techniques [50]. Alternatively,

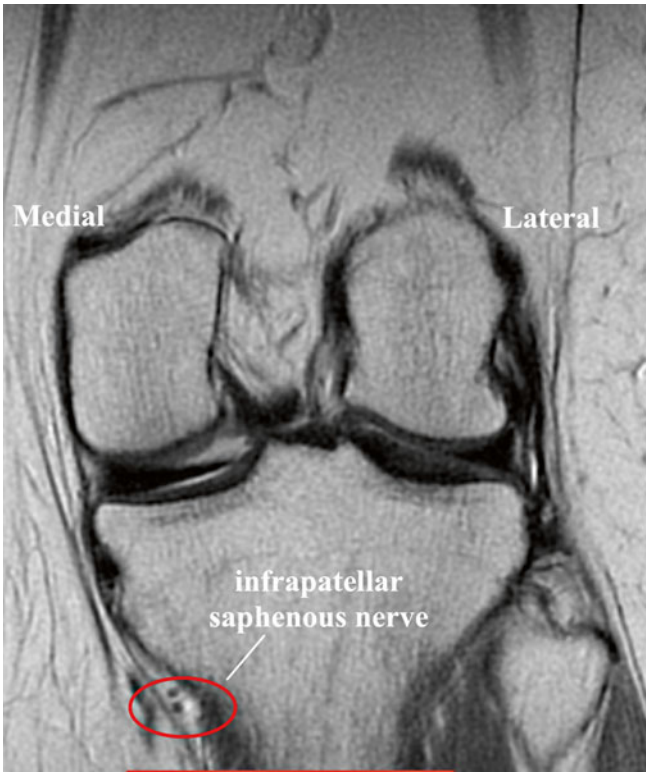

Fig. 58.14 Infrapatellar saphenous nerve on MRI (Image courtesy of Andrea Trescot, MD)

the US probe can be placed transversely across the nerve at the tibial plateau (Fig. 58.21a); the nerve will be seen in cross section as it passes from medial to lateral (Fig. 58.21b) and injected with just 1 cc of local anesthetic and deposteroid.

Clendenen et al. [51] retrospectively reviewed 16 consecutive patients with persistent knee pain after total knee replacement (arthroplasty). These patients underwent hydrodissection of the IPS under US guidance; 9 of the 16 noted post procedure scores of 0–1 for a minimum of 9 months.

Neurolytic Techniques

Cryoneuroablation

The SN can be treated with cryoneuroablation anywhere along its distribution where it can be identified [52], and successful cryoneuroablation of the IPS has been reported [23, 29]. Because of the built-in nerve stimulator, cryoneuroablation can be performed by landmark guidance (Fig. 58.22), fluoroscopic guidance (Fig. 58.23), or direct US visualization. Ackmann et al. [29], based on their dissections, noted that because of the great variability of the nerve, the optimum cryoneuroablation site varied with the anatomic type, so that no common cryoneuroablation site could be universally applied. Hence, US visualization should be preferable to using landmarks or fluoroscopy.

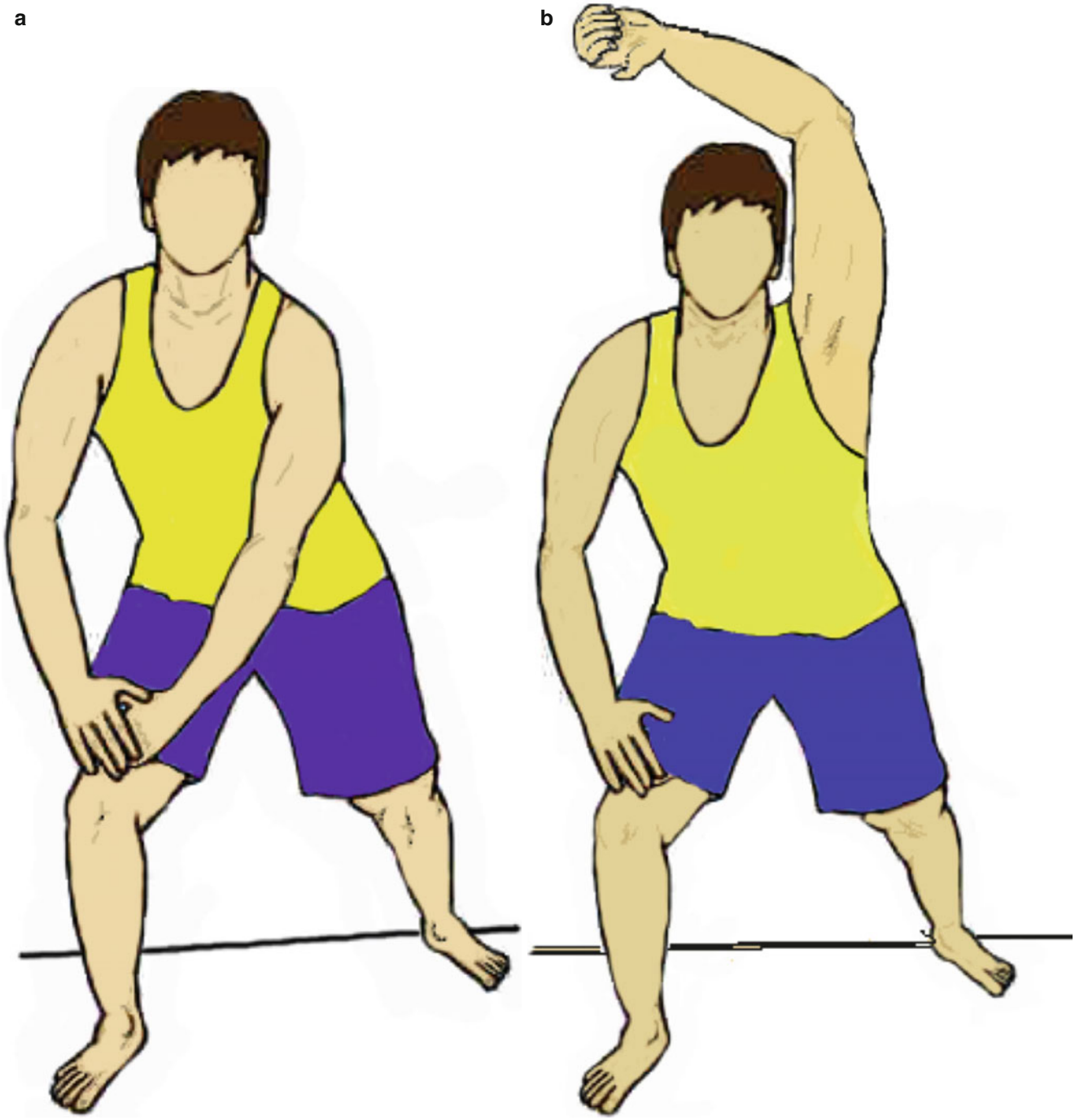

Fig. 58.15 Saphenous nerve (**a**) and femoral nerve (**b**) mobilization (Image courtesy of Michael Brown, MD)

Radiofrequency

Akbas et al. [44] described the use of pulsed radiofrequency (PRF) lesioning of the IPS for treatment of the pain of osteoarthritis of the knee. All of the 115 treated patients were "substantially improved" by this therapy.

Nerve Stimulation

Spinal Cord Stimulation

Spinal cord stimulation (SCS), as described in Chap. 9, can be used to treat lower limb pain, such as seen with saphenous

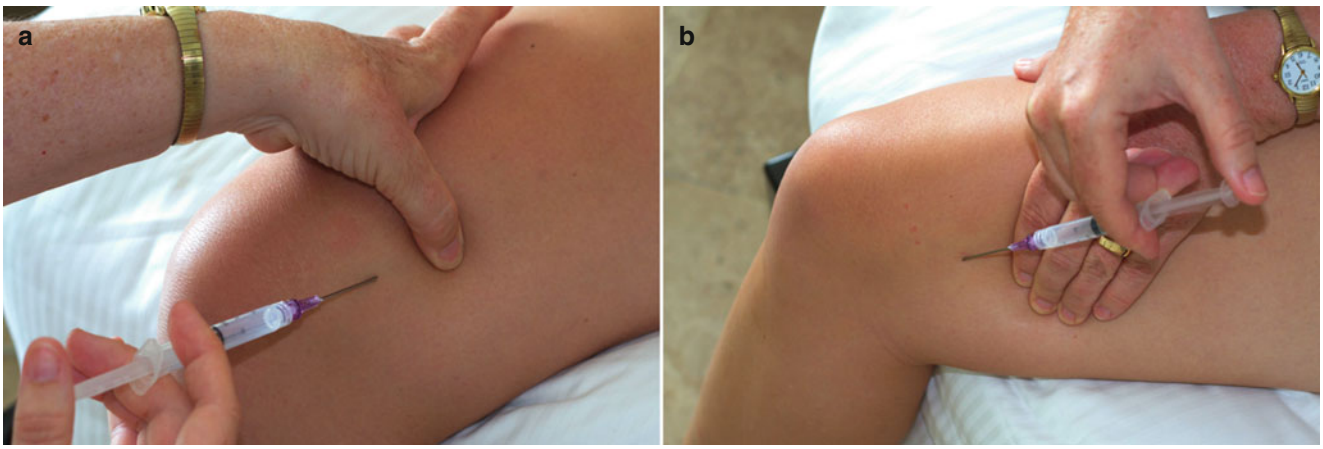

Fig. 58.16 Injection of the saphenous nerve in the adductor canal. (**a**) Distal to proximal; (**b**) proximal to distal (Image courtesy of Andrea Trescot, MD)

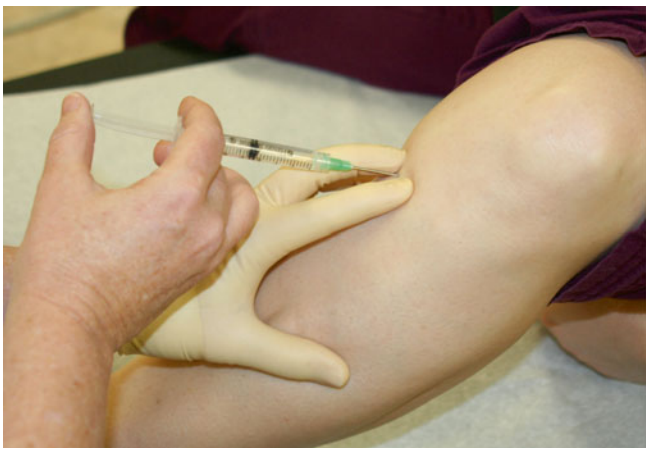

Fig. 58.17 Landmark-guided injection of the infrapatellar saphenous nerve (Image courtesy of Andrea Trescot, MD)

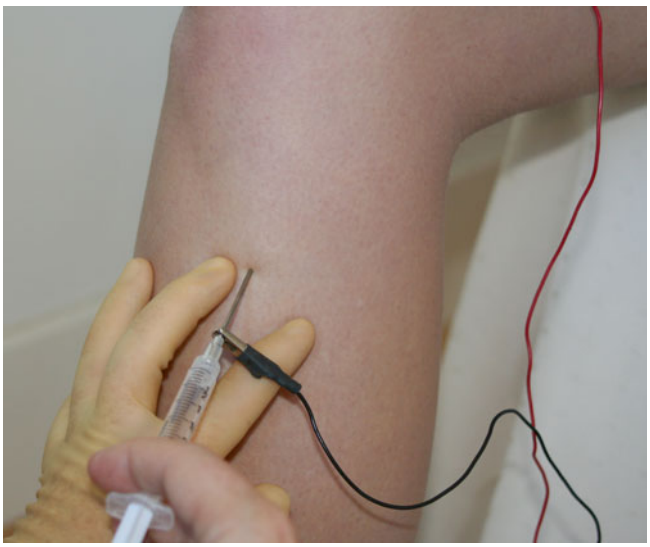

Fig. 58.18 Infrapatellar saphenous nerve landmark-guided injection with peripheral nerve stimulator use (Image courtesy of Andrea Trescot, MD)

nerve pathology. Lowry and Simopoulos described the use of SCS to treat a patient with knee pain after a total knee replacement (TKR); the pattern of pain seen on Fig. 58.24a is consistent with IPS neuralgia, while Fig. 58.24b shows the location of the stimulation lead.

Peripheral Nerve Stimulation

Although not yet reported in the literature, Dr. Porter McRoberts shared with us images (Fig. 58.25) showing the use of peripheral nerve field stimulation (see Chap. 9) for knee pain after TKR (personal communication).

Surgery

If there is a temporary but not sustained response to injections for adductor canal entrapment, some authors have recommended splitting the roof of the adductor canal [7, 19]. In one study, 14 out of 16 patients (88 %) who underwent this procedure improved [19], though other authors

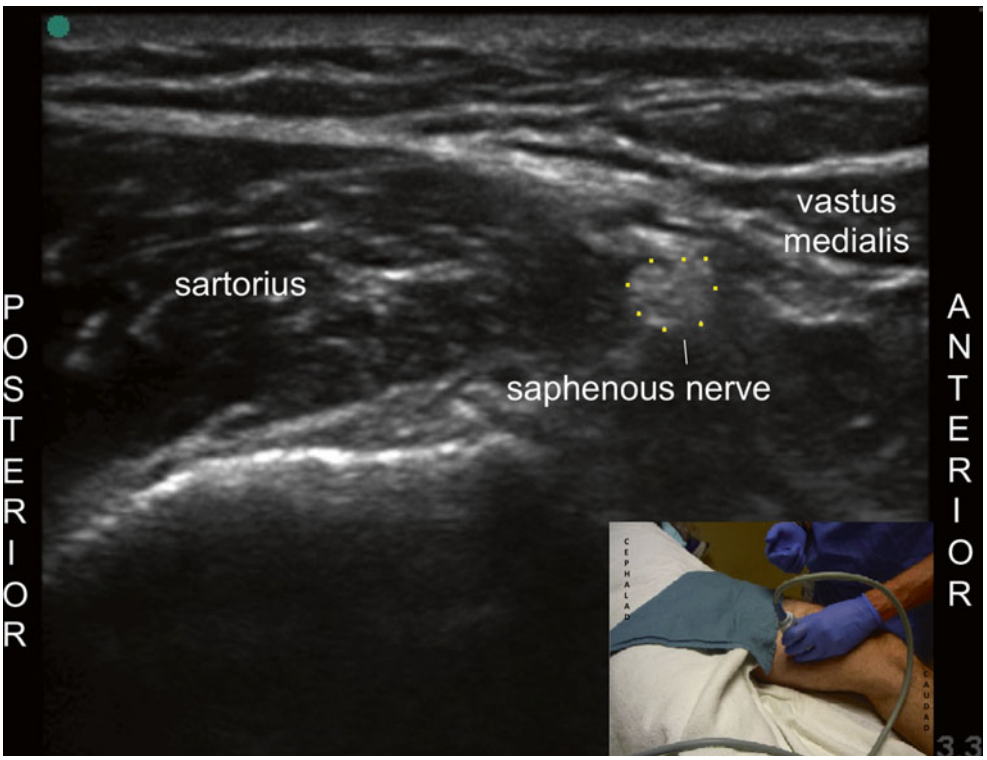

Fig. 58.19 Ultrasound identification and injection of the proximal saphenous nerve in the adductor canal (Image courtesy of Thiago Nouer Frederico, MD)

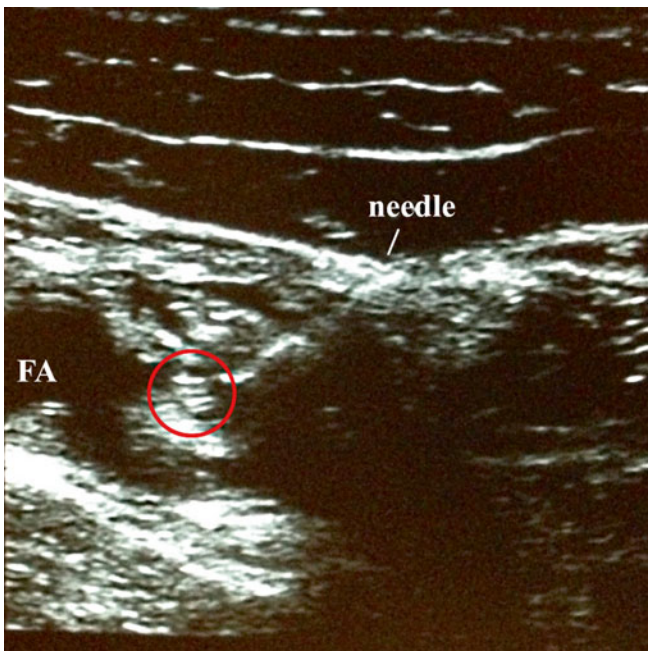

Fig. 58.20 Ultrasound injection of the proximal saphenous nerve, using the femoral artery as a landmark, with the saphenous nerve *circled*. Note needle approaching from the *right*. *FA* femoral artery (Image courtesy of Agnes Stogicza, MD)

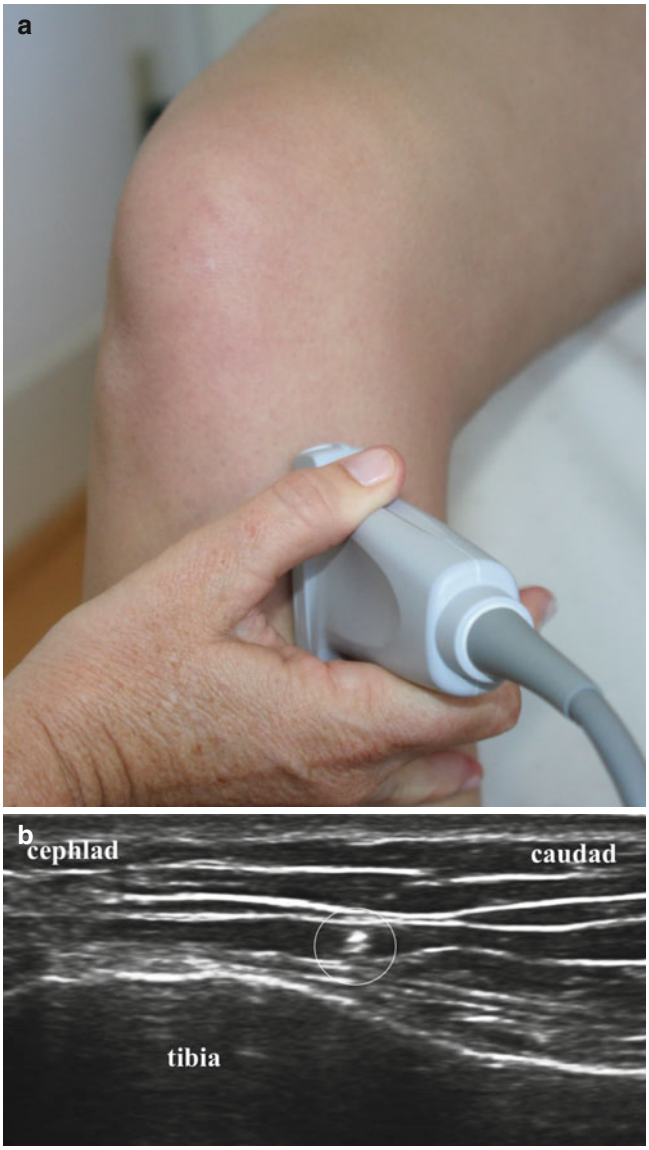

Fig. 58.21 Ultrasound identification of the infrapatellar saphenous nerve. (**a**) Ultrasound probe location for infrapatellar nerve identification; (**b**) ultrasound image of the infrapatellar saphenous nerve; nerve is *circled* (Image courtesy of Andrea Trescot, MD)

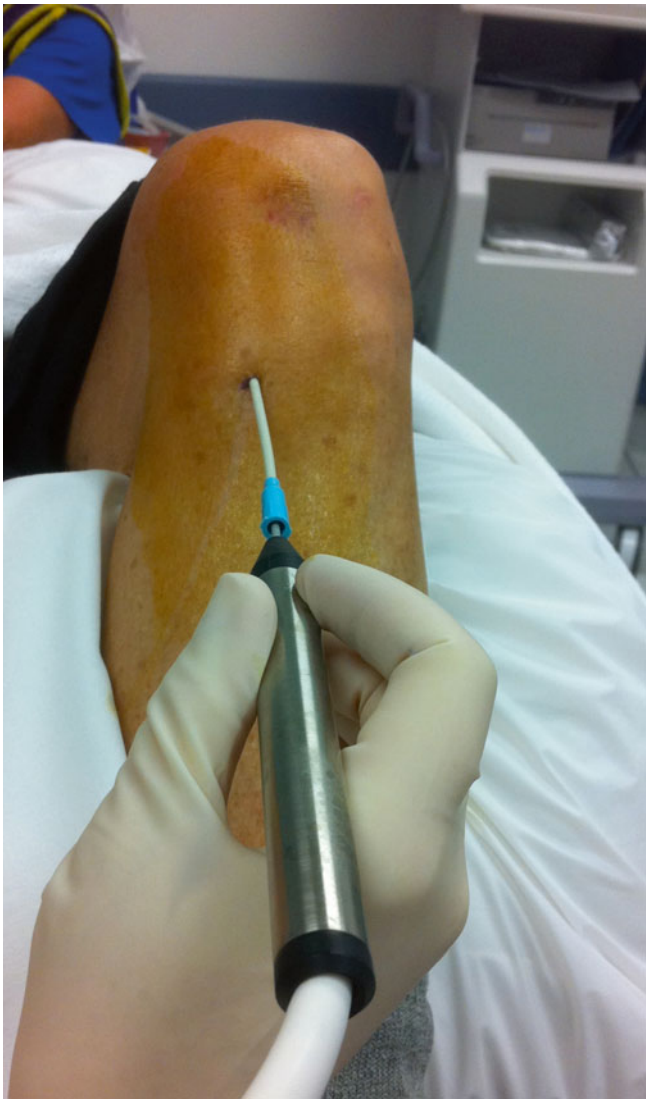

Fig. 58.22 Cryoneuroablation of the infrapatellar saphenous nerve under landmark guidance (Image courtesy of Andrea Trescot, MD)

have been less successful (50 %) [7]. Neuromas or mass lesions are most likely to respond to surgical excision [8] or cryoneuroablation.

Complications

The most likely complication of injections is skin atrophy due to the steroid injected. This is avoided by depositing the injectate as deep in the tissues as possible. Large volume blocks of the proximal saphenous nerve may lead to quadriceps weakness, putting patients at risk for falls. Dysesthesias and neuropathic pain can occur with incomplete radiofrequency or cryoneuroablation treatment of any nerve. A repeat procedure may be needed if such a complication is encountered.

Summary

The saphenous nerve has a very long course and multiple sites of entrapment. Knowing the proximal saphenous nerve entrapment syndromes can aid the treatment of patients with thigh, knee, and lower leg pain.

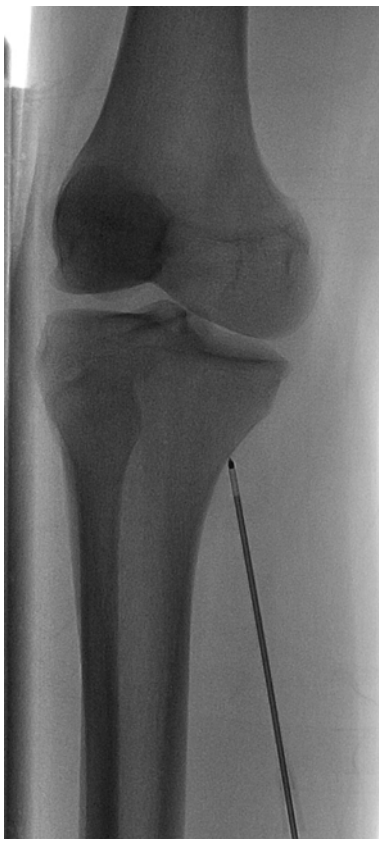

Fig. 58.23 Cryoneuroablation of the infrapatellar saphenous nerve under fluoroscopic guidance (Image courtesy of Andrea Trescot, MD)

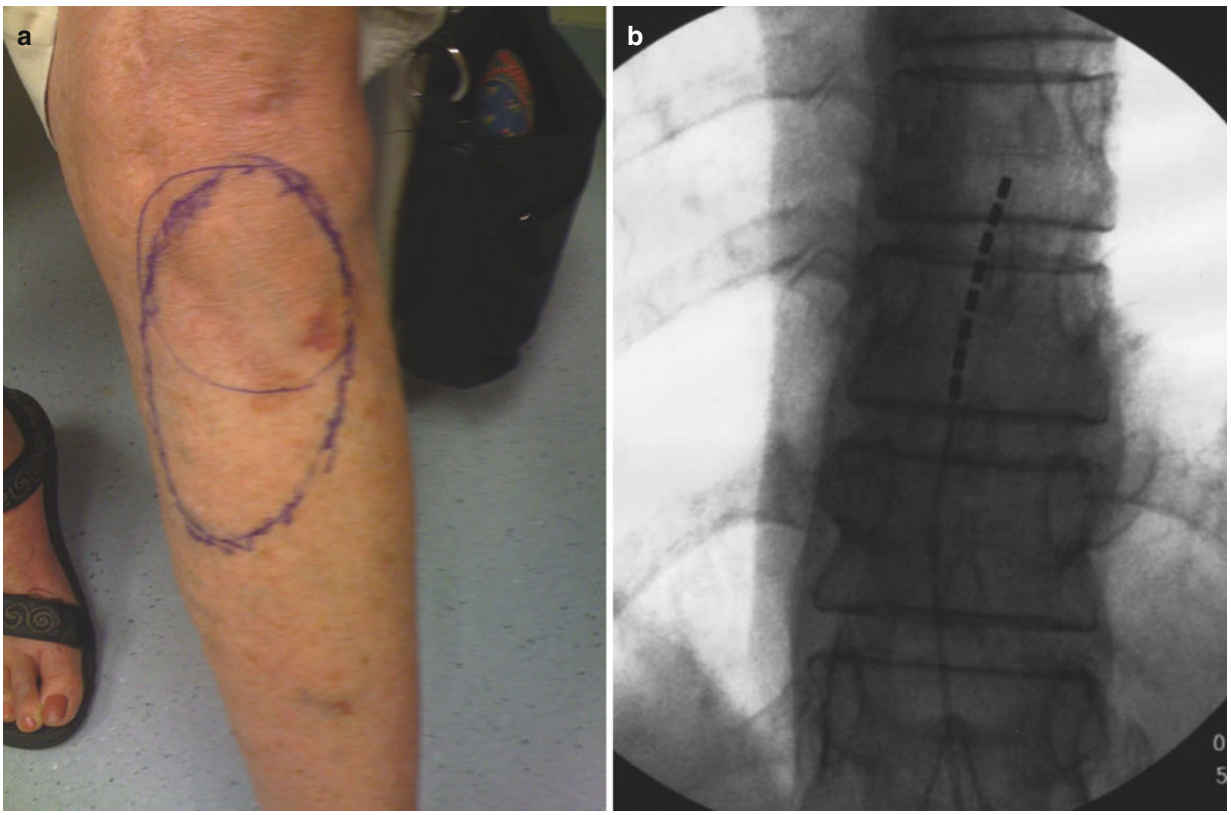

Fig. 58.24 Spinal cord stimulation for knee pain, most likely from infrapatellar saphenous pathology. (**a**) Pattern of pain; (**b**) fluoroscopic image of spinal cord stimulator (From Lowry and Simopoulos [53]. Reprinted with permission from American Society of Interventional Pain Physicians)

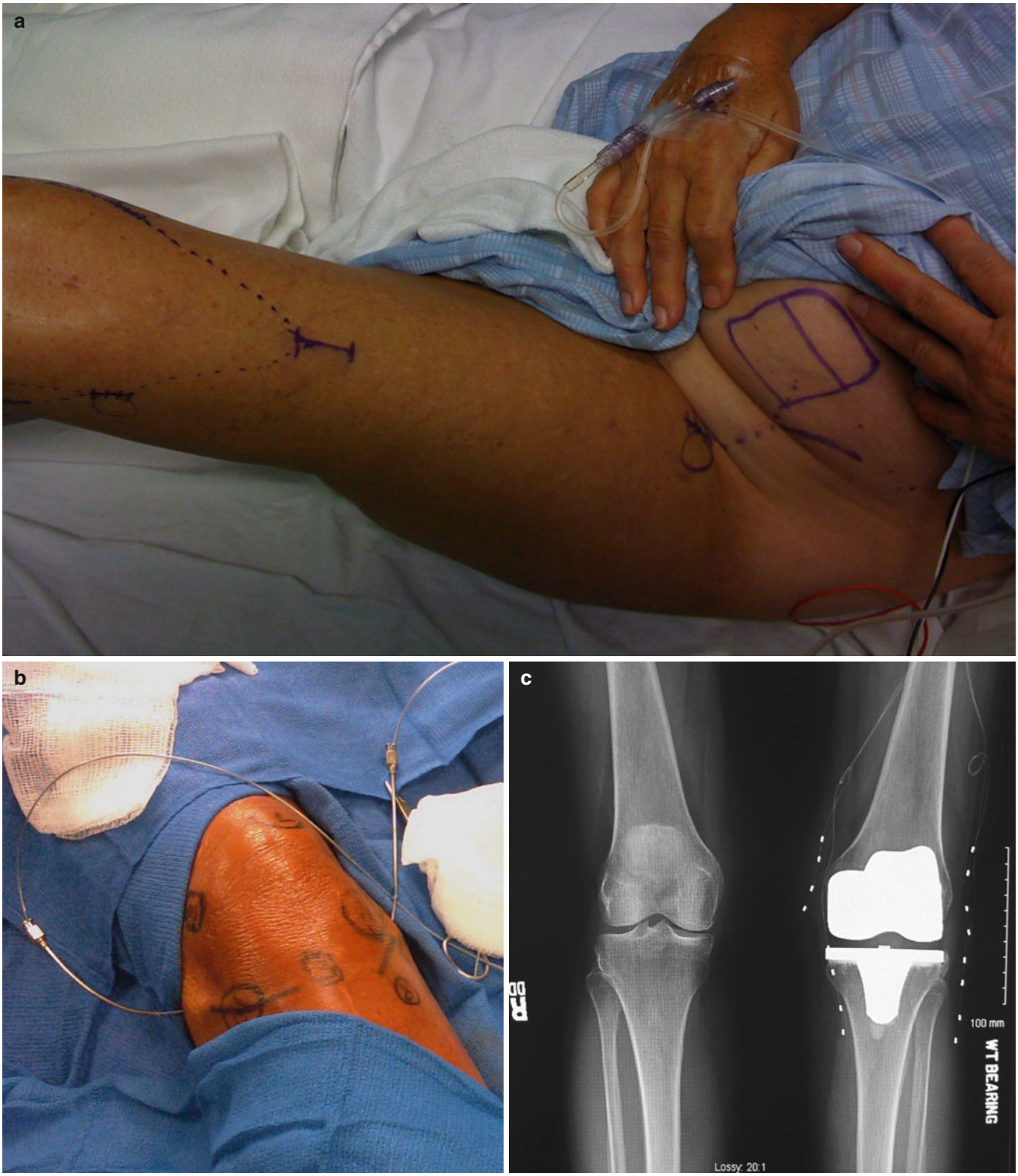

Fig. 58.25 Images of peripheral nerve field stimulation for knee pain after total knee replacement. (**a**) Preoperative planning; (**b**) trial technique of knee peripheral nerve stimulation; (**c**) postoperative AP knee X-rays after peripheral nerve stimulation (Images courtesy of Dr. W. Porter McRoberts)

References

1. Kurtz S, Ong K, Lau E, Mowat F, Halpern M. Projections of primary and revision hip and knee arthroplasty in the United States from 2005 to 2030. J Bone Joint Surg. 2007;89:780–5.
2. Lavee J, Schneiderman J, Yorav S, Shewach-Millet M, Adar R. Complications of saphenous vein harvesting following coronary artery bypass surgery. J Cardiovasc Surg (Torino). 1989;30(6): 989–91.
3. Adar R, Meyer E, Zweig A. Saphenous neuralgia: a complication of vascular reconstructions below the inguinal ligament. Ann Surg. 1979;190(5):609–13.
4. Morrison C, Dalsing MC. Signs and symptoms of saphenous nerve injury after greater saphenous vein stripping: prevalence, severity, and relevance for modern practice. J Vasc Surg. 2003;38(5): 886–90.
5. Busis NA. Femoral and obturator neuropathies. Neurol Clin. 1999;17(3):633–53.
6. Fabian RH, Norcross KA, Hancock MB. Surfer's neuropathy. N Engl J Med. 1987;316(9):555.
7. Luerssen TG, Campbell RL, Defalque RJ, Worth RM. Spontaneous saphenous neuralgia. Neurosurgery. 1983;13(3):238–41.
8. Dellon AL, Mont MA, Krackow KA, Hungerford DS. Partial denervation for persistent neuroma pain after total knee arthroplasty. Clin Orthop Relat Res. 1995;316:145–50.
9. Toms AD, Mandalia V, Haigh R, Hopwood B. The management of patients with painful total knee replacement. J Bone Joint Surg Br Vol. 2009;91-B:143–50.
10. Beaufils P, Gaudot F, Drain O, Boisrenoult P, Pujol N. Mini-invasive technique for bone patellar tendon bone harvesting: its superiority in reducing anterior knee pain following ACL reconstruction. Curr Rev Musculoskelet Med. 2011;4(2):45–51.
11. Papastergiou SG, Voulgaropoulos H, Mikalef P, Ziogas E, Pappis G, Giannakopoulos I. Injuries to the infrapatellar branch(es) of the saphenous nerve in anterior cruciate ligament reconstruction with four-strand hamstring tendon autograft: Vertical versus horizontal incision for harvest. Knee Surg Sports Traumatol Arthrosc. 2006;14(8):789–93.
12. Bertram C, Porsch M, Hackenbroch MH, Terhaag D. Saphenous neuralgia after arthroscopically assisted anterior cruciate ligament reconstruction with a semitendinosus and gracilis tendon graft. Arthrosc: J Arthrosc Relat Surg. 2000;16:763–6.
13. Sherman OH, Fox JM, Snyder SJ, Del Pizzo W, Friedman MJ, Ferkel RD, et al. Arthroscopy – "no-problem surgery". An analysis of complications in two thousand six hundred and forty cases. J Bone Joint Surg Am. 1986;68(2):256–65.
14. Detenbeck LC. Infrapatellar traumatic neuroma resulting from dashboard injury. J Bone Joint Surg. 1972;54:170–2.
15. Lippitt AB. Neuropathy of the saphenous nerve as a cause of knee pain. Bull Hosp Joint Dis. 1993;52(2):31–3.
16. Iizuka M, Yao R, Wainapel S. Saphenous nerve injury following medial knee joint injection: a case report. Arch Phys Med Rehabil. 2005;86(10):2062–5.
17. House JH, Ahmed K. Entrapment neuropathy of the infrapatellar branch of the saphenous nerve. Am J Sports Med. 1977;5(5): 217–24.
18. Kopell HP, Thompson WAL. Knee pain due to saphenous nerve entrapment. N Engl J Med. 1960;263(7):351–3.
19. Mozes M, Ouaknine G, Nathan H. Saphenous nerve entrapment simulating vascular disorder. Surgery. 1975;77(2):299–303.
20. Nir-Paz R, Luder AS, Cozacov JC, Shahin R. Saphenous nerve entrapment in adolescence. Pediatrics. 1999;103(1):161–3.
21. Romanoff ME, Cory PC, Kalenak A, Keyser GC, Marshall WK. Saphenous nerve entrapment at the adductor canal. Am J Sports Med. 1989;17:478–81.
22. Worth RM, Kettelkamp DB, Defalque RJ, Duane KU. Saphenous nerve entrapment. A cause of medial knee pain. Am J Sports Med. 1984;12(1):80–1.
23. Trescot AM, Brown MN, Karl HW. Infrapatellar saphenous neuralgia – diagnosis and treatment. Pain Physician. 2013;16(3): E315–24.
24. Tubbs RS, Loukas M, Shoja MM, Apaydin N, Oakes WJ, Salter EG. Anatomy and potential clinical significance of the vastoadductor membrane. Surg Radiol Anat. 2007;29(7):569–73.
25. Tsai P, Karnwal A, Kakazu C, Tokhner V, Julka I. Efficacy of an ultrasound-guided subsartorial approach to saphenous nerve block: a case series. Can J Anesth/J Can d'anesthésie. 2010;57(7):683–8.
26. Horner G, Dellon AL. Innervation of the human knee joint and implications for surgery. Clin Orthop Relat Res. 1994;301:221–6.
27. Arthornthurasook A, Gaew-Im K. Study of the infrapatellar nerve. Am J Sports Med. 1988;16(1):57–9.
28. Arthornthurasook A, Gaew-Im K. The sartorial nerve: its relationship to the medial aspect of the knee. Am J Sports Med. 1990;18:41–2.
29. Ackmann T, Von During M, Teske W, Ackermann O, Muller P, Pellengahr C. Anatomy of the infrapatellar branch in relation to skin incisions and as the basis to treat neuropathic pain by percutaneous cryodenervation. Pain Physician. 2014;17(3):E339–48.
30. Spicer DDM, Blagg SE, Unwin AJ, Allum RL. Anterior knee symptoms after four-strand hamstring tendon anterior cruciate ligament reconstruction. Knee Surg Sports Traumatol Arthrosc. 2000;8(5):286–9.
31. Mochida H, Kikuchi S. Injury to infrapatellar branch of saphenous nerve in arthroscopic knee surgery. Clin Orthop Relat Res. 1995; 320:88–94.
32. Murayama K, Takeuchi T, Yuyama T. Entrapment of the saphenous nerve by branches of the femoral vessels. A report of two cases. J Bone Joint Surg Am. 1991;73(5):770–2.
33. Hemler DE, Ward WK, Karstetter KW, Bryant PM. Saphenous nerve entrapment caused by pes anserine bursitis mimicking stress fracture of the tibia. Arch Phys Med Rehabil. 1991;72(5):336–7.
34. Bianchi S, Martinoli C, Demondion X. Ultrasound of the nerves of the knee region: Technique of examination and normal US appearance. J Ultrasound. 2007;10(2):68–75.
35. Rayegani SM. Saphenous nerve conduction study techniques. Arch Phys Med Rehabil. 2007;88:1542. author reply 3.
36. Le Corroller T, Lagier A, Pirro N, Champsaur P. Anatomical study of the infrapatellar branch of the saphenous nerve using ultrasonography. Muscle Nerve. 2011;44:50–4.
37. Bademkiran F, Obay B, Aydogdu I, Ertekin C. Sensory conduction study of the infrapatellar branch of the saphenous nerve. Muscle Nerve. 2007;35(2):224–7.
38. Ellis RF, Hing WA. Neural mobilization: a systematic review of randomized controlled trials with an analysis of therapeutic efficacy. J Man Manip Ther. 2008;16(1):8–22.
39. Santos FM, Silva JT, Giardini AC, Rocha PA, Achermann AP, Alves AS, et al. Neural mobilization reverses behavioral and cellular changes that characterize neuropathic pain in rats. Mol Pain. 2012;8:57.
40. van der Wal M, Lang SA, Yip RW. Transsartorial approach for saphenous nerve block. Can J Anaesth. 1993;40(6):542–6.
41. Benzon HT, Sharma S, Calimaran A. Comparison of the different approaches to saphenous nerve block. Anesthesiology. 2005;102(3): 633–8.
42. De Mey JC, Deruyck LJ, Cammu G, De Baerdemaeker LE, Mortier EP. A paravenous approach for the saphenous nerve block. Reg Anesth Pain Med. 2001;26(6):504–6.
43. Chi J, Greensmith JE. Saphenous nerve block technique with neurostimulation. Reg Anesth Pain Med. 2007;32:548–9.
44. Akbas M, Luleci N, Dere K, Luleci E, Ozdemir U, Toman H. Efficacy of pulsed radiofrequency treatment on the saphenous

nerve in patients with chronic knee pain. J Back Musculoskelet Rehabil. 2011;24(2):77–82.
45. Horn JL, Pitsch T, Salinas F, Benninger B. Anatomic basis to the ultrasound-guided approach for saphenous nerve blockade. Reg Anesth Pain Med. 2009;34:486–9.
46. Kent ML, Hackworth RJ, Riffenburgh RH, Kaesberg JL, Asseff DC, Lujan E, et al. A comparison of ultrasound-guided and landmark-based approaches to saphenous nerve blockade: a prospective, controlled, blinded, crossover trial. Anesth Analg. 2013;117(1):265–70.
47. Krombach J, Gray AT. Sonography for saphenous nerve block near the adductor canal. Reg Anesth Pain Med. 2007;32:369–70.
48. Kirkpatrick JD, Sites BD, Antonakakis JG. Preliminary experience with a new approach to performing an ultrasound-guided saphenous nerve block in the mid to proximal femur. Reg Anesth Pain Med. 2010;35:222–3.
49. Lundblad M, Kapral S, Marhofer P, Lonnqvist PA. Ultrasound-guided infrapatellar nerve block in human volunteers: description of a novel technique. Br J Anaesth. 2006;97(5):710–4.
50. Hsu LP, Oh S, Nuber GW, Doty Jr R, Kendall MC, Gryzlo S, et al. Nerve block of the infrapatellar branch of the saphenous nerve in knee arthroscopy: a prospective, double-blinded, randomized, placebo-controlled trial. J Bone Joint Surg Am. 2013;95(16):1465–72.
51. Clendenen S, Greengrass R, Whalen J, O'Connor MI. Infrapatellar saphenous neuralgia after TKA can be improved with ultrasound-guided local treatments. Clin Orthop Relat Res. 2015;473(1):119–25.
52. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
53. Lowry AM, Simopoulos TT. Spinal cord stimulation for the treatment of chronic knee pain following total knee replacement. Pain Physician. 2010;13:251–56.

Distal Saphenous Nerve Entrapment

59

Michael N. Brown, Beth S. Pearce, Helen W. Karl,
and Andrea M. Trescot

Introduction

The *saphenous nerve* (SN) at the ankle is called the *superficial saphenous nerve* (SSN) or *distal saphenous nerve*. Its entrapment is the least common of the entrapment syndromes in the foot and ankle [1].

Clinical Presentation (Table 59.1)

Patients with SSN neuralgia at the ankle present with classic neuropathic pain symptoms in the distribution of the nerve. They have burning, tingling, and pain sensations along the anteromedial calf and ankle, usually proximal to the metatarsophalangeal (MTP) joints (Fig. 59.1) [6]. There can be skin hypoesthesia from the medial knee to the foot along the SN pattern of distribution, as well as tenderness with manipulation of the nerve distally along the anteromedial lower third of the leg (Fig. 59.2). SSN pathology affects the medial ankle and heel, and it may present first to the podiatrist with complaints of "foot pain" (Fig. 59.3).

Table 59.1 Occupation/exercise/trauma history relevant to distal saphenous nerve entrapment/injury

Ankle arthroscopy [2-4] 5 SN injuries in 612 ankle arthroscopies (0.8%); less common than superficial peroneal (2.4%) (Chapter 68) or sural (1%) (Chapter 71) nerve injuries during arthroscopy [2] Varicose vein stripping [5] Particularly when extended below the knee CABG [6,7] May be injured during saphenous vein harvest Direct non-surgical trauma	Saphenous vein cannulation, displaced ankle fracture, ankle joint dislocation
Compression	Tight shoes, lower extremity casts

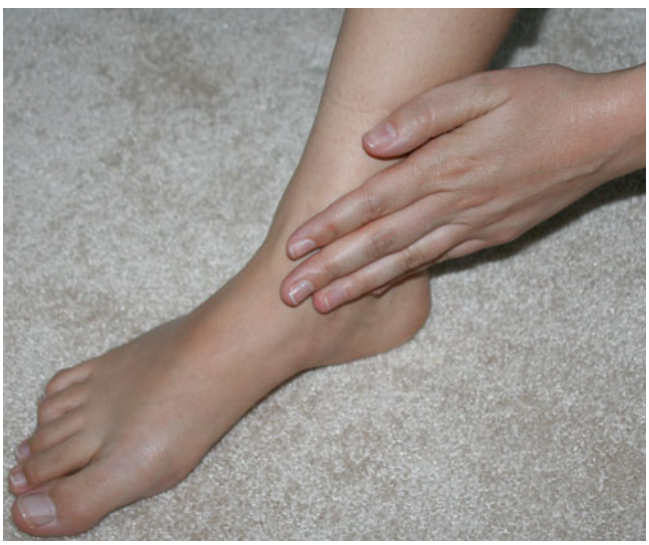

Fig. 59.1 Patient's pain complaint from distal saphenous nerve entrapment (Image courtesy of Andrea Trescot, MD)

Injury, including surgery, can damage the SSN. Eleven percent of nerve injuries after ankle arthroscopy (1/9 nerve injuries in 260 arthroscopies) were found to be of the SSN [5]. The SSN is also frequently injured during *saphenous vein stripping* (10 % of 280 patients in one study showed evidence of SN injury after vein stripping) [5]. In another

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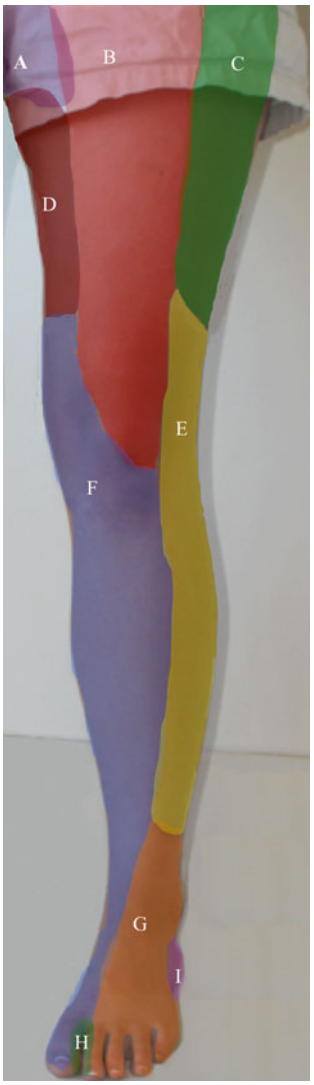

Fig. 59.2 Pattern of pain from anterior lower extremity nerve entrapments. *A* genitofemoral nerve, *B* femoral nerve, *C* lateral femoral cutaneous nerve, *D* obturator, *E* lateral sural cutaneous nerve, *F* saphenous nerve, *G* superficial peroneal nerve, *H* deep peroneal nerve, *I* sural nerve (Image courtesy of Andrea Trescot, MD)

study, 15 % of the 526 patients who underwent saphenous vein graft harvest for coronary artery bypass graft (CABG) had significant postoperative neuropathic pain from injury to the distal SN [7].

It appears that the greatest risk of SSN injury after vein stripping occurs distally. In a prospective study comparing saphenous vein stripping down only to the knee versus stripping continued down to the ankle; at 1 month it was noted that 5.7 % of the patients with stripping to the knee had neurologic symptoms, compared to 15 % of the patients with surgery to the ankle, and only 1.5 % versus 7 % at 1 year follow-up [5].

When this nerve has been injured, it may also develop cutaneous allodynia and a complex *regional pain syndrome* (CRPS) picture [8]. Patients who present with CRPS symptoms in the foot should be carefully questioned as to how and where the pain started and where the pain is most intense. If the pain started in the distribution of the SN, diagnostic blockade and treatment of this nerve may be of benefit.

Anatomy (Table 59.2)

The SN is the longest, as well as the terminal, branch of the *femoral nerve* (Chaps. 57 and 58). The fibers of the SN originate at L3 and L4 (Fig. 59.4), and the SN has several cutaneous branches, including the *infrapatellar saphenous* (IPS) nerve and the *superficial saphenous nerve* (SSN) (Fig. 59.5). The SN also has multiple potential entrapment sites along its long path from the back to the ankle; the specific entrapments of the SSN are discussed in the "Entrapments" section. The SN becomes subcutaneous just distal to the knee (Fig. 59.6) and proceeds to the ankle beside the *greater saphenous vein.* It gives off *crural cutaneous branches* to the medial and anterior distal leg, then often branches just above the medial malleolus to provide sensation to the dorsal medial aspect of the foot, rarely extending as far down as the first metatarsophalangeal joint. The course of the distal SN is highly variable [3, 10, 11], and there has been some question as to whether or not it should be included in an ankle block before forefoot surgery [11, 12, 13]. Others think that even though the distribution of the SN in the foot is less extensive than previously described, its fibers can reach into the first metatarsal and therefore should be included in routine surgical ankle blocks [3].

Entrapment

The saphenous nerve has multiple sites of entrapment (Fig. 59.7), including the distal medial shin (due to surgical vein stripping trauma) and the medial malleolus (where the SSN is tethered by the thin skin of the medial ankle and likely to be stretched with ankle eversion or extension). Footwear, tight laces, elastic bandages, socks, straps, and other sources of repeated microtrauma can affect distal SN function. SSN injury near the ankle may be an uncommon complication of displaced ankle fracture, ankle joint dislocation, sprained ankles (eversion), ankle arthroscopy [6], or tarsal tunnel surgery [14]. SN abnormalities can also be related to structural and functional anomalies of the leg or foot, ganglion cyst, varicosities in the leg, bone and joint abnormalities, tumors, tenosynovitis, or hypertrophic muscles. For this reason, ultrasonography (US) or magnetic resonance imaging (MRI) can often be a very helpful tool in identifying the potential source of SN compression [15, 16, 17].

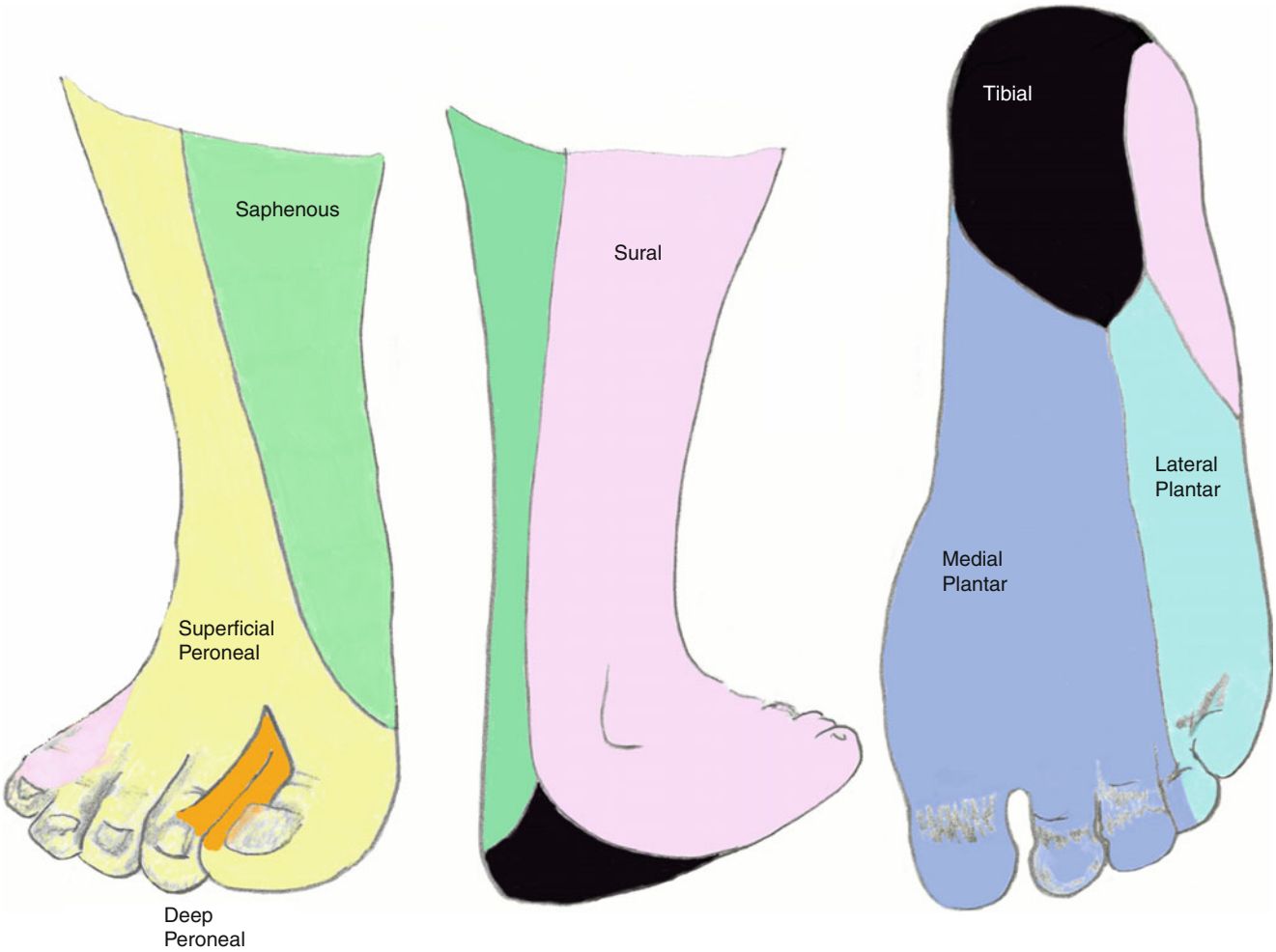

Fig. 59.3 Distribution of the nerves of the foot (Image courtesy of Michael Brown, MD)

Table 59.2 Superficial saphenous nerve anatomy

Origin	L3 and L4 nerve roots
General route	For nerve course proximal to the tibia, see Proximal Saphenous (Chap. 58)
	Accompanies the saphenous vein; very superficial in the leg
	Variable branches near the ankle
	Variable termination, often proximal to the foot
Sensory distribution	Cutaneous branches to the skin of the medial leg and often foot
Motor innervation	None
Anatomic variability	Presence at the medial malleolus 15/20 (75 %) [9] to 24/29 (83 %) [3]
	Extension into the foot: 19/29 (66 %) [3]
	Number of branches at the ankle: 1–3 [3]
	Position in relation to the medial malleolus: 21/24 over anterior third [3]
	Extent of the branches on the foot: 0 reached the toe [3, 10, 11] and very few (3 %) reached the first tarsometatarsal joint [11]
Other relevant structures	Saphenous vein

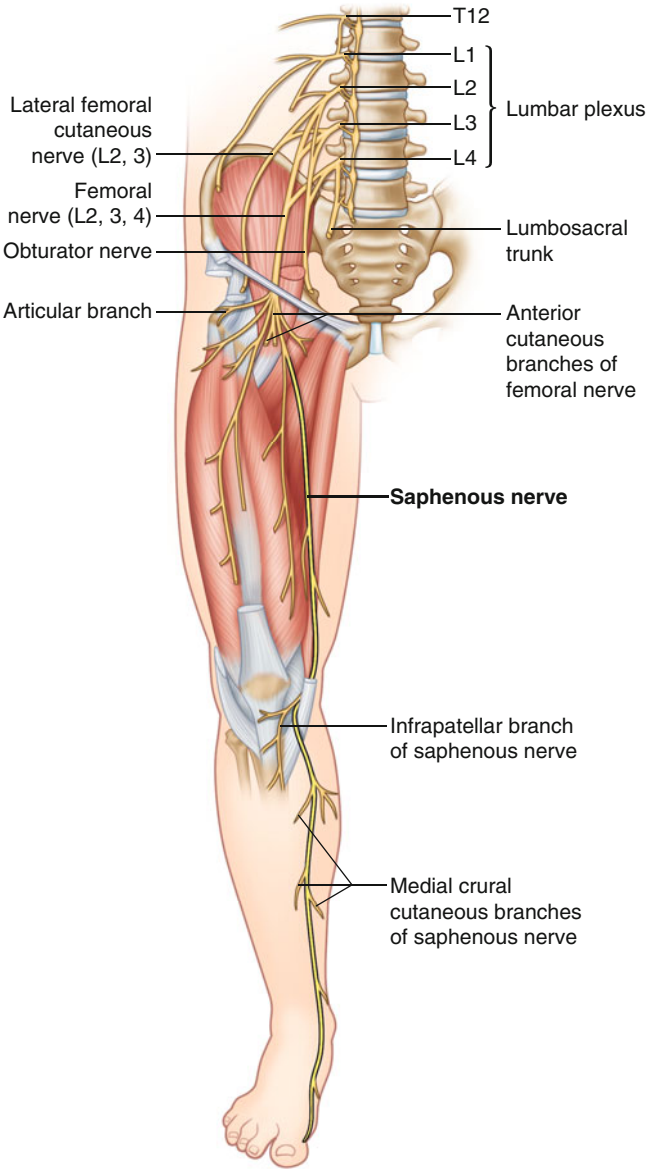

Fig. 59.4 Saphenous nerve anatomy (Image by Springer)

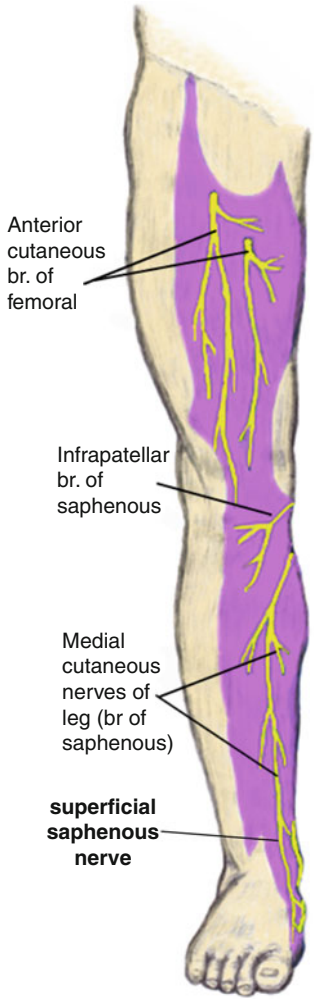

Fig. 59.5 Cutaneous saphenous nerve branches (Image courtesy of Michael Brown, MD)

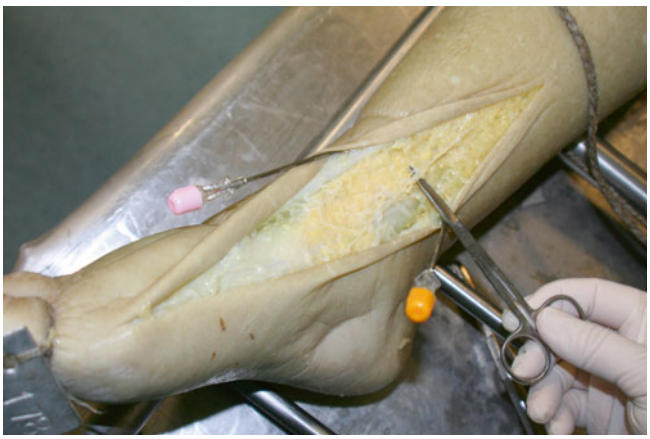

Fig 59.6 Dissection of the superficial saphenous nerve at the ankle (Image courtesy of Andrea Trescot, MD)

Physical Exam

Physical examination is considered the principal method to diagnose distal SN neuropathies. Tenderness to palpation at any area along the nerve is key to the diagnosis [6]. Local pressure may aggravate sensory symptoms (*Hoffman-Tinel's sign*) and thus indicate the site of the lesion. The patient suffering from SN entrapment neuropathy has normal motor function of the extremity. This is a key distinction between SN symptoms and lumbar radiculopathy: SN involvement creates only sensory alterations, while patients with lumbar radiculopathy may have associated motor, sensory, and deep tendon reflex alterations. However, patients with long-standing pain may have muscle wasting due to disuse atrophy [6].

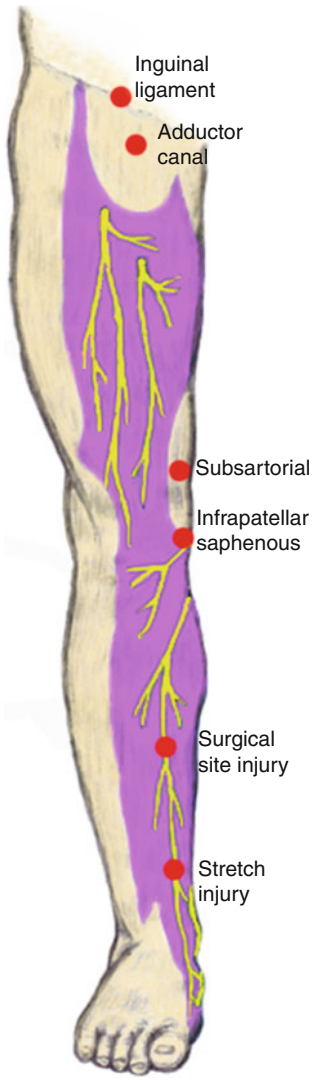

Fig. 59.7 Sites of saphenous nerve entrapment (Image by Michael Brown, MD)

The most common clinical entrapment is at the level of the extensor retinaculum at the medial ankle. The patient reports exquisite tenderness with tangential palpation across the nerve (Video 59.1) (Fig. 59.8), which may be palpable as a string-like structure just above the retinaculum.

Differential Diagnosis (Table 59.3)

The most likely common misdiagnosis of medial leg and ankle pain is radiculopathy; unlike radiculopathy, SN entrapment should not have muscle weakness. A patient with *claudication* from vascular compromise will have abnormal pulses, and an MRI of a patient with shin splints will show periosteal changes.

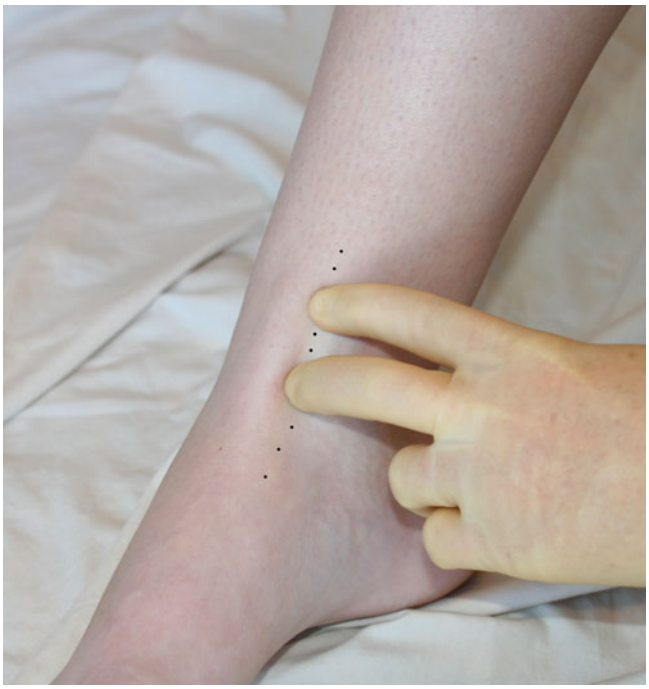

Fig. 59.8 Physical exam of the distal saphenous nerve at the ankle (Image courtesy of Andrea Trescot, MD)

Table 59.3 Differential diagnosis of medial leg and ankle pain

	Potential distinguishing features
Radiculopathy	Abnormal motor responses
Vascular compromise/PAD	Abnormal pulses
Medial tibial stress syndrome (shin splints)	MRI will show early changes [18]

Table 59.4 Diagnostic tests for distal saphenous entrapment

	Potential distinguishing features
Physical exam	Tenderness along the course of the saphenous nerve
Diagnostic injection	Use very low volume so as to not block one of the other four nerves at the ankle
Ultrasound	May be useful to rule out nerve impingement by a soft tissue mass
MRI	May be useful to rule out nerve impingement by a soft tissue mass [15, 16, 17]
Arteriography	Not useful
X-ray	To rule out a fracture or SN impingement by displaced bone [6]
Electrodiagnostic studies	Side to side comparisons are important; negative EMG

Diagnostic Tests (Table 59.4)

Because of the variable anatomy of the distal SN, US or MRI can often be very helpful in identifying the potential

source of compression [15, 16, 17]. Nerve conduction studies can be used to help diagnose distal SN neuropathy, but these studies can be problematic. Even in healthy volunteers, the response rate at the ankle (50 %) is substantially less than it is at the knee (77 %), and a useful response is less likely if the patient is obese [19]. Rayegani et al. suggested using a proximal technique first to find the nerve, but in women, the elderly, or those with high BMI, a distal technique is preferred [20].

Identification and Treatment of Contributing Factors

Tight braces or casts, tight high-top shoes, and trauma (such as fractures or surgery) can contribute to distal saphenous entrapment. Adjustment of tight hose or other footwear will decrease local irritation of the nerve. Diabetics or those patients with peripheral vascular disease (because of compromise of the *vasa vasorum*) are at particular risk.

As with all entrapments, optimizing the patient's general health, including reduction of chronic edema or obesity and smoking cessation, should benefit their symptoms.

Injection Technique

Since the sensory area of the SN in the foot is variable, there is ongoing discussion as to whether or not it should be included in an ankle block before forefoot surgery. Some believe it is not necessary because of the infrequent (3 %) presence of SN innervation of the forefoot [11], while others feel that even though the distribution of the SN in the foot is less extensive than previously described, fibers may reach the first metatarsal and therefore should be included in preoperative ankle blocks [3].

Landmark-Guided Injection

Descriptions of landmark-guided injection of the distal SN rely on subcutaneous infiltration of local anesthetic near the medial malleolus [21]. The needle should be placed parallel to the nerve (Video 59.2) (Fig. 59.9), and a peripheral nerve stimulator may be useful. For diagnostic purposes, infiltration of 1 cc of local anesthetic at the point of maximum tenderness is used. This should result in relief of pain, with numbness in the small area of SN territory distal to the point of injection, and no motor block. Deposteroids in the injectate can give good, long-term relief, but care must be used to avoid skin atrophy from injections too close to the surface of the skin.

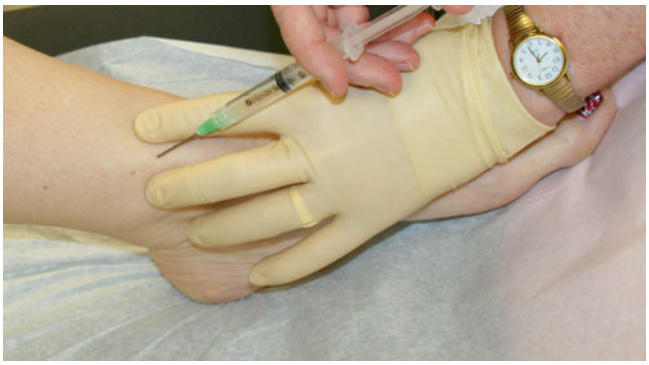

Fig. 59.9 Landmark-guided distal saphenous nerve injection (Image courtesy of Andrea Trescot, MD)

Fluoroscopy-Guided Injections

Fluoroscopy is not useful for this procedure.

Ultrasound-Guided Injections

The distal SN may be hard to visualize with ultrasound, due to its small size. Holding the probe perpendicular to the nerve, start at the adductor canal and try to trace the nerve distally. Injections with US rely on the nerve's location adjacent to the *saphenous vein*. A specific injection at this level is challenging because of the nerve's small size and variable position, as well as the presence of bony prominences in the area (which make it difficult to achieve good probe contact). Using a small "*hockey stick*" probe above

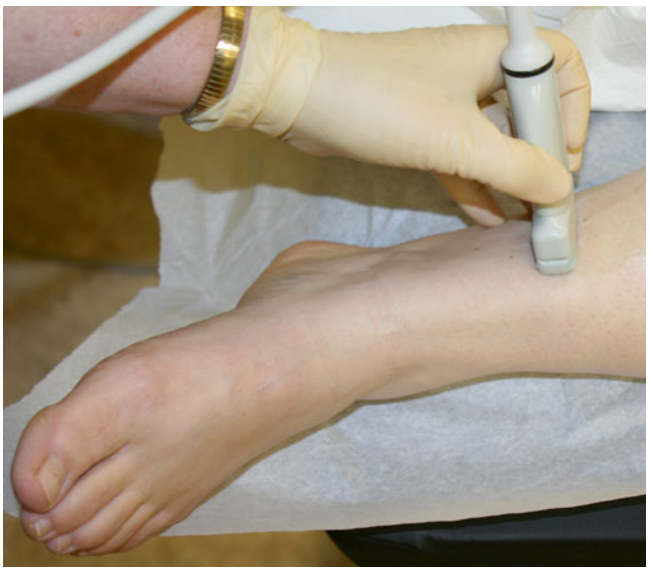

Fig. 59.10 Ultrasound probe on the distal saphenous nerve at the distal tibia (Image courtesy of Andrea Trescot, MD)

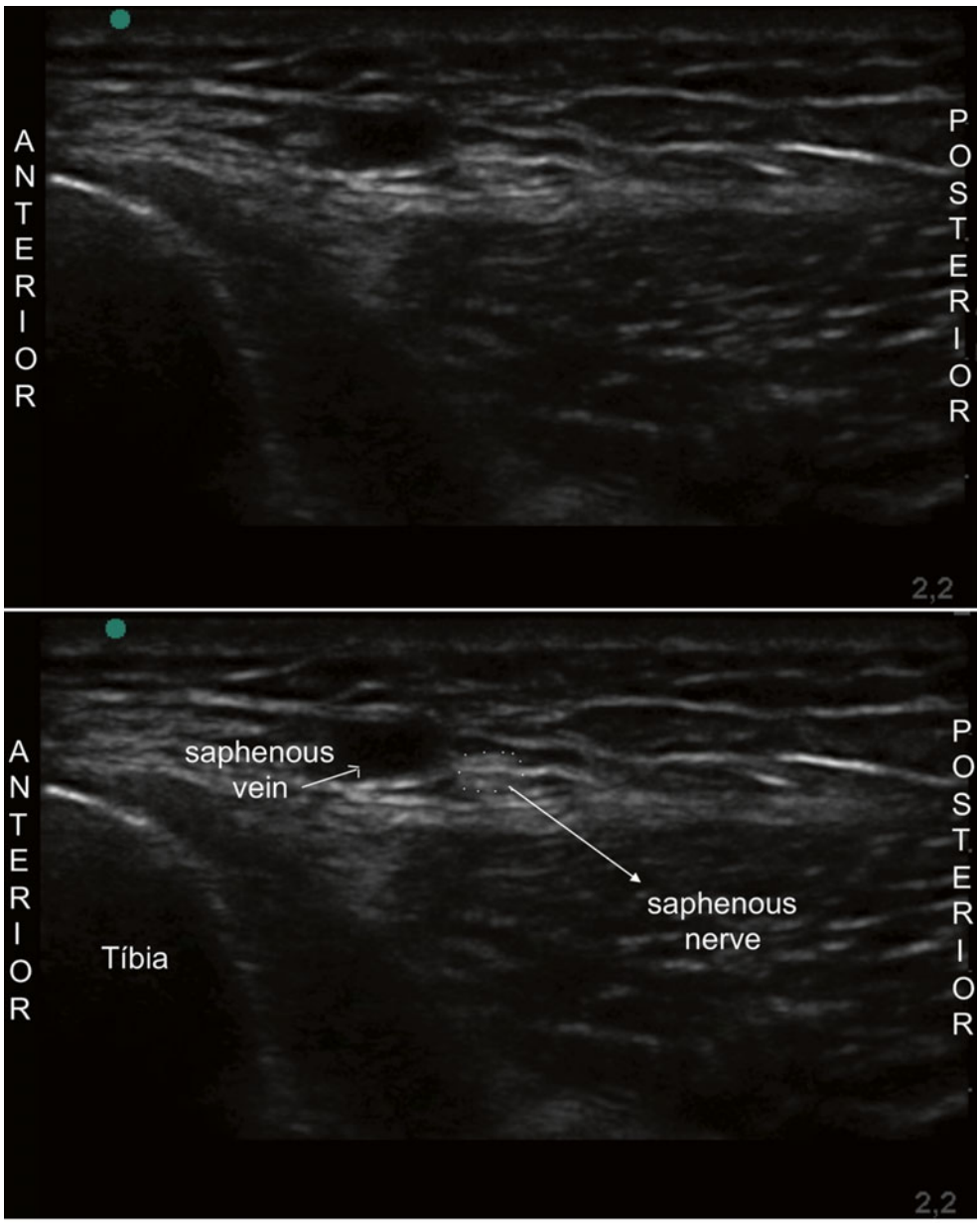

Fig. 59.11 Ultrasound image of the distal saphenous nerve at the tibia, unlabeled and labeled (Image courtesy of Thiago Nouer Frederico, MD)

the malleolus will improve the chances of success [22, 23], but a standard linear probe can be used if the region of contact is over the nerve. The patient is placed supine with the leg externally rotated. The nerve can be palpated, and the probe is placed perpendicular to the nerve over the distal tibia (Fig. 59.10) and is moved in order to identify the *greater saphenous vein* (Fig. 59.11). Note that the vein begins to move more medially with the saphenous nerve next to the vessel as the probe is traced distally (Fig. 59.12). A tourniquet placed proximally can distend the vein, making identification easier. After a sterile prep and drape, a 27-gauge 1.5-in., or 25-gauge 2-in., needle is then advanced in-plane (Fig. 59.13) or out-of-plane (Fig. 59.14), with or without a peripheral nerve stimulator. One cc of local anesthetic and deposteroid should provide at least temporary relief. It is important to avoid injections that are too superficial, so as to avoid skin atrophy.

A retrospective review by Chin et al. [21] looked at the difference between landmark-guided (LG) and US-guided ankle blocks (including superficial saphenous nerves) and found that the LG patients needed more opioids intraoperatively, indicative of an inadequate injection.

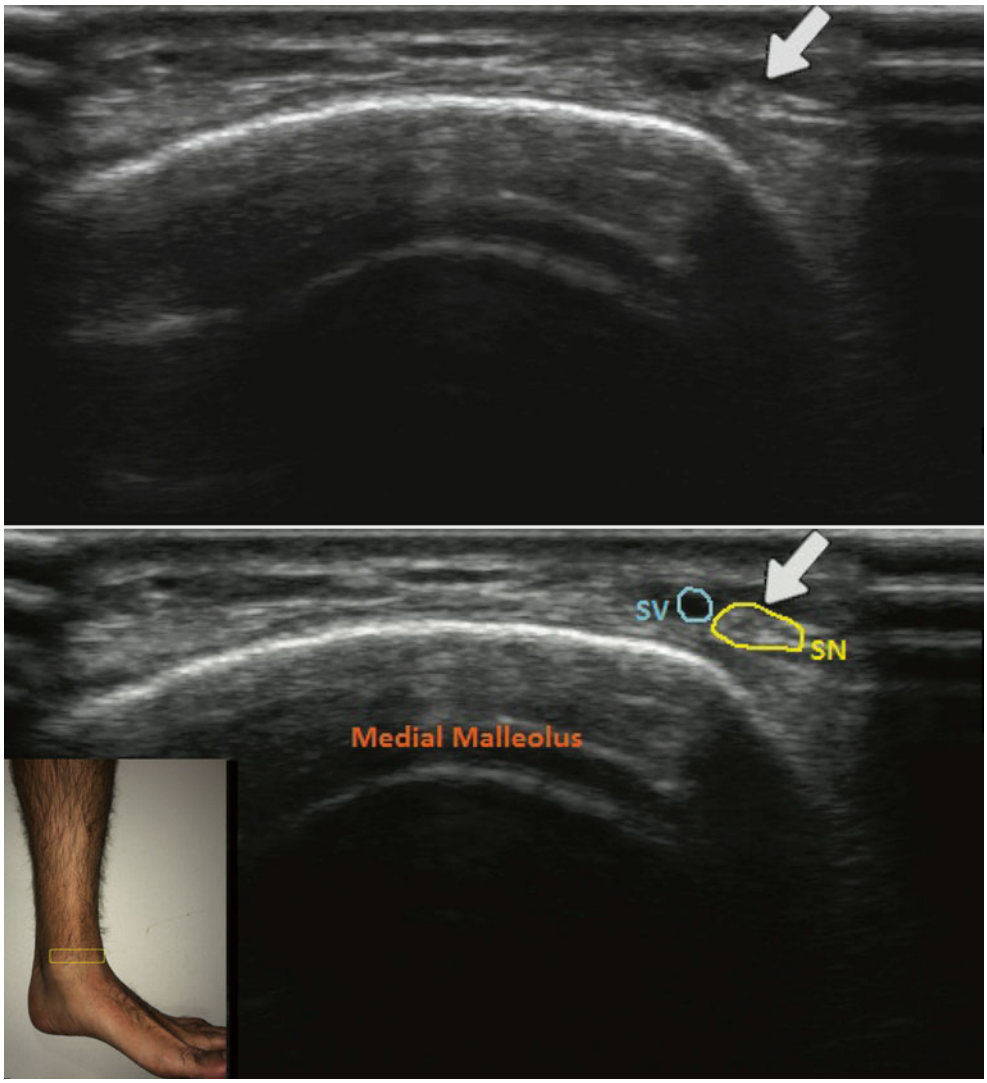

Fig. 59.12 Ultrasound image of the distal saphenous nerve at the malleolus, unlabeled and labeled. SV saphenous vein, SN saphenous nerve (Image courtesy of Agnes Stogicza, MD)

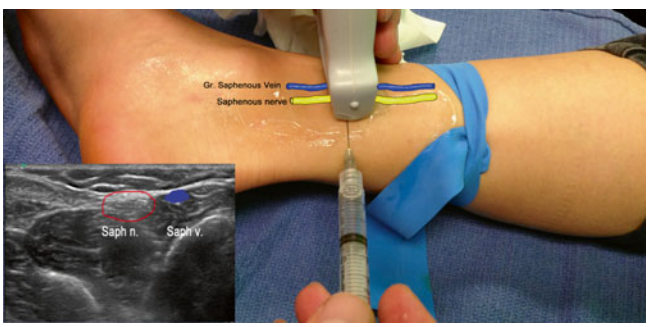

Fig. 59.13 In-plane ultrasound injection of the distal saphenous nerve. Note the tourniquet to increase the size of the saphenous vein for easier localization (Image courtesy of Michael Brown, MD)

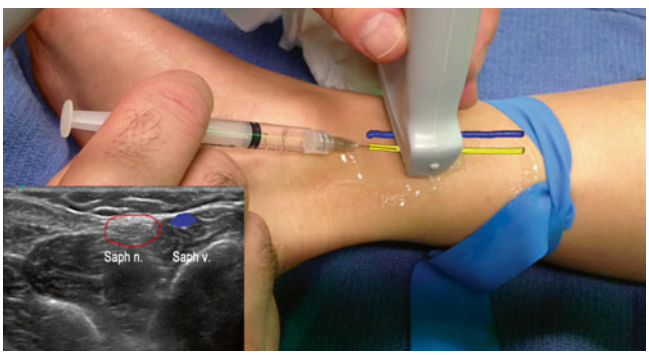

Fig. 59.14 Out-of-plane ultrasound injection of the distal saphenous nerve. Note the tourniquet to increase the size of the saphenous vein for easier localization (Image courtesy of Michael Brown, MD)

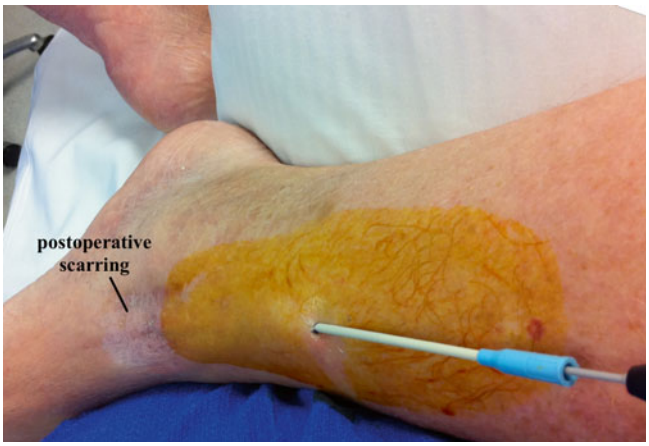

Fig. 59.15 Cryoneuroablation of the distal saphenous nerve (Image courtesy of Andrea Trescot, MD)

Neurolytic Technique

Cryoneuroablation

If a low volume diagnostic distal saphenous nerve injection gives excellent but only temporary relief, this is a nerve well suited for cryoneuroablation [24]. In fact, the saphenous nerve can be accessed for cryoneuroablation anywhere along its distribution. The probe can be placed percutaneously using landmarks and the built-in nerve stimulator, (Fig. 59.15) or under US guidance. If the nerve can be directly visualized under US, the cryoprobe can be easily positioned directly on to the nerve with or without the built-in nerve stimulator.

Surgery

Surgical options include decompression, neurectomy, and neurolysis [25]. Lower extremity nerve injuries (including the saphenous nerve) have also been treated by wrapping the nerve with fetal umbilical vein material [26].

Complications

The most likely complication of injections is skin atrophy due to the steroid injected. This is particularly likely with distal SN injections because of the superficial location of the nerve, and it can be avoided by depositing the injectate as deep in the tissues as possible.

Summary

Entrapment of the SN can cause burning pain and dysesthesias along the medial calf and foot, mimicking or triggering CRPS. Recognition of the distribution of pain can help in the diagnosis and treatment of distal saphenous neuralgia.

References

1. Schon LC, Baxter DE. Neuropathies of the foot and ankle in athletes. Clin Sports Med. 1990;9(2):489–509.
2. Ferkel RD, Heath DD, Guhl JF. Neurological complications of ankle arthroscopy. Arthroscopy. 1996;12(2):200–8.
3. Marsland D, Dray A, Little NJ, Solan MC. The saphenous nerve in foot and ankle surgery: its variable anatomy and relevance. Foot Ankle Surg Off J Eur Soc Foot Ankle Surg. 2013;19(2):76–9.
4. Deng DF, Hamilton GA, Lee M, Rush S, Ford LA, Patel S. Complications associated with foot and ankle arthroscopy. J Foot Ankle Surg. 2012;51(3):281–4.
5. Jia GL, Xi HL, Wang XK, Feng S, Tian ZL. Selective retention of the great saphenous vein to prevent saphenous nerve injury during varicose vein surgery. Eur Rev Med Pharmacol Sci. 2014;18(22):3459–63.
6. Flanigan RM, DiGiovanni BF. Peripheral nerve entrapments of the lower leg, ankle, and foot. Foot Ankle Clin. 2011;16(2):255–74.
7. Hakim SM, Narouze SN. Risk factors for chronic saphenous neuralgia following coronary artery bypass graft surgery utilizing saphenous vein grafts. Pain Pract. 2015;15(8):720–9.
8. Oaklander AL. Role of minimal distal nerve injury in complex regional pain syndrome-I. Pain Med. 2010;11(8):1251–6.
9. Williams RP, Sugars W. Lumbar foot innervation of the medial foot and ankle region. Aust N Z J Surg. 1998;68(8):565–7.
10. Saito A, Kikuchi S. Anatomic relations between ankle arthroscopic portal sites and the superficial peroneal and saphenous nerves. Foot Ankle Int. 1998;19(11):748–52.
11. López AM, Sala-Blanch X, Magaldi M, Poggio D, Asuncion J, Franco CD. Ultrasound-guided ankle block for forefoot surgery: the contribution of the saphenous nerve. Reg Anesth Pain Med. 2012;37(5):554–7.
12. Bugamelli S, Zangheri E, Montebugnoli M, Borghi B, Ricci A, De Simone N, et al. One-day surgery for acquired forefoot deformity: sciatic nerve blockade with mepivacaine vs mepivacaine + ropivacaine: a prospective, randomized study. Minerva Anestesiol. 2007;73(1–2):57–64.
13. Migues A, Slullitel G, Vescovo A, Droblas F, Carrasco M, PT H. A peripheral foot blockade versus popliteal fossa nerve block: a prospective randomized trial in 51 patients. J Foot Ankle Surg. 2005;44:354–7.
14. Kim J, Dellon AL. Pain at the site of tarsal tunnel incision due to neuroma of the posterior branch of the saphenous nerve. J Am Podiatr Med Assoc. 2001;91(3):109–13.
15. Donovan A, Rosenberg ZS. MRI of ankle and lateral hindfoot impingement syndromes. AJR Am J Roentgenol. 2010;195(3):595–604.

16. Donovan A, Rosenberg Z, Cavalcanti C. MR imaging of entrapment neuropathies of the lower extremity: part 2. The knee, leg, ankle, and foot. Radiographics. 2010;30:1001–19.
17. Martinoli C, Court-Payen M, Michaud J, Padua L, Altafini L, Marchetti A, et al. Imaging of neuropathies about the ankle and foot. Semin Musculoskelet Radiol. 2010;14(3):344–56.
18. Aoki Y, Yasuda K, Tohyama H, Ito H, Minami A. Magnetic resonance imaging in stress fractures and shin splints. Clin Orthop Relat Res. 2004;421:260–7.
19. Chiodo A, Spiegelberg T, Tong HC. Comparing saphenous nerve conduction study techniques at the knee and at the ankle and their relationship to body mass index. Arch Phys Med Rehabil. 2007;88(4):477–80.
20. Rayegani SM. Saphenous nerve conduction study techniques. Arch Phys Med Rehabil. 2007;88:1542; author reply 3.
21. Chin KJ, Wong NW, Macfarlane AJ, Chan VW. Ultrasound-guided versus anatomic landmark-guided ankle blocks: a 6-year retrospective review. Reg Anesth Pain Med. 2011;36(6):611–8.
22. Fredrickson MJ. Ultrasound-guided ankle block. Anaesth Intensive Care. 2009;37(1):143–4.
23. Purushothaman LA, Anthony GL, Bedforth N. Ultrasound-guided ankle block. Contin Educ Anaesth Crit Care Pain. 2013;13:174–8.
24. Trescot AM. Cryoneuroablation. In: Manchikanti L, Singh V, editors. Interventional techniques in chronic non-spinal pain. Paducah: ASIPP Publishing; 2009. p. 69–86.
25. Schon LC, Anderson CD, Easley ME, Lam PW, Trnka HJ, Lumsden DB, et al. Surgical treatment of chronic lower extremity neuropathic pain. Clin Orthop Relat Res. 2001;389:156–64.
26. Easley ME, Schon LC. Peripheral nerve vein wrapping for intractable lower extremity pain. Foot Ankle Int. 2000;21(6):492–500.

Genitofemoral Nerve Entrapment: Lower Extremity

60

Thais Khouri Vanetti, Alexandra Tavares Raffaini Luba, Fabrício Dias Assis, and Charles Amaral de Oliveira

Introduction

The *genitofemoral nerve* (GFN) is a branch of *lumbar plexus* and may become entrapped in the abdominal region, causing pain in the distribution of this nerve, i.e., lower abdomen/pelvic region, groin, scrotum or labia majora, and anterior proximal thigh area. The pelvic presentation is discussed in Chap. 45, and the abdominal presentation is discussed in Chap. 41. In this chapter, we will focus on the lower extremity aspect of GFN entrapment.

It is also very important to remember that the *genitofemoral*, *ilioinguinal*, and *iliohypogastric* (see Chaps. 40 and 44) nerves originate from similar levels of spinal nerve roots; therefore, it is often difficult to distinguish which of the three nerves is causing the pain. In addition, the *lateral femoral cutaneous nerve* (LFCN) (see Chap. 61) also has an overlapping sensory distribution, making diagnosis even more difficult. Diagnostic injections are a critical tool for differentiation. Benes et al. [1] have proposed using the term *abdomino-inguinal syndrome* to describe the collection of nerve pathologies in this region.

The femoral branch of the genitofemoral nerve, also known as the *lumboinguinal nerve*, enters the sheath of the femoral artery to provide medial thigh innervation [2]. Zempoalteca et al. [3] described the GFN as contributing to male reproductive performance by transmitting sensory information during sexual activity, via contraction of the *cremaster muscle*, to promote ejaculation, the protective displacement of the testes into the abdominal cavity during fighting, and as a sperm-protecting thermoregulatory measure.

Clinical Presentation (Table 60.1)

Magee first reported the syndrome of *genitofemoral neuralgia* in 1942, but it remains a rarely encountered (or diagnosed) clinical entity [4]. The most common clinical presentation of GFN entrapment consists of intermittent or constant pain, burning dysesthesia, and sensory changes in the inguinal region. Paresthesias and persistent pain in the lower abdomen and *groin* (Fig. 60.1), medial proximal thigh (Fig. 60.2), as well as *scrotum* or *labia majora*, and *anterior proximal thigh* (Fig. 60.3) area may indicate GFN neuropathy. Pain is aggravated with thigh flexion because of psoas compression of the nerve; activities such as walking and hyperextension of the thigh tend to exacerbate the pain, while lying down and thigh flexion will help to relieve it [10]. In contrast to ilioinguinal neuralgia, *Tinel's sign* cannot usually be obtained [11].

Mesh hernia repair is particularly associated with GFN entrapment at the pubic tubercle, since the mesh has to be secured on *Poupart's ligament*, at the pubic attachment of the inguinal ligament, which is the site of the GFN's transit across the pubic tubercle. The GFN is also traumatized by C-sections (at the pubic level), as well as lumbar fusions, retroperitoneal surgery (at the psoas level), and varicocelectomy [12].

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Table 60.1 Occupation/exercise/trauma history relevant to genitofemoral nerve entrapment

Compression	Psoas spasm
	Psoas abscess
	Pelvic hematoma
	Lymphoma
	Obesity (compression of the femoral branch at the inguinal ligament)
Trauma	Pubic ramus fracture
	Pubic symphysis irritation (pubic synovitis)
	Needle trauma during lumbar sympathetic injections [5]
Surgery	Inguinal hernia repair (especially with mesh) [6]
	Laparoscopic inguinal hernia repair [7]
	C-section/appendectomy [6]
	Lumbar fusion
	Retroperitoneal surgery
Neuritis	Alcohol/phenol/RF to the lumbar sympathetic chain [8, 9] or celiac plexus
	Thermal damage from renal radiofrequency lesioning (renal cell CA) [9]

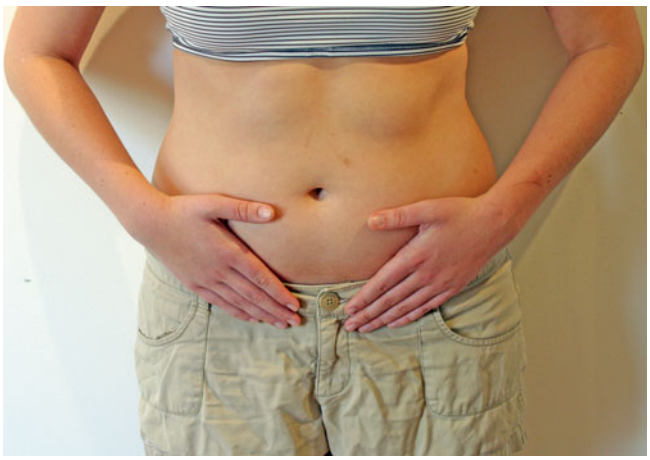

Fig. 60.1 Groin pattern of genitofemoral pain (Image courtesy of Andrea Trescot, MD)

Fig. 60.2 Medial thigh pattern of genitofemoral pain (Image courtesy of Andrea Trescot, MD)

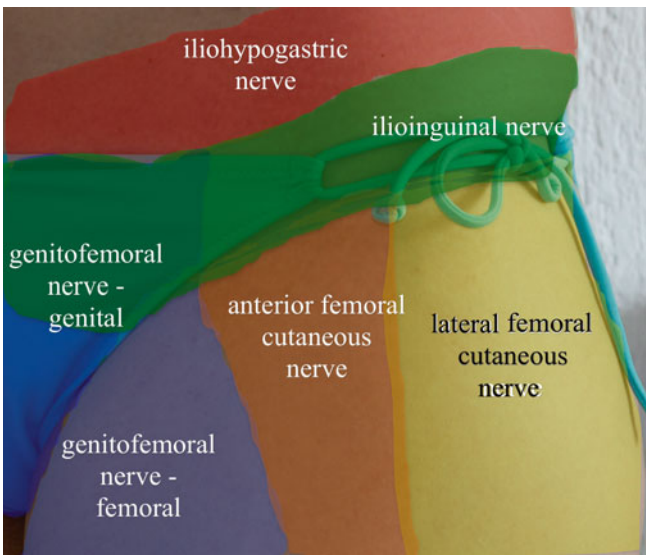

Fig. 60.3 Groin and anterior thigh pain patterns from iliohypogastric, ilioinguinal, genitofemoral, lateral femoral cutaneous, and anterior femoral cutaneous nerves (Image courtesy of Andrea Trescot, MD)

Anatomy (Table 60.2)

The GFN arises from the upper *lumbar plexus* (see Chap. 49), emerges at L1 and L2, and consists mainly of sensory fibers, with a motor component for the *cremaster muscle* (*cremasteric reflex*). The nerve penetrates the psoas muscle anteriorly at the L3 and L4 levels and descends to the medial border of the psoas muscle (Fig. 60.4), where it divides into a genital and femoral branch, just above the inguinal ligament (Fig. 60.5). In contrast, the ilioinguinal and iliohypogastric nerves are found most consistently on the surface of the quadratus lumborum muscle [13].

The medial *genital branch* of the GFN accompanies the psoas muscle (Fig. 60.6). In males, it passes inside the *internal inguinal ring* together with the *spermatic cord*, supplying motor fibers to the cremaster muscle and sensation to the scrotum. In females, it accompanies the *round ligament*, innervating the mons pubis and the labia majora. The *femoral branch* of the GFN is located caudally and laterally to the genital branch and travels alongside the external iliac artery, behind the inguinal ligament, passing through the fasciae latae to the femoral sheath to innervate the skin of the anterior medial superior part of the thigh [14, 15].

Though the course of this nerve and its branches is similar in men and women, anatomical studies suggest great

Table 60.2 Genitofemoral nerve anatomy

Origin	L1 and L2
General route	Perforates the psoas at L3 and L4, descends along the medial psoas border, divides into genital and femoral branches just above the inguinal ligament
	Genital branch
	Males: inside internal inguinal ring with spermatic cord to scrotum
	Females: accompanies the round ligament to the mons pubis and labia majora
	Femoral branch
	Located caudally and laterally to the genital branch, traveling caudally with external iliac artery, behind inguinal ligament through fasciae latae to femoral sheath
Sensory distribution	Anterior medial thigh, scrotum, and mons pubis or labia majora
Motor innervation	Cremaster muscle
Anatomic variability	Location where genital and femoral branches split; location of spermatic cord relative to genital branch; communication between ilioinguinal and GFN
Other relevant structures	Pubic tubercle

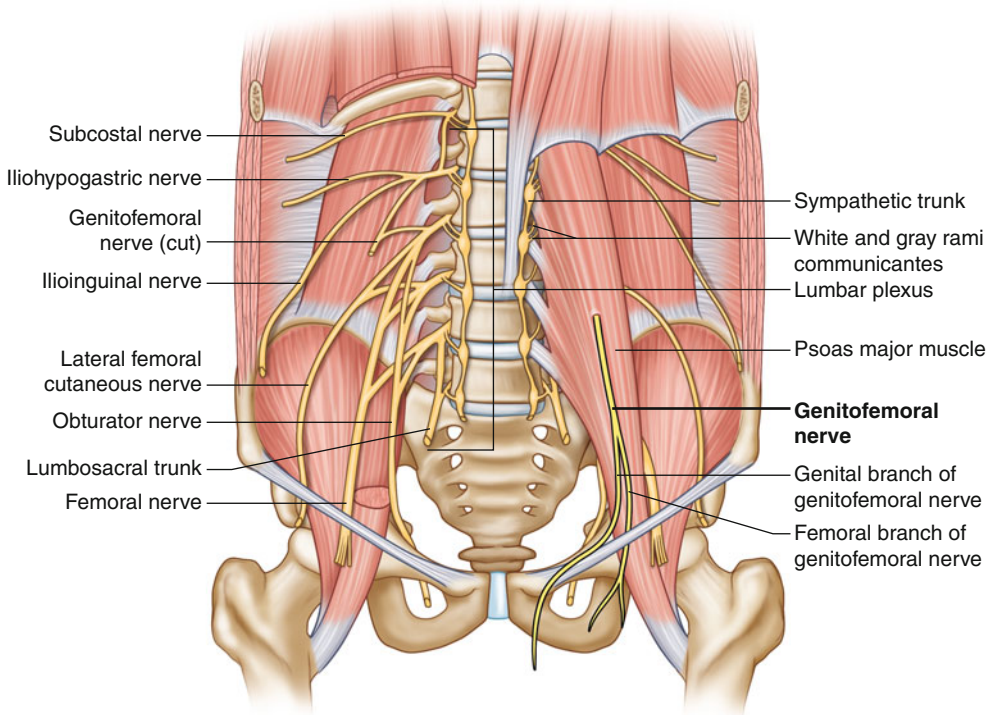

Fig. 60.4 Lumbar plexus (Image courtesy of Springer)

variability among individuals, with the minority of nerves following the course traditionally described [15]. Moreover, the location where genital and femoral branches split before reaching the inguinal ligament is quite variable. Also, the relation between the genital branch and the spermatic cord varies considerably; it can travel dorsally, ventrally, inferiorly, or even outside the spermatic cord [11].

Both the GFN and the lateral femoral cutaneous nerve (LFCN) (see Chap. 61) are at risk for injury during lumbar sympathetic injections and neurolysis. Feigi and colleagues [8] dissected the sympathetic trunk of 118 cadavers and evaluated the distance from the *lumbar sympathetic trunk* (LST) to the GFN (in 20 cases, they could only measure from the femoral branch). There were 186 dissections where the GFN passed the LST at a distance of 0–28 mm (mean distance 8.5) at the level of L3/4, 0–13 mm at L4/5 in 55 cases, and 9–19 mm at L2/3 in 19 cases. The authors suggested that, due to the close proximity of the two structures (especially at

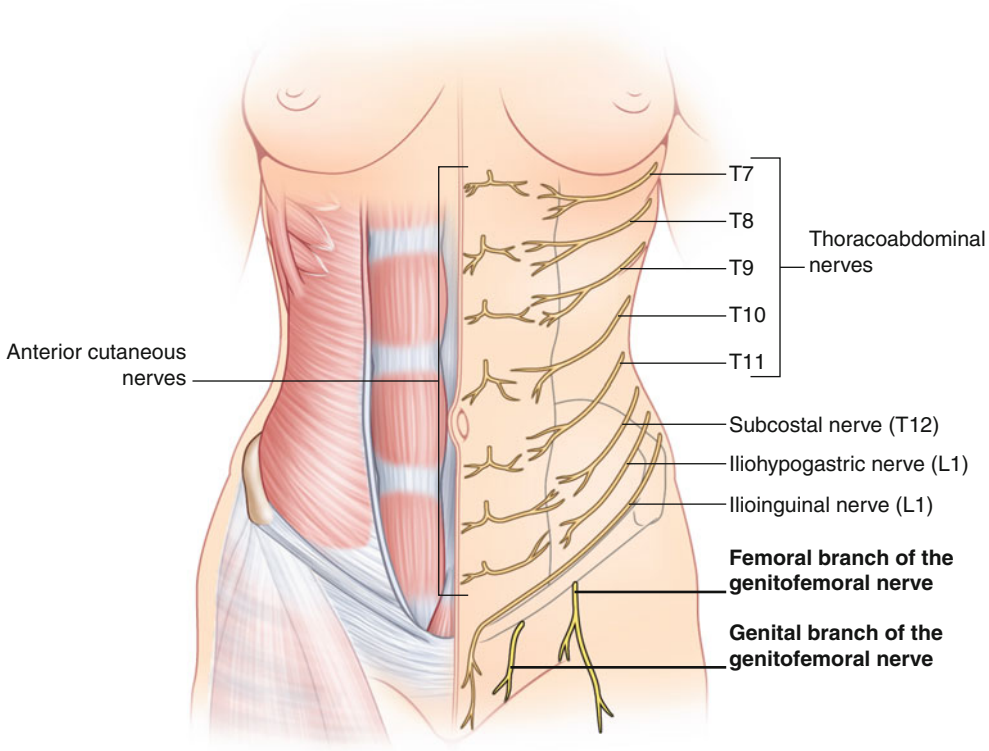

Fig. 60.5 Abdominal wall and groin nerves (Image courtesy of Springer)

L3/4 and L4/5), needle trauma or neurolytic damage could easily affect the GFN during lumbar sympathetic treatment. Interestingly, the authors found that the GFN was fused with the LFCN in three cases.

Special attention to the great variation of the nerves in the groin region (ilioinguinal, iliohypogastric, and genitofemoral nerves, as well as lateral femoral cutaneous nerves) is warranted, including noting the free communication between these branches. According to a cadaver study, the ilioinguinal nerve was solely responsible for cutaneous innervation of the genital branch of the genitofemoral nerve in 28 % of the dissections, and it shared innervation with the genital branch of the genitofemoral nerve in 8 % [16].

Entrapment

The GFN passes anteriorly through the psoas major muscle and may therefore become entrapped on its path, or by reflex spasm of the psoas muscle [17]. Although entrapment symptoms of this primarily sensory lumbosacral nerve have not been specifically related to psoas major trigger points, this possibility should be considered when the patient suffers from pain and sensory alterations along the distribution of this nerve [17]. The GFN can be entrapped in the presence of hematoma, abscess, or trigger points of the psoas muscle. Retroperitoneal hematoma, lymphoma, and failed lumbar surgery can also cause entrapment of this nerve [17].

Operations such as C-section, appendectomy, inguinal hernia repair, and laparoscopic procedures may contribute to injury of the GFN and therefore result in pain arising along the distribution of the GFN [6, 18].

In addition, more distal entrapment at the pubic tubercle or spermatic cord (after hernia repairs or groin surgeries) or under the inguinal ligament (after pelvic surgeries, similar to the LFC) can occur.

Physical Examination

A thorough neurologic exam may find sensory changes, which can be subtle, in the lower abdomen/pelvic area, groin area, anterior proximal thigh, or scrotum or labia majora. Sensory changes can range from hypoesthesia to total loss of sensation, paresthesias, or even allodynia. Pain may also worsen on performing a *Valsalva maneuver*, coughing, rising, and peristaltic movements. Female patients may observe worsening of pain during menstruation and sexual intercourse [19, 20]. In advanced neuropathy, loss of cremasteric reflex can occur. Maneuvers to activate or tighten the psoas muscle may also aggravate the pain.

Except in the very thin areas, it is difficult to palpate the anterior aspect of the psoas muscle where the GFN runs. The most common site of tenderness to palpation is on the pubic tubercle (Fig. 60.7), which can be confirmed by fluoroscopy (Fig. 60.8).

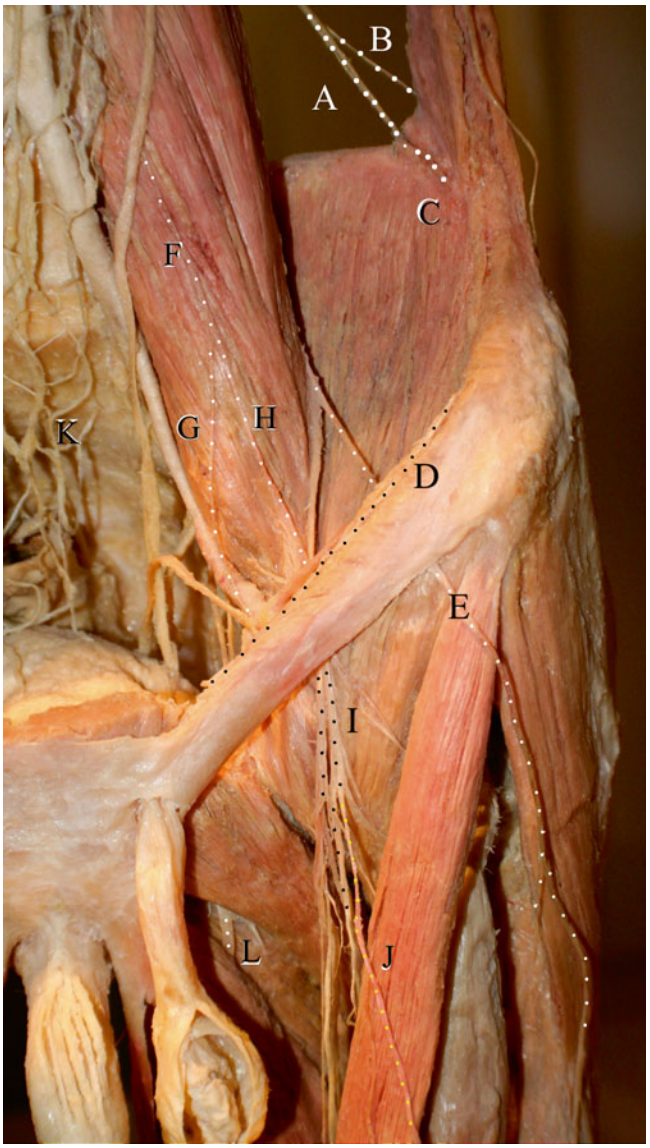

Fig. 60.6 Pelvic and groin dissection, modified from an image from *Bodies*, *The Exhibition*, with permission. *A* ilioinguinal nerve, *B* iliohypogastric nerve, *C* site of ilioinguinal nerve entrapment at the external oblique, *D* ilioinguinal nerve over the inguinal ligament, *E* lateral femoral cutaneous nerve, *F* genitofemoral nerve, *G* genital branch of the genitofemoral nerve, *H* femoral branch of the genitofemoral nerve, *I* femoral nerve, *J* saphenous nerve, *K* inferior hypogastric plexus, *L* obturator nerve (Image courtesy of Andrea Trescot, MD)

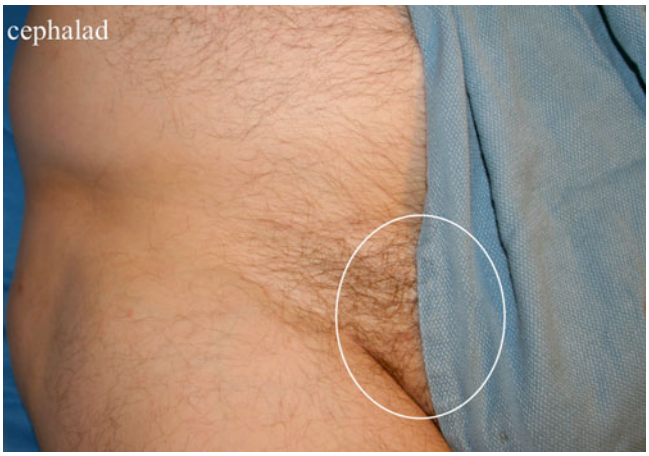

Fig. 60.7 Site of physical exam tenderness from genitofemoral pathology (Image courtesy of Andrea Trescot, MD)

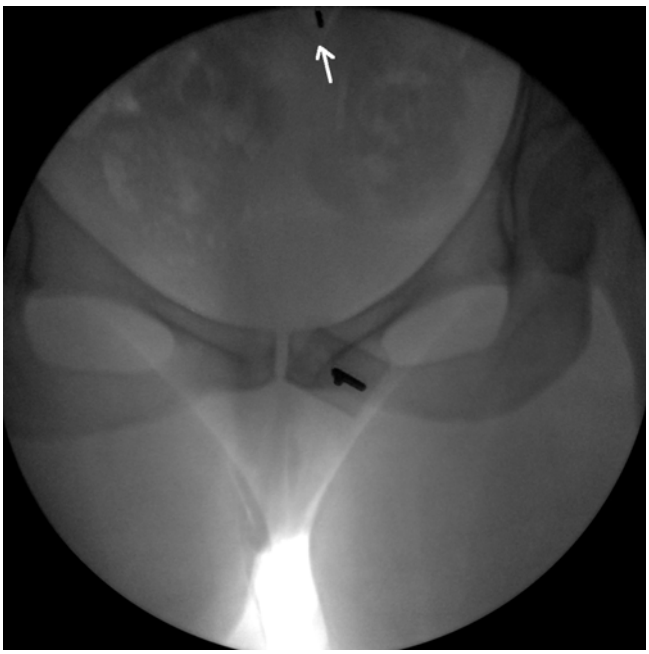

Fig. 60.8 Fluoroscopic confirmation of the site of physical exam tenderness from genitofemoral pathology. Note the *white arrow* showing the InterStim® placed for the wrong diagnosis (interstitial cystitis pain) that offered no relief (Image courtesy of Andrea Trescot, MD)

Differential Diagnosis (Table 60.3)

There is considerable overlapping of the causes of anterior proximal thigh pain related to GFN, ilioinguinal, and LFC due to the overlapping in nerve distribution and/or communication among those nerves. It may be quite difficult to differentiate between these nerves [11]. In this situation, it is important to perform specific diagnostic injections to exclude pain originating from these other nerves (Table 60.4) [24].

Identification and Treatment of Contributing Factors

Strenuous exercise and overstretching of psoas muscle will trigger spasm of the psoas, entrapping the GFN. Past abdominal or pelvic surgeries, scars, mesh placement, neuroma or hematoma formation are associated GFN entrapment. Pregnancies, with pressure on the GFN at the psoas or at the pelvic rim from the gravid uterus, can result in paresthesias

Table 60.3 Differential diagnosis of groin and lower extremity pain

	Potential distinguishing features
Adductor or rectus abdominis tendonitis/myofascial spasm	Tenderness at the pubis; palpable spasm of the muscle
Avascular necrosis femoral head	X-ray and MRI show femoral head collapse
Pubic symphysis/osteitis [21]	Bone scan positive
L1–L3 radiculopathy [22]	Sensory loss, weakness, EMG positive
Distal psoas tendonitis [22]	Tenderness on the lesser trochanter
Abdominal wall hernia [21]	Abdominal wall defect
Hip joint pathology [23]	Stiffness, limited range of motion, crepitus, clicking
Ilioinguinal nerve injury/ entrapment [21]	Increased abdominal wall tension can result in groin pain; may be tender near ASIS; increased pain with hip hyperextension
Adductor strain [23]	Tenderness over adductors; usual site is at the muscle-tendon intersection and sometimes at tendon-bone

Table 60.4 Diagnostic tests for genitofemoral nerve entrapment

	Potential distinguishing features
Physical exam	Tenderness at the pubic tubercle
Diagnostic injection	At the pubic tubercle, the psoas muscle, or the L1 or L2 foramen
Ultrasound	Not useful for diagnosis
MRI	Not useful for diagnosis
Arteriography	Not useful for diagnosis
X-ray	Not useful for diagnosis
Electrodiagnostic studies	Not useful for diagnosis

or hypoesthesias that usually resolve with delivery. Obesity can entrap the femoral branch under the inguinal ligament.

Treatment of underlying conditions that caused nerve entrapment, such as physical therapy or PT-guided exercise [17], can help to alleviate iliopsoas muscle dysfunction or spasm; in addition, scar release, ablation of neuroma, and timely drainage of hematoma can relieve the entrapment. If compression is caused by muscle hypertrophy, the treatment of choice is to inject local anesthetic to enable muscle relaxation and, if necessary, botulinum toxin for longer-lasting relaxation.

Injection Technique

Landmark-Guided Technique

Because the pubic tubercle is superficial in all but the most obese patients, the landmark-guided injection of the GFN at the pubic tubercle can be useful, especially as a screening tool. Tenderness to palpation at the tubercle identifies the injection site. The use of a peripheral nerve stimulator (PNS) can aid in locating the nerve.

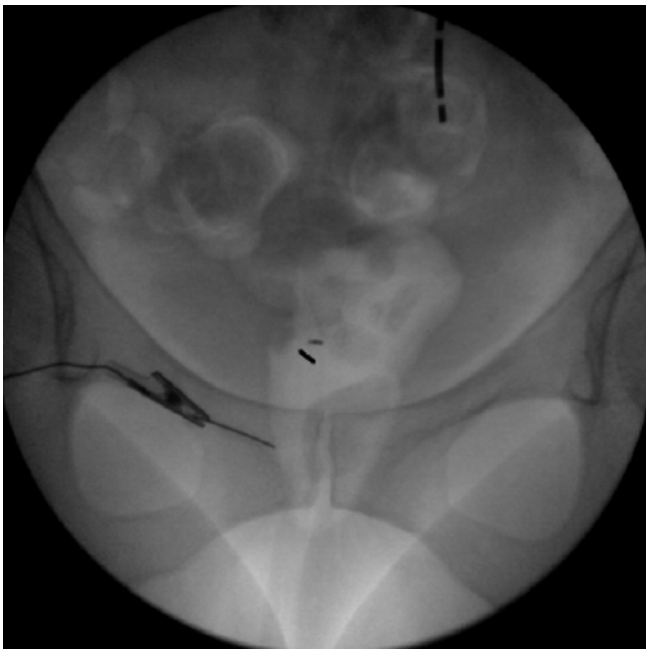

Fig. 60.9 Fluoroscopy-guided injection of the genitofemoral nerve using a peripheral nerve stimulator (Image courtesy of Andrea Trescot, MD)

Trescot described a landmark-guided ("blind") technique to inject the genital branch of the GFN [25]. The patient is placed in the supine position, with a pillow under the knees if extending the lower limbs evokes pain. After a sterile skin prep, the pubic tubercle is palpated, and 1 cc of local anesthetic and deposteroid is injected via a 25-gauge needle just superior and lateral to the tubercle. The use of a PNS can facilitate the accuracy of the injection. When performing the landmark-guided technique, the utmost care must be taken in relation to important spermatic cord structures (such as the testicular artery) and to peritoneal cavity transgression.

Fluoroscopy-Guided Technique

With the patient in the supine position, the area of maximal tenderness (just lateral to the pubic tubercle) is identified by fluoroscopy (Fig. 60.8). After a sterile skin prep and local anesthetic infiltration subcutaneously, a 22-guage needle is advanced to the periosteum. The use of a PNS will facilitate identification of the nerve (Fig. 60.9), and 1 cc of local anesthetic and deposteroid is then injected.

Another technique for fluoroscopic GFN diagnosis and treatment is the dorsal root ganglion (DRG) local anesthetic block at T12, L1, and L2, ipsilateral to the pain (Fig. 60.10). T12 DRG should be included because it is common for the

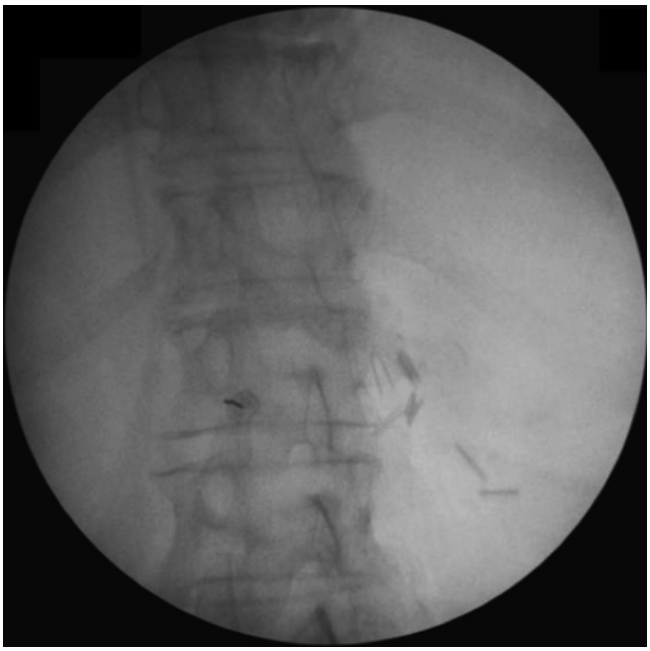

Fig. 60.10 Injection of the dorsal root ganglia at L1 (Image courtesy of Fabrício Assis, MD)

ilioinguinal and genitofemoral nerves to communicate, and the ilioinguinal nerve can come from T12. If the block results in significant pain relief (at least 50 % improvement), but is short lived, cryoneuroablation or pulsed radiofrequency may be applied to these ganglia (see Neurolytics below) [11].

Ultrasound-Guided Injection (US)

Ultrasound can be used to perform a selective block of the genital or femoral branch of the GFN. The *genital branch* is slender and cannot be directly visualized, but the femoral/external iliac artery is easily found at the inguinal canal, and the genital branch is immediately medial to it, within the internal inguinal ring (Fig. 60.11) [19, 26]. The *femoral branch* runs superficially outside of the inguinal canal; the target is superficial and lateral to the femoral artery, caudal to the inguinal ligament, and approximately a third of the distance between the pubic tubercle and the anterior superior iliac spine. A high-frequency linear probe is utilized, oriented perpendicular to the inguinal ligament with its tip about one fingerbreadth lateral to the pubic tubercle. It is suggested that the practitioner starts in the internal inguinal ring, in which it is possible to visualize the longitudinal (lengthwise) section of the artery. At this point, an oval or round structure (the cremaster in males, the round ligament in females), superficial to the femoral artery, can easily be visualized (Fig. 60.11). The probe is then rotated transversely, advanced in the lateral direction, slowly, moving away from the femoral artery, toward the sartorius muscle. The femoral branch of the GFN can be found above the space between the femoral artery and femoral nerve (Fig. 60.12). The needle may be inserted out-of-plane, injecting local anesthetic without vasoconstrictors so as to avoid the adverse effects of testicular artery vasoconstriction (Fig. 60.13). Because of anatomical variability, it is recommendable to use 5 mL inside and 5 mL outside the spermatic cord [6], though use of a peripheral nerve stimulator should decreased the need for large volumes.

CT-Guided Injection

The genitofemoral nerve is retroperitoneal before entering the inguinal canal. This position increases the risk of transgressing the peritoneum in the event of an anterior approach. Because of this, there is a computer tomography (CT)-guided transpsoas technique capable of selectively blocking the genitofemoral nerve [15] while avoiding injury to the ureters and intestines (Fig. 60.14). This technique can be used for diagnostic and therapeutic purposes. Since the genitofemoral nerve is difficult to visualize, when this technique is done with the aid of a PNS, small volumes of local anesthetic can be used, thus preventing the spread of medication into the lumbar sympathetic chain. Needle entry point is just above the L4 transverse process, and utilizing the PNS, stimulation radiating to the groin and the upper ipsilateral thigh should be achieved [15].

Neurolytic Technique

Cryoneuroablation

Cryoneuroablation at the pubic tubercle can be performed by fluoroscopy-guided or ultrasound-guided techniques. Because the tissue at the pubis is usually relatively thin, it is conceivable that one could identify the GFN by just landmarks and the built-in nerve stimulator on the cryoprobe, but this is not a recommended technique. Trescot [27] described placement of the cryoprobe onto the pubic tubercle, using fluoroscopy and the nerve stimulator to find the nerve (Fig. 60.15). In that same publication, Trescot also described cryoneuroablation at the L1 foramen to treat the GFN proximally (Fig. 60.16). Campos et al. [28] described cryoneuroablation of the femoral branch of the GFN under US guidance (Fig. 60.17). The target location was superficial and lateral to the femoral artery (identified by US), caudal to the inguinal ligament, and approximately a third of the distance from the pubic tubercle to the anterior superior iliac spine. The catheter introducer and then the cryoprobe were advanced under US control with confirmation with

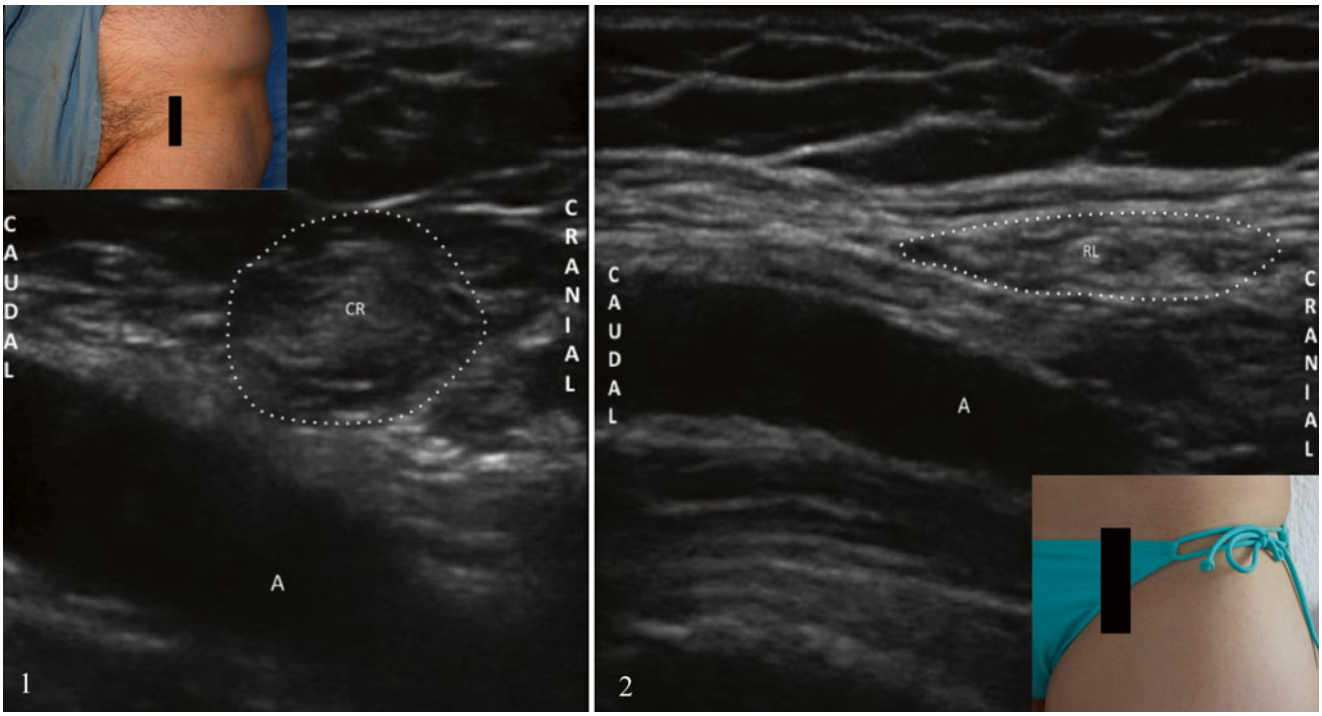

Fig. 60.11 Ultrasound image of the male internal inguinal ring and female round ligament. *Dotted line* represents the internal inguinal ring; *CR* cremaster, *RL* round ligament, *A* femoral artery at the site of bifurcation (superficially, the vessel is the femoral artery, while the external iliac artery would be deep) (Image courtesy of Thiago Nouer Frederico, MD)

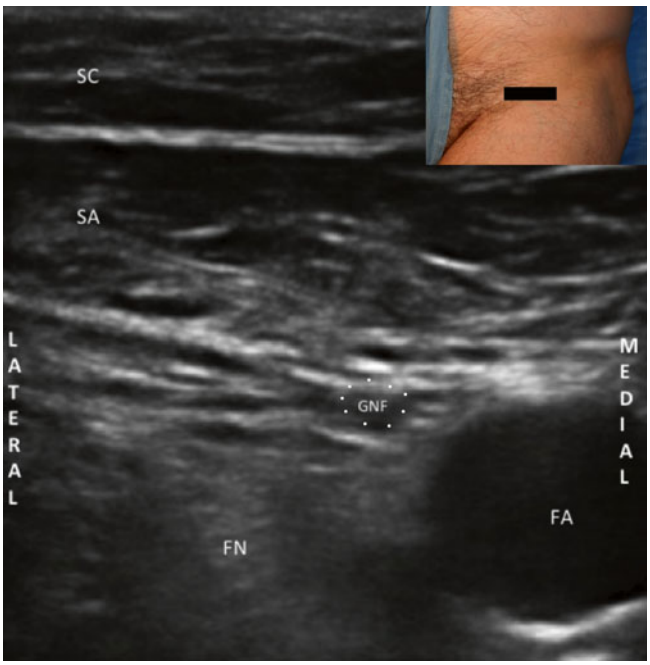

Fig. 60.12 Ultrasound image taken transversely below the inguinal ligament, showing the femoral branch of the genitofemoral nerve. *FA* femoral artery, *FN* femoral nerve, *GNF* femoral branch genitofemoral nerve, *SC* subcutaneous tissue, *SA* sartorius muscle (Image courtesy of Thiago Nouer Frederico, MD)

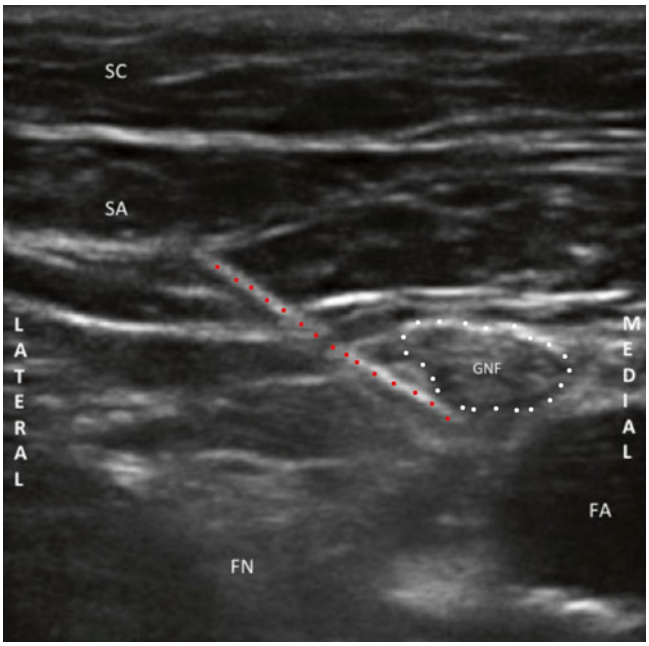

Fig. 60.13 Ultrasound image taken transversely below the inguinal ligament, showing injection of the femoral branch of the genitofemoral nerve. Note the needle (*red dots*) and the space distension by the local anesthetic (*white dots*). *FA* femoral artery, *FN* femoral nerve, *GNF* femoral branch genitofemoral nerve, *SC* subcutaneous tissue, *SA* sartorius muscle (Image courtesy of Thiago Nouer Frederico, MD)

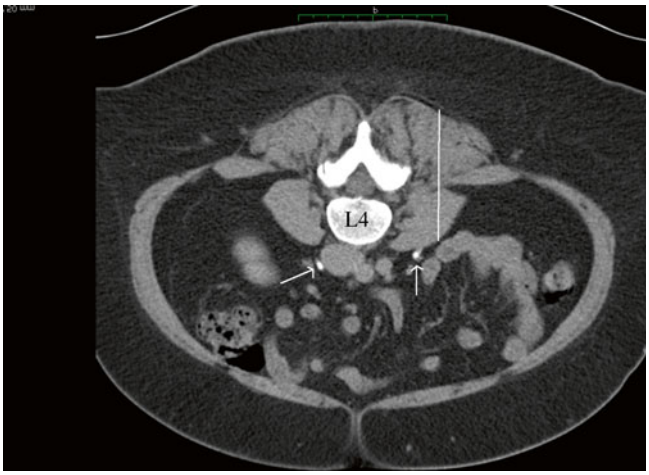

Fig. 60.14 Transpsoas contrast CT-guided technique (simulated). As per Parris et al. [14], the needle (simulated by *white line*) is placed through the psoas muscle; *arrows* = ureters (with contrast) (Image courtesy of Andrea Trescot, MD)

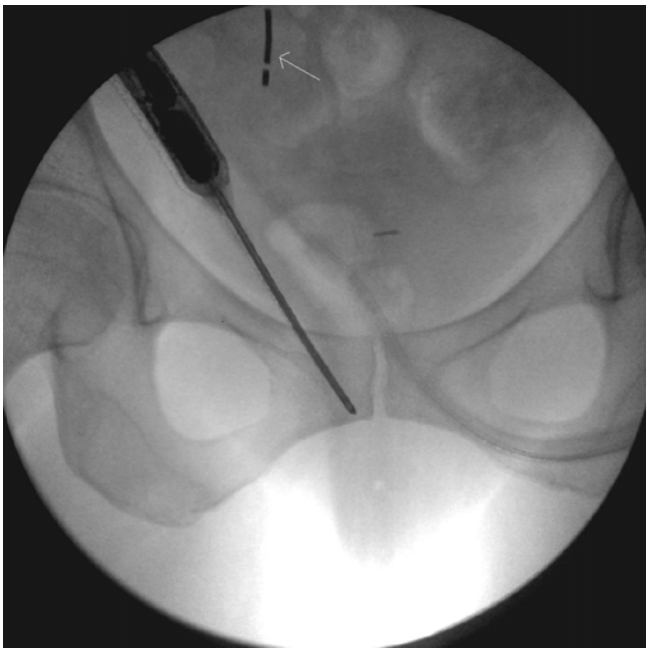

Fig. 60.15 Cryoneuroablation of the genitofemoral nerve at the pubic tubercle (*white arrow* indicates prior InterStim® implant) (Image courtesy of Andrea Trescot, MD)

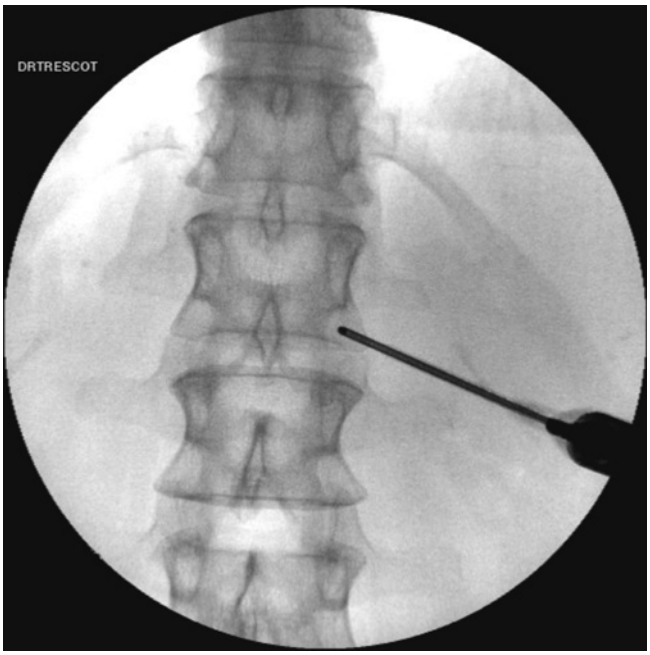

Fig. 60.16 Cryoprobe positioned on the proximal genitofemoral nerve at L1 (Image courtesy of Andrea Trescot, MD)

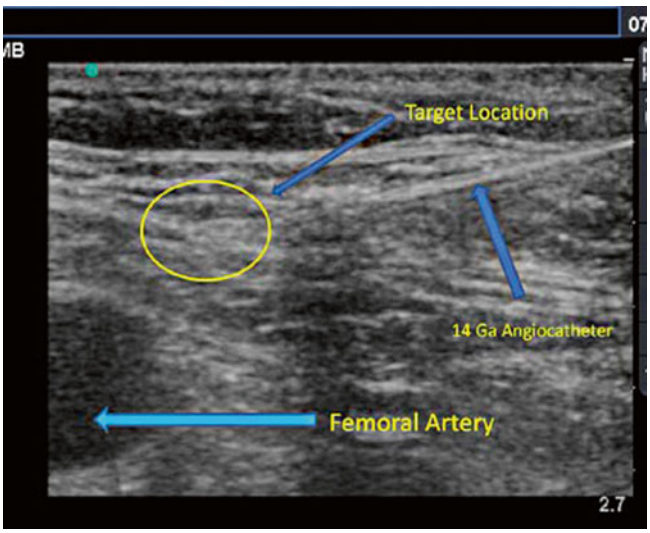

Fig. 60.17 Cryoneuroablation under ultrasound of the femoral branch of the genitofemoral nerve (Image courtesy of John Chiles, MD)

sensory stimulation and no motor stimulation. Patient underwent two 3-min freeze cycles, with immediate and sustained relief.

Radiofrequency Lesioning

Although conventional radiofrequency (RF) should be strongly discouraged in this area because of the risk of neuritis and neuroma, pulsed radiofrequency treatment of the GFN has been described. Terkawi and Romdhane [29] treated a young man suffering from chronic orchialgia; a diagnostic injection of the genital branch under US guidance (using the technique similar to that described above) gave excellent, but only temporary, relief. The patient underwent a pulsed RF lesion, again using the same ultrasound technique. At 7 months follow-up, the patient was still noting excellent relief.

Rozen and Ahn [30] described five patients with chronic groin pain treated with pulsed RF at T12, L1, and L2.

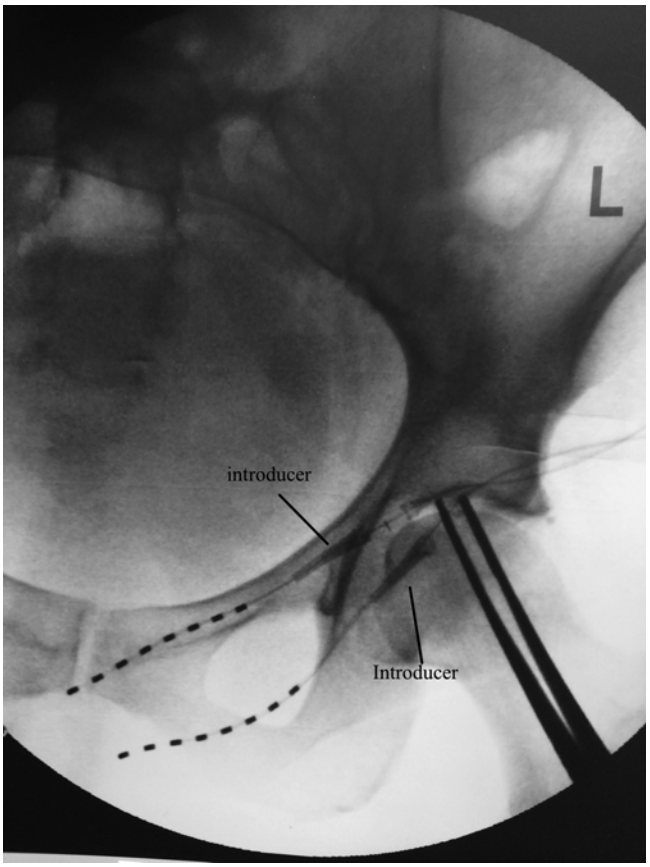

Fig. 60.18 Peripheral nerve stimulation abdominal wall (Image courtesy of Gladstone MacDowell, MD)

Although they described the treatment of ilioinguinal neuralgia after inguinal hernia repair, because of the great variability distally and the shared origin at L1 and L2, the nerves treated could well have been the GFN.

Phenol

Weksler and colleagues [31] described injecting 4 % phenol onto a variety of painful structures (including the GFN) in 35 patients; they noted good relief and no complications.

Neurostimulation

Peripheral nerve stimulation has been used to treat chronic groin pain (Fig. 60.18) [32, 33]. In a technique similar to that used for inguinal nerve stimulation (see Chap. 44), the trial electrodes are placed percutaneously through introducers, and if there is significant temporary relief, the leads can be placed permanently.

Surgery

Starling and colleagues reviewed 30 patients with ilioinguinal or GFN abdominal pain over a 7-year period. Patients were diagnosed with local anesthetic injections; 10 of the 13 GFN patients treated with neurectomy proximal to the entrapment noted relief. Triple neurectomy (ilioinguinal, iliohypogastric, and GFN) has also been advocated, with a reported 80 % success rate in relieving postsurgical groin pain [34]. According to Muto et al. retroperitoneal endoscopic lumbar neurectomy is a simple, minimally invasive technique with low morbidity and mortality. In this technique, the genitofemoral nerve is easily visualized where it penetrates the psoas muscle and can be dissected and resected at this site [14].

Acar et al. [34] described 20 patients with ilioinguinal and GFN neuralgia; 14 of these patients (70 %) were treated with injections. Of the six patients that did not respond to those injections (30 %), all underwent neurectomy with "pain relief."

Complications

Any injection may cause the usual complications of bleeding, infection, and nerve damage. Psoas hematoma is a potentially serious complication of nerve block, but is usually encountered in the setting of anticoagulation. In the CT-guided transpsoas technique, the main complications are ureter or intestine perforation, but retroperitoneal or psoas hematoma can also occur. At any rate, in the CT-guided technique, there is theoretically a lower risk of this happening than in the blind or fluoroscopy-guided techniques [15].

Though uncommon, after the endoscopic surgical procedure in men, there may be loss of cremasteric reflex and in women a loss of sensation in the mons pubis and labia majora [14].

Summary

The GFN is an under-recognized cause of lower extremity pain, as well as abdominal pain and pelvic pain. A careful history and physical examination, as well as a high index of suspicion, will help the clinician to begin to recognize and then treat GFN entrapments.

Acknowledgments We would like to thank Camille Khan for translation from Brazilian Portuguese to English.

References

1. Benes J, Nádvornik P, Dolezel J. Abdominoinguinal pain syndrome treated by centrocentral anastomosis. Acta Neurochir (Wien). 2000;142(8):887–91.
2. Drake RL, Vogl W, Tibbitts AWM. Gray's anatomy for students. Philadelphia: Elsevier/Churchill Livingstone; 2005.
3. Zempoalteca R, Martínez-Gómez M, Hudson R, Cruz Y, Lucio RA. An anatomical and electrophysiological study of the genitofemoral nerve and some of its targets in the male rat. J Anat. 2002;201(6):493–505.
4. Magee RK. Genitofemoral causalgia: (a new syndrome). Can Med Assoc J. 1942;46(4):326–9.
5. Peng PW, Tumber PS. Ultrasound-guided interventional procedures for patients with chronic pelvic pain – a description of techniques and review of literature. Pain Physician. 2008;11(2):215–24.
6. Rho RH, Lamer TJ, Fulmer JT. Treatment of genitofemoral neuralgia after laparoscopic inguinal herniorrhaphy with fluoroscopically guided tack injection. Pain Med. 2001;2(3):230–3.
7. Feigl GC, Dreu M, Ulz H, Breschan C, Maier C, Likar R. Susceptibility of the genitofemoral and lateral femoral cutaneous nerves to complications from lumbar sympathetic blocks: is there a morphological reason? Br J Anaesth. 2014;112(6):1098–104.
8. Boss A, Clasen S, Kuczyk M, Anastasiadis A, Schmidt D, Claussen CD, et al. Thermal damage of the genitofemoral nerve due to radiofrequency ablation of renal cell carcinoma: a potentially avoidable complication. AJR Am J Roentgenol. 2005;185(6):1627–31.
9. Starling JR, Harms BA. Diagnosis and treatment of genitofemoral and ilioinguinal neuralgia. World J Surg. 1989;13(5):586–91.
10. Rozen D, Parvez U. Pulsed radiofrequency of lumbar nerve roots for treatment of chronic inguinal herniorraphy pain. Pain Physician. 2006;9(2):153–6.
11. Muensterer OJ. Genitofemoral nerve injury after laparoscopic varicocelectomy in adolescents. J Urol. 2008;180(5):2155–7.
12. Reinpold W, Schroeder AD, Schroeder M, Berger C, Rohr M, Wehrenberg U. Retroperitoneal anatomy of the iliohypogastric, ilioinguinal, genitofemoral, and lateral femoral cutaneous nerve: consequences for prevention and treatment of chronic inguinodynia. Hernia. 2015;19(4):539–48.
13. Muto CM, Pedana N, Scarpelli S, Galardo R, Guida G, Schiavone V. Inguinal neurectomy for nerve entrapment after open/laparoscopic hernia repair using retroperitoneal endoscopic approach. Surg Endosc. 2005;19(7):974–6.
14. Parris D, Fischbein N, Mackey S, Carroll I. A novel CT-guided transpsoas approach to diagnostic genitofemoral nerve block and ablation. Pain Med. 2010;11(5):785–9.
15. Rab M, Ebmer, Dellon AL. Anatomic variability of the ilioinguinal and genitofemoral nerve: implications for the treatment of groin pain. Plast Reconstr Surg. 2001;108(6):1618–23.
16. Ingber RS. Iliopsoas myofascial dysfunction: a treatable cause of "failed" low back syndrome. Arch Phys Med Rehabil. 1989;70(5):382–6.
17. Reinpold WM, Nehls J, Eggert A. Nerve management and chronic pain after open inguinal hernia repair: a prospective two phase study. Ann Surg. 2011;254(1):163–8.
18. Parnell BA, Johnson EA, Zolnoun DA. Genitofemoral and perineal neuralgia after transobturator midurethral sling. Obstet Gynecol. 2012;119(2 Pt 2):428–31.
19. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
20. Ekberg O, Persson NH, Abrahamsson PA, Westlin NE, Lilja B. Longstanding groin pain in athletes. A multidisciplinary approach. Sports Med. 1988;6(1):56–61.
21. Malik A, Simopolous T, Elkersh M, Aner M, Bajwa ZH. Percutaneous radiofrequency lesioning of sensory branches of the obturator and femoral nerves for the treatment of non-operable hip pain. Pain Physician. 2003;6(4):499–502.
22. Tammareddi K, Morelli V, Reyes Jr M. The athlete's hip and groin. Prim Care. 2013;40(2):313–33.
23. Rigaud J, Riant T, Delavierre D, Sibert L, Labat JJ. Somatic nerve block in the management of chronic pelvic and perineal pain. Prog Urol. 2010;20(12):1072–83.
24. Trescot AM. Cryoneurolysis and the healing response. Annual meeting American Academy of Pain Management, San Diego. 2005.
25. Recker DC, Perry PM. Postsurgical pain syndromes: chronic pain after hysterectomy and cesarean section. Tech Reg Anesth Pain Manag. 2011;15(3):133–9.
26. Peng PW, Narouze S. Ultrasound-guided interventional procedures in pain medicine: a review of anatomy, sonoanatomy, and procedures: part I: nonaxial structures. Reg Anesth Pain Med. 2009; 34(5):458–74.
27. Campos NA, Chiles JH, Plunkett AR. Ultrasound-guided cryoablation of genitofemoral nerve for chronic inguinal pain. Pain Physician. 2009;12(6):997–1000.
28. Terkawi AS, Romdhane K. Ultrasound-guided pulsed radiofrequency ablation of the genital branch of the genitofemoral nerve for treatment of intractable orchalgia. Saudi J Anaesth. 2014;8(2): 294–8.
29. Rozen D, Ahn J. Pulsed radiofrequency for the treatment of ilioinguinal neuralgia after inguinal herniorrhaphy. Mt Sinai J Med N Y. 2006;73(4):716–8.
30. Weksler N, Klein M, Gurevitch B, Rozentsveig V, Rudich Z, Brill S, et al. Phenol neurolysis for severe chronic nonmalignant pain: is the old also obsolete? Pain Med. 2007;8(4):332–7.
31. Rosendal F, Moir L, de Pennington N, Green AL, Aziz TZ. Successful treatment of testicular pain with peripheral nerve stimulation of the cutaneous branch of the ilioinguinal and genital branch of the genitofemoral nerves. Neuromodulation. 2013;16(2):121–4.
32. Banh DP, Moujan PM, Haque Q, Han TH. Permanent implantation of peripheral nerve stimulator for combat injury-related ilioinguinal neuralgia. Pain Physician. 2013;16(6):E789–91.
33. Amid P. Causes, prevention, and surgical treatment of postherniorrhaphy neuropathic inguinodynia: triple neurectomy with proximal end implantation. Hernia. 2004;8(4):343–9.
34. Acar F, Ozdemir M, Bayrakli F, Cirak B, Coskun E, Burchiel K. Management of medically intractable genitofemoral and ilioinguinal neuralgia. Turk Neurosurg. 2013;23(6):753–7.

Lateral Femoral Cutaneous Nerve Entrapment

61

Lisa Rochelle Witkin, Amitabh Gulati, Tiffany Zhang, and Helen W. Karl

Introduction

The *lateral femoral cutaneous nerve (LFCN)* is a sensory nerve that is susceptible to compression as it courses from the lumbosacral plexus, through the retroperitoneum, under the inguinal ligament, and into the subcutaneous tissue of the thigh. *Meralgia paresthetica* (MP), or *Bernhardt-Roth syndrome*, is the clinical syndrome of dysesthesia, pain, or both in the anterolateral thigh associated with compression of this nerve. MP was first described in 1885, by the German surgeon Werner Hager [1], and later named "meralgia," from the Greek words "meros" meaning thigh and "algos" meaning pain. Nontraumatic MP is not uncommon, with an overall incidence of 3.4–4.3/10,000 person-years [2, 3]. Sigmund Freud confessed to suffering from this condition [4].

It is significantly related to carpal tunnel syndrome [3] (see Chap. 37), pregnancy [3], high body mass index, and diabetes mellitus (DM) [2]. MP occurs seven times more frequently in patients with DM, and patients with MP are twice as likely to develop DM after their MP diagnosis than those in a matched population [2]. Earlier studies reported a male predominance [5].

Clinical Presentation (Table 61.1)

MP classically presents with a subacute onset of burning pain, dysesthesia (paresthesia and hypesthesia), or both in the anterolateral thigh (Fig. 61.1). The pain can be located anywhere from the anterior to the lateral hip, the anterior and

Table 61.1 Occupation/exercise/trauma history relevant to lateral femoral cutaneous entrapment

Compression	Obesity [2]
	Pregnancy [3, 6]
	Abdominal masses (uterine myoma, retroperitoneal lipofibrosarcoma)
	Ascites, large abdomen [7]
	Tight garments or seat belts, especially in thin individuals [8]
	Leg length discrepancy [9]
	Lumbar herniated disk [10, 11]
	Psoas tumor/infection/spasm [12]
Trauma	"Hip-checked" [1]
Surgery	Laparoscopic appendectomy, cholecystectomy [13], hernia repair [14, 15]
	Iliac crest graft [16–18]
	Lumbar sympathetic block or neurolysis [19]
	Femoral artery catheterization [20]
	Occurs in 20 % of spinal surgeries [16]
	Total hip arthroplasty [21]
	Ilioinguinal repair of a pelvic fracture [22]
Infection/inflammation	Diabetes mellitus
	Periostitis of the ilium
	Retrocecal tumor
	Appendicitis [23]
Exercise	Strenuous abdominal or lower body exercise [24]

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Fig. 61.1 Patient pain complaint from lateral femoral cutaneous nerve entrapment (Image courtesy of Andrea Trescot, MD)

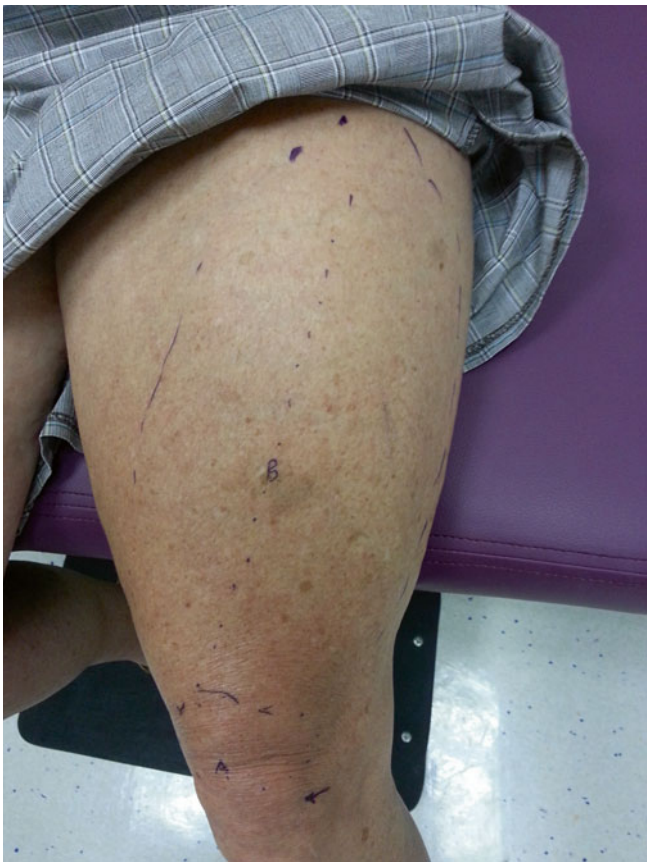

Fig. 61.2 Pattern of pain from lateral femoral cutaneous nerve entrapment (Image courtesy of Eric Wilson, MD)

lateral thigh, and distally to the anterior knee (Fig. 61.2). Patients may also describe coldness, deep muscle ache, frank anesthesia in a discrete area, or hair loss in the anterolateral thigh from stroking the area [25, 26]. Symptoms are often worse with prolonged standing and walking and alleviated by sitting down, though some report that sitting exaggerates the pain. Furthermore, patients may have secondary hip, knee, and calf pain as they try to modify their gait and physical activities to minimize their symptoms. Importantly, neurologic symptoms are restricted to sensory changes, since the LFCN does not contain motor fibers.

Anatomy (Table 61.2)

The most striking aspect of LFCN anatomy is its variability, particularly as it transitions from the pelvis into the thigh. The LFCN originates at L2 and L3, as part of the *lumbar plexus* (see Chap. 66), and then passes obliquely between the superficial and deep parts of the *psoas muscle* and between the two layers of fascia over the surface of the *iliacus muscle* (Figs. 61.3 and 61.4). It then travels through an *aponeurotic-cofascial tunnel* from the *iliopubic tract* to the *inguinal ligament* (IL) [27], under the IL or through a split in its most lateral part at the *anterior superior iliac spine (ASIS)*. As it approaches the ASIS, it courses under the *deep circumflex iliac vessels* and exits into the thigh. It may cross into the thigh anywhere between 6 cm medial to the ASIS and over 2 cm lateral to it [30]. The LFCN may enter the thigh superficial to or within the substance of the *sartorius muscle* (Fig. 61.5), or it may cross over the iliac crest lateral and posterior to the ASIS, where it is particularly susceptible to pressure from tight garments or belts [8, 35]. The LFCN usually divides into two branches: a smaller posterior branch that innervates the greater trochanter area (Fig. 61.6) and a larger anterior branch, which innervates the anterolateral thigh to the knee [36].

Furthermore, it may leave the pelvis in as many as five branches; entrapment of even a single branch can lead to the classic MP symptoms [31]. Some of the variability in the reported findings reflects the different interests of the investigators; for example, some of the dissections have been performed through the perspective of a specific surgical incision [22, 32]. Other variability may be due to differences in labeling the complex musculo-fascial layers that weave around

Table 61.2 Lateral femoral cutaneous nerve anatomy

Origin	L2 and L3
General route	From between the superficial and deep parts of the psoas and around the pelvis on the *iliacus muscle* between two layers of fascia. Then travels through an "aponeuroticofascial tunnel" from the *iliopubic tract* to the inguinal ligament (IL) [27], under the IL or through a split in its most lateral part at the ASIS. About 10 cm below the IL, it emerges through the superficial fascia of the thigh, divides into anterior and posterior branches, and ends in the skin of the anterolateral thigh
Sensory distribution	Anterior branch (larger): anterolateral thigh to the knee
	Posterior branch (smaller): greater trochanter to the area supplied by the anterior division
Motor innervation	None
Anatomic variability	*Fused with the genitofemoral nerve (GFN) in about 2 %*: if so, there is increased vulnerability to lumbar sympathetic block [19]. Earlier authors, focused on a more distal area, reported more frequent merging with the GFN or femoral nerve [28, 29]
	Intra-abdominal branching: has one vertebral origin and one distributing branch at the level of the inguinal ligament in 86 % of cases [30]
	Site at which the LFCN leaves the abdomen: *A* (4 %) = posterior to ASIS, across the iliac crest; *B* (27 %) = medial to the ASIS, superficial to the origin of the sartorius; *C* (23 %) = medial to the ASIS, within the origin of the sartorius muscle; *D* (26 %) = medial to the origin of the sartorius muscle, between its tendon and the thick fascia of the iliopsoas muscle, deep to the inguinal ligament; and *E* (20 %) = the most medial origin, also deep to the inguinal ligament, but superficial to the iliopsoas fascia [28]
	Distance from the ASIS at the level of the inguinal ligament: 1.4 ± 1.5 cm with a range of 2.3 cm lateral to 6.2 cm medial [30]
	Inguinal branching: As many as five branches identified; in 8 of 29 cadavers (28 %), the LFCN branched before traversing the inguinal ligament [31]
	Angle between the pelvic and femoral portions: 100 ± 10° [27]
	Relationship to the sartorius muscle: exit through the muscle (11/50 = 22 %) [8] or its tendon of origin (24/104 = 23 %) [28]
	Relationship to the superficial thigh fascia: 88 % are deep to the superficial fascia below the ASIS, 3 % are superficial to it, and 9 % were not found [32]
	Thigh branching: 27/50 (54 %) bifurcation to anterior and posterior branches; 18/50 (36 %) had no posterior branch [27]. The area of the skin over the lateral and anterior thigh innervated by the LFCN is highly variable [33, 34]
	Side to side symmetry: present in 34/52 (65 %) cadavers [28]
Other relevant structures	*Transversus abdominis muscle*: may originate in part from the iliacus fascia; therefore abdominal muscle contraction may increase LFCN stress [9]
	Iliopubic tract: compression here is analogous to entrapment of the median nerve in the carpal tunnel [27]
	Inguinal ligament: the inferior border of the external oblique aponeurosis. Many structures converge here, including the fascia lata and the origin of the sartorius muscle
	Muscular compartment of the inguinal region: between the inguinal ligament, the iliopectineal arch, and the ilium, through which the LFCN, femoral nerve, and iliopsoas muscle reach the leg
	Deep circumflex vessels: the LFCN is deep to these structures as it approaches the ASIS

the inguinal area. For example, the "sharp ridge of *iliacus fascia*,…causing a bowstring deformity of the nerve when the patient is in the supine position" [35] is likely what has been described by others as the *iliopubic tract* [27]. None of this confusion should detract from the inherent inconstancy and variability of the LFCN itself.

The high degree of LFCN anatomical variation has significant implications for determining the location and etiology of entrapment, treating the symptoms, and protecting the nerve during surgical procedures [14, 27, 28]. Since one early study showed a five times greater incidence of MP after *laparoscopic herniorrhaphy* than after *open herniorrhaphy*, the subsequent increase in laparoscopic surgery has stimulated interest in LFCN anatomy [30].

Entrapment

The LFCN may be compressed or injured at any point during its long path in the retroperitoneum or pelvis. However, there are two areas at which it is particularly vulnerable. The *iliopubic tract* (IPT) is an area of dense connective tissue at the junction of the anterior lamina of the iliac fascia and the *transversalis fascia* that invests the *transversus abdominis muscle*. The LFCN always passes deep to and juxtaposed to this structure; the presence of *pseudoneuromas* proximal to the IPT is a strong clue to the potential for compression of the LFCN at the IPT [27].

As the nerve passes from the pelvis to the thigh, there is a 100 ± 10° angle of the path of the nerve that makes it

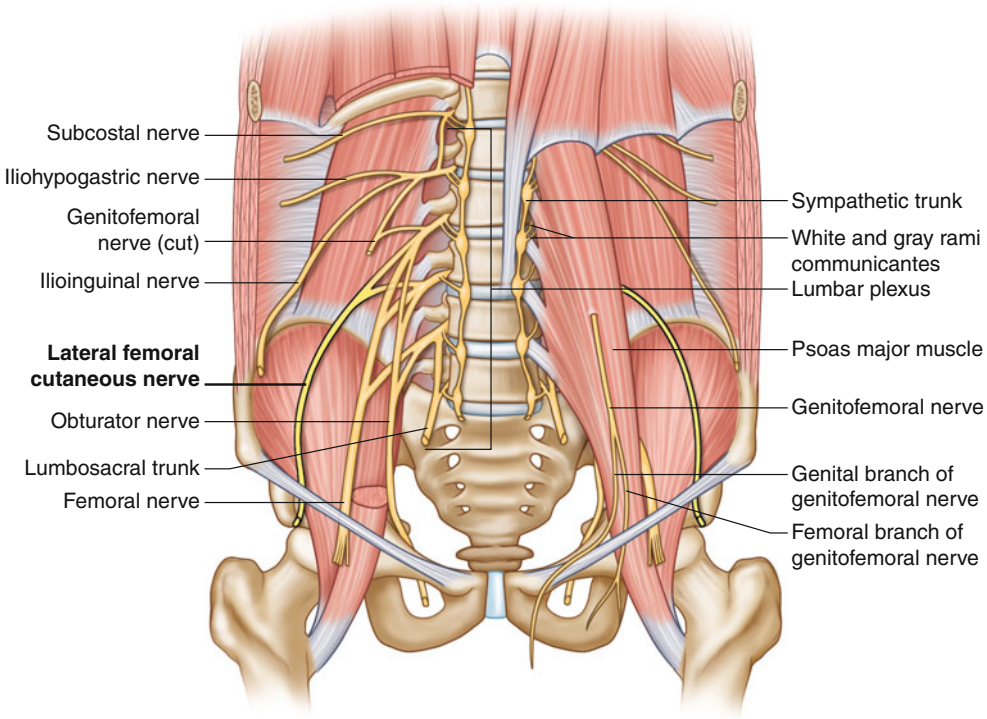

Fig. 61.3 Anatomy of the lumbar plexus, including the lateral femoral cutaneous nerve (Image by Springer)

susceptible to entrapment, compression, and stretching injuries (Fig. 61.7) [27]. This angle increases with movement and hip extension; however, hip flexion and abduction as seen in the lithotomy position does not increase strain on the LFCN [10]. The tendency for entrapment at the path between the pelvis and thigh, as well as in the thigh itself, is exacerbated by obesity, pregnancy, ascites, tight garments, seat belts, braces, direct trauma, leg length changes, scoliosis, and muscle spasm [25].

Many authors have described enlargements of the LFCN proximal to [27] or at [25, 37, 38] the inguinal ligament. The presence of significant enlargement of the nerve (46/90 adult nerves = 51 %, 0/20 fetal nerves) [37] or occasional *pseudoganglions* (also called *pseudoneuromas* [28]) at the level of the inguinal ligament suggests that repetitive trauma results in enlargement of the nerve. A study by Aszmann et al. [28] suggests that the LFCN is most susceptible to mechanical trauma when the nerve takes the path they have classified as type A, B, or C (see Table 61.2); however, MP has been reported in all known variants [28, 39].

Physical Exam

A comprehensive neurologic exam should be conducted on patients with possible entrapment of the LFCN. Typical findings of LFCN dysfunction include sensory changes such as abnormal pinprick and light touch over the anterolateral upper thigh; but because of the variation in the distribution of the LFCN, abnormalities may be present only in the lateral thigh. Standing or lying straight or extending the hip can trigger the pain; it may be alleviated by sitting or by compression of the lateral pelvis. The remainder of the lower extremity neurologic examination, particularly straight leg raising, deep tendon reflexes, and motor strength should be normal. There should also be no evidence of hip, back, or sacroiliac joint abnormality. The most reliable physical finding is tenderness over the ASIS (Fig. 61.8).

Entrapment neuropathies must be distinguished from other mononeuropathies, particularly those due to vasculitis and subsequent ischemia or infarction. Their onset is typically acute, associated with pain, and usually self-limited; often, onset resolves over 6 weeks and may only require symptomatic treatment. Entrapment syndromes, on the other hand, typically start slowly, progress gradually, and persist without intervention [40].

The differential diagnosis of MP requires a high index of suspicion; other pathologies in the abdomen, lower back, pelvis, or hip may mimic LFCN entrapment (Table 61.3), and limb pain may be due to many other peripheral nerve entrapments (Fig. 61.9). It is important to note that MP can be a presenting symptom of more serious underlying conditions due to compression or injury of the LFCN within the pelvis. A pelvic mass [41], chronic appendicitis [23], and hemangiomas [425] have all been

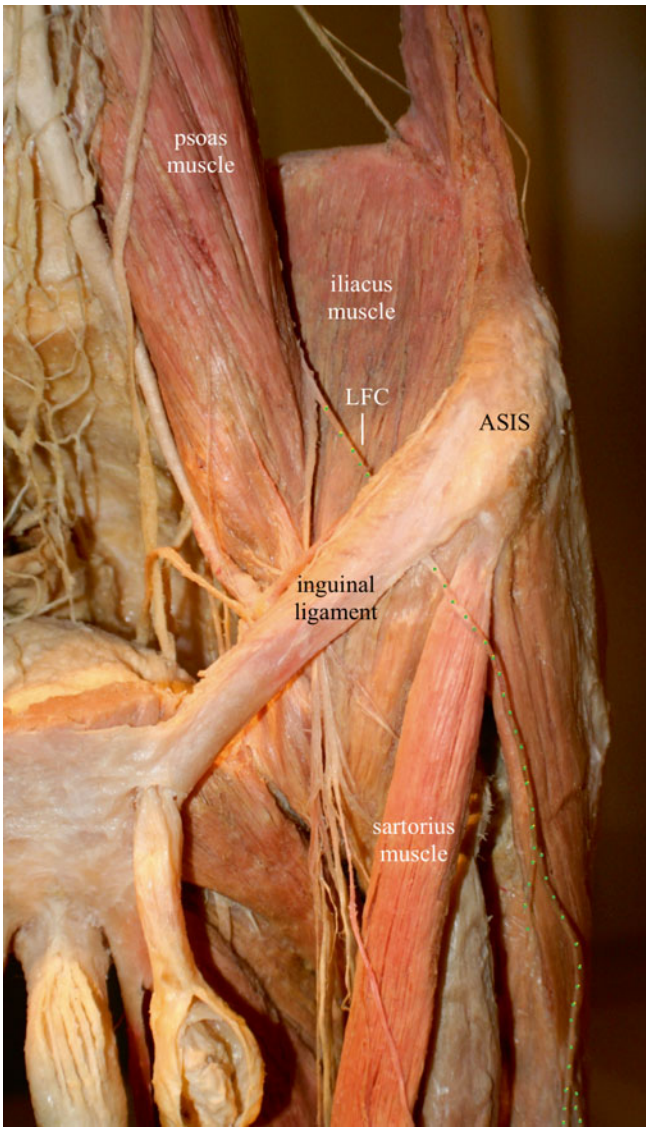

Fig. 61.4 Dissection of the abdomen and upper thigh. *LFC* lateral femoral cutaneous nerve (*highlighted*), *ASIS* anterior superior iliac spine (Modified from an image from *Bodies*, *The Exhibition*, with permission. Image courtesy of Andrea Trescot, MD)

reported to present with pain and dysesthesias in LFCN distribution. MP has also been reported in association with uterine myoma, cecal tumor, retroperitoneal lipofibrosarcoma, periostitis of the ilium, and traction during retroperitoneal procedures [16].

Lumbar radiculopathy can also cause leg numbness; but it is rare that the symptoms or signs are as localized as LFCN entrapment, and back pain is usually present [11]. Pain due to peripheral nerve irritation is usually referred both proximally and distally; therefore, pain in MP may be referred to the gluteal region, resulting in the incorrect diagnosis of lumbar radiculopathy [43].

Diagnostic Tests (Table 61.4)

Plain X-rays of the hip and pelvis are not necessary in patients with the characteristic findings of MP on history and physical examination. Radiographic studies of the lumbar spine should be obtained, however, when the clinical findings are equivocal, in order to exclude spondylolisthesis, spinal stenosis, or disk disease. MRI may be useful in detecting space-occupying lesions in the region of the LFCN (Fig. 61.5) and in identifying peripheral nerve changes in clinically and electrodiagnostically inconclusive cases [44]. The LFCN has a nearly horizontal intrapelvic course along the anterior surface of the iliacus (Fig. 61.10), so it is rarely specifically identified on axial images until it leaves the pelvis [45]. Interestingly, the use of 3-tesla MRI imaging was recently shown to have a 94 % predictive value in diagnosing MP [46].

Sensory nerve conduction studies have been well described for the LFCN [47–50]. However, responses are extremely variable, and it is often difficult to perform this study because of the variation of the location of the LFCN and the physical characteristics of the typical patients (i.e., overweight individuals). Therefore, if a nerve conduction study is to be performed, it is essential that the contralateral side be studied as well. *Somatosensory-evoked potential* (SSEP) recordings of electrical signals on the scalp following LFCN stimulation have been utilized to confirm the diagnosis LFCN of neuropathy [12]. *Electromyography* (EMG) has no role in MP diagnosis, as there are no motor manifestations; however, EMG and nerve conduction studies are occasionally useful to rule out radiculopathy or plexopathy [11].

Because of the difficulty with traditional electrodiagnostic studies, others have verified LFCN entrapment by using non-painful and noninvasive computer-assisted neurosensory testing with the *pressure-specified sensory device* (PSSD). Coert et al. [51] used the PSSD to prospectively compare 24 consecutive patients with clinical signs of MP with 10 normal controls. Patients with LFCN symptoms had significantly decreased skin sensitivity.

Identification and Treatment of Contributing Factors

While most cases of MP are idiopathic, several contributing and risk factors have been identified. Large, retrospective population-based studies have demonstrated an increased risk in patients with a higher mean body mass index (BMI) and in patients with diabetes mellitus. The mean age at MP diagnosis was 50 years, and its frequency is similar for men and women [2].

Additional associations include a large abdomen with overlying panniculus, compression due to tight belts or gar-

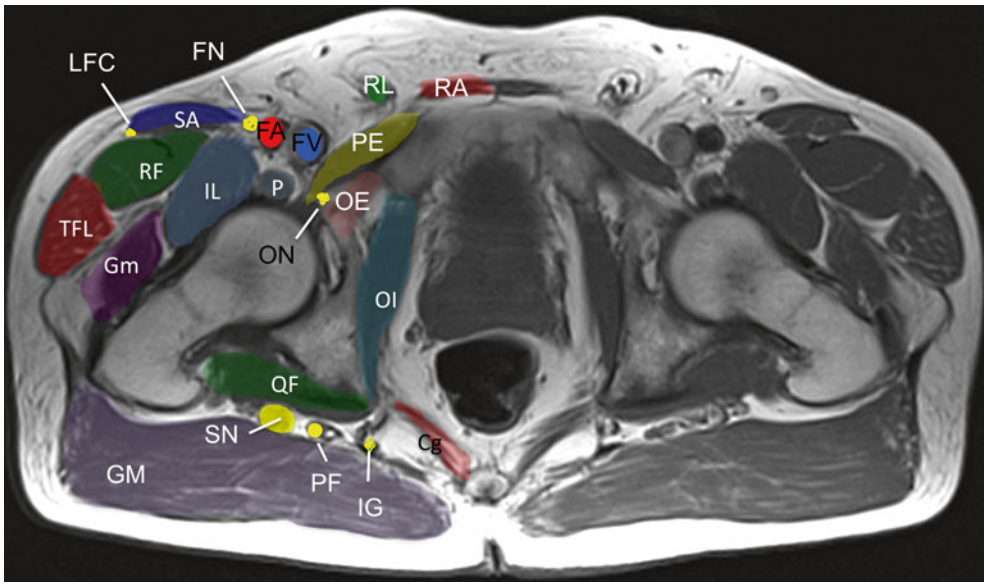

Fig. 61.5 MRI axial image of the pelvis. *Cg* coccygeus muscle, *FA* femoral artery, *FN* femoral nerve, *FV* femoral vein, *GM* gluteus maximus muscle, *Gm* gluteus medius, *IG* inferior gluteal nerve, *IL* iliopsoas muscle, *IT* ischial tuberosity, *ITT* iliotibial tract, *LFC* lateral femoral cutaneous nerve, *OI* obturator internus muscle, *P* psoas muscle, *PE* pectineus muscle, *PF* posterior femoral cutaneous nerve, *QF* quadratus femoris muscle, *RA* rectus abdominis muscle, *RF* rectus femoris muscle, *RL* round ligament, *SA* sartorius muscle, *SN* sciatic nerve, *TFN* tensor fascia lata muscle (Image courtesy of Andrea Trescot, MD)

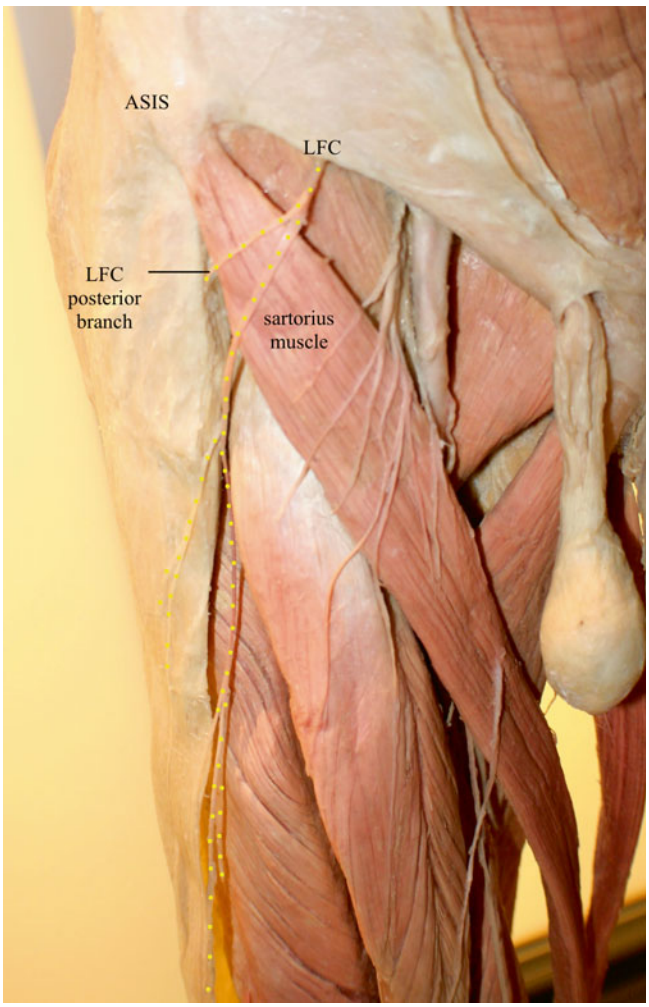

Fig. 61.6 Dissection of the upper thigh. *LFC* lateral femoral cutaneous nerve, *ASIS* anterior superior iliac spine (Modified from an image from *Bodies*, *The Exhibition*, with permission. Image courtesy of Andrea Trescot, MD)

ments around the waist, scar tissue near the lateral aspect of the inguinal ligament, alcohol and lead poisoning, and pregnancy [3, 6, 8, 47]. Trauma (i.e., avulsion fracture of the ASIS, seat belt injuries after a motor vehicle accident), pelvic and retroperitoneal tumors, and leg length discrepancies have also been associated with increased risk of developing MP [52, 53].

A large, retrospective population-based study found that the pain was unchanged with positions including walking or standing [2]. However, others have observed that pain can be aggravated by stretching of the nerve due to prolonged hip and thigh extension, prolonged standing [54], or long-distance walking and cycling, perhaps due to local ischemia during repetitive muscle stretching [24].

Injury during local or regional surgery is another important cause of MP (see Table 61.1). These surgical complications are not trivial, accounting for 17 % of cases in one series of 120 patients [47]. Several causes have been postulated, including direct nerve trauma or injury due to incisions or dissections, harvesting a bone graft, inadequate padding of the posts of the Relton-Hall frame that is often used to support the torso during surgery in the prone position, and excessive traction on the psoas muscle during retroperitoneal dissection [16]. Pennekamp et al. [55] reported a temporary genitofemoral neuralgia but a permanent lesion of the LFCN after the use of alcohol for a lumbar sympathetic denervation.

When contributing factors have been identified, they may be modified with the goal of reducing pressure over the LFCN in the groin. Weight loss, particularly in patients with large abdomens and overlying panniculus, can be helpful, and many pregnant patients see resolution or improvement of their symptoms after delivery. Lifestyle changes such as avoidance of compression due to tight belts or garments

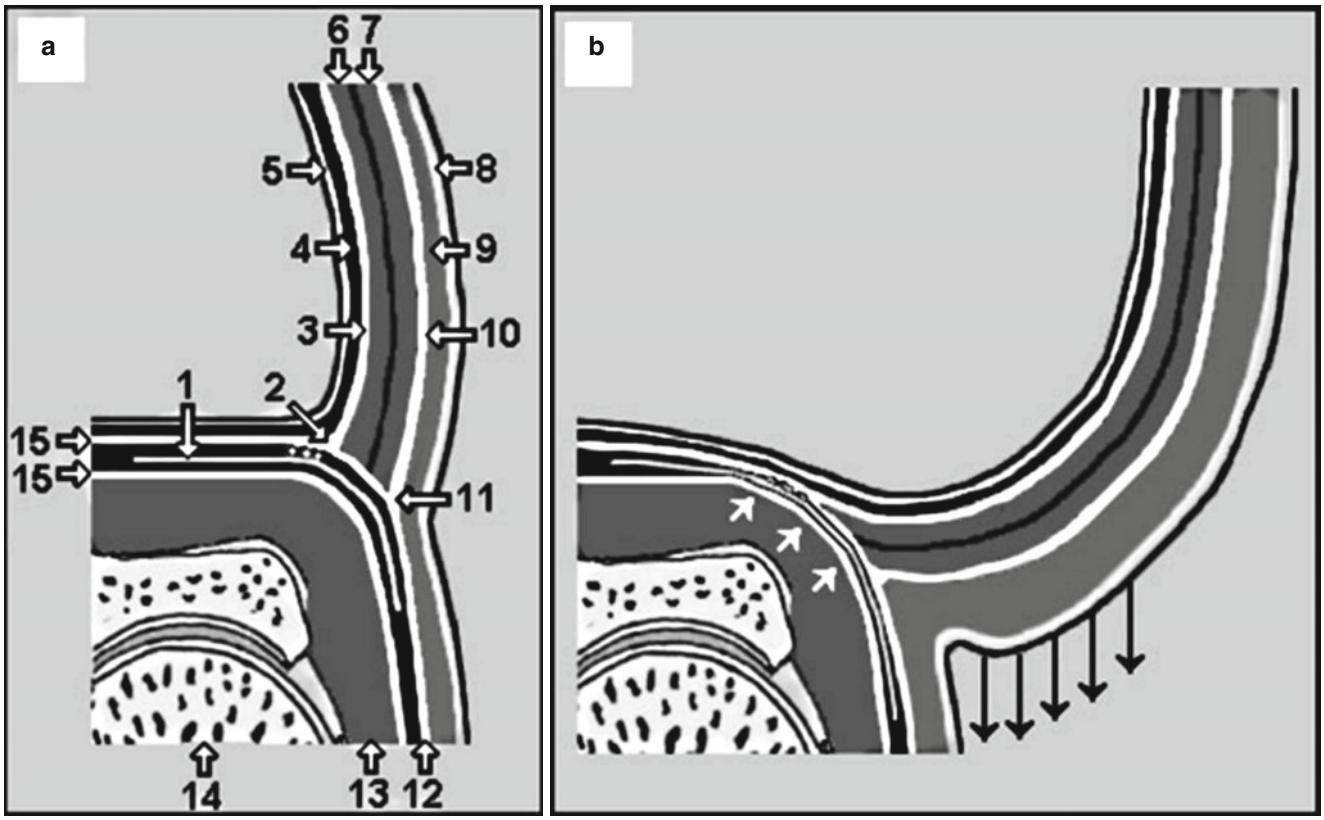

Fig. 61.7 Parasagittal diagram of the potential site of entrapment of the lateral femoral cutaneous nerve, near the anterior superior iliac spine. (**a**) The path of the nerve in a flat abdomen. (**b**) The path of the nerve in a distended abdomen; *black arrows* indicate the downward traction on the nerve, while the *white arrows* show the critical area of nerve compression. *1* Lateral femoral cutaneous nerve; *2* iliopubic tract; *3* transversalis fascia; *4* subserous tissue; *5* parietal peritoneum; *6* transversus abdominis muscle; *7* internal oblique muscle; *8* skin; *9* subcutaneous tissue; *10* aponeurosis of external oblique muscle; *11* inguinal ligament; *12* fascia lata; *13* iliacus muscle; *14* head of femur; *15* laminae of iliac fascia (From Dias et al. [27]. Reprinted with permission from John Wiley and Sons)

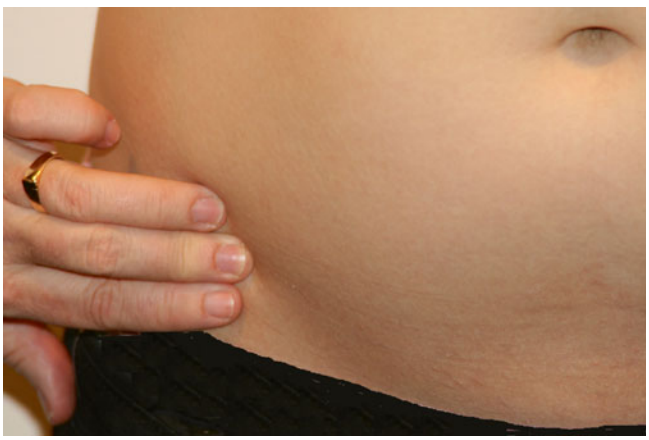

Fig. 61.8 Physical exam showing tenderness at the ASIS (Image courtesy of Andrea Trescot, MD)

around the waist are recommended, as well as correcting metabolic derangements associated with diabetes, alcohol misuse, and lead poisoning. A hiatus from long-distance walking and cycling may also be recommended if it is thought they are contributing to the MP symptoms. If MP has developed due to an intra-abdominal tumor or musculoskeletal pathology, then the goal is to treat those underlying causes.

Most patients achieve satisfactory pain relief from noninterventional treatment modalities and analgesic and antiinflammatory drugs. Other agents for treatment of neuropathic pain, such as tricyclic antidepressants, anticonvulsants, topical local anesthetic patches, or TENS [6], can also be considered. Although it is a benign condition, often with spontaneous remission, recurrence is common [2].

Injection Technique

In patients with symptoms which persist for more than 1–2 months despite conservative measures, interventional therapies may be considered. Relief of pain and paresthesias after injection of a local anesthetic and deposteroid is helpful in establishing the diagnosis, may help restore normal conduction across the nerve by breaking the afferent-efferent

Table 61.3 Differential diagnosis of anterior and lateral thigh pain

	Potential distinguishing features
L2 or L3 radiculopathy [6, 11]	Low back pain, sensory and/or motor changes below the knee; disk herniation visible on spinal MRI or CT
Pelvic or iliac crest mass [41]	History of tumor; palpable mass; positive MRI or CT findings
Chronic appendicitis [23]	RLQ pain; low grade fever; chronic fatigue; abdominal CT or US is diagnostic
Superior gluteal nerve entrapment [9]	Pain from the lateral buttock down to the knee; tender at mid-lateral buttock; no cutaneous sensory findings (see Chap. 53)
Femoral neuropathy [6]	Quadriceps weakness (see Chap. 57)
Hemangioma of pelvis [42]	Imaging (CT, MRI) is diagnostic
Inguinal hernia	Defect of inguinal canal; palpable hernia
Hip joint pain	Pain and crepitus of hip joint; arthritic findings on imaging; pain is usually referred to the medial thigh [21]
Greater trochanter bursitis	Tenderness at lateral hip over great trochanter area; relief after GT bursa local anesthetic injection; pain is usually referred to the lateral thigh [21]

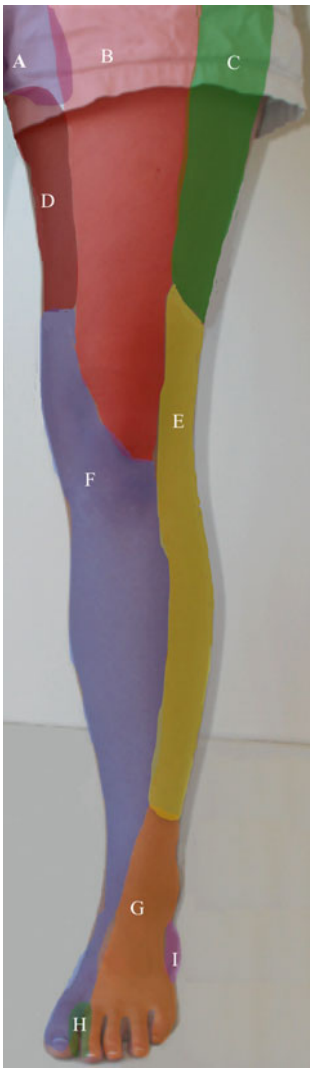

Fig. 61.9 Pattern of pain from anterior lower extremity nerve entrapments. *A* genitofemoral nerve, *B* femoral nerve, *C* lateral femoral cutaneous nerve, *D* obturator, *E* lateral sural cutaneous nerve, *F* saphenous nerve, *G* superficial peroneal nerve, *H* deep peroneal nerve, *I* sural nerve (Image courtesy of Andrea Trescot, MD)

Table 61.4 Diagnostic tests for lateral femoral cutaneous neuralgia

	Potential distinguishing features
Physical exam	Characteristic sensory changes over the anterolateral thigh with no motor findings; tenderness and Tinel's sign adjacent to the ASIS; often the history and physical are sufficient for diagnosis [6]
Provocative maneuvers	Hip extension [6]
Diagnostic injection	Pain relief from LFCN nerve block [25]
Ultrasound	Assists in localizing LFCN for diagnosis and therapeutic nerve block [8]
MRI	Important to exclude other diseases
Arteriography	Not useful
X-ray	Exclude other disease
Electrodiagnostic studies	EMG may rule out other diseases

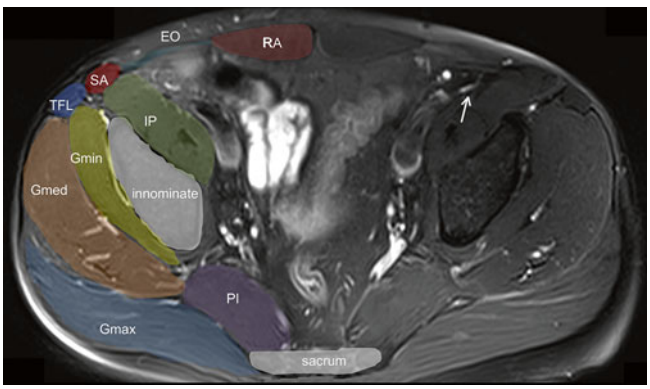

Fig. 61.10 MRI showing the lateral femoral cutaneous nerve at the iliacus muscle (*white arrow*). *EO* external oblique muscle, *Gmax* gluteus maximus muscle, *Gmed* gluteus medius muscle, *Gmin* gluteus minimus, *IP* iliopsoas muscle, *PI* piriformis muscle, *RA* rectus abdominus muscle, *SA* sartoris muscle, *TFL* tensor fascia lata muscle. (Image courtesy of Andrea Trescot, MD)

neuronal loop at the spinal segmental level, and will allow the patient to experience the anticipated results of a neurolytic procedure [56]. However, if there is no improvement of symptoms after the injection and clinical suspicion remains high, a more proximal LFCN irritation or compression site should be sought [57].

There are several injection techniques available, including the traditional landmark-guided, fluoroscopic-guided, and ultrasound-guided methods with or without the use of a nerve stimulator.

Landmark-Guided Technique

The conventional technique employs a fanned-out injection through the fascia lata and along the inguinal ligament without eliciting a paresthesia [58]. The *fascia iliaca compartment block* attempts to address the anatomical variability of the LFCN by relying on a field approach. The target point is identified approximately 2–3 cm inferior to and 2–3 cm medial to the ASIS (Fig. 61.11), and a skin wheal is made with local anesthetic. Then, a short bevel 22-gauge, 4 cm needle with a syringe attached is inserted perpendicularly and advanced until a release (a "pop") occurs, signifying passage through the fascia lata. After negative aspiration, two-thirds of the injectate is injected, and then the needle is withdrawn slightly above the fascia lata, where the other one-third of the solution is injected in a fan-like manner medially and laterally. Conventional injection is effective in most cases, likely due to the diffusion of the local anesthetic through the fascia layer; however, failure rates have been reported at up to 60 %, and slow onset has been noted [59, 60].

The success rate of an LFCN block can be increased to 85 % by using a peripheral nerve stimulator; however, this technique depends on patient factors, may be more time-consuming, and may cause more patient discomfort [60]. Reports recommend first identifying the likely location of the LFCN below the inguinal ligament, using a transdermal nerve stimulator to maximize the paresthesia. An uninsulated needle is then attached to a nerve stimulator, inserted at the marked location, and advanced until a paresthesia over the lateral thigh is demonstrated with a current <0.6 mA. The injection is then given after a negative aspiration.

Nonetheless, a landmark-guided technique has the disadvantage of using more local anesthetic than an ultrasound-guided injection, and there have been several reports of obtaining a "3-in-1 block" after injection of about 20 mL of local anesthetic with resultant blockade of the obturator, femoral, and LFC nerves [58]. It is therefore recommended to use no more than 10 mL of solution and to examine the patient for evidence of femoral and obturator nerve blockade prior to discharge [58]. A landmark-guided technique has additional disadvantages including the potential for needle trauma causing nerve damage, as well as block failure.

Fluoroscopic-Guided Technique

Fluoroscopic guidance is typically unnecessary for this procedure, as the ASIS is usually palpable, and it is difficult to target the LFCN because of its anatomical variations. The ASIS can be identified by fluoroscopy when the body habitus precludes palpation (Fig. 61.12). There are reports of pulsed

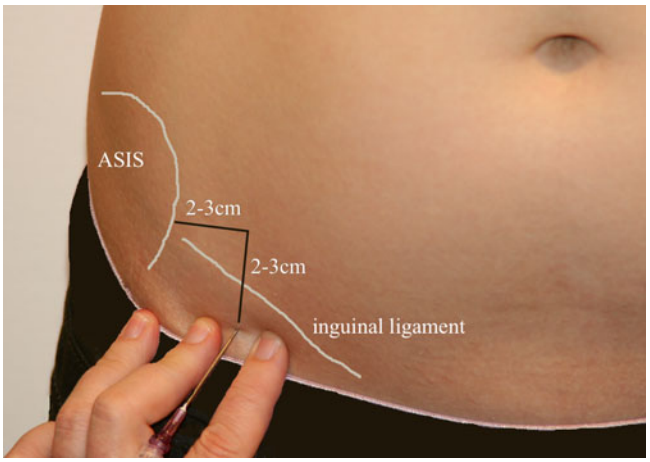

Fig. 61.11 Landmark-directed lateral femoral cutaneous nerve injection (Image courtesy of Andrea Trescot, MD)

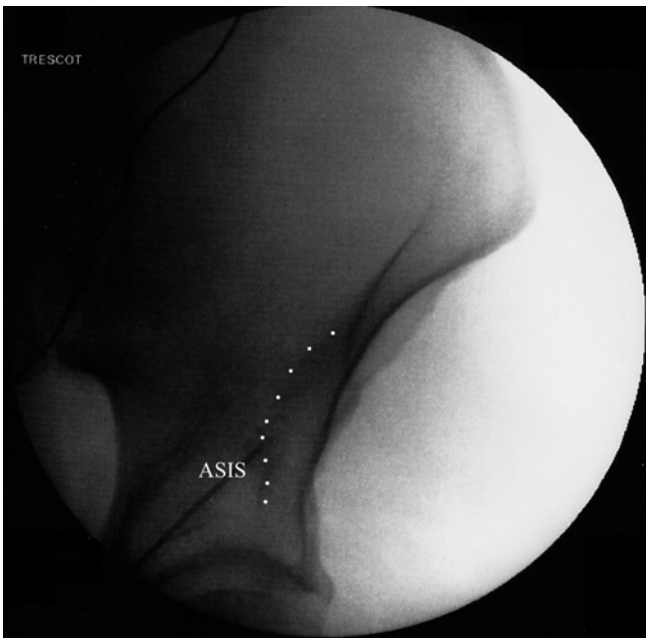

Fig. 61.12 Fluoroscopic landmarks for lateral femoral cutaneous nerve injections (Image courtesy of Andrea Trescot, MD)

radiofrequency (see *Neurolytic*s below) using fluoroscopy to locate the target approximately 1 cm medial and cephalad to the ASIS or inferior to the ASIS and under the inguinal ligament. The needle is then advanced to obtain concordant sensory stimulation [61, 62].

Ultrasound-Guided Technique (US)

Ultrasound can be used to identify the variable position of the LFCN in relation to the ASIS [63] and evaluate the LFCN both at the inguinal ligament and distally. Palpate the ASIS with the patient supine. Because the nerve is very superficial (underneath the fascia lata and sometimes above the sartorius), a high frequency linear array ultrasound probe is necessary to identify the structures. The lateral end of the probe is placed on the ASIS, and the medial end of the probe is angled in a slightly caudal direction, so the transducer is parallel with the inguinal ligament [64]. The ASIS is visualized as a hyperechoic structure with posterior acoustic shadowing. The probe is then moved inferior to the ASIS at the insertion site of the sartorius muscle on the ASIS and swept medially and inferiorly below the inguinal ligament until two continuous hyperechoic lines are seen under the subcutaneous tissue (the fascia lata and the fascia iliaca, which are typically about 0.5–1 cm apart). The LFCN is seen in cross section in the space between the two fascial layers, appearing as a hyperechoic, elliptical fibrillar structure described as having the appearance of an "eye" (Fig. 61.13).

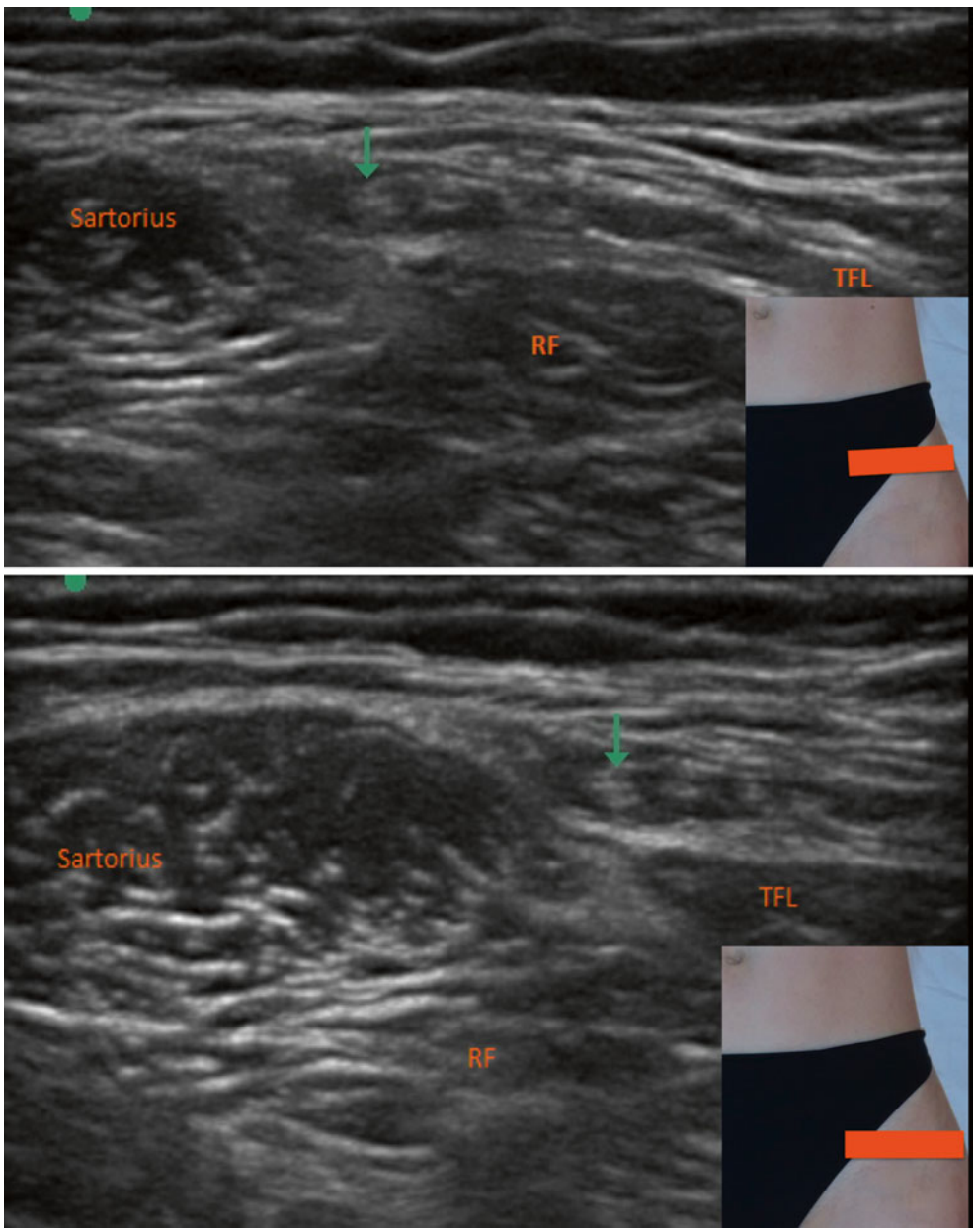

Fig. 61.13 Ultrasound evaluation of the lateral femoral cutaneous nerve. Scanning distally from the ASIS, the sartorius muscle tendon turns into the sartorius muscle belly. Lateral to the sartorius, the lateral femoral cutaneous nerve (*green arrow*) is visualized as a hyperechoic structure. *RF* rectus femoris muscle, *TFL* tensor fasciae latae muscle (Image courtesy of Agnes Stogicza, MD)

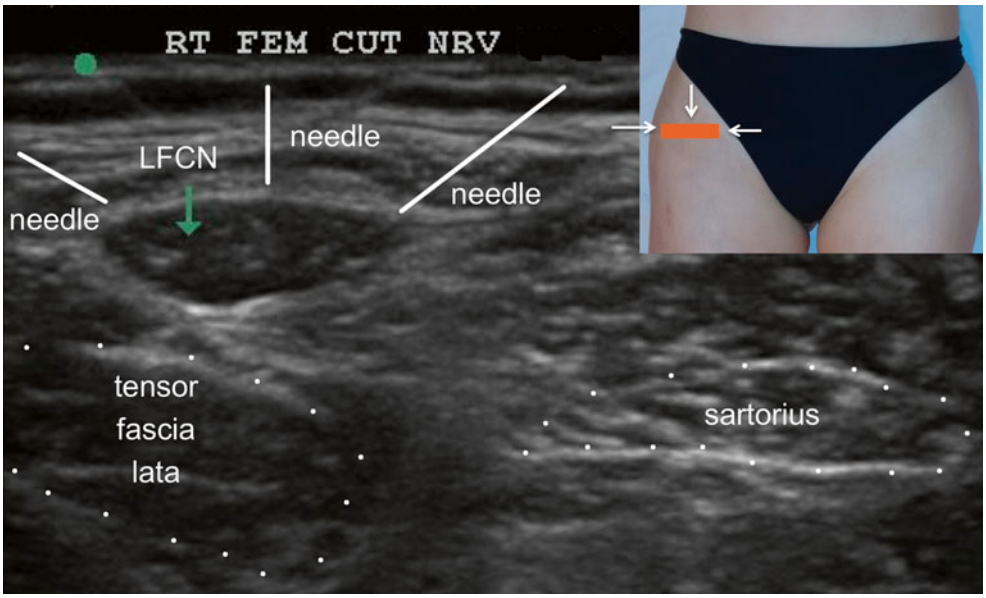

Fig. 61.14 Ultrasound localization of the lateral femoral cutaneous nerve with simulated needle for an in-plane (lateral to medial, medial to lateral) and out-of-plane injection. *LFCN* lateral femoral cutaneous nerve (Image courtesy of Andrea Trescot, MD)

Alternatively, the medial end of the ultrasound probe may be angled slightly caudal so the transducer is parallel to the inguinal ligament (Fig. 61.14). In this position, the LFCN in cross section looks like an oval structure on short-axis images and tubular on longitudinal ones [64]. Trace the course of the nerve by scanning it proximally and distally to confirm its appropriate course toward the lateral thigh; then, follow the nerve proximally to perform the injection at the most proximal location possible [65]. The needle is advanced medially to laterally in-plane; hydrodissection between the fascia lata and fascia iliaca with dextrose 5 % can be utilized to better visualize the nerve [65]. Advance the needle under real-time ultrasound, targeting the nerve by an in-plane or out-of-plane approach, depending on the position of the LFCN relative to the ASIS [66, 67].

Ultrasound guidance has several advantages. It overcomes the anatomical variability of the LFCN by allowing direct visualization of the nerve, and it enables the user to trace the nerve from the ASIS down to the inguinal region, in order to identify the best possible transverse view. Moreover, it allows for visual guidance of the needle to the target and confirmation of the deposition of local anesthetic around the nerve [28, 31, 63]. Recent studies also showed that the use of ultrasound reduces patient discomfort [68]. Ultrasound also reduces the amount of local anesthetic agent required, which can reduce the risk of systemic toxicity, as well as the risk of unintended blockage of the femoral and obturator nerves, or a "3-in-1" block [58]. The disadvantages of ultrasound are that it is more time-consuming, requires a trained technician, and is more costly compared to landmark-guided injections.

Neurolytic Technique

A small proportion of patients demonstrate intractable pain refractory to noninterventional therapies who note only temporary relief from repeat injections of local anesthetic with or without steroid, so that more aggressive interventions are considered [25].

These potential treatment modalities included cryoneurolysis, radiofrequency lesioning, chemical or surgical neurolysis, peripheral nerve or spinal cord stimulation, and surgical decompression or nerve transaction. Any of the neurolytic techniques (other than cryoneuroablation) and surgical nerve transection, however, have significant potential adverse side effects of persistent numbness and anesthesia dolorosa. Moreover, because of the anatomical variations in the location of the LFCN and the proximity to other vital structures including the femoral and obturator nerves, there may be an increased risk of collateral damage if neuroablation is performed using radiofrequency or chemical neurolysis [69].

Cryoneuroablation

Denervation of the LFCN with *cryoneuroablation* was described by Trescot [70]. Using the built-in nerve stimulator and US guidance can increase the success of the

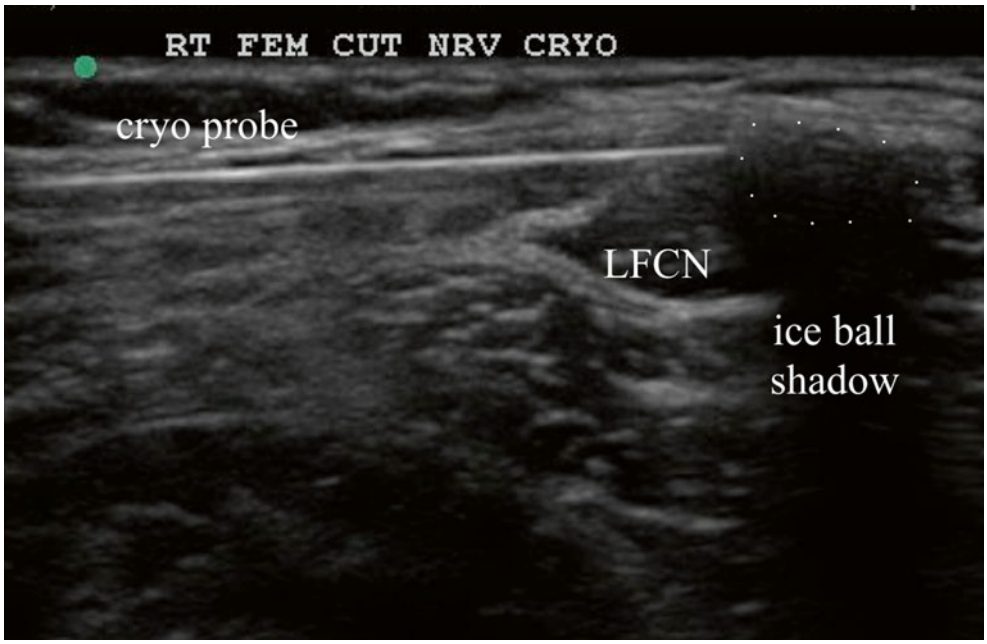

Fig. 61.15 Cryoneuroablation of the lateral femoral cutaneous nerve. *LFCN* lateral femoral cutaneous nerve (Image courtesy of Agnes Stogicza, MD)

procedure. A 12-gauge introducer is advanced in either a medial to lateral, lateral to medial, or cephalad to caudad direction toward the visualized nerve under US, and the probe advanced through the catheter. The nerve stimulator and US are used to find the nerve, and two freeze cycles are used. Under US, the iceball itself is easily visualized (Fig. 61.15).

Radiofrequency Lesioning

In a few case reports, patients with refractory symptoms noted improvement after pulsed radiofrequency nerve ablation of the LFCN [61, 62, 71]. *Pulsed radiofrequency* (PRF) has been used successfully for the treatment of many other neuropathic pain syndromes, including mono-radiculopathies, trigeminal neuralgia, postherpetic neuralgia, phantom limb pain, chronic postsurgical thoracic pain, and peripheral neuropathies [72–78]. As a minimally invasive and relatively nontraumatic procedure, PRF does not destroy the nerve or impair function; rather, it modulates the way the nerve fires. Its use, therefore, may be recommended prior to other neuroablative techniques as an effective, low-risk treatment option in patients refractory to medical management who are unwilling or unfit to undergo surgery. Further data, particularly prospective randomized controlled trials, are needed to determine the full benefit of this procedure for treating MP.

Spinal and Peripheral Nerve Stimulation

Another option is implantation of a spinal cord or peripheral nerve stimulator. These techniques have advantages over other surgical options, as they are not destructive, can be trialed first with a temporary device to predict success, are unlikely to worsen the pain, and can be removed without significant permanent adverse effects [79]. The spinal cord stimulator lead tip is placed at the mid-T10 level (Fig. 61.16) [79], while peripheral nerve stimulator leads are placed based on tissue mapping and/or direct nerve stimulation with US guidance [80].

Surgery

Rarely, open neurosurgical procedures are necessary in patients with severe chronic symptoms that are refractory to other measures, but there is no consensus as to the best approach [81]. One proposed algorithm for surgical treatment is as follows:

- Adults with less than 1 year of symptoms and all pediatric patients should undergo simple decompression.
- If these patients have persistent or recurrent symptoms, they should be considered for resection.
- Adult patients with symptoms present more than 1 year should be considered for primary resection [26].

Decompression of the nerve, by sectioning the inferior slip of the attachment of the inguinal ligament to the ASIS, may provide long lasting relief in some patients while preserving sensory function. However, it is not uniformly successful and may lead to recurrence.

Therefore, the definitive surgical procedure is neurolysis of the LFCN as it exits the pelvis with transposition, and it remains the most common surgical procedure performed for this condition [25]. This, however, has the disadvantage of permanent anesthesia and risk of anesthesia dolorosa.

Complications

Blocking the LFCN is very safe; however, there is a risk of bleeding/hematoma, infection, injury to surrounding structures including vessels and nerves (i.e., femoral, obturator), spread of medication that blocks or destroys nearby nerves, and failure to relieve pain. After neuroablative or surgical procedures, there is also a risk of permanent anesthesia, recurrence, or worsening pain.

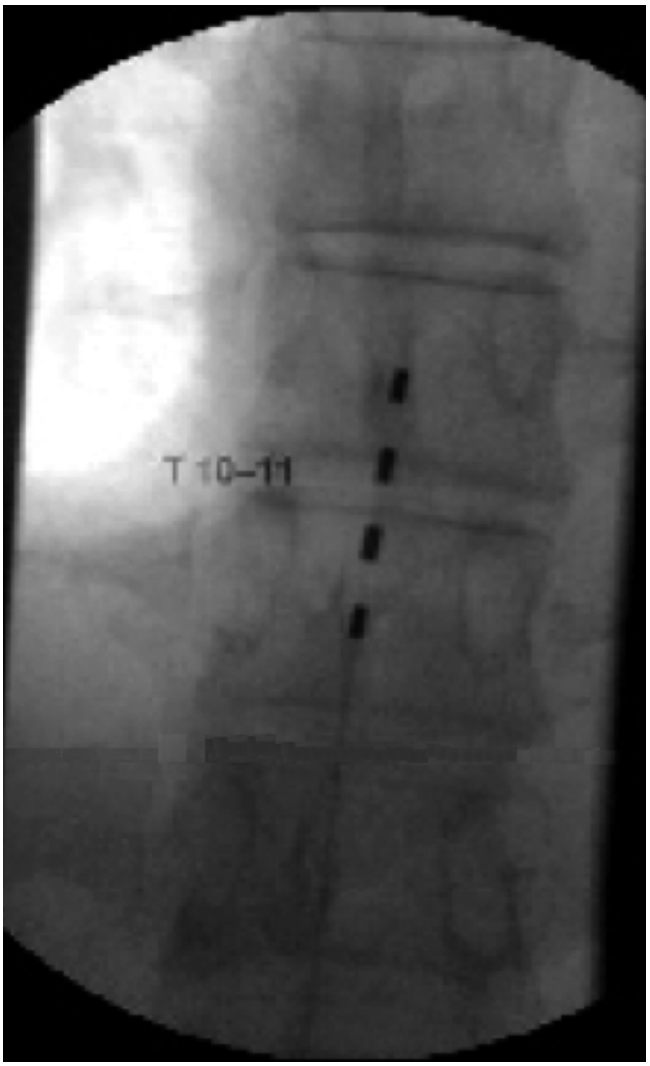

Fig. 61.16 Spinal cord stimulation for meralgia paresthetica, with the distal tip of the lead at T10 (From Barna et al. [79]. Reprinted with permission from American Society of Interventional Pain Physicians)

Summary

The lateral femoral cutaneous nerve (LFCN) has a variable course and presentation. Although the presentation may sometimes be classic, a high index of suspicion is necessary to diagnose and treat LFCN entrapment and meralgia paresthetica (MP). The strong association of MP and diabetes mellitus (DM) underlines the importance of ongoing screening and lifestyle changes in patients with MP to mitigate subsequent development of DM.

References

1. Hager W. Neualgia femoria. Resection des nervous cutaneous femoris anterior externus. Heilung Disch Med Wochenschr. 1885;11:218.
2. Parisi TJ, Mandrekar J, Dyck PJ, Klein CJ. Meralgia paresthetica: relation to obesity, advanced age, and diabetes mellitus. Neurology. 2011;77(16):1538–42.
3. van Slobbe AM, Bohnen AM, Bernsen RM, Koes BW, Bierma-Zeinstra SM. Incidence rates and determinants in meralgia paresthetica in general practice. J Neurol. 2004;251(3):294–7.
4. Kitchen C, Simpson J. Meralgia paresthetica. A review of 67 patients. Acta Neurol Scand. 1972;48(5):547–55.
5. Reichert FL. Meralgia paresthetica; a form of causalgia relieved by interruption of the sympathetic fibers. Surg Clin N Am. 1933;13:1443.
6. Van Diver T, Camann W. Meralgia paresthetica in the parturient. Int J Obstet Anesth. 1995;4(2):109–12.
7. Deal CL, Canoso JJ. Meralgia paresthetica and large abdomens. Ann Intern Med. 1982;96(6 Pt 1):787–8.
8. Park JW, Kim DH, Hwang M, Bun HR. Meralgia paresthetica caused by hip-huggers in a patient with aberrant course of the lateral femoral cutaneous nerve. Muscle Nerve. 2007;35(5):678–80.
9. Kopell HP, Thompson WAL. Peripheral entrapment neuropathies. 2nd ed. Malabar, FL: R. E. Krieger Pub. Co; 1976.
10. Litwiller JP, Wells Jr RE, Halliwill JR, Carmichael SW, Warner MA. Effect of lithotomy positions on strain of the obturator and lateral femoral cutaneous nerves. Clin Anat. 2004;17(1):45–9.
11. Trummer M, Flaschka G, Unger F, Eustacchio S. Lumbar disc herniation mimicking meralgia paresthetica: case report. Surg Neurol. 2000;54(1):80–1.
12. Amoiridis G, Wohrle J, Grunwald I, Przuntek H. Malignant tumour of the psoas: another cause of meralgia paraesthetica. Electromyogr Clin Neurophysiol. 1993;33(2):109–12.
13. Yamout B, Tayyim A, Farhat W. Meralgia paresthetica as a complication of laparoscopic cholecystectomy. Clin Neurol Neurosurg. 1994;96(2):143–4.
14. Dibenedetto LM, Lei Q, Gilroy AM, Hermey DC, Marks Jr SC, Page DW. Variations in the inferior pelvic pathway of the lateral femoral cutaneous nerve: implications for laparoscopic hernia repair. Clin Anat. 1996;9(4):232–6.
15. Broin EO, Horner C, Mealy K, Kerin MJ, Gillen P, O'Brien M, et al. Meralgia paraesthetica following laparoscopic inguinal hernia repair. An anatomical analysis. Surg Endosc. 1995;9(1):76–8.

16. Mirovsky Y, Neuwirth M. Injuries to the lateral femoral cutaneous nerve during spine surgery. Spine (Phila Pa 1976). 2000;25(10):1266–9.
17. Massey EW. Meralgia paresthetica secondary to trauma of bone graft. J Trauma. 1980;20(4):342–3.
18. Yamamoto T, Nagira K, Kurosaka M. Meralgia paresthetica occurring 40 years after iliac bone graft harvesting: case report. Neurosurgery. 2001;49(6):1455–7.
19. Feigl GC, Dreu M, Ulz H, Breschan C, Maier C, Likar R. Susceptibility of the genitofemoral and lateral femoral cutaneous nerves to complications from lumbar sympathetic blocks: is there a morphological reason? Br J Anaesth. 2014;112(6):1098–104.
20. Butler R, Webster MW. Meralgia paresthetica: an unusual complication of cardiac catheterization via the femoral artery. Cathet Cardiovasc Interv Off J Soc Card Angiography Interv. 2002;56(1):69–71.
21. Dellon AL, Mont M, Ducic I. Involvement of the lateral femoral cutaneous nerve as source of persistent pain after total hip arthroplasty. J Arthroplasty. 2008;23(3):480–5.
22. Hospodar PP, Ashman ES, Traub JA. Anatomic study of the lateral femoral cutaneous nerve with respect to the ilioinguinal surgical dissection. J Orthop Trauma. 1999;13(1):17–9.
23. Ghavanini MR, Ghavanini AA. Meralgia paresthetica as the presenting feature of chronic appendicitis. Am J Phys Med Rehabil. 2001;80(9):703–5.
24. Kho KH, Blijham PJ, Zwarts MJ. Meralgia paresthetica after strenuous exercise. Muscle Nerve. 2005;31(6):761–3.
25. Williams PH, Trzil KP. Management of meralgia paresthetica. J Neurosurg. 1991;74(1):76–80.
26. Ivins GK. Meralgia paresthetica, the elusive diagnosis: clinical experience with 14 adult patients. Ann Surg. 2000;232(2):281–6.
27. Dias Filho LC, Valença MM, Guimarães Filho FA, Medeiros RC, Silva RA, Morais MG, et al. Lateral femoral cutaneous neuralgia: an anatomical insight. Clin Anat. 2003;16(4):309–16.
28. Aszmann OC, Dellon ES, Dellon AL. Anatomical course of the lateral femoral cutaneous nerve and its susceptibility to compression and injury. Plast Reconstr Surg. 1997;100(3):600–4.
29. Keegan JJ, Holyoke EA. Meralgia paresthetica. An anatomical and surgical study. J Neurosurg. 1962;19:341–5.
30. Majkrzak A, Johnston J, Kacey D, Zeller J. Variability of the lateral femoral cutaneous nerve: an anatomic basis for planning safe surgical approaches. Clin Anat. 2010;23(3):304–11.
31. Grothaus MC, Holt M, Mekhail AO, Ebraheim NA, Yeasting RA. Lateral femoral cutaneous nerve: an anatomic study. Clin Orthop Relat Res. 2005;437:164–8.
32. Carai A, Fenu G, Sechi E, Crotti FM, Montella A. Anatomical variability of the lateral femoral cutaneous nerve: findings from a surgical series. Clin Anat. 2009;22(3):365–70.
33. Hopkins PM, Ellis FR, Halsall PJ. Evaluation of local anaesthetic blockade of the lateral femoral cutaneous nerve. Anaesthesia. 1991;46(2):95–6.
34. Enneking FK, Chan V, Greger J, Hadzic A, Lang SA, Horlocker TT. Lower-extremity peripheral nerve blockade: essentials of our current understanding. Reg Anesth Pain Med. 2005;30(1):4–35.
35. Ghent WR. Further studies on meralgia paresthetica. Can Med Assoc J. 1961;85:871–5.
36. Ghent WR. Meralgia paraesthetica. Can Med Assoc J. 1959;81:631–3.
37. Edelson JG, Nathan H. Meralgia paresthetica. An anatomical interpretation. Clin Orthop Relat Res. 1977;122:255–62.
38. Nathan H. Gangliform enlargement on the lateral cutaneous nerve of the thigh. Its significance in the understanding of the etiology of meralgia paresthetica. J Neurosurg. 1960;17:843–50.
39. Jefferson D, Eames RA. Subclinical entrapment of the lateral femoral cutaneous nerve: an autopsy study. Muscle Nerve. 1979;2(2):145–54.
40. Vinik A, Mehrabyan A, Colen L, Boulton A. Focal entrapment neuropathies in diabetes. Diabetes Care. 2004;27(7):1783–8.
41. Suber DA, Massey EW. Pelvic mass presenting as meralgia paresthetica. Obstet Gynecol. 1979;53(2):257–8.
42. Yamamoto T, Kurosaka M, Marui T, Mizuno K. Hemangiomatosis presenting as meralgia paresthetica. J Pediatr Surg. 2001;36(3):518–20.
43. Jones RK. Meralgia paresthetica as a cause of leg discomfort. Can Med Assoc J. 1974;111(6):541–2.
44. Kim S, Choi JY, Huh YM, Song HT, Lee SA, Kim SM, et al. Role of magnetic resonance imaging in entrapment and compressive neuropathy – what, where, and how to see the peripheral nerves on the musculoskeletal magnetic resonance image: part 1. Overview and lower extremity. Eur Radiol. 2007;17(1):139–49.
45. Petchprapa CN, Rosenberg ZS, Sconfienza LM, Cavalcanti CFA, Vieira RLR, Zember JS. MR imaging of entrapment neuropathies of the lower extremity. RadioGraphics. 2010;30(4):983–1000.
46. Chhabra A, Del Grande F, Soldatos T, Chalian M, Belzberg AJ, Williams EH, et al. Meralgia paresthetica: 3-Tesla magnetic resonance neurography. Skelet Radiol. 2013;42(6):803–8.
47. Seror P, Seror R. Meralgia paresthetica: clinical and electrophysiological diagnosis in 120 cases. Muscle Nerve. 2006;33(5):650–4.
48. Shin YB, Park JH, Kwon DR, Park BK. Variability in conduction of the lateral femoral cutaneous nerve. Muscle Nerve. 2006;33(5):645–9.
49. Deimel GW, Hurst RW, Sorenson EJ, Boon AJ. Utility of ultrasound-guided near-nerve needle recording for lateral femoral cutaneous sensory nerve conduction study: does it increase reliability compared with surface recording? Muscle Nerve. 2013;47(2):274–6.
50. Boon AJ, Bailey PW, Smith J, Sorenson EJ, Harper CM, Hurdle MF. Utility of ultrasound-guided surface electrode placement in lateral femoral cutaneous nerve conduction studies. Muscle Nerve. 2011;44(4):525–30.
51. Coert JH, Dellon AL. Documenting neuropathy of the lateral femoral cutaneous nerve using the pressure-specified sensory testing device. Ann Plast Surg. 2003;50(4):373–7.
52. Nahabedian MY, Dellon AL. Meralgia paresthetica: etiology, diagnosis, and outcome of surgical decompression. Ann Plast Surg. 1995;35(6):590–4.
53. van Eerten PV, Polder TW, Broere CA. Operative treatment of meralgia paresthetica: transection versus neurolysis. Neurosurgery. 1995;37(1):63–5.
54. Harney D, Patijn J. Meralgia paresthetica: diagnosis and management strategies. Pain Med. 2007;8(8):669–77.
55. Pennekamp W, Krumova EK, Feigl GP, Frombach E, Nicolas V, Schwarzer A, et al. Permanent lesion of the lateral femoral cutaneous nerve after low-volume ethanol 96% application on the lumbar sympathetic chain. Pain Physician. 2013;16(4):391–7.
56. Dureja GP, Gulaya V, Jayalakshmi TS, Mandal P. Management of meralgia paresthetica: a multimodality regimen. Anesth Analg. 1995;80(5):1060–1.
57. Grossman MG, Ducey SA, Nadler SS, Levy AS. Meralgia paresthetica: diagnosis and treatment. J Am Acad Orthop Surg. 2001;9(5):336–44.
58. Sharrock NE. Inadvertent "3-in-1 block" following injection of the lateral cutaneous nerve of the thigh. Anesth Analg. 1980;59(11):887–8.
59. Capdevila X, Biboulet P, Bouregba M, Barthelet Y, Rubenovitch J, d'Athis F. Comparison of the three-in-one and fascia iliaca compartment blocks in adults: clinical and radiographic analysis. Anesth Analg. 1998;86(5):1039–44.
60. Shannon J, Lang SA, Yip RW, Gerard M. Lateral femoral cutaneous nerve block revisited. A nerve stimulator technique. Reg Anesth. 1995;20(2):100–4.

61. Choi HJ, Choi SK, Kim TS, Lim YJ. Pulsed radiofrequency neuromodulation treatment on the lateral femoral cutaneous nerve for the treatment of meralgia paresthetica. J Korean Neurosurg Soc. 2011;50(2):151–3.
62. Philip CN, Candido KD, Joseph NJ, Crystal GJ. Successful treatment of meralgia paresthetica with pulsed radiofrequency of the lateral femoral cutaneous nerve. Pain Physician. 2009;12(5):881–5.
63. Hurdle MF, Weingarten TN, Crisostomo RA, Psimos C, Smith J. Ultrasound-guided blockade of the lateral femoral cutaneous nerve: technical description and review of 10 cases. Arch Phys Med Rehabil. 2007;88(10):1362–4.
64. Tagliafico A, Serafini G, Lacelli F, Perrone N, Valsania V, Martinoli C. Ultrasound-guided treatment of meralgia paresthetica (lateral femoral cutaneous neuropathy): technical description and results of treatment in 20 consecutive patients. J Ultrasound Med. 2011;30(10):1341–6.
65. Soneji N, Peng PW. Ultrasound-guided pain interventions – a review of techniques for peripheral nerves. Korean J Pain. 2013;26(2):111–24.
66. Ng I, Vaghadia H, Choi PT, Helmy N. Ultrasound imaging accurately identifies the lateral femoral cutaneous nerve. Anesth Analg. 2008;107(3):1070–4.
67. Kim JE, Lee SG, Kim EJ, Min BW, Ban JS, Lee JH. Ultrasound-guided lateral femoral cutaneous nerve block in meralgia paresthetica. Korean J Pain. 2011;24(2):115–8.
68. Tagliafico A, Bodner G, Rosenberg I, Palmieri F, Garello I, Altafini L, et al. Peripheral nerves: ultrasound-guided interventional procedures. Semin Musculoskelet Radiol. 2010;14(5):559–66.
69. Khalil N, Nicotra A, Rakowicz W. Treatment for meralgia paraesthetica. Cochrane Database Syst Rev. 2012;12:Cd004159.
70. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
71. del Rey ML, Canuel JF, Skouvakis D. Successful treatment of bilateral meralgia paresthetica using pulsed radiofrequency. NYNJSIPP annual meeting; Jersey City; 2014.
72. Wu H, Groner J. Pulsed radiofrequency treatment of articular branches of the obturator and femoral nerves for management of hip joint pain. Pain Pract. 2007;7(4):341–4.
73. Abejon D, Garcia-del-Valle S, Fuentes ML, Gomez-Arnau JI, Reig E, van Zundert J. Pulsed radiofrequency in lumbar radicular pain: clinical effects in various etiological groups. Pain Pract. 2007;7(1):21–6.
74. Balogh SE. Transcutaneous application of pulsed radiofrequency: four case reports. Pain Pract. 2004;4(4):310–3.
75. Van Zundert J, Brabant S, Van de Kelft E, Vercruyssen A, Van Buyten JP. Pulsed radiofrequency treatment of the Gasserian ganglion in patients with idiopathic trigeminal neuralgia. Pain. 2003;104(3):449–52.
76. Kim YH, Lee CJ, Lee SC, Huh J, Nahm FS, Kim HZ, et al. Effect of pulsed radiofrequency for postherpetic neuralgia. Acta Anaesthesiol Scand. 2008;52(8):1140–3.
77. Wilkes D, Ganceres N, Solanki D, Hayes M. Pulsed radiofrequency treatment of lower extremity phantom limb pain. Clin J Pain. 2008;24(8):736–9.
78. Cohen SP, Sireci A, Wu CL, Larkin TM, Williams KA, Hurley RW. Pulsed radiofrequency of the dorsal root ganglia is superior to pharmacotherapy or pulsed radiofrequency of the intercostal nerves in the treatment of chronic postsurgical thoracic pain. Pain Physician. 2006;9(3):227–35.
79. Barna SA, Hu MM, Buxo C, Trella J, Cosgrove GR. Spinal cord stimulation for treatment of meralgia paresthetica. Pain Physician. 2005;8(3):315–8.
80. Slavin KV. Peripheral nerve stimulation. Lunsford LD, editor. Pittsburgh: Karger; 2011.
81. de Ruiter GC, Wurzer JA, Kloet A. Decision making in the surgical treatment of meralgia paresthetica: neurolysis versus neurectomy. Acta Neurochir (Wien). 2012;154(10):1765–72.

Posterior Femoral Cutaneous Nerve Entrapment: Pelvic

62

Natalia Murinova, Daniel Krashin, and Andrea M. Trescot

Introduction

The *posterior femoral cutaneous nerve* (PFCN) neuropathy is an uncommon, potentially under-recognized cause of posterior and lateral thigh pain and numbness. PFCN provides sensation to the posterior thigh and perineal region, mimicking sciatic and pudendal nerve pathologies. It is rare to present with an isolated neuropathy. In the literature, the most common reported causes of PFCN injury have been intramuscular injections or pressure. The alternate names for this nerve and syndrome include *lesser sciatic nerve entrapment* or *posterior cutaneous nerve of the thigh*. The perineal branch of the PFCN has been called the *inferior pudendal nerve*, *pudendal longus inferior*, *long pudendal nerve*, or *nerve of Soemmering*. It is common to have injury of the inferior pudendal nerve in the presentation of PFCN neuropathy. Consider PFCN entrapment neuropathy in a patient presenting with posterior and lateral thigh pain.

Clinical Presentation (Table 62.1)

PFCN entrapment presents as posterior thigh pain, from the gluteal fold to the back of the knee, down to a variable amount of the posterior calf (Fig. 62.1). There can be sensory abnormalities localized to the lower buttock and the posterior thigh and the posterior thigh of the upper leg (Fig. 62.2).

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The PFCN, through its perineal branch, also innervates the perineal region (Fig. 62.3). PFCN injury has been reported due to trauma from intramuscular gluteal injections [1]. This

Table 62.1 Clinical presentation of posterior femoral cutaneous nerve entrapment

Branches of PFCN	Sensory innervation and pain to the skin of particular PFCN branch
Major branch of PFCN	Sensation to the back and medial side of the thigh, popliteal fossa, and upper part of the back of the leg
Inferior cluneal nerve of PFCN	Sensation to the inferior buttocks
Perineal branch of PFCN	Sensation to the lateral perineum, upper and medial thigh, posterolateral scrotum, labia majora, part of the penis/clitoris
Gluteal branches of PFCN (inferior cluneal nerves)	Sensation to the lower and lateral part of gluteus maximus
Collateral branches from the anterior divisions	Sensation to the quadratus femoris, inferior gemellus muscles (L4, L5, S1), internal obturator, and superior gemellus muscles (L5, S1, S2)

Fig. 62.1 Patient complaints of pain from posterior femoral cutaneous entrapment (Image courtesy of Andrea Trescot, MD)

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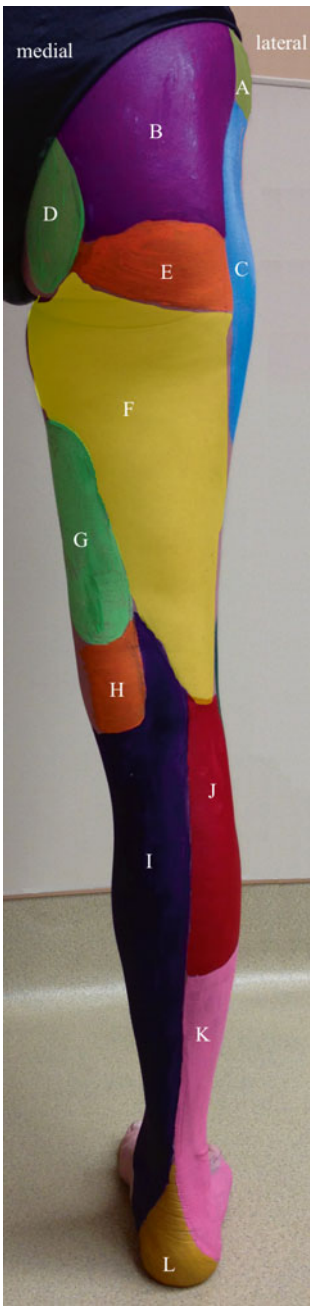

Fig. 62.2 Pattern of pain from nerves of posterior leg. *A* lateral branch iliohypogastric nerve, *B* superior cluneal nerve, *C* lateral femoral cutaneous nerve, *D* middle cluneal/sacral nerve, *E* inferior cluneal nerve, *F* posterior femoral cutaneous nerve, *G* obturator nerve, *H* femoral nerve, *I* saphenous nerve, *J* lateral sural cutaneous nerve, *K* superficial peroneal nerve, *L* medial calcaneal nerve (Image courtesy of Terri Dallas-Prunskis, MD)

nerve is traumatized by poor gluteal injections, prolonged bicycle rides, pelvic tumors, venous malformation, trauma, and idiopathic causes (Table 62.1) [2, 3]. Other cases may be due to pressure on the nerve inferior margin of the gluteus maximus muscle due to sitting on hard surfaces [4]. It can also present as perineal pain, including the rectum, scrotum/

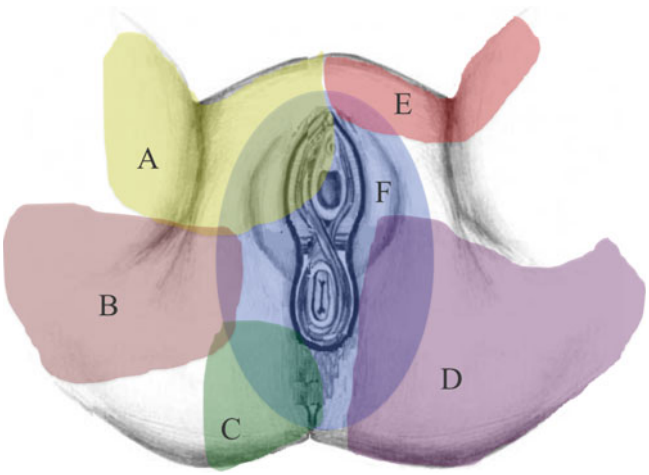

Fig. 62.3 Innervation of perineum: *A* genitofemoral nerve, *B* obturator nerve, *C* inferior cluneal nerve, *D* peroneal branch of the posterior femoral cutaneous nerve, *E* ilioinguinal nerve, and *F* pudendal nerve (Image inspired by Hibner et al. [20], courtesy of Andrea Trescot, MD)

labia majora, and penis/clitoris. Isolated lesions of PFCN are described in a small number of individual case studies in the literature (Table 62.2).

Anatomy (Table 62.3)

The PFCN is a pure sensory nerve, which originates from the anterior and posterior rami of the first three sacral nerves [9], as well as the anterior divisions from S2, S3, and the posterior rami of S1–S3 (Table 62.3). The anatomy of the PFCN is very variable, and it may involve branches as high as L4 or as low as S4 [10]. The PFCN exits the pelvis beneath the gluteus maximus (Fig. 62.4) with the inferior gluteal artery, through the sciatic foramen, below the piriformis muscle, and passes down the buttock and thigh on the medial aspect of the sciatic nerve (Figs. 62.4 and 62.5); it is sometimes referred to as the *lesser sciatic nerve.* Deep to the gluteus maximus muscle, the PFCN gives off the *inferior cluneal nerve* (Chap. 63) and the *perineal branch of the PFCN* (PBPFCN) (Fig. 62.6). The inferior cluneal nerve provides the cutaneous innervation of the inferior buttocks, while the perineal branch innervates the lateral perineum, the proximal medial thigh, the posterolateral scrotum/labia majora, and part of the penis/clitoris (Fig. 62.7) [11]. The perineal branch courses medially, staying about 4 cm inferior to the attachment of the sacrotuberous ligament onto the ischial tuberosity, parallel to the ischial ramus [11]. However, Bergman et al. [12] described the perineal branch piercing the sacrotuberous ligament, which would place it close to the *pudendal nerve* in *Adcock's canal* (Chap. 47).

The rest of the nerve then continues inferiorly from the lower edge of the gluteus maximus in a muscular groove formed by the medial and lateral hamstring muscles, with

Table 62.2 Cases in the literature regarding different causes of neuropathy of posterior femoral cutaneous nerve

Symptoms of patients presenting with PFCN	Causes of PFCN neuropathy	Article
54-year-old man developed hyperalgesia of posterior thigh and lateral scrotum	Injury of the PFCN after gluteal injection	Iyer VG et al. Isolated injection injury. 1989. [5]
137 cases of sciatic nerve injury, including two cases with PFCN lesions	Injury of the PFCN after gluteal injection – article describes the *infrapiriformis foramen syndrome*	Obach J et al. The infrapiriformis foramen syndrome from intragluteal injection. 1983. [6]
37-year-old female compressed her PFCN with gymnastics; 47-year-old sedentary male with bilateral hyperpathia; 54-year-old male rode a bicycle 30 miles daily with pain in the posterior thigh and paresthesias in lower buttocks and lateral scrotum	Injury to PFCN from compression of the nerve by the ischial tuberosity, thought to arise during exercise, sitting, and biking	Arnoldussen WJ. Pressure neuropathy. 1980. [4]
40-year-old female with decreased sensation in the right posterior thigh after a left putamen hemorrhage 4 years earlier	Unknown etiology	Dumitru D et al. Posterior femoral cutaneous nerve neuropathy and somatosensory evoked potentials. 1988. [7]
25-year-old female with decreased sensation in posterior/lateral thigh	Injury of PFCN after two gluteal intramuscular injections	Tong H et al. Posterior femoral cutaneous nerve mononeuropathy: A case report. 2000. [1]
22-year-old female with pain, lack of sensation in posterior thigh and lower half of buttock	Injury of PFCN after gluteal intramuscular injection	Kim JE, et al. Isolated posterior femoral cutaneous neuropathy following intragluteal injection. 2009. [8]

Table 62.3 Posterior femoral cutaneous nerve anatomy

Origin	Posterior rami S1 to S3 (but may include up to L4 and down to S4)
General route	Exits the pelvis anterior to the piriformis but posterolateral to the sciatic nerve; gives off inferior cluneal nerve and perineal branch of the PFCN; travels down the posterior thigh between the medial and lateral hamstring muscles, joining the sural nerve, occasionally down to calcaneus
Sensory distribution	Inferior buttocks, lateral perineum, proximal medial thigh, posterolateral scrotum/labia, and part of the penis/clitoris
Motor innervation	None
Anatomic variability	Perineal branch can come from the inferior cluneal nerve
Other relevant structures	Sacrotuberous ligament, piriformis muscle, and pudendal nerve

branches providing the sensation to the posterior thigh. The PFCN extends a variable distance into the calf, becoming superficial near the popliteal fossa, joining with the *lateral sural* (Chap. 72) and the *distal saphenous nerves* (Chap. 59) to innervate the posterior calf. Kosinski [13] described the PFCN as occasionally extending all the way to the calcaneal region.

The *main trunk of the PFCN* to the back of the thigh and leg consists of numerous filaments derived from both sides of the nerve and distributes to the skin covering the back and medial side of the thigh, the popliteal fossa, and the upper part of the back of the leg.

A study by Nakanishi et al. [14] described the dissection of 37 Japanese subjects, which showed that the origin of PFCN was variable. They found that the PFCN may receive root components from the segments S1 through S4 and does not arise from S1 alone.

Tubbs et al. [11] dissected 20 cadavers and found that the perineal branch of the PFCN (PBPFCN) arose directly from the PFCN in 55 % of the sides; in 30 % of the sides, it arose from the inferior cluneal nerve, and it was absent in 15 % of sides (Table 62.4). The PBPFCN is described to provide two to three branches to the medial thigh and then innervate the scrotum and labia majora. In males, one nerve branch traveled inferior to the corpora cavernosa and anterior to the spermatic cord to cross the midline. Communications between the PBPFCN and the perineal branch of the pudendal nerve are common.

Entrapment

The PFCN lies adjacent to the sciatic nerve and can be injured by prolonged tourniquet time; like the sciatic nerve, the PFCN may also be compressed by the piriformis muscle. The *sciatic foramen* is divided into two parts by the piriformis muscle (*foramina suprapiriformis* and *foramina infrapiriformis*). The PFCN reaches the dorsal part of the pelvis in close proximity to the ischial tuberosity as it passes though the infrapiriformis foramen. Compression of the nerve can occur at the ischial tuberosity, such as by prolonged sitting on the edge of a chair [4]. Gluteal intramuscular injections have been reported to injure the PFCN and present as thigh and scrotal/labial pain [5].

Both the pudendal nerve and the PFCN leave the pelvis together through the greater sciatic foramen after passing through the infrapiriformis canal, so they can be trapped together along that path. They also share innervation of the

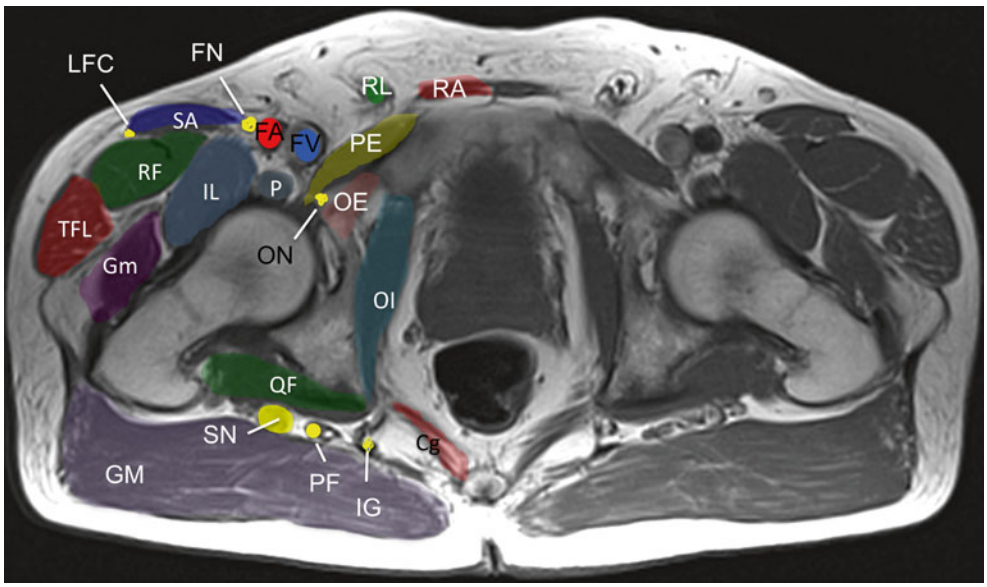

Fig. 62.4 MRI axial image of the pelvis. *Cg* coccygeus muscle, *FA* femoral artery, *FN* femoral nerve, *FV* femoral vein, *GM* gluteus maximus muscle, *Gm* gluteus medius, *IG* inferior gluteal nerve, *IL* iliopsoas muscle, *IT* ischial tuberosity, *ITT* iliotibial tract, *LFC* lateral femoral cutaneous nerve, *OI* obturator internus muscle, *P* psoas muscle, *PE* pectineus muscle, *PF* posterior femoral cutaneous nerve, *QF* quadratus femoris muscle, *RA* rectus abdominis muscle, *RF* rectus femoris muscle, *RL* round ligament, *SA* sartorius muscle, *SN* sciatic nerve, *TFN* tensor fascia lata muscle (Image courtesy of Andrea Trescot, MD)

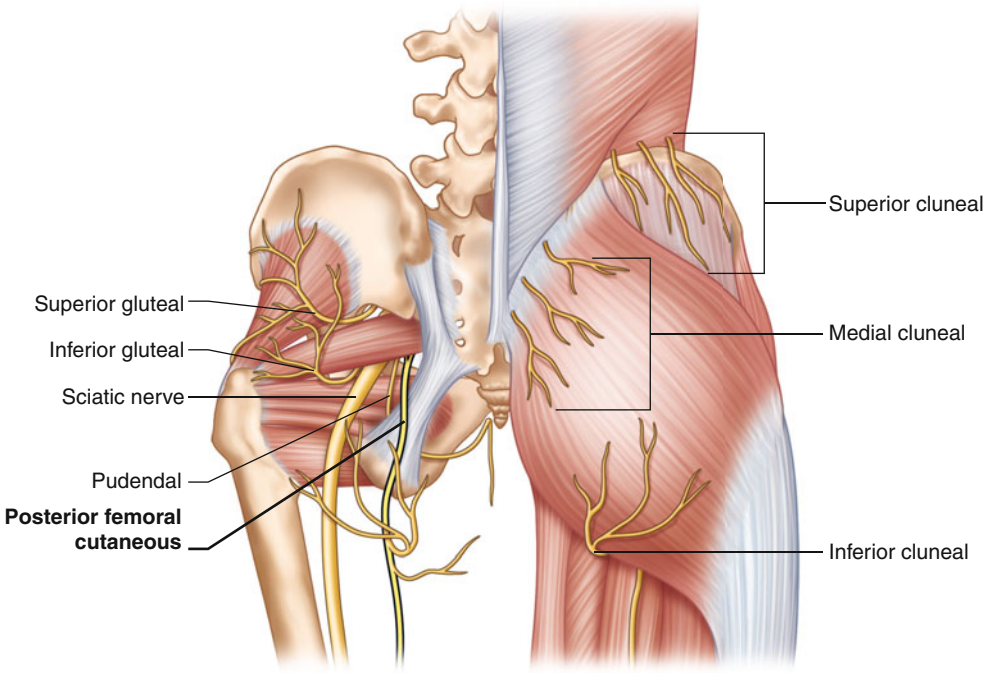

Fig. 62.5 Anatomy of the buttocks and lower extremity (Image courtesy of Springer)

perineal region. As early as 1900, Cushing [15] suggested that pain after ligation of testicular veins might be due to trauma of the perineal branch of PFCN.

Physical Exam

Because the nerve lies deep in the thigh, it is relatively hard to examine the PFCN by palpation of the thigh (Fig. 62.8). However, at the level of the ischium, the nerve can be palpated between the heads of the hamstring muscles (Fig. 62.9). In the case reports, only two mentioned that there was numbness in the region of the PFCN [4, 7]. Two cases had decreased sensation in the posterior thigh and lower buttock. This suggests that in a significant number of posterior femoral cutaneous neuropathies, the inferior cluneal nerve is also affected. On physical examination, look for distribution of pain and sensory abnormality localized in the area corresponding to the particular branch of the PFCN that is injured, such as the lower buttock, the posterior thigh, and the dorsal surface of the upper leg. The rest of the physical examination should be normal, including motor strength and reflexes.

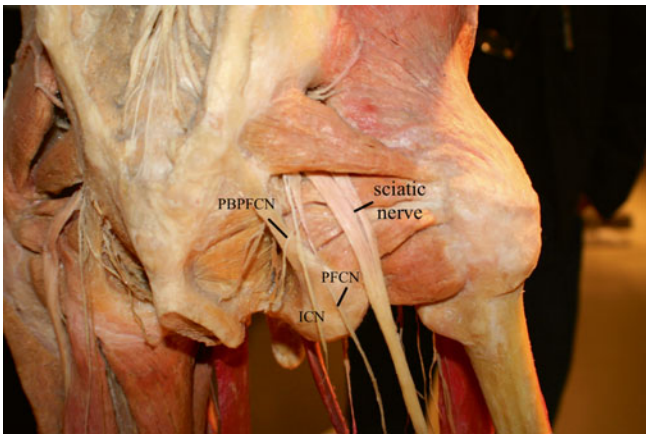

Fig. 62.6 Buttocks dissection, modified from an image from *Bodies, The Exhibition*, with permission. *PFCN* posterior femoral cutaneous nerve, *PBPFCN* perineal branch posterior femoral cutaneous nerve, *ICN* inferior cluneal nerve (Image courtesy of Andrea Trescot, MD)

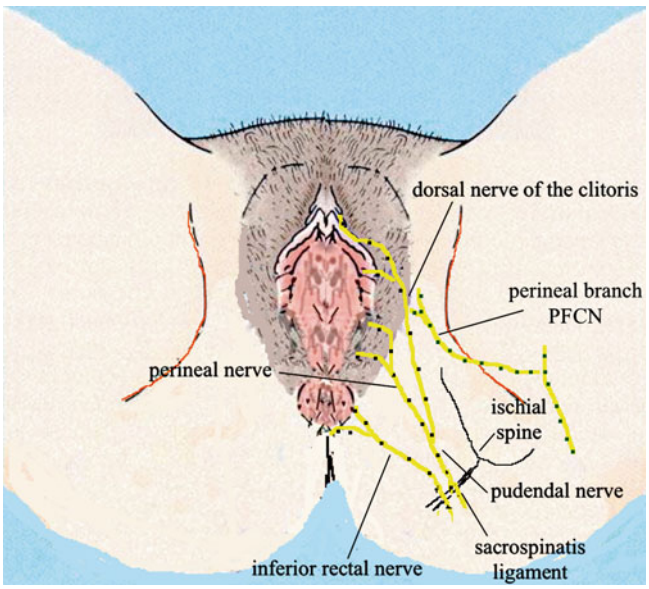

Fig. 62.7 Schematic image of the perineal nerves. *PFCN* posterior femoral cutaneous nerve (Image courtesy of Andrea Trescot, MD)

Table 62.4 Origin of the perineal branch of PFCN (PBPFCN)

From PFCN directly	55 %
From inferior cluneal nerve	30 %
Absent	15 %

Table was constructed using results of data from Tubbs et al. [11] study of 20 dissected adult cadavers (40 sides)

Differential Diagnosis (Table 62.5)

The PFCN may be responsible for many symptoms that are attributed to sciatic nerve and "piriformis syndrome" (Table 62.5). The piriformis syndrome presents with pain in

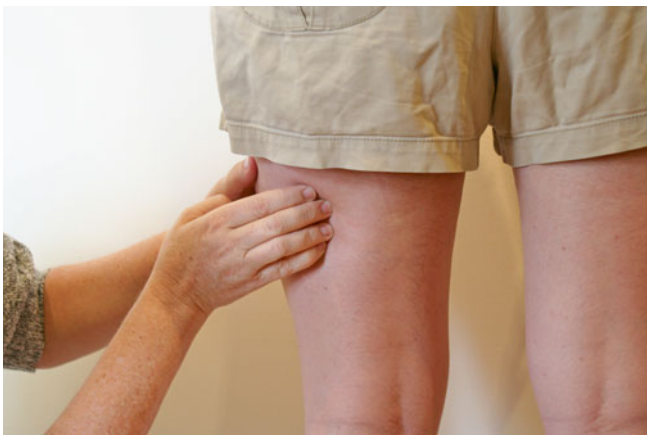

Fig. 62.8 Palpation of the posterior femoral cutaneous nerve at the posterior thigh (Image courtesy of Andrea Trescot, MD)

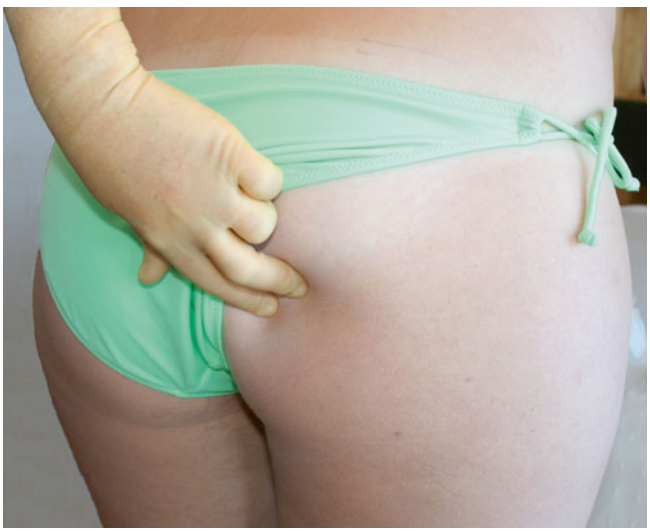

Fig. 62.9 Palpation of the posterior femoral cutaneous nerve at the ischium (Image courtesy of Andrea Trescot, MD)

Table 62.5 Differential diagnosis of low back and buttocks pain

	Potential distinguishing features
Lumbar spine disorders	Physical exam and MRI scan should confirm the diagnosis
Sacroiliac joint disorders	Pain may be present in the lower back, back of hips, groin, and thighs; tenderness at PSIS; relief of pain from an SI joint injection will confirm the diagnosis
Lower lumbar disk problems	Local pain in the affected area; abnormal X-ray tests or MRI scans
Sciatic entrapment	Pain worse with internal rotation, foot externally rotated
Myofascial pain syndromes, especially piriformis	Visible hypertonicity or knots in the muscle may be seen; trigger point injections into the muscle will alleviate the symptoms
Inferior gluteal neuralgia	Pain usually does not radiate down the leg

Table 62.6 Diagnostic tests for posterior femoral cutaneous nerve

	Potential distinguishing features
Physical exam	Tenderness between the heads of the hamstring muscles at the ischium
Diagnostic injection	At the ischium
Ultrasound	Not described
MRI	Not useful
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies	SSEP

the gluteal area, which may mimic PFCN neuropathy. However, in the piriformis syndrome and sciatic entrapment, the pain can be worsened by internal rotation and flexion at the hip. Pressure over the greater sciatic foramen also elicits local pain with piriformis syndrome. Often, there is tenderness to palpation along the course of the nerve at the level of the thigh. The *perineal branch of the PFCN* can mimic *pudendal nerve* pathology, and it shares much of the same innervation. Table 62.5 discusses the distinguishing features of the differential diagnosis, and Table 62.6 describes the diagnostic tests.

Dumitru and Marquis [7] described somatosensory evoked potential (SSEP) evaluation of the PFCN. A recording electrode is placed 6 cm proximal to the midpopliteal fossa, and the nerve is stimulated supramaximally 12 cm proximally on a line between the active electrode and the ischial tuberosity. A ground electrode is placed just proximal to the active recording electrode. The lower extremities of 40 individuals with a mean age of 34 years (20–78 years) were examined. The mean peak latency of the response is 2.8 (2.3–3.4) ms ± 0.2 ms with a mean amplitude of 6.5 (4.1–12.0) microV ± 1.5 microV.

Treatment of Contributing/Perpetuating Factors

- Iatrogenic trauma with gluteal injections
- Pressure of ischial area from prolonged bicycle riding or prolonged sitting
- Anatomic variations
- Smoking
- Diabetes
- Poor circulation
- Cachexia/excessive muscle loss that functions as a nerve cushion
- Obesity

Iatrogenic trauma can be prevented with careful intramuscular injections in the gluteal area. Tourniquet times and pressures should be closely monitored to prevent ischemia of the nerve. Avoid pressure into the ischial area from prolonged bicycle riding or prolonged sitting, especially in people who are prone to peripheral nerve injuries due to genetic and other medical conditions such as diabetes, poor circulation, and smoking. It is important to educate patients that if they experience recurrent pain in the gluteal area due to exercise, they might be overstretching the PFCN or causing entrapment of PFCN. Also, for bikers with gluteal pain, the recommendation is to use a cushion to prevent compression due to excessive pressure. It is likely that athletes such as rowers, bikers, and other athletes with excessive compression of the gluteal area are especially prone to this injury and might be misdiagnosed with sciatica.

Injection Technique

The choice of approach to the PFCN depends on the proposed site of entrapment; in other words, entrapment in the sciatic foramen would be targeted differently than pathology at the ischium, perineum, or distal thigh.

Landmark-Guided Technique

There was only one study found on PubMed describing using the blind injection technique of PFCN. Hughes et al. [16] described blocking the PFCN at the point where its branches come from below the medial border of gluteus maximus. This location is found by Hughes to be a quarter of the distance from the ischial tuberosity to the greater trochanter, in the gluteal fold (Fig. 62.10 Site A). They suggest feeling two distinct losses of resistance as superficial and deep fascia are penetrated with a short-beveled needle. The perineal branch of the PFCN is usually injected at the ischial tuberosity (Fig. 62.10 Site B) [11].

The choice of approach to the PFCN depends on the proposed site of entrapment. For patients presenting with pain and paresthesia in the perineal area, consider injecting the PFCN 2 cm below the ischial tuberosity, proximal to the PBPFCN (Fig. 62.10 Site C) [17]. In patients with inferior lateral buttock area pain and numbness, target the inferior cluneal nerves under the gluteus maximus muscle. In patients presenting with pain in the posterior thighs, the most likely area of involvement is the distal portion of the PFCN [17]. This area is located between the gluteus maximus and the long head of the biceps femoris and is the area that should be considered for injection [17]. Based on cadaver studies, Tubbs et al. suggested that the PBPFCN was on average located 4 cm inferior to the ischial tuberosity, and they recommend injecting this area with anesthetic to block the nerve (Fig. 62.10 Site D) [11].

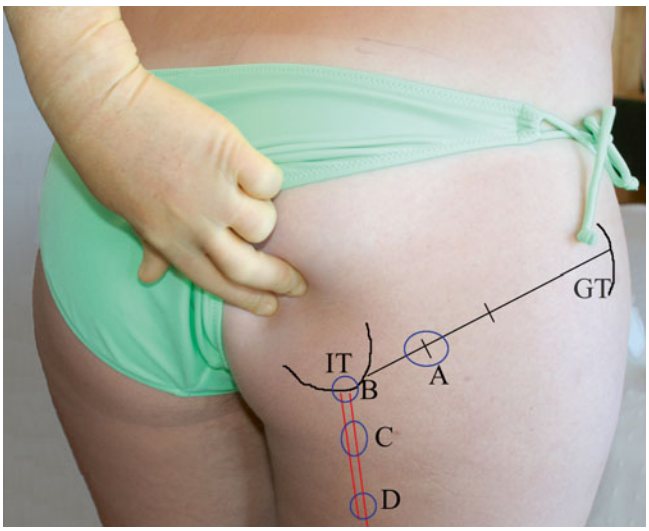

Fig. 62.10 Landmark-guided injection of the posterior femoral cutaneous nerve at the ischium. *IT* ischial tuberosity, *GT* greater trochanter. *A* site described by Hughes [11], *B* injection at the ischium, *C* site recommended by Fritz [17], 2 cm below the IT, *D* site recommended by Tubbs et al. [11], 4 cm below the IT (Image courtesy of Andrea Trescot, MD)

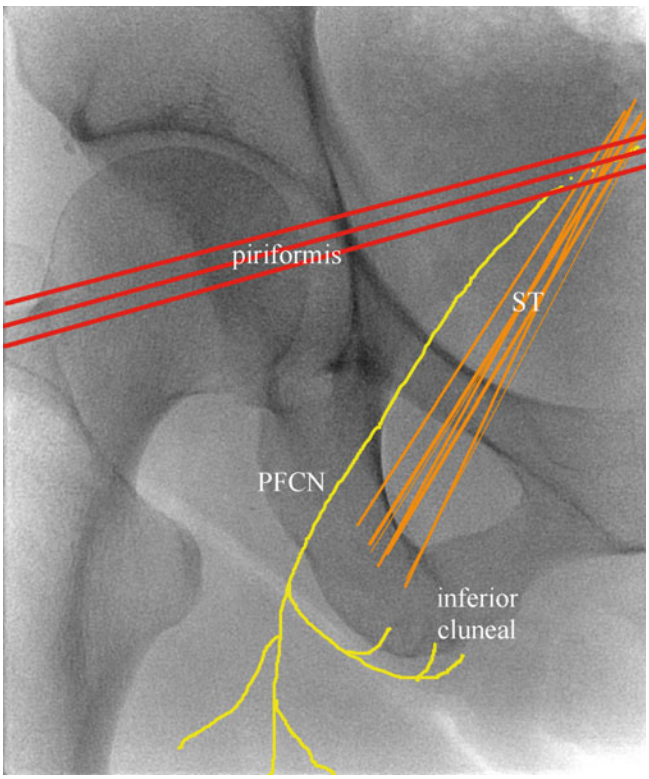

Fig. 62.11 Fluoroscopic landmarks of the posterior femoral nerve (Image courtesy of Andrea Trescot, MD)

Fluoroscopy-Guided Technique

There are no good fluoroscopic landmarks other than the ischium (Fig. 62.11).

Ultrasound-Guided Technique (US)

The sciatic nerve is well visualized by ultrasound, but there are no reported US approaches to injecting the PFCN.

CT-Guided Technique

Kasper et al. [18] described CT-guided PFCN blocks in two patients. The PFCN neurovascular bundle was traced from the inferior margin of the ipsilateral gluteus maximus muscle, in between the hamstring tendon origin and the sciatic nerve. Under aseptic precautions, a 22-gauge needle was advanced to the above-described position, and the location was documented by injecting 1 cc of 50 % diluted, nonionic water-soluble contrast. One patient had decreased sharp sensation in the inferior gluteal region as well as posterior thigh (the inferior cluneal nerve and PFCN distribution) [18]. Neither of the two patients had improvement of their pain intensity.

MRI-Guided Technique

Fritz et al. [17] performed MR-guided PFCN injections technically successful in 12/12 cases (100 %), with uniform perineural distribution of the injectant.

All procedures were performed using a clinical, wide-bore 1.5-Tesla MR imaging system with patients in prone position and the table landmark centered at the inferior buttock area. The course of the PFCN was mapped. Superficial local anesthesia was administered, and a 20-gauge needle of 10 or 15 cm in length was used in the immediate vicinity of the PFCN. In all patients and locations, a total amount of 4 mL was injected around the PFCN, consisting of 1 mL of 1 % preservative-free lidocaine, 1 mL of 0.5 % bupivacaine, and 1 mL of non-particulate dexamethasone (10 mg/mL).

Neurolytic Technique

There are no described neurolytic techniques, although cryoneuroablation or radiofrequency lesioning would theoretically be reasonable options.

Surgical Technique

Mobbs et al. [19] described one case of successful surgery of the PFCN in a 51-year-old healthy male that presented with a 12-month history of shooting pains down the posterior and lateral aspect of his thigh and buttock. The pain extended to the knee and to the lateral thigh. Lower limb power was

intact. The somatosensory evoked potentials of the PFCN demonstrated response differences consistent with entrapment neuropathy. Tubbs et al. [11] performed an open exploration of the PFCN. After identification of the sciatic nerve, they identified the PFCN, as it lies superficial to the sciatic nerve in its proximal course. Although no site of entrapment was obvious, release of the fascia over the nerve was successful in their patient, with complete resolution of his symptoms at 12 months follow-up.

Complications

There were no complications of the blind technique described in the literature; however, since there are multiple nerves in this area, the main concerns are neurological complications. There were no complications using CT-guided or MRI-guided injections of the PFCN during the procedure or during follow-up [17].

Summary

PFCN is rarely considered as a cause of pelvic pain, which probably contributes to the limited available literature. PFCN entrapment should be considered in the patient with pelvic pain and posterior thigh pain.

References

1. Tong HC, Haig A. Posterior femoral cutaneous nerve mononeuropathy: a case report. Arch Phys Med Rehabil. 2000;81(8):1117–8.
2. Schuchmann JA. Sural nerve condition: a standardized technique. Arch Phys Med Rehabil. 1977;58:166–8.
3. Chutkow JG. Posterior femoral cutaneous neuralgia. Muscle Nerve. 1988;11:1146–8.
4. Arnoldussen W, Korten J. Pressure neuropathy of the posterior femoral cutaneous nerve. Clin Neurol Neurosurg. 1980;82(1):57–60.
5. Iyer VG, Shields CB. Isolated injection injury to the posterior femoral cutaneous nerve. Neurosurgery. 1989;25(5):835–8.
6. Obach J, Aragones JM, Ruano D. The infrapiriformis foramen syndrome resulting from intragluteal injection. J Neurol Sci. 1983; 58(1):135–42.
7. Dumitru D, Marquis S. Posterior femoral cutaneous nerve neuropathy and somatosensory evoked potentials. Arch Phys Med Rehabil. 1988;69(1):44.
8. Kim JE, Kang JH, Chol Choi J, Seok Lee J, Kang SY. Isolated posterior femoral cutaneous neuropathy following intragluteal injection. Muscle Nerve. 2009;40(5):864–6.
9. Enneking FK, Chan V, Greger J, Hadzic A, Lang SA, Horlocker TT. Lower-extremity peripheral nerve blockade: essentials of our current understanding. Reg Anesth Pain Med. 2005;30(1):4–35.
10. Hollinshead WH. Anatomy for surgeons: the back and limbs. 3rd ed. Philadelphia: Harper & Row; 1982.
11. Tubbs RS, Miller J, Loukas M, Shoja MM, Shokouhi G, Cohen-Gadol AA. Surgical and anatomical landmarks for the perineal branch of the posterior femoral cutaneous nerve: implications in perineal pain syndromes. Laboratory investigation. J Neurosurg. 2009;111(2):332–5.
12. Bergman RA, Afifi AK, Miyauchi R. Posterior femoral cutaneous nerve 2014 [Jan 4, 2014]. Available from: http://www.anatomyatlases.org/AnatomicVariants/NervousSystem/Text/PosteriorFemoralCutaneous.shtml.
13. Kosinski C. Course, mutual relations and distribution of the cutaneous nerves of the metazonal regions of leg and foot. J Anat. 1926; 60:274–97.
14. Nakanishi T, Kanno Y, Kaneshige. Comparative morphological remarks on the origin of the posterior femoral cutaneous nerve. Anat Anz. 1976;139(1–2):8–23.
15. Cushing HI. The employment of local anaesthesia in the radical cure of certain cases of hernia, with a note upon the nervous anatomy of the inguinal region. Ann Surg. 1900;31(1):1–34.
16. Hughes P, Brown T. An approach to posterior femoral cutaneous nerve block. Anaesth Intensive Care. 1986;14(4):350–1.
17. Fritz J, Bizzell C, Kathuria S, Flammang AJ, Williams EH, Belzberg AJ, et al. High-resolution magnetic resonance-guided posterior femoral cutaneous nerve blocks. Skelet Radiol. 2013;42(4):579–86.
18. Kasper JM, Wadhwa V, Scott KM, Chhabra A. Clunealgia: CT guided therapeutic posterior femoral cutaneous nerve block. Clin Imag. 2014;38(4):540–42.
19. Mobbs R, Szkandera B, Blum P. Posterior femoral cutaneous nerve entrapment neuropathy: operative exposure and technique. Br J Neurosurg. 2002;16(3):309–11.
20. Hibner M, Desai N, Robertson LJ, Nour M. Pudendal neuralgia. J Minim Invasive Gynecol. 2010;17(2):148–53.

Inferior Cluneal Nerve Entrapment: Lower Extremity

63

Terri Dallas-Prunskis

Introduction

Neuropathic gluteal pain is usually associated with sciatic entrapment or radiculopathy. However, some patients present with pain located at the caudal and medial parts of the buttocks and the upper part of the posterior thigh, as well as in the perineal area (including the scrotum or the labia majora). These pains do not match the pattern of a *sciatic nerve* entrapment (see Chap. 65) but rather may represent entrapment of the *inferior cluneal nerve*, which emerges from the *posterior femoral cutaneous nerve* (PFCN) (see Chap. 62), innervating the buttocks and upper posterior thigh, as well as the perineum. Careful attention to the description of the pain, combined with a directed physical exam, is necessary for accurate diagnosis and treatment.

Clinical Presentation (Table 63.1)

Patients with inferior cluneal nerve (ICN) entrapment complain of a burning, tingling, or numbness sensation along the inferior and medial aspects of the buttocks (Fig. 63.1) and/or along the dorsal and proximal thigh, as well as the lateral anal margin and the skin of the scrotum or labia majora (Fig. 63.2). Pain will increase with sitting on hard surfaces, such as chairs or bicycle seats, and it may mimic or be triggered by piriformis spasm. As a branch of the PFCN, the ICN is entrapped by the same mechanisms and may present in a similar way.

Table 63.1 Occupation/exercise/trauma history relevant to inferior cluneal entrapment

Trauma	Fall onto buttocks, hamstring injury, intramuscular injection into gluteal muscle, piriformis trauma or injury
Direct compression of nerve	Sitting on a hard seat, bicycle riding
Myofascial compression	Piriformis spasm, gluteal muscle spasm

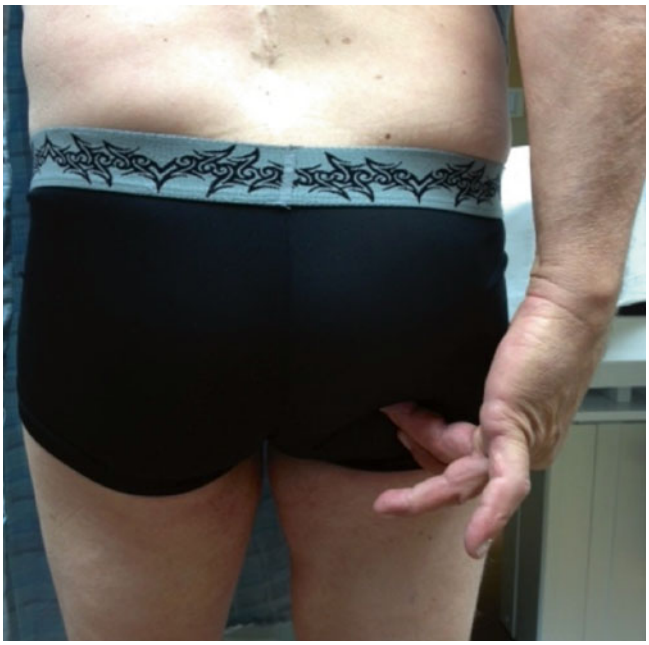

Fig. 63.1 Pain location from inferior cluneal neuralgia (Image courtesy of Terri Dallas-Prunskis, MD)

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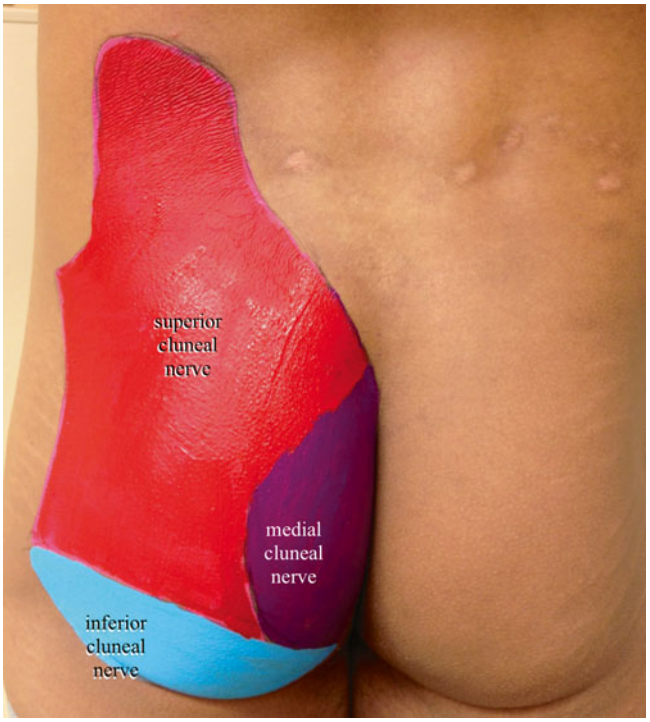

Fig. 63.2 Cutaneous distribution of the cluneal nerves (Image courtesy of Terri Dallas-Prunskis, MD)

Table 63.2 Inferior cluneal nerve anatomy

Origin	Posterior rami S1 to S3 (but may include up to L4 and down to S4)
General route	The PFCN exits the pelvis anterior to the piriformis but posterolateral to the sciatic nerve and gives off inferior cluneal nerve and perineal branch of the PFCN
Sensory distribution	Provides cutaneous innervation to the inferior part of the buttocks, lateral anus region (but not the anus), and the lateral region of the labia majora
Motor innervation	None
Anatomic variability	Only a few variations are noted in the literature, giving a description of the inferior cluneal nerves going through the gluteus maximus muscle; other sources indicate that the nerves reach the caudal edge of the gluteus maximus then circumvent it at various levels

Anatomy (Table 63.2)

The cluneal nerves are divided into three groups: the *superior cluneal nerve* (see Chap. 51), the *middle cluneal nerve (sacral nerves)*, and the *inferior or lateral cluneal nerves* (Fig. 63.3). The ICN arises from the inferior portion of the *posterior femoral cutaneous nerve* (PFCN) (see Chap. 62). This nerve is made up of sensory branches of S1, S2, and S3, traveling parallel with the sciatic nerve and the pudendal nerve through the sciatic notch (Fig. 63.4). After reaching the subgluteal area, the PFCN gives rise to the *inferior cluneal branch* and the *perineal branch* (Fig. 63.5). These nerves then go to the inferior edge of the gluteus maximus muscle and follow a recurrent course behind the muscle (Fig. 63.6). The ICN provides cutaneous innervation to the inferior part of the buttocks (Fig. 63.7), the lateral anal region (but not the anus), and the lateral region of the labia majora (but not the labia minora or the vagina) [1]. It also does not innervate the penis or clitoris (Fig. 63.2) [2].

Tubbs et al. [3] dissected 20 cadavers to study the PFCN and its branches. The perineal branch of the PFCN arose directly from the PFCN in 55 % of the dissections and from the ICN in 30 %. It was absent in 15 % of the bodies studied (Table 63.3).

Entrapment

There are two common areas where entrapment may occur. The first one would extend from the passage of the perineal ramus under the ischium to the perineum (Fig. 63.6, site A). This entrapment may be due to nerve compression by the ischium on the gluteus maximus and the hamstring muscles in a sitting position and stretching of the perineal ramus with internal rotation of the thigh.

The second site of entrapment is more proximal, at the level of the sciatic spine and the piriformis. At this point, the roots of the PFCN, which gives rise to the ICN, may be encircled by the piriformis against the sciatic notch (Fig. 63.6, site B). However, whatever the etiology, it is the sitting position that triggers the entrapment, giving the *inferior cluneal entrapment* syndrome the same general appearance as a *pudendal syndrome* or *ischial bursitis* [4].

Physical Examination

The examination should begin by evaluating the entire back to rule out other causes for the pain. Palpation should elicit non-radiating pain increasing with deep pressure over the sciatic notch. Palpate the inferior edge of the gluteus maximus between the ischium and the greater trochanter (Fig. 63.8). Hyperesthesia to pin scratch and decreased sensation to touch over the inferior buttocks corresponding to the distribution of the inferior cluneal nerve will also be noted (Fig. 63.7); no evidence of motor involvement should be appreciated. Pain may be induced on digital rectal examination of the ischium more superficial than that associated with pudendal canal syndrome (at the pelvic head of the obturator internus) [5].

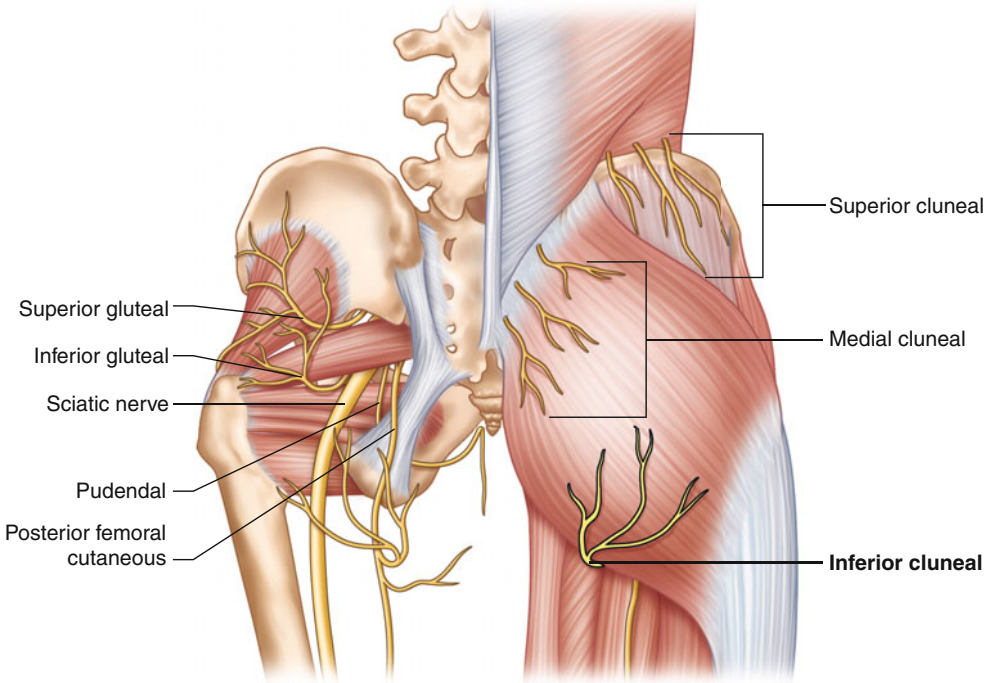

Fig. 63.3 Anatomy of the buttocks nerves (Image courtesy of Springer)

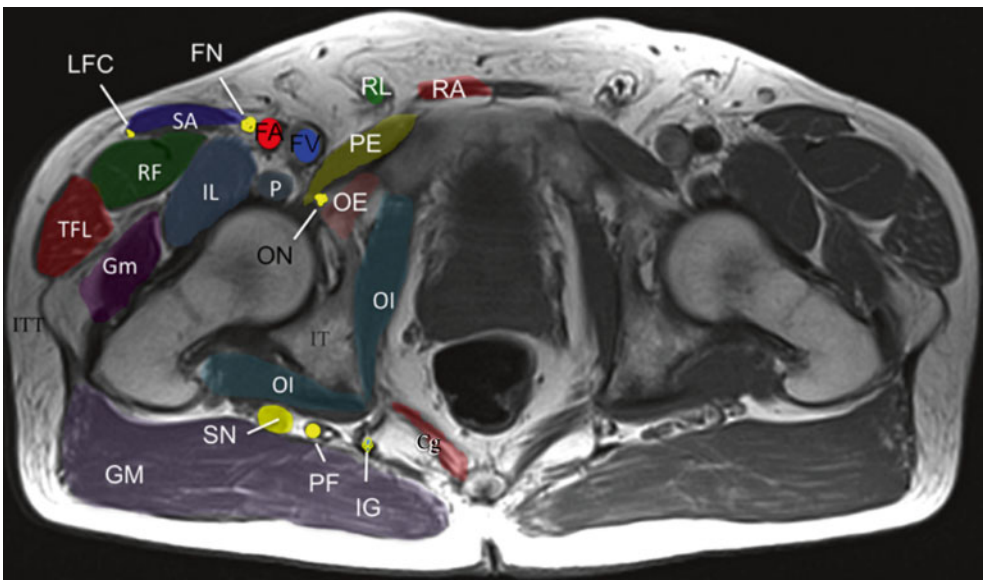

Fig. 63.4 MRI axial image of the pelvis. *Cg* coccygeus muscle, *FA* femoral artery, *FN* femoral nerve, *FV* femoral vein, *GM* gluteus maximus muscle, *Gm* gluteus medius, *IG* inferior gluteal nerve, *IL* iliopsoas muscle, *IT* ischial tuberosity, *ITT* iliotibial tract, *LFC* lateral femoral cutaneous nerve, *OI* obturator internus muscle, *P* psoas muscle, *PE* pectineus muscle, *PF* posterior femoral cutaneous nerve, *QF* quadratus femoris muscle, *RA* rectus abdominis muscle, *RF* rectus femoris muscle, *RL* round ligament, *SA* sartorius muscle, *SN* sciatic nerve, *TFN* tensor fascia lata muscle (Image courtesy of Andrea Trescot, MD)

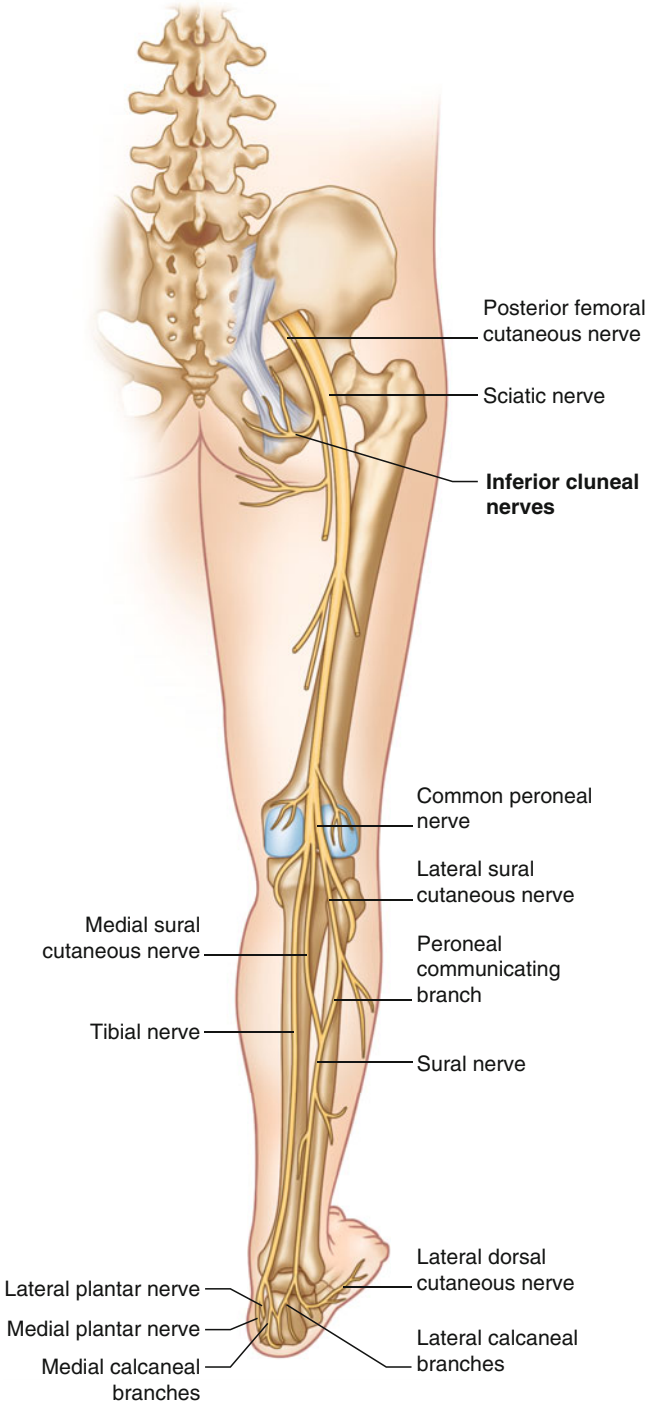

Fig. 63.5 Anatomy of the lower extremity nerves (Image courtesy of Springer)

Differential Diagnosis (Table 63.4)

Inferior cluneal nerve entrapment must be differentiated from other lower limb pain disorders such as entrapment of the *sciatic nerve* (see Chap. 65), the *posterior femoral cutaneous nerve* (see Chap. 62), or the *obturator nerve*

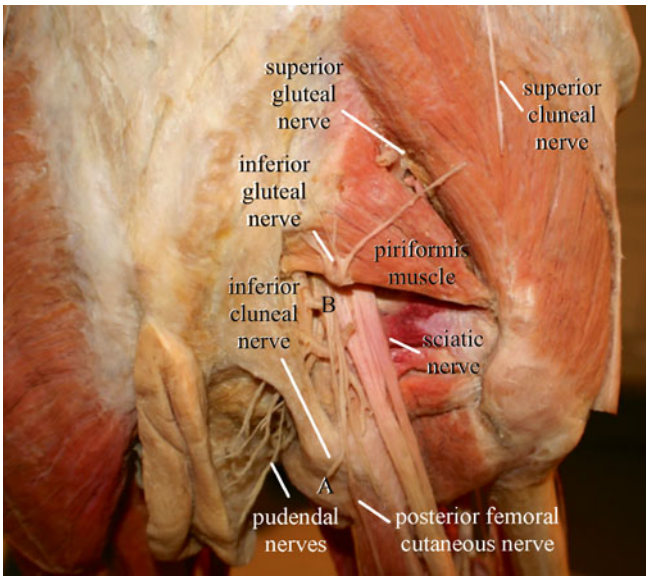

Fig. 63.6 Gluteal muscle dissection showing sites of entrapment of the inferior cluneal nerve, modified from an image from *Bodies*, *The Exhibition*, with permission. *A* distal entrapment, *B* proximal entrapment (Image courtesy of Andrea Trescot, MD)

(see Chap. 64), caused by muscle spasms of the piriformis and obturator internus muscles. This entrapment is also misdiagnosed as *pudendalgia* (*pudendal canal syndrome*) (see Chap. 47) [6]. Table 63.5.

Identification and Treatment of Contributing Factors

The inferior cluneal nerves typically may be injured by a fall onto the buttocks or by a hamstring injury, but sometimes it is not clear what caused the cluneal nerves to be symptomatic. Intramuscular injections into the medial inferior quadrant of the buttocks leading to muscle spasm, myositis, and entrapment of nerves within the muscle have also been reported.

Sitting on a hard seat will increase the compression of the nerves in the buttocks or underneath the ischium, and there is the possibility of a subischial tunnel syndrome where the nerves can be trapped at the insertion of the hamstring muscles [8]. Mechanical damage to the inferior cluneal nerves can occur during their course through the piriformis [7].

Injection Technique

Landmark-Guided Technique

The procedure may be difficult to perform blindly and will depend on the ability to adequately palpate the patient's anatomy. The patient should be placed either in the prone position

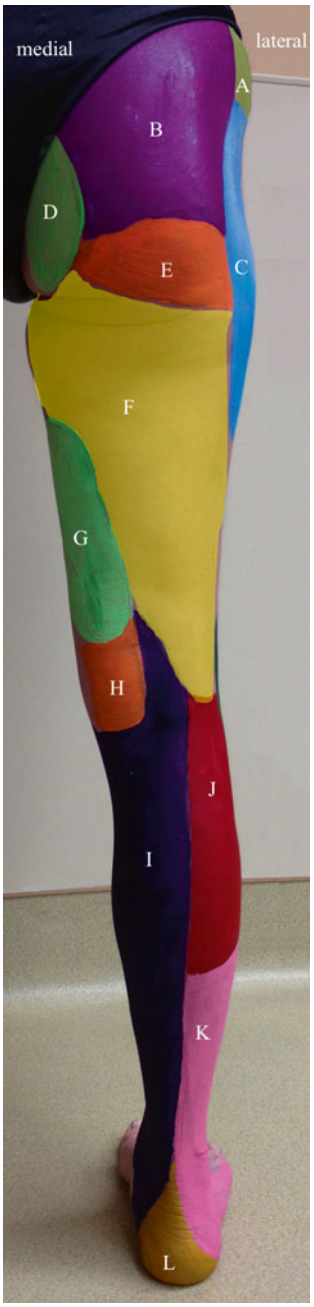

Fig. 63.7 Pain pattern from nerves of the posterior leg. *A* lateral branch iliohypogastric nerve, *B* superior cluneal nerve, *C* lateral femoral cutaneous nerve, *D* middle cluneal/sacral nerve, *E* inferior cluneal nerve, *F* posterior femoral cutaneous nerve, *G* obturator nerve, *H* femoral nerve, *I* saphenous nerve, *J* lateral sural cutaneous nerve, *K* superficial peroneal nerve, *L* medial calcaneal nerve (Image courtesy of Terri Dallas-Prunskis, MD)

Table 63.3 Origin of the perineal branch of posterior femoral cutaneous nerve (PBPFCN)

From PFCN directly	55 %
From inferior cluneal nerve	30 %
Absent	15 %

Table was constructed using results of data from Tubbs et al. [3] study of 20 dissected adult cadavers (40 sides)

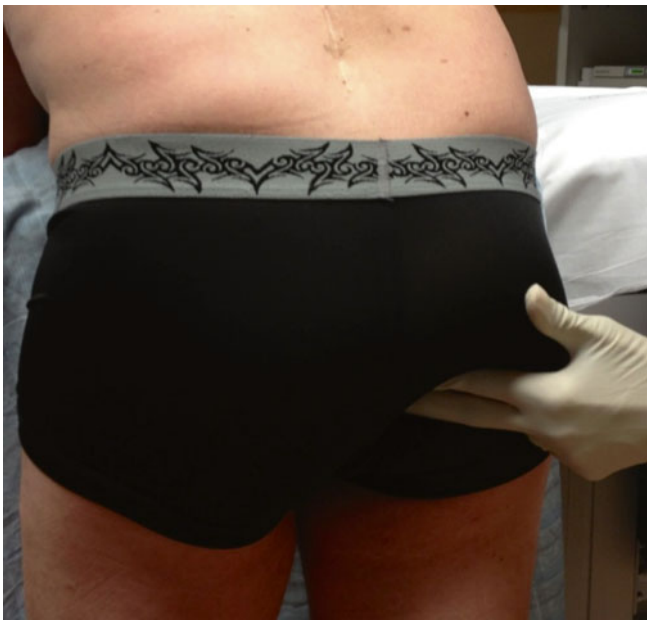

Fig. 63.8 Physical exam of the inferior cluneal nerve, showing palpation over the inferior cluneal nerve near the sciatic notch (Image courtesy of Terri Dallas-Prunskis, MD)

Table 63.4 Differential diagnosis of buttock pain

	Potential distinguishing features
Sciatica	Weakness, numbness, or difficulty moving the leg or foot
Posterior femoral cutaneous neuritis	Innervates the lateral and lower portions of the gluteus maximus muscle and the posterior parts of the leg and thigh and the skin of the perineum
Obturator neuritis	Medial thigh or groin pain, weakness with leg adduction, and sensory loss in the medial thigh
Piriformis syndrome	Pain, tingling, and numbness in the buttocks and along the path of the sciatic nerve descending down the lower thigh and into the leg and hypertonicity of the piriformis muscle
Obturator internus muscle spasm	Difficulty with lateral rotation of the femur with hip extension and abduction of the femur with hip flexion, instability of the femoral head in the acetabulum, and hypertonicity of the obturator internus muscle

or standing while leaning securely over either a cart or a bed. Utilizing aseptic technique, prep the buttocks, then palpate the inferior aspect of the ischium, and mark the site. Next, localize the gluteus maximus muscle and the lateral edge of the hamstring muscle insertion. After local infiltration to the skin and subcutaneous tissue, insert a 22-gauge 3.5-in. needle through the gluteus maximus on the lateral edge of the hamstring muscle insertion at the lateral and inferior edges of the ischium (Fig. 63.9). Following a negative aspiration, inject 2–3 cc of a local anesthetic and steroid solution. Utilizing a

Table 63.5 Diagnostic tests for inferior cluneal neuralgia

	Potential distinguishing features
Physical exam	Palpation should elicit non-radiating pain increasing with deep pressure over the sciatic notch; hyperesthesia to pin scratch and decreased sensation to touch over the inferior buttocks corresponding to the nerve distribution
Diagnostic injection	Utilizing landmark or fluoroscopic-guided technique local anesthetic and steroid solution may be injected through the gluteus maximus directed toward the lateral and inferior edge of the ischium
Ultrasound	The nerves are located by identifying the inferior border of the ischium, the gluteus maximus muscle, and the lateral edge of the hamstring muscle insertion; the block needle is placed on the lateral and inferior edges of the ischium
MRI	Not useful
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies	Not useful

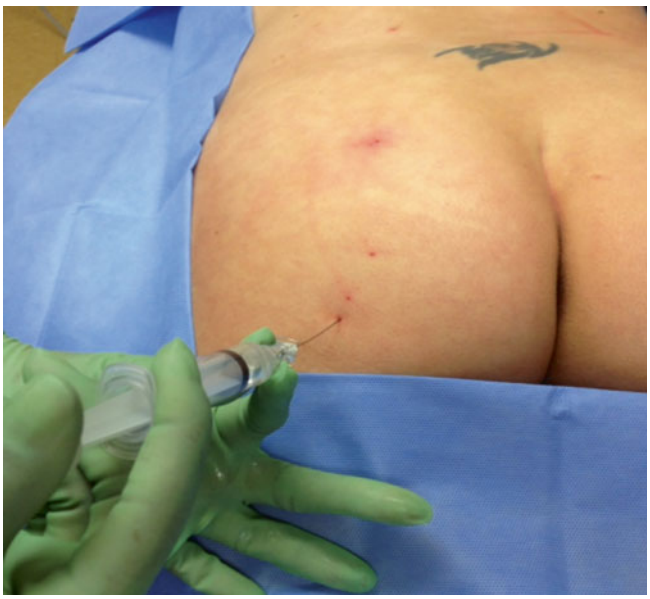

Fig. 63.9 Landmark-guided injection of the inferior cluneal nerve; the injection is performed through the gluteus maximus, lateral and inferior to the ischium (Image courtesy of Terri Dallas-Prunskis, MD)

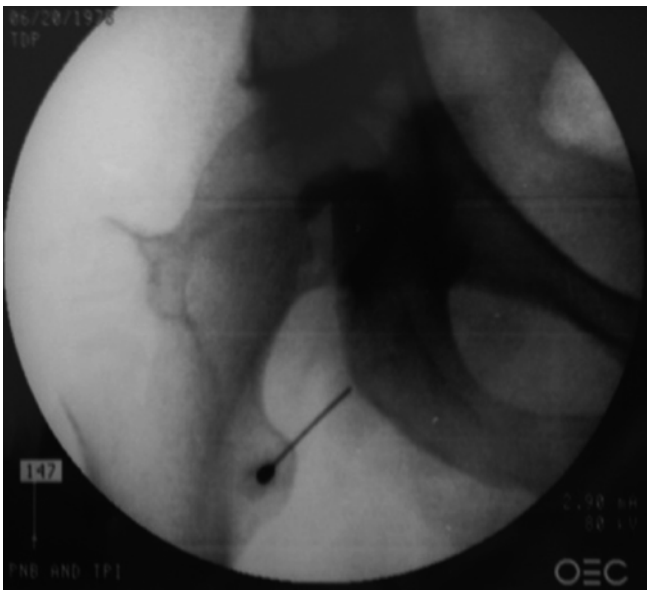

Fig. 63.10 Fluoroscopic injection of the inferior cluneal nerve (Image courtesy of Terri Dallas-Prunskis, MD)

peripheral nerve stimulator would be appropriate for this procedure.

Fluoroscopic-Guided Technique

With the patient in the prone position, the lateral and inferior edges of the ischium are identified utilizing the fluoroscopic image in an AP view. Using aseptic technique, 1 % lidocaine is infiltrated in the skin and subcutaneous tissue over the targeted point. Next, a 22-gauge 3.5-in. needle is inserted 1 cm laterally from the caudal edge of the ischium, which is under the gluteus maximus and on the lateral edge of the hamstring muscle insertion (Fig. 63.10). Following a negative aspiration, inject 2–3 cc of a local anesthetic and steroid solution. A peripheral nerve stimulator may also be used to confirm proper needle placement.

Ultrasound (US)-Guided Technique

For the US-guided injection, the patient is placed in the prone position. Palpate the inferior edge of the ischium. A high frequency (7–12 MHz) linear array probe is appropriate for this block, and an in-plane or an out-of-plane approach may be used. Locate the inferior border of the ischium (which casts a bony shadow on the US image), the gluteus maximus muscle, and the lateral edge of the hamstring muscle insertion. The skin is infiltrated with lidocaine, and a

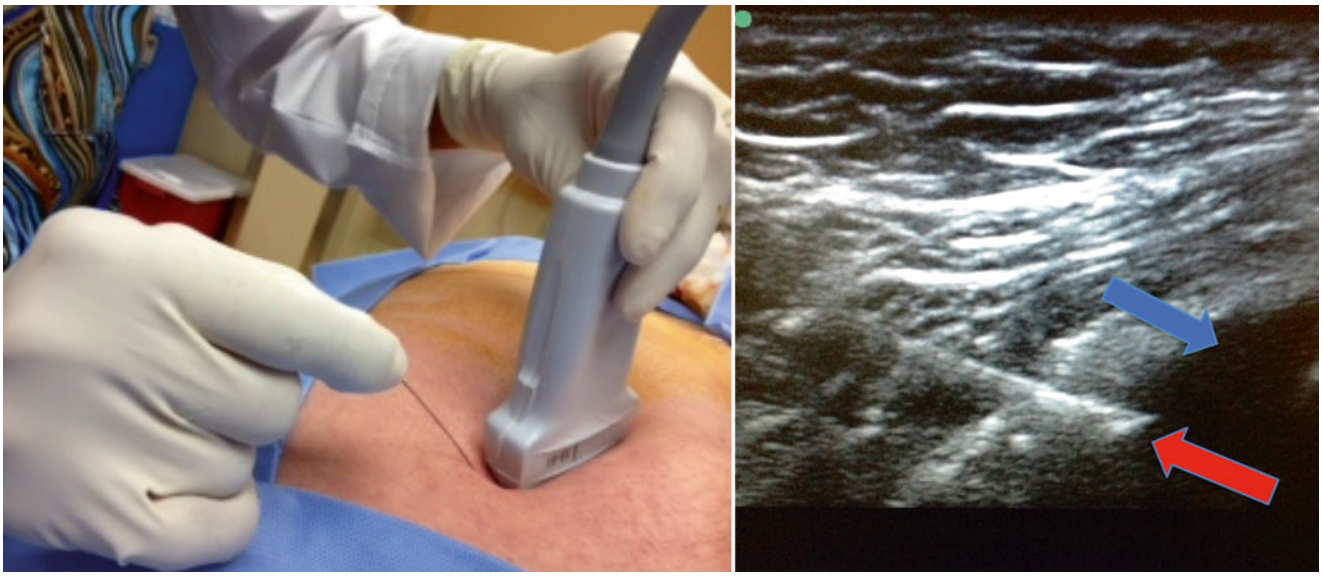

Fig. 63.11 Ultrasound image of the inferior cluneal nerve. *Blue arrow* ischium, *red arrow* injectate (Image courtesy of Terri Dallas-Prunskis, MD)

22-gauge 3.5-in. needle is inserted through the gluteus maximus on the lateral and inferior edges of the ischium (Fig. 63.11). Using US guidance, the ICN can be blocked with 2–3 cc of a local anesthetic and steroid solution. Peripheral nerve stimulation can confirm the proper needle placement.

Neurolytic Techniques

After the injections, if there is only temporary relief of pain, neurolytic or surgical techniques may be considered. All of the neurolytic techniques should be performed using a choice of imaging.

Cryoneuroablation

Cryoneuroablation may be performed at the lateral and inferior edges of the ischium, with the patient in the prone position. Utilizing an aseptic technique, a small amount of local anesthetic is infiltrated subcutaneously using a 25-gauge 1.5-in. needle. A small incision is made into the skin, and an introducer needle (size 12 or 14 gauge, depending on the probe size) is advanced to the target area. The stylet is removed, and the cryoprobe is then advanced through the catheter. The tip of the probe is exposed by withdrawing the catheter back into the subcutaneous tissues. The probe placement should be confirmed with maximal sensory stimulation and negative motor stimulation. This should be followed by a series of three 2-min freezes, with a 30 s defrosting between each cycle. The patient may experience burning pain initially during the first freeze cycle, which often replicates the pain, that should resolve within approximately 30 s.

Radiofrequency Lesioning (RF)

Radiofrequency lesioning has also been utilized for extended pain relief of inferior cluneal neuropathies following successful infiltration. The patient is placed in a prone position, and, utilizing imaging in an AP view, the lateral and inferior edges of the ischium are identified. Using aseptic technique, the skin is anesthetized subcutaneously, followed by insertion of the radiofrequency cannula, which is advanced to the target site at the ischium. After the radiofrequency probe is advanced through the cannula appropriately, maximal sensory and negative motor stimulation is used to confirm that the tip of the probe is placed adequately. Pulsed RF may provide relief, but conventional RF should be discouraged because of the risk of neuritis.

Surgical Technique

Surgery may be considered after the infiltration provides improvement or temporary pain relief. Two surgical approaches have been discussed in the literature. A transgluteal approach for decompression and transposition of the ICN is described when the clunealgia is caused by a piriformis syndrome [6]. The second approach is used when there is an isolated clunealgia with an ischial entrapment. This surgical approach is from the dorsal and cranial parts of the thigh [4].

Complications

General complications may occur, based on the location of the needle placement, including neural trauma, hematoma formation, infectious complications including abscess, and side effects related to the administration of local anesthetic and/or steroid and other drugs. Caution must be exercised when performing the procedures blindly to make sure to come into contact with bone, so as not to place the needle too deeply.

When performing cryoneuroablation, depigmentation or hyperpigmentation at the cryolesion site has been reported, though cryoneuroablation at this site is relatively deep [8].

The most common complications of radiofrequency include those related to the placement of the needle and those related to the neurolysis. The majority of problems are short lived and self limited, and they include local swelling and pain at the site of the needle insertion, as well as somatic pain from the site of insertion. Other reported complications of radiofrequency thermoneurolysis include a worsening of the usual pain, burning or dysesthesias, decreased sensation, and allodynia over the skin [9].

Summary

The inferior cluneal nerve is a cause of pelvic pain, low back pain, and upper leg pain. It is rarely diagnosed and even more rarely taught. Understanding the clinical presentation and the physical exam will perhaps increase the awareness and therefore treatment of the entrapment syndrome of this potentially debilitating problem.

References

1. Hibner M, Desai N, Robertson LJ, Nour M. Pudendal neuralgia. J Minim Invasive Gynecol. 2010;17(2):148–53.
2. Netter FH. Atlas of human anatomy. ICON Learning Systems, 1997, pp. 468–509.
3. Tubbs RS, Miller J, Loukas M, Shoja MM, Shokouhi G, Cohen-Gadol AA. Surgical and anatomical landmarks for the perineal branch of the posterior femoral cutaneous nerve: implications in perineal pain syndromes. Laboratory investigation. J Neurosurg. 2009;111(2):332–5.
4. Darnis B, Robert R, Labat JJ, Riant T, Gaudin C, Hamel A, Hamel O. Perineal pain and inferior cluneal nerves: anatomy and surgery. Surg Radiol Anat. 2008;30:177–83.
5. Labat JJ. Pudendal neuralgia: clinical signs and diagnosis. In: Urogenital iain in clinical practice. First edition. London: Informa Healthcare Publishing; 2007. p. 361–372.
6. Robert R, Prat-Pradal D, Labatt JJ. Anatomic basis of chronic perineal pain: role of the pudendal nerve. Surg Radiol Anat. 1998;20:93–8.
7. Hanson D. Intramuscular injection injuries and complications. Gen Pract. 1963;27:109–15.
8. Trescot A. Cryoneurolysis. In: Manchikanti L, Singh V, editors. Interventional techniques in chronic non-spinal pain. Paducah: ASIPP Publishing; 2009. p. 69–86.
9. Trescot A, Hansen HC. Radiofrequency neurolysis. In: Manchikanti L, Singh V, editors. Interventional techniques in chronic non-spinal pain. Paducah: ASIPP Publishing; 2009. p. 59–68.

Obturator Nerve Entrapment: Lower Extremity

64

Andrea M. Trescot and Helen W. Karl

Introduction

Groin and medial thigh pain are often difficult to diagnose due to the many structures that pass through that region, and *obturator nerve* (ON) entrapment is not commonly identified or considered. An alternate name for this entrapment is *obturator tunnel syndrome*. ON dysfunction can present as groin pain (see Chap. 48) and/or lower extremity pain. This chapter will discuss the presentation, diagnosis, and treatment of ON entrapment as a cause of lower extremity pain.

Clinical Presentation (Table 64.1)

Patients with obturator entrapment present with medial thigh pain (Fig. 64.1) often related to participation in sports, especially those involving kicking or twisting [19]. Patients complain of paresthesias, sensory loss, or pain, from the hip to the knee along the medial aspect of the thigh (and occasionally to the calf). Males seem to be much more affected than females, perhaps because of the sports association [1]. In fact, in one study, 81 % of athletes with ON entrapment were soccer players [19]. Patients note exercise-induced medial thigh pain and adductor muscle weakness and may complain of paresthesias along the medial or anteromedial thigh and knee (Fig. 64.2), with pain on adduction or a monopedal stance [20]. Other common symptoms include sensory loss or a deep ache in the medial thigh from the pubis to the medial knee, occasionally to the ipsilateral anterior superior iliac spine (ASIS) [1] and very rarely to the medial calf [21]. Pain is worsened by maneuvers, such as extension or lateral rotation of the leg, which stretch the nerve [4], and medial knee pain can be induced by forced hip abduction, extension, and internal rotation (*Howship-Romberg's sign*) [22]. There may be a sense of "lack of propulsion… during running but numbness is very rarely reported" [23]. As with claudication, the symptoms subside with rest but reoccur with the resumption of activity. According to Sorenson, 73 % of patients with known ON entrapment complained of groin and thigh pain [20].

Pain and restriction of hip movement is often attributed to osteoarthritis ("coxarthrosis"). However, obturator pathology may mimic or contribute to hip pathology, and ON

Table 64.1 Occupation/exercise/trauma history relevant to obturator nerve entrapment

Sports injuries	Kicking sports, such as soccer [1]
Trauma	Stress fracture pubic ramus or fracture of the pelvis [2, 3]
	Gunshot wounds [4]
Infection/inflammation	Pubic osteitis [5]
Surgery	Intra-abdominal surgery [6]
	Laparoscopic pelvic lymphadenectomy [7]
	Genitourinary surgery [8]
	Urologic surgery [9]
	Hip replacement [10–14]
	Cardiac catheterization [4]
Mechanical	Prolonged lithotomy position [15]
	Improper leg position holder [4]
Genitourinary pathology	Endometriosis [4, 16]
Compression	Acetabular cyst/obturator foramen tumor [17, 18]
	Obturator hernia [4]
	Pelvic hematomas, retroperitoneal masses [1]
	Pregnancy [4]
	Pelvic tumor [4]
	Aneurysm of hypogastric artery [4]

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Fig. 64.1 Patient complaints of pain from obturator nerve entrapment (Image courtesy of Andrea Trescot, MD)

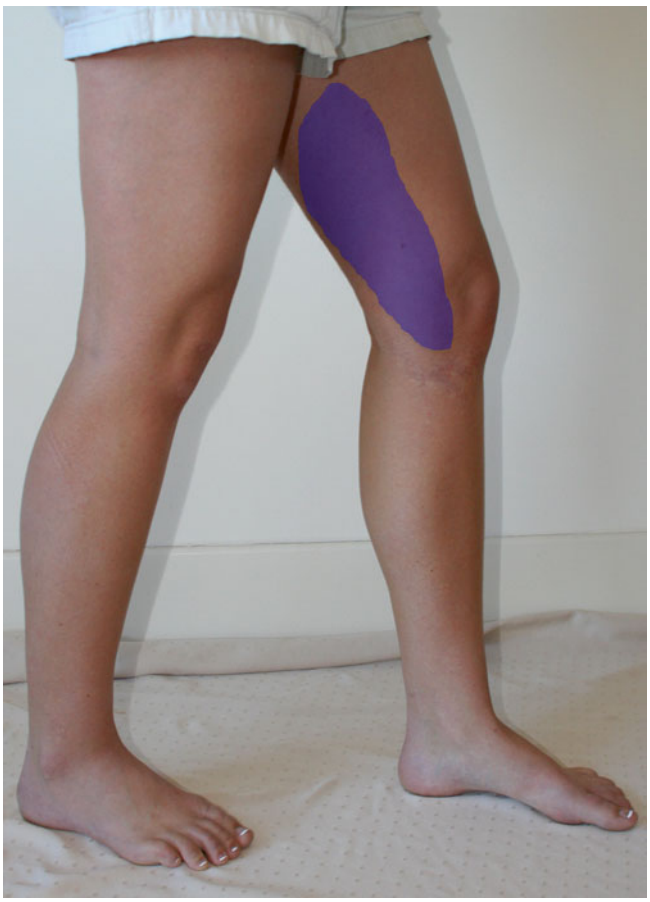

Fig. 64.2 Pattern of pain from obturator entrapment (Image courtesy of Andrea Trescot, MD)

injections with subsequent denervation may offer relief to patients who are not hip arthroplasty candidates (see the *RF section* below). In addition, although nerve injuries after hip arthroplasty are not common (0.7–1 % of all postoperative complications), ON pathology after total hip replacement (THR) can be a cause of persistent hip or groin pain as well as varying degrees of adductor weakness [11]. Unwin and Scott described potential obturator damage from any surgery that violates the floor of the acetabulum (including cement extravasation) and suggested that obturator pathology may account for some of the "obscure patterns of pain" following hip replacement [24].

Bradshaw et al. [1] described 32 athletes who had obturator neuropathy from fascial entrapment at the thigh; the patients complained of exercise-induced medial thigh pain. Busis noted that the obturator nerve can be injured by gunshot wounds and pelvic fractures, during laparoscopic pelvic lymph node dissections, by pelvic hemorrhage, by endometriosis or pelvic tumors, or by aneurysm of the hypogastric artery, as well as by cardiac catheterizations that cause retroperitoneal hematoma [4].

Anatomy (Table 64.2)

The ON arises from the anterior divisions of the ventral rami of L2, L3, and L4, which join within the *psoas muscle,* as part of the *lumbar plexus*, to form the ON (Table 64.2). This is in contrast to the *femoral nerve*, which is formed by the posterior divisions of the ventral rami of those same nerves; the ON travels more medially than the femoral nerve. The ON exits the medial aspect of the psoas at the level of the sacroiliac joint (Fig. 64.3) and travels over the pelvic brim, curving anterior-inferiorly along the lateral pelvic wall. The nerve then passes into the *obturator foramen* through a fibro-osseous tunnel (*obturator canal* or *obturator tunnel*), formed superiorly by the *obturator sulcus* of the pubic bone and inferiorly by the *internal and external obturator muscles* [28] (Fig. 64.4). Within the tunnel, the nerve splits into an anterior branch and a posterior branch (Fig. 64.5), as well as a branch to the external obturator muscle (which crosses the *obturator artery*) and then exits through the obturator tunnel to enter the thigh. Kumka [28] noted that the ON could bifurcate within the pelvic cavity, as well as at the entrance, within, or at the exit of obturator canal.

The anterior branch travels anterior to the *external obturator muscle* and posterior to the *pectineal muscle* and gives a branch to the hip joint, innervating the *adductor longus*, *adductor brevis*, and *gracilis* muscles, with a cutaneous branch descending in the adductor canal to innervate the skin of the distal two-thirds of the medial thigh, extending to and occasionally below the knee. The larger posterior branch pierces and supplies the *external obturator muscle* and the *adductor magnus muscle* (which is also innervated by the *sciatic nerve*) and then runs between the *short and great adductors*, with a sensory branch that also descends through the *adductor canal* to supply the *knee capsule*, *cruciate*

Table 64.2 Anatomy of the obturator nerve

Origin	Anterior divisions of ventral rami of L2–L4 nerve roots (femoral nerve is the posterior divisions of ventral rami of the same nerves)
General route	Formed within the psoas muscle, leaves its medial border and travels through the pelvis medial to the femoral nerve; follows the iliopectineal line into the lesser pelvis and through the fat-filled obturator canal with the obturator vessels
	Variable anatomy; division into the anterior and posterior branches may occur in the pelvis (23 %), in the canal (52 %), or in the thigh (25 %) [25]; an accessory ON is present in 13 % of the population [26] and may communicate with the femoral nerve
Sensory distribution	Articular branch to the anteromedial hip joint from anterior branch
	Articular branch to medial knee joint from posterior branch
	Cutaneous branch to a patch of skin on the inner thigh just above the medial knee from anterior branch; absent in 20 % of the population [27]
Motor innervation	Obturator externus
	Anterior division: adductor longus, brevis, gracilis, and sometimes pectineus (pectineus also gets motor from femoral nerve)
	Posterior division: adductor magnus (also gets motor from sciatic)
	Only the adductor brevis is innervated solely by ON
Sonoanatomy	Nerve found in the triangle formed by the lower border of the superior pubic ramus, posterior pectineus, and anterior surface of obturator externus [27]

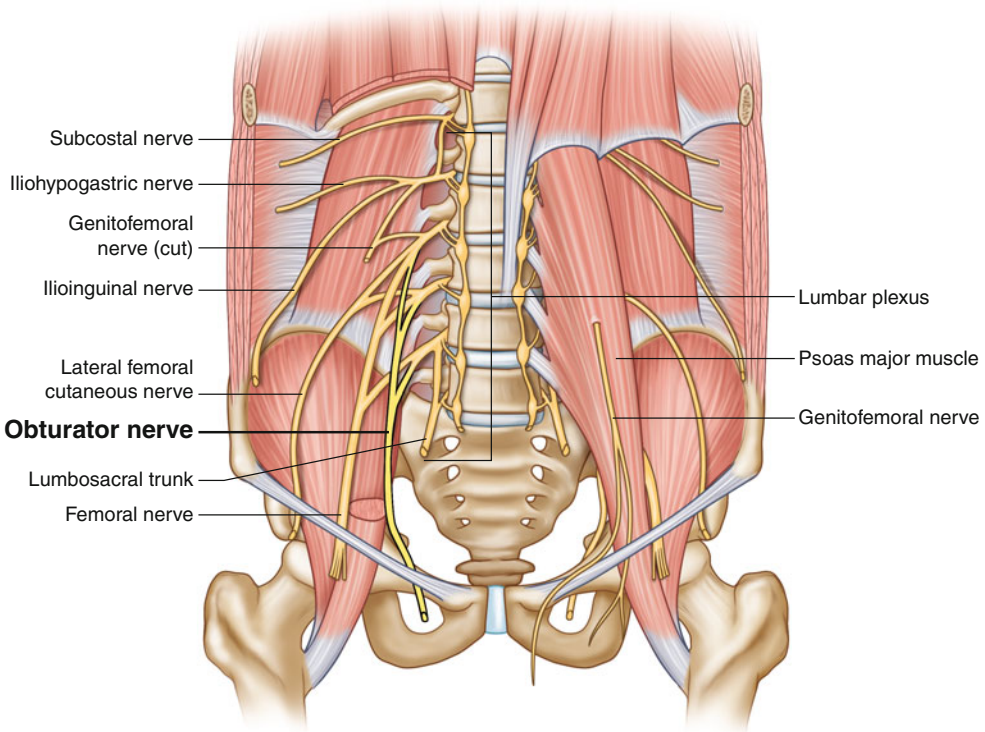

Fig. 64.3 Anatomy of the lumbar plexus nerves (Image by Springer)

ligaments, and *synovial membranes* (Table 64.3). The posterior branch occasionally innervates the *adductor brevis* and usually was found by dissection to be in immediate contact with the musculotendinous aponeurotic arch [28]. There is also an *accessory obturator nerve*, arising from L3 and L4, present in about 13 % of the population [26] that descends along the medial psoas and crosses the superior pubic ramus behind the *pectineal muscle*, providing the nerve supply to that muscle and to the hip joint [1]. The adductor magnus and longus are also partially innervated by the sciatic and femoral nerves [29]. The adductor brevis is the only muscle solely innervated by the ON [30].

The ON is seen by ultrasound (US) in the triangle formed by the lower border of the superior pubic ramus, posterior pectineus, and anterior surface of obturator externus [27]. The anterior division is easier to visualize by US than the posterior division, which is within fascia [31, 32] (see the *US injection* section below).

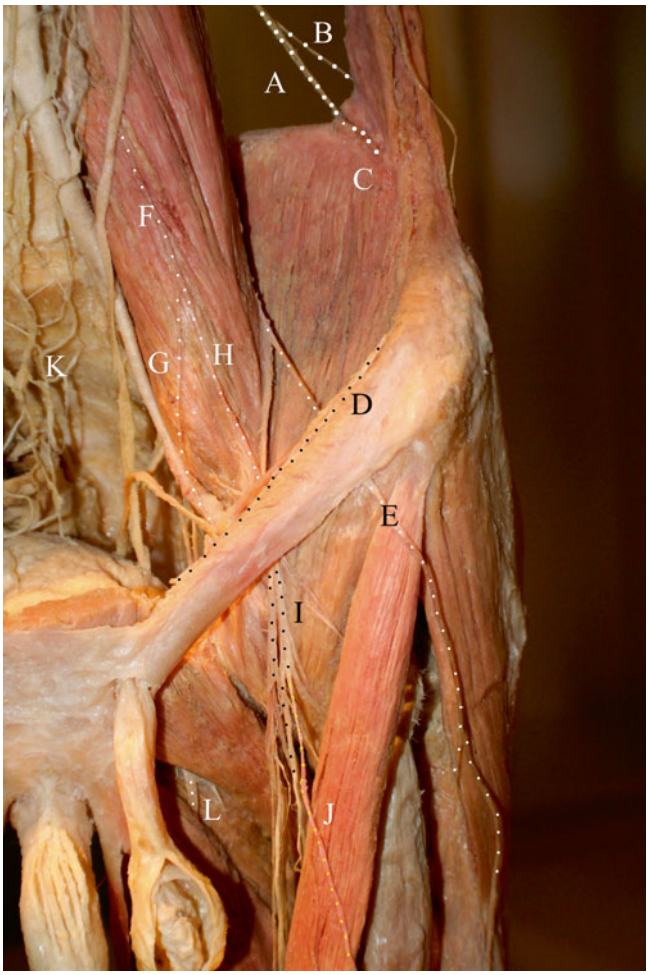

Fig. 64.4 Lumbar plexus nerves, modified from an image from *Bodies*, *The Exhibition*, with permission. *A* ilioinguinal nerve, *B* iliohypogastric nerve, *C* site of ilioinguinal nerve entrapment at the external oblique, *D* ilioinguinal nerve over the inguinal ligament, *E* lateral femoral cutaneous nerve, *F* genitofemoral nerve, *G* genital branch of the genitofemoral nerve, *H* femoral branch of the genitofemoral nerve, *I* femoral nerve, *J* saphenous nerve, *K* inferior hypogastric plexus, *L* obturator nerve (Image courtesy of Andrea Trescot, MD)

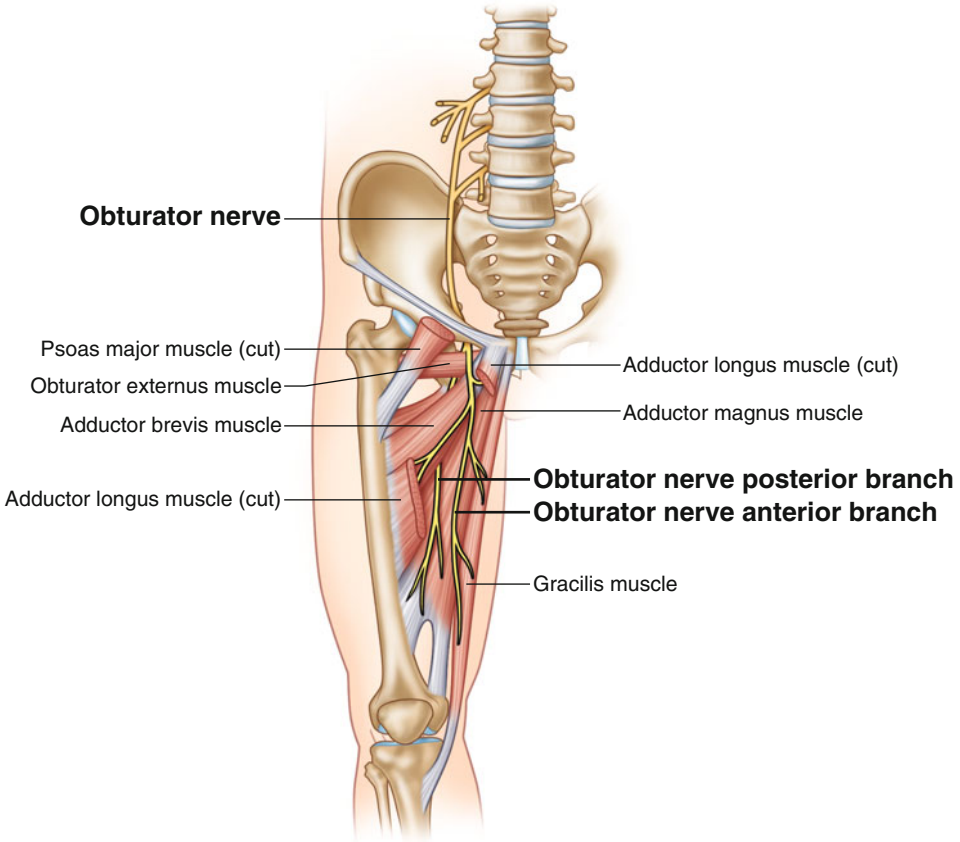

Fig. 64.5 Anatomy of the obturator nerve in the leg (Image by Springer)

Table 64.3 Kumka [28] dissected 56 lower limbs and found the following variations

Posterior division crosses over the aponeurotic arch, anterior to obturator externus	32/56 (57 %)
Posterior division emerges from obturator externus muscle, perforates it, and descends anterior to its distal part	22/56 (39 %)
Posterior division descends entirely posterior to the obturator externus muscle, emerging from its inferior border	2/56 (4 %)

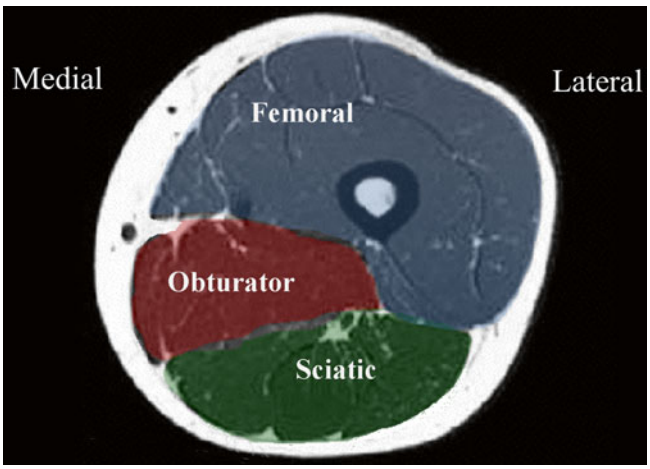

Fig. 64.6 Distribution of innervation of the proximal thigh (Image courtesy of Andrea Trescot, MD)

There have been a variety of areas described as having sensory innervation by the ON, including the medial thigh, medial knee, and superior calf. However, Bouaziz et al. [33] found that the sensory hypoesthesia was not a reliable indicator of an obturator block; adductor weakness was the only reliable sign of adequate blockade. The ON enervates the medial compartment of the thigh, the sciatic nerve enervates the posterior compartment and the femoral nerve enervates the anterior compartment (Fig. 64.6).

Entrapment

The ON is rarely injured in isolation. Prolonged, acute hip flexion, such as seen in urologic and gynecologic surgery, has been associated with *obturator palsy* [9]. The course of the obturator nerve through the pelvis puts it at risk for entrapment, compression, and damage at many sites. Several authors [5, 34] have described an *obturator tunnel syndrome*. Bradshaw and McCrory [1] described a fascial entrapment of the obturator in the adductor compartment, induced by exercise, confirmed by anatomic dissection [35]. They also suggested that the male predominance of this condition may be related to anatomy, since males have higher iliac crests, a smaller transverse diameter of the pelvic inlet, and a narrower subpubic angle, which may put a greater bend in the nerve as it passes through the obturator canal. There may also be entrapment at the fibrous edge of the *obturator membrane* [36].

Based on the dissections by Kumka [28] described above, the likely sites of entrapment include:

- Within the obturator canal, by the vascular bundle of the obturator vessels, or by complications of gynecological or orthopedic surgery (see above)
- In the fibromuscular canal formed by the anterior surface of the obturator membrane and the posterior surface of the obturator externus muscle
- In the muscular tunnel where the posterior division perforates the obturator externus muscle
- Within the distinct fascial plane situated deep to the pectineus and adductor brevis muscles and superficial to the obturator externus and the proximal one-third of the adductor magnus muscles

Several authors [10–14] described ON entrapment and/or heat damage of the femoral and obturator nerves caused by cement extrusion from hip arthroplasty. There may also be entrapment from fibrous bands formed due to chronic *adductor tendinopathy* or *pubic osteitis (osteitis pubis)*.

Physical Exam

The hallmark of obturator pathology is adductor weakness (Fig. 64.7), with muscle atrophy in severe cases [21]. The *obturator externus* laterally rotates the thigh, while the *gracilis* provides flexion and internal rotation. With loss of adduction and internal rotation, the hip will be externally rotated and abducted, which results in a wide-based, circumducting gait [37]. Ipsilateral loss of the hip adductor tendon reflex suggests, but does not prove, obturator pathology, since this reflex is not always present even in healthy people [21]. There may be tenderness to deep palpation in the proximal adductor canal (Fig. 64.8), as well as increased pain when the nerve is stretched by extension and/or lateral leg movement [4]. A *pectineus stretch* (where the patient actively externally rotates and abducts the hip) will stretch the obturator nerve [29]; pain may also be reproduced with internal rotation of the hip against resistance [30] (known as an *obturator sign*) (Fig. 64.9).

Objective measures of adductor strength were devised by Lang [38] and used by several subsequent investigators [39, 40].

Fig. 64.7 Adduction of thigh against resistance (Image courtesy of Andrea Trescot, MD)

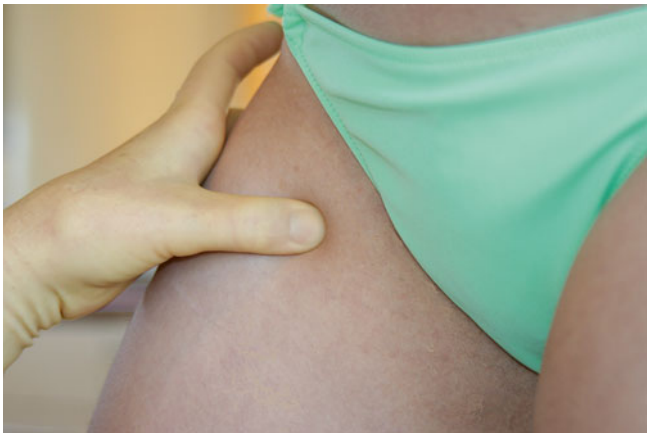

Fig. 64.8 Physical exam of the obturator nerve (Image courtesy of Andrea Trescot, MD)

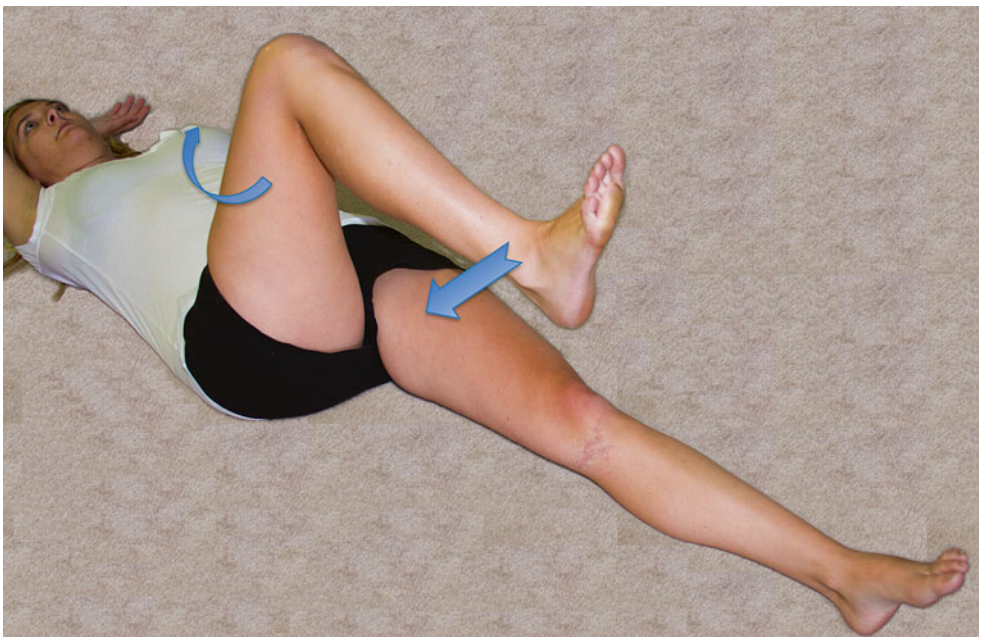

Fig. 64.9 Obturator sign – internal rotation of the hip against resistance (Image courtesy of David Trescot)

Differential Diagnosis (Table 64.4)

The innervation of the hip joint includes the femoral, sciatic, superior gluteal, and obturator nerves (Fig. 64.10). Groin and hip pain can be due to hip osteoarthritis (OA), avascular necrosis, adductor or rectus abdominis tendonitis, greater trochanter bursitis, adductor spasm, pubic osteitis, stress fracture, pregnancy (by the fetal head, forceps, or hematoma), aneurysm of the hypogastric artery, lumbar radiculopathy, cardiac catheterization (from retroperitoneal hematoma), and sports hernia (*obturator hernia*) [43]. Weakness of the hip adductors can be caused by a lumbar radiculopathy or plexopathy [4]. Anterolateral thigh pain can come from the articular branches of the femoral nerve. Travell and Simons [44] described trigger points (myofascial pain) of the long adductor muscle as "perhaps the most common cause of groin pain," though Bradshaw and McCrory [1] point out that at least one of the patients injected by Travell and Simons had a "clear-cut obturator block." Because the ON innervates both the knee and hip, the evaluation of chronic hip or knee pain needs to include ON pathology in the differential diagnosis.

Plain films can show small bone spurs in the region of the ipsilateral pubic tubercle. In the 32 athletes with groin pain studied by Bradshaw and McCrory [1], 21 of 24 patients showed bone scan uptake on the ipsilateral pubic tubercle at the origins of the short and long adductors. MRI may show atrophy of the adductor brevis and longus or gracilis muscles [45], as well as identify intrapelvic masses that might entrap the obturator nerve [37] (Fig. 64.11). EMG/NCV studies can confirm the entrapment, showing fibrillation or complex motor unit potentials of the short and long adductor

Table 64.4 Differential diagnosis of hip and groin pain

	Potential distinguishing features
Osteoarthritis hip or knee	X-ray and MRI show DJD
Adductor or rectus abdominis tendonitis/myofascial spasm	Tenderness at pubis; palpable spasm
Avascular necrosis	X-ray and MRI show femoral head collapse
Pubic osteitis or stress fracture	Bone scan positive
Facet arthropathy [41]	Paravertebral tenderness, lumbar spondylosis
SI joint [41]	PSIS tenderness
Piriformis muscle syndrome [41]	Increased pain with hip internal rotation
L1–L3 radiculopathy [41]	EMG positive
Hip flexor tendonitis [41]	Tenderness of tendon attachment
Abdominal wall hernia [19];	Abdominal wall tenderness
Sports hernia [42]	
Knee pathology [41]	Knee tenderness
Hip joint pathology [29]	Stiffness, limited range of motion, crepitus, clicking
Ilioinguinal N injury/entrapment [19]	Increased abdominal wall tension can result in groin pain; may be tender near ASIS; increased pain with hip hyperextension
Pubic symphysitis [19]	Pain moves from side to side
Adductor strain [29]	Tenderness over adductors; usual site is at the muscle-tendon intersection; sometimes at tendon-bone

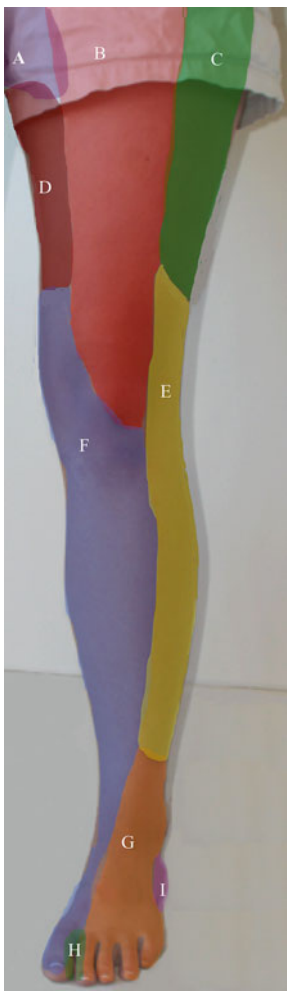

Fig. 64.10 Pattern of pain from anterior lower extremity nerve entrapments. *A* Genitofemoral nerve, *B* femoral nerve, *C* lateral femoral cutaneous nerve, *D* obturator, *E* lateral sural cutaneous nerve, *F* saphenous nerve, *G* superficial peroneal nerve, *H* deep peroneal nerve, *I* sural nerve (Image courtesy of Andrea Trescot, MD)

muscles, while the iliopsoas and quadriceps are normal. Bradshaw et al. [46] reported that all 32 of the groin pain patients they studied had EMG evidence of denervation changes to the obturator nerve with normal paravertebral muscles. The diagnostic tests for ON entrapment are listed on Table 64.5.

Identification and Treatment of Contributing Factors

Transurethral prostate resections (TURP) are occasionally associated with adductor spasms, which can be relieved with ON injections.

Bradshaw and McCrory [1] noted that several of the patients in their series had symptoms of *inguinal hernia* prior to the development of medial thigh weakness, and they proposed the possibility of a mechanical entrapment. Kashuk [36] felt that inflammatory changes and edema from *pubic osteitis* were a major cause of obturator pathology.

Entrapment of the ON by the external obturator muscle may cause a persistent pathology. Kassolik et al. [47] describe the use of massage of the obturator and piriformis muscles to treat ON entrapment.

Abelson and Abelson [48] describe a *neural mobilization* ("*neural flossing*") (see Chap. 5) technique of treating obturator entrapment. The patient is positioned seated with the hands behind the back and neck flexed; as the affected leg is abducted, there will be an increased tension on the nerve, causing increased pain, which should resolve with raising the head. The technique initially involves having the patient extend the neck as they abduct the leg; having the patient flex the neck as they abduct the leg will provide more aggressive mobilization of the nerve.

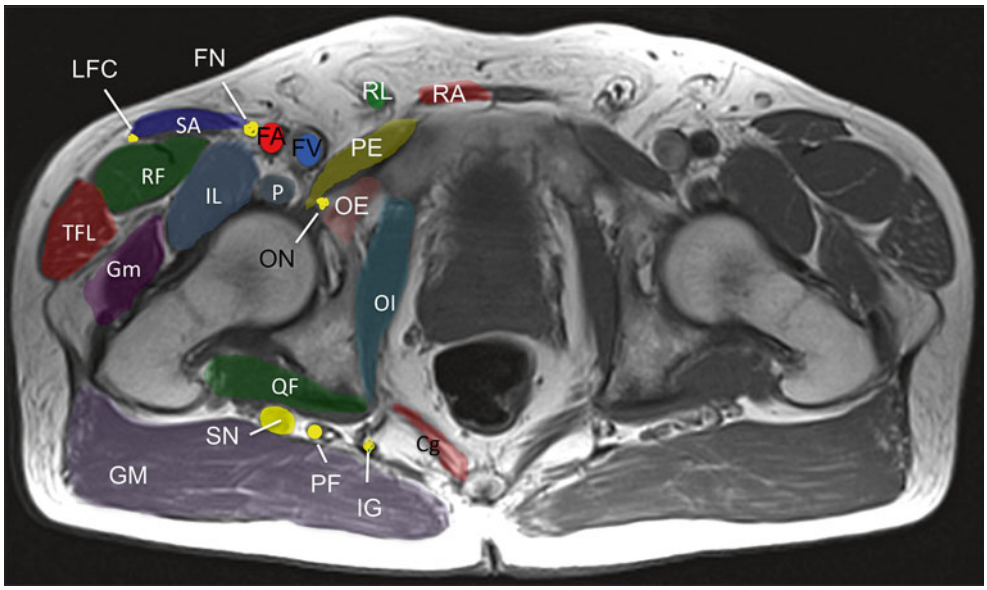

Fig. 64.11 Axial MRI of the pelvic structures. *Cg* coccygeus muscle, *FA* femoral artery, *FN* femoral nerve, *FV* femoral vein, *GM* gluteus maximus muscle, *Gm* gluteus medius, *IG* inferior gluteal nerve, *IL* iliopsoas muscle, *LFC* lateral femoral cutaneous nerve, *OI* obturator internus muscle, *P* psoas muscle, *PE* pectineus muscle, *PF* posterior femoral cutaneous nerve, *QF* quadratus femoris muscle, *RA* rectus abdominis muscle, *RF* rectus femoris muscle, *RL* round ligament, *SA* sartorius muscle, *SN* sciatic nerve, *TFN* tensor fascia lata muscle (Image courtesy of Andrea Trescot, MD)

Table 64.5 Diagnostic tests for obturator nerve entrapment

Test	Potential distinguishing features
Physical exam	Adductor weakness; wide-based, circumducting gait [37]
Diagnostic injection	At the obturator foramen
X-rays	Pubic ramus spurring; rule out other causes
Bone scan	May show increased uptake at pubic tubercle
MRI	Atrophy of adductor brevis and longus or gracilis [45]; rule out other causes
Arteriography	Not useful
Ultrasound	Rule out intrapelvic mass lesions
Electrodiagnostic studies	Needle EMG is consistent with acute and chronic denervation of the adductor muscles with normal readings in other lower extremity muscles

Injection Technique

ON injections are used to help to confirm the diagnosis of obturator neuropathy. ON injections have been used to treat adductor spasms from cerebral palsy or during and after TURP [49], to supplement analgesia for knee surgery, and to treat chronic hip pain. Bouaziz et al. [33] stated that the only way to reliably confirm obturator anesthesia is to assess adductor strength.

Landmark-Guided Technique

Winnie et al. [50] introduced a "three in one" anesthesia injection, designed to anesthetize the femoral, lateral femoral cutaneous, and obturator nerves at the same time (Fig. 64.12).

More specifically, the ON can be blocked either proximal or distal to the division into the anterior and posterior branches. Proximally, only one injection is necessary, while after the division, it is necessary to inject both nerves separately. The standard landmark-guided ON nerve injection involves positioning the patient supine, with the leg slightly abducted and externally rotated. The needle is then placed 2 cm caudad and 2 cm lateral to the pubic tubercle, advanced onto the inferior border of superior pubic ramus, and dropped into the obturator canal (Fig. 64.13) [51], with or without the use of a peripheral nerve stimulator (PNS).

The more distal, inguinal approach has the patient positioned supine with the leg slightly abducted. The patient is asked to flex the hip, and a line is drawn to mark the inguinal crease. The adductor longus tendon will be the most superficial palpable tendon in the medial thigh. A mark is made at the midpoint of the adductor longus and the femoral artery, at the site of the groove between the vascular bundle and the adductor. The needle is inserted at this site and advanced at a 30° cephalad direction with a PNS until contractions of the gracilis or adductor muscle are identified (which identifies the anterior branch). Five cc of local anesthetic is injected here and then the needle is advanced deeper and 5° laterally to obtain contractions of the adductor magnus (posterior

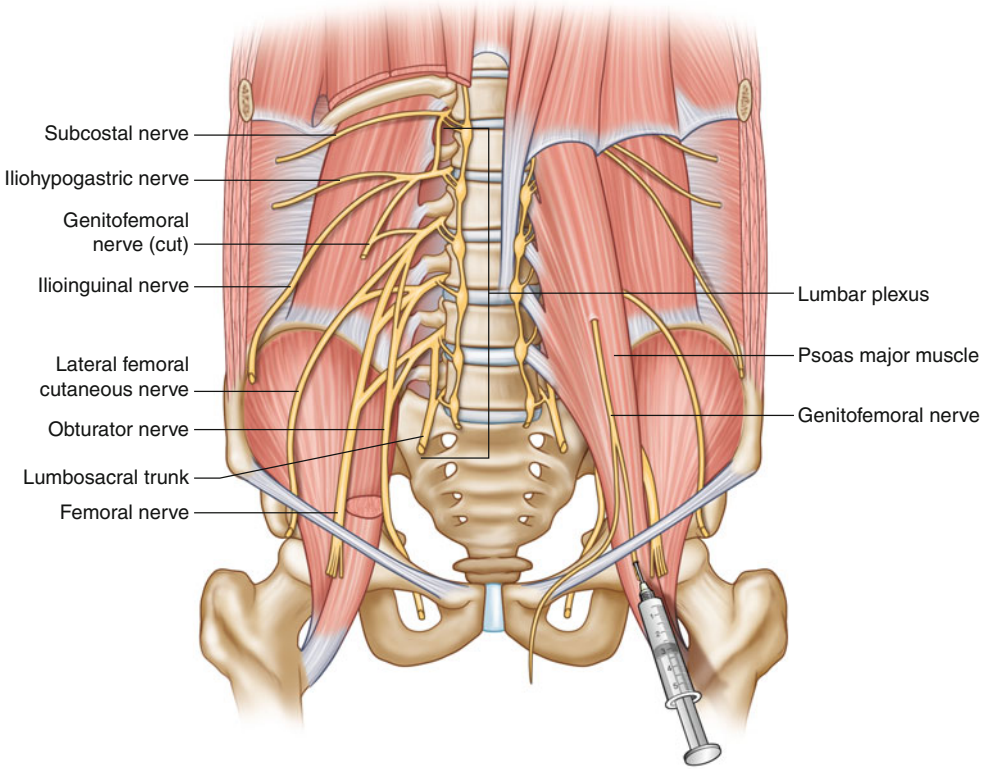

Fig. 64.12 Needle location for inguinal perivascular injection (Image from Springer)

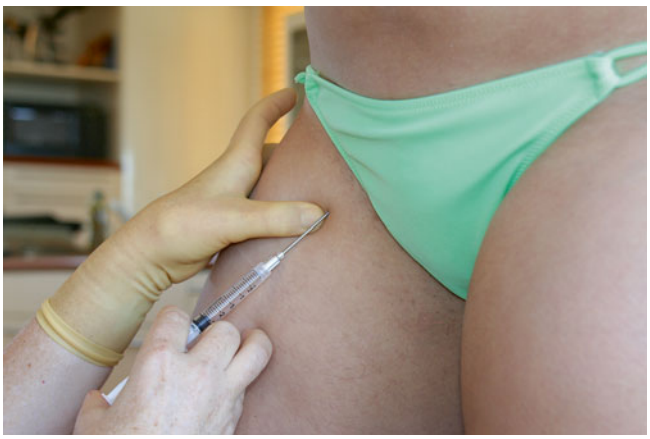

Fig. 64.13 Landmark-guided obturator injection (Image courtesy of Andrea Trescot, MD)

branch), where another 5 cc of local anesthetic is injected. In 2005, Choquet et al. [40] performed a randomized trial of 50 patients undergoing knee arthroscopy, comparing an inguinal approach versus the standard approach to the obturator nerve. Patient in the inguinal group reported less pain during the procedure, and there were no failures. This group also confirmed that motor strength, not sensory hypoesthesia, was the only reliable way to evaluate nerve function, and they noted that 48 % of their patients reported no areas of sensory hypoesthesia 20 min after the injection.

Fluoroscopy-Guided Technique

Using fluoroscopy to aid the needle placement for an ON injection involves the fluoroscopic visualization of the superior and inferior pubic rami. The patient is placed supine, and the inferior portion of the obturator foramen is identified. A peripheral nerve stimulator can help to identify the nerve (Fig. 64.14).

The hip joint is innervated by sensory branches of the obturator and femoral nerve (see Chap. 57). For diagnostic ON injections of the hip joint, the patient is placed supine, and the needle is placed just medial to the femoral artery below the inguinal ligament; the needle tip is then directed under fluoroscopy to the inferior junction of the ischium and pubis, where the "teardrop" landmark is made up of the wall of the acetabulum, the lesser pelvis, and the acetabular notch (Figs. 64.14 and 64.15).

Ultrasound (US)-Guided Technique

Some authors use US alone, while others use US with a nerve stimulator [39]. Soong et al. [52] described two techniques of finding the nerve under US. The patient is examined supine, with the thigh slightly externally rotated. The first technique involves scanning laterally from the pubic tubercle until the three muscle layers (adductor longus,

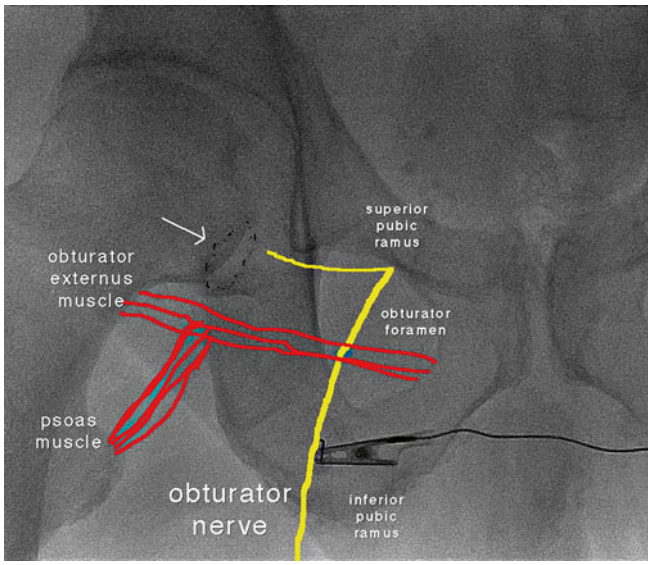

Fig. 64.14 Fluoroscopic anatomy and obturator injections sites. *White arrow* identifies the "teardrop" target for obturator hip denervation technique. Note the peripheral nerve stimulator on the needle, used to find the main obturator nerve (Image courtesy of Andrea Trescot, MD)

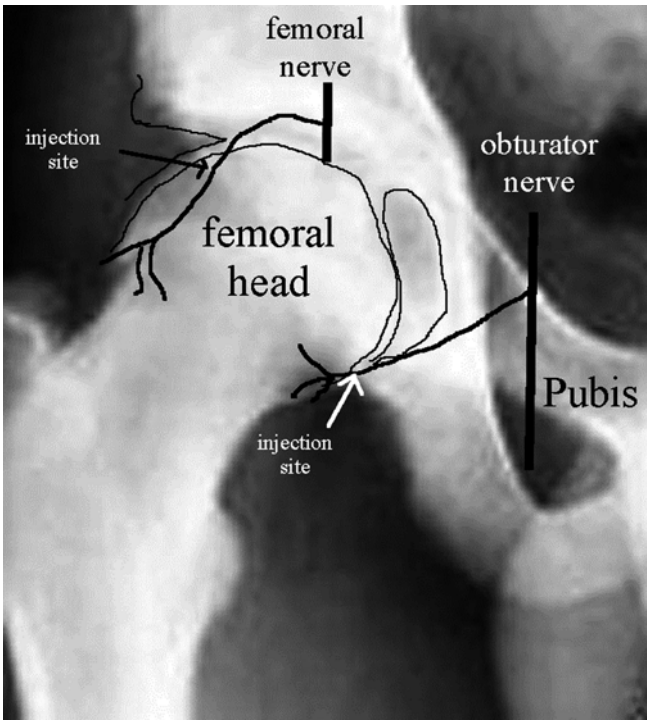

Fig. 64.15 Injection sites for denervation of the hip joint. *White arrow* identifies the injection site for the obturator nerve; black arrow identifies the femoral branch site (Image courtesy of Andrea Trescot, MD)

adductor brevis, and adductor magnus) were identified, moving the probe in a medial/lateral or proximal/distal direction to find the anterior and posterior divisions of the obturator nerve (Fig. 64.16). In the second technique, the femoral artery/vein/nerve is identified at the femoral crease, and the probe is moved medially toward the pubis to visualize the obturator nerve, which, unlike other nerves, appears flat instead of honeycombed.

Manassero et al. [53] compared two types of US-directed adductor injections – using either a peripheral nerve stimulator (PNS) or an intrafascial injection – on 716 patients undergoing TURP in a lithotomy position. The symptomatic leg was lowered from the lithotomy position, extended, and slightly externally rotated. An ultrasound probe for one group (interfascial) was positioned at 90 degrees to the skin, parallel to, and 2–3 cm below the inguinal crease. The probe was positioned medially from the femoral nerve until the muscle layers of the adductor longus, adductor brevis, and adductor magnus could be visualized. A 22-guage insulated needle was advanced using an in-plane approach, first to the fascial layer between the adductor longus and brevis (where 5 cc of local anesthetic was injected) and then advanced between the adductor brevis and magnus (where another 5 cc of local anesthetic was injected). For the PNS group, the US probe was positioned to visualize the anterior and posterior divisions of the ON between the adductor longus, brevis, and magnus muscles. Using an in-plane approach, the needle was advanced to the posterior division of the ON, looking for adductor magnus contractures of the posterior thigh. After 5 cc of local anesthetic was injected at this site, the needle was redirected to the anterior division; once there was adductor brevis and longus contraction at low amplitude, another 5 cc of local anesthetic was injected. They found no difference in success rates, but the PNS stimulation took slightly longer; however, when there was a failure of the initial interfascial technique, adding the PNS improved the success rate to 100 %.

Using a more proximal approach has the potential advantage of more reliable blockade of branches to the hip joint. Akkaya and colleagues [27] described a more proximal approach, first dissecting the obturator structures and then identifying the structures with US. They described a "triangle," with the superior pubic ramus as the superior border, the pectineus muscle as the anterior border, and the external obturator as the posterior border. They then used eight volunteers to confirm that the structures could be identified on live patients; they were able to identify the ON in 12 out of 16 groins. This was followed by ON injections under US guidance in 15 patients, which was deemed successful in all the patients. The area around the ON is very vascular; even with US, 1 out of 15 patients had a puncture of the obturator vein. Soong and colleagues [52] felt that it was easier to see the branches (≥85 %) with US than to see the main nerve (25 %).

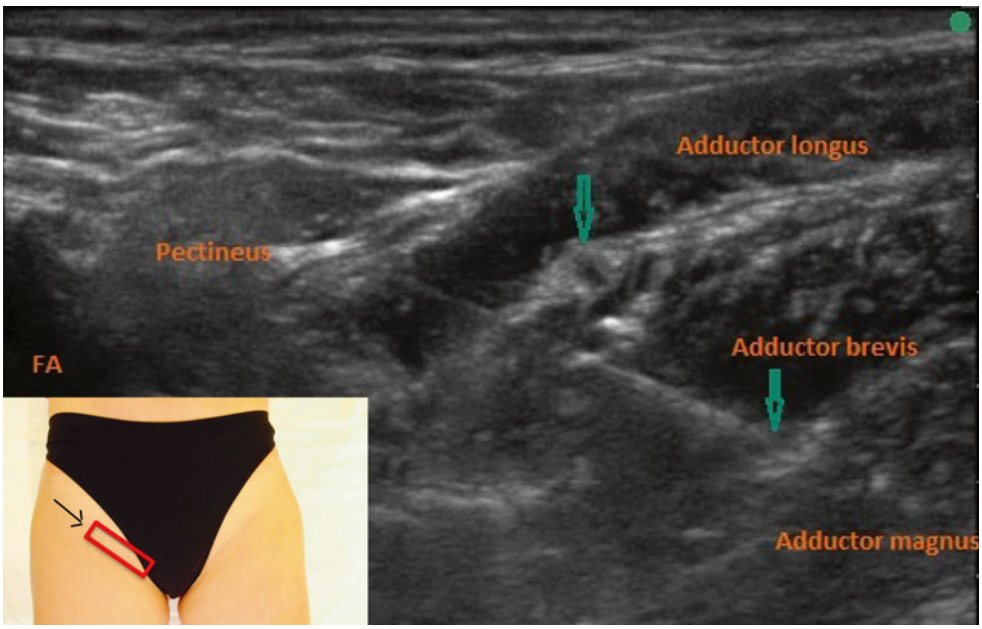

Fig. 64.16 Ultrasound needle placement for anterior and posterior branches of the obturator nerve. *Upper green arrow* marks the anterior branch of the obturator nerve, below the inguinal ligament. *Lower green arrow* identifies the needle injecting the posterior branch of the obturator nerve. *FA* femoral artery. Femoral vein is compressed and therefore not visualized (Image courtesy of Agnes Stogicza, MD)

Neurolytic Technique

Cryoneuroablation

Trescot [54] described the following cryoneuroablation technique for the ON. The patient is placed supine, with the affected limb abducted slightly. Fluoroscopy may be useful in the obese or severely spastic patient. The pubic tubercle is palpated, and local anesthetic is infiltrated subcutaneously approximately one fingerbreadth laterally and inferiorly to the tubercle. After saline with epinephrine infiltration, the 12-gauge catheter is carefully and gently advanced to the inferior border of the ramus. If done blindly, hitting the edge of the ramus will confirm depth. If done under fluoroscopy, the catheter can be directed to just below the inferior border of the ramus (Fig. 64.17). Kim and Ferrante [55] reported cryoneuroablation of the obturator nerve for the treatment of adductor spasticity and obturator neuropathy. To treat spasticity, this is one of the few times that motor stimulation for localization is appropriate. Adduction of the thigh at low voltages (0.5–1 mV) will confirm position. Spastic muscles should relax quickly, usually during the first freeze cycle. For pain, on the other hand, localization with the sensory mode is more effective, and an effort is made to avoid strong motor simulation if repositioning is possible.

Radiofrequency Lesioning (RF)

Because the sensory branches of the obturator and femoral nerves supply sensation to the hip joint, percutaneous RF of these branches was described in 1997 [56, 57] to denervate the painful degenerated hip in patients who were not candidates for hip arthroplasty. Akatov et al. [56] looked at RF denervation of 15 hips, followed for up to 3 years, noting pain relief in all but 1 patent. Rivera et al. [58] prospectively studied 18 patients with DJD of the hip who were not surgical candidates. Several days after a positive diagnostic injection, each patient underwent RF of the ON and femoral sensory branches to the hip. The ON was identified at the junction of the inferior ischium and the pubis (see *Fluoroscopic injection* above for details of placement). Sensory stimulation resulted in groin and thigh pain; after negative motor stimulation, the site was lesioned at 90 °C for 90 s. The femoral sensory branch was also lesioned. Eight patients had >50 % pain relief at 6 months. Kawaguchi et al. reported the results of RF of the obturator and femoral branches for 14 cases of hip pain, using lesions of 80 °C for 90 s; 12 patients (86 %) noted at least 50 % relief for 1–11 months [57]. Locher et al. [59] dissected the articular branches of the obturator nerve in 20 cadavers and compared the fluoroscopic and MRI images. They concluded that multiple lesions were necessary. Because of concerns regarding conventional RF and neuroma formation, Wu and Groner [60] proposed using pulsed RF; they treated two patients, both of whom noted >50 % pain relief at 3 months.

Phenol

The above techniques of neurolysis (cryoneuroablation and radiofrequency lesioning) require special equipment,

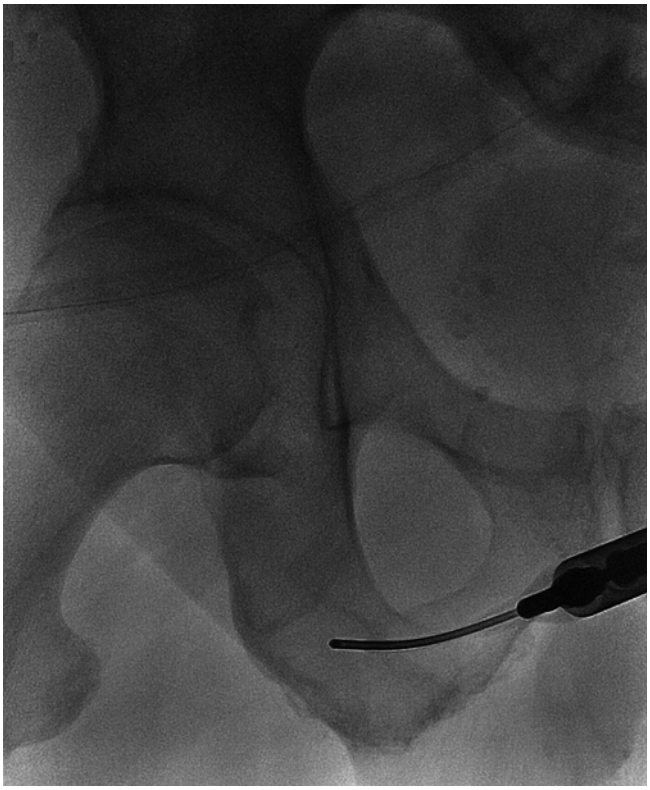

Fig. 64.17 Cryoneuroablation of the obturator nerve (Image courtesy of Andrea Trescot, MD)

which is not available in all parts of the world. Phenol has been described for many years to treat nerves causing pain and spasticity; Akkaya and colleagues [61] retrospectively reviewed 80 phenol ON injections performed for spasticity from spinal cord injuries, cerebral palsy, and traumatic brain injury, using a peripheral nerve stimulator, fluoroscopy, and 6 % phenol. These patients were followed for 3 months, and the patients responded with a dramatic increase in range of motion that started to decrease by the third month. They describe a "100 %" success rate, and there were no complications, though they did note that the literature describes dysesthesias in about 15 % of adults and 5 % of children undergoing neurolysis with phenol [62].

Surgical Technique

Bradshaw and McCrory [1] reported 32 cases of "obturator neuropathy" treated by surgical release using an inguinal incision; several of their patients also underwent inguinal hernia repairs at the same time. They found a fascial entrapment of the ON overlying the short adductor muscle. Rigaud et al. [63] developed a laparoscopic surgical treatment of the ON entrapment. Surgical recovery appears to be related to the onset of symptoms, so Tipton [64] recommended only a limited trial of conservative therapy before surgical intervention.

Complications

Zwolak et al. [14] described an obturator and femoral palsy after cement extrusion from a hip arthroplasty. Mittal and Bhandarkar [65] described temporary ON palsy after local anesthetic spray during a laparoscopic hernia repair; the patient complained of difficulty getting out of bed postoperatively with weakness of the hip adductors. Because of the vascular structures in the area, there is a significant risk of life-threatening arterial puncture [66] as well as venous puncture [27]. Rivera et al. [58] noted three transient hematomas after hip RF.

Summary

The obturator nerve can be a cryptic cause of hip, thigh, and knee pain. ON injections can also be a useful tool for treating adductor or hip pathology. Knowledge of the anatomy and clinical presentation will increase the likelihood of accurate diagnosis and treatment of obturator entrapments.

References

1. Bradshaw C, McCrory P. Obturator nerve entrapment. Clin J Sport Med. 1997;7(3):217–9.
2. Pavlov H, Nelson TL, Warren RF, Torg JS, Burstein AH. Stress fractures of the pubic ramus. A report of twelve cases. J Bone Joint Surg Am. 1982;64(7):1020–5.
3. Barrick EF. Entrapment of the obturator nerve in association with a fracture of the pelvic ring. A case report. J Bone Joint Surg Am. 1998;80(2):258–61.
4. Busis NA. Femoral and obturator neuropathies. Neurol Clin. 1999;17(3):633–53.
5. Leno C, Combarros O, Polo JM, Rebollo M, Berciano J. Bilateral obturator neuralgia secondary to osteitis of the pubis. Arch Neurobiol (Madr). 1984;47(6):347–52.
6. Bischoff C, Schonle PW. Obturator nerve injuries during intra-abdominal surgery. Clin Neurol Neurosurg. 1991;93(1):73–6.
7. Fishman JR, Moran ME, Carey RW. Obturator neuropathy after laparoscopic pelvic lymphadenectomy. Urology. 1993;42(2):198–200.
8. Crews DA, Dohlman LE. Obturator neuropathy after multiple genitourinary procedures. Urology. 1987;29(5):504–5.
9. Pellegrino MJ, Johnson EW. Bilateral obturator nerve injuries during urologic surgery. Arch Phys Med Rehabil. 1988;69(1):46–7.
10. Melamed NB, Satya-Murti S. Obturator neuropathy after total hip replacement. Ann Neurol. 1983;13(5):578–9.
11. Schmalzried TP, Noordin S, Amstutz HC. Update on nerve palsy associated with total hip replacement. Clin Orthop Relat Res. 1997;344:188–206.

12. Siliski JM, Scott RD. Obturator-nerve palsy resulting from intrapelvic extrusion of cement during total hip replacement. Report of four cases. J Bone Joint Surg Am. 1985;67(8):1225–8.
13. Weber ER, Daube JR, Coventry MB. Peripheral neuropathies associated with total hip arthroplasty. J Bone Joint Surg Am. 1976;58(1):66–9.
14. Zwolak P, Eysel P, William-Patrick MJ. Femoral and obturator nerves palsy caused by pelvic cement extrusion after hip arthroplasty. Orthop Rev (Pavia). 2011;3(1), e6.
15. Petchprapa CN, Rosenberg ZS, Sconfienza LM, Cavalcanti CF, Vieira RL, Zember JS. MR imaging of entrapment neuropathies of the lower extremity. Part 1. The pelvis and hip. Radiographics. 2010;30(4):983–1000.
16. Redwine DB, Sharpe DR. Endometriosis of the obturator nerve. A case report. J Reprod Med. 1990;35(4):434–5.
17. Yukata K, Arai K, Yoshizumi Y, Tamano K, Imada K, Nakaima N. Obturator neuropathy caused by an acetabular labral cyst: MRI findings. AJR Am J Roentgenol. 2005;184(3 Suppl):S112–4.
18. Schwabegger AH, Shafighi M, Gurunluoglu R. An unusual case of thigh adductor weakness: Obturator nerve ganglion. J Neurol Neurosurg Psychiatry. 2004;75(5):775.
19. Ekberg O, Persson NH, Abrahamsson PA, Westlin NE, Lilja B. Long standing groin pain in athletes. A multidisciplinary approach. Sports Med. 1988;6(1):56–61.
20. Sorenson EJ, Chen JJ, Daube JR. Obturator neuropathy: causes and outcome. Muscle Nerve. 2002;25(4):605–7.
21. Stewart JD. Obturator nerve. Focal peripheral neuropathies. 3rd ed. Philadelphia: Lippincott, Williams & Wilkins; 2000. p. 483–8.
22. Saurenmann P, Brand S. Obturator neuralgia (Howship-Romberg phenomenon). Schweiz Med Wochenschr. 1984;114(42):1462–4.
23. Brukner P, Khan KM. Groin Pain. In: Bahr R, Blair S, Cook J, Crossley K, McConnell J, McCrory P, et al., editors. Brukner and Khan's clinical sports medicine. 4th ed. Sydney: McGraw-Hill; 2012. p. 545–78.
24. Unwin A, Scott J. Nerve palsy after hip replacement: medico-legal implications. Int Orthop. 1999;23(3):133–7.
25. Anagnostopoulou S, Kostopanagiotou G, Paraskeuopoulos T, Chantzi C, Lolis E, Saranteas T. Anatomic variations of the obturator nerve in the inguinal region: implications in conventional and ultrasound regional anesthesia techniques. Reg Anesth Pain Med. 2009;34(1):33–9.
26. Katritsis E, Anagnostopoulou S, Papadopoulos N. Anatomical observations on the accessory obturator nerve (based on 1000 specimens). Anat Anz. 1980;148(5):440–5.
27. Akkaya T, Ozturk E, Comert A, Ates Y, Gumus H, Ozturk H, et al. Ultrasound-guided obturator nerve block: a sonoanatomic study of a new methodologic approach. Anesth Analg. 2009;108(3): 1037–41.
28. Kumka M. Critical sites of entrapment of the posterior division of the obturator nerve: anatomical considerations. J Can Chiropr Assoc. 2010;54(1):33–42.
29. Tammareddi K, Morelli V, Reyes Jr M. The athlete's hip and groin. Prim Care. 2013;40(2):313–33.
30. Morelli V, Weaver V. Groin injuries and groin pain in athletes: part 1. Prim Care. 2005;32(1):163–83.
31. Saranteas T, Paraskeuopoulos T, Alevizou A, Kouskouri A, Zogojiannis J, Anagnostopoulou S, et al. Identification of the obturator nerve divisions and subdivisions in the inguinal region: a study with ultrasound. Acta Anaesthesiol Scand. 2007;51(10):1404–6.
32. Yamauchi M, Sato Y. Ultrasound-guided obturator nerve block. Masui. 2008;57(5):588–95.
33. Bouaziz H, Vial F, Jochum D, Macalou D, Heck M, Meuret P, et al. An evaluation of the cutaneous distribution after obturator nerve block. Anesth Analg. 2002;94(2):445–9. table of contents.
34. Kopell HP, Thompson WA. Peripheral entrapment neuropathies of the lower extremity. N Engl J Med. 1960;262:56–60.
35. Harvey G, Bell S. Obturator neuropathy. An anatomic perspective. Clin Orthop Relat Res. 1999;363:203–11.
36. Kashuk K. Proximal peripheral nerve entrapment syndromes in the lower extremity. J Am Podiatry Assoc. 1977;67(8):529–44.
37. Dawson DM, Hallett M, Wilbourn AJ. Miscellaneous uncommon syndromes of the lower extremity. In: Dawson DM, Hallett M, Wilbourn AJ, editors. Entrapment neuropathies. 3rd ed. Philadelphia: Lippincott-Raven; 1999. p. 369–79.
38. Lang SA, Yip RW, Chang PC, Gerard MA. The femoral 3-in-1 block revisited. J Clin Anesth. 1993;5(4):292–6.
39. Helayel PE, da Conceicao DB, Pavei P, Knaesel JA, de Oliveira Filho GR. Ultrasound-guided obturator nerve block: a preliminary report of a case series. Reg Anesth Pain Med. 2007;32(3):221–6.
40. Choquet O, Capdevila X, Bennourine K, Feugeas JL, Bringuier-Branchereau S, Manelli JC. A new inguinal approach for the obturator nerve block: anatomical and randomized clinical studies. Anesthesiology. 2005;103(6):1238–45.
41. Malik A, Simopolous T, Elkersh M, Aner M, Bajwa ZH. Percutaneous radiofrequency lesioning of sensory branches of the obturator and femoral nerves for the treatment of non-operable hip pain. Pain Physician. 2003;6(4):499–502.
42. Garvey JF, Hazard H. Sports hernia or groin disruption injury? Chronic athletic groin pain: a retrospective study of 100 patients with long-term follow-up. Hernia. 2013: on line.
43. Kozlowski JM, Beal JM. Obturator hernia: an elusive diagnosis. Arch Surg. 1977;112(8):1001–2.
44. Travell J, Simons D. In: Simmons DC, Travell JC, Simmons LS, editors. Travell & Simmons' myofascial pain and dysfunction: the trigger point manual, Lower Extremity, vol. 2. Baltimore: Williams & Wilkins; 1992. p. 289–314.
45. Marhofer P, Nasel C, Sitzwohl C, Kapral S. Magnetic resonance imaging of the distribution of local anesthetic during the three-in-one block. Anesth Analg. 2000;90(1):119–24.
46. Bradshaw C, McCrory P, Bell S, Brukner P. Obturator nerve entrapment. A cause of groin pain in athletes. Am J Sports Med. 1997;25(3):402–8.
47. Kassolik K, Kurpas D, Wilk I, Uchmanowicz I, Hyzy J, Andrzejewski W. The effectiveness of massage in therapy for obturator nerve dysfunction as complication of hip joint alloplasty-case report. Rehabil Nurs. 2014;39(6):311–20.
48. Abelson B, Abelson K. Release your pain: resolving soft tissue injuries with exercise and active release techniques. 2nd ed. Calgary: Rowan Tree Books Ltd; 2012.
49. Tatlisen A, Sofikerim M. Obturator nerve block and transurethral surgery for bladder cancer. Minerva Urol Nefrol. 2007;59(2):137–41.
50. Winnie AP, Ramamurthy S, Durrani Z. The inguinal paravascular technique of lumbar plexus anesthesia: the "3-in-1 block". Anesth Analg. 1973;52:989–96.
51. Feigl GC, Ulz H, Pixner T, Dolcet C, Likar R, Sandner-Kiesling A. Anatomical investigation of a new vertical obturator nerve block technique. Ann Anat. 2012;195(1):82–7.
52. Soong J, Schafhalter-Zoppoth I, Gray AT. Sonographic imaging of the obturator nerve for regional block. Reg Anesth Pain Med. 2007;32(2):146–51.
53. Manassero A, Bossolasco M, Ugues S, Palmisano S, De Bonis U, Coletta G. Ultrasound-guided obturator nerve block: interfascial injection versus a neurostimulation-assisted technique. Reg Anesth Pain Med. 2011;37(1):67–71.
54. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
55. Kim PS, Ferrante FM. Cryoanalgesia: a novel treatment for hip adductor spasticity and obturator neuralgia. Anesthesiology. 1998;89(2):534–6.
56. Akatov OV, Dreval ON. Percutaneous radiofrequency destruction of the obturator nerve for treatment of pain caused by coxarthrosis. Stereotact Funct Neurosurg. 1997;69(1–4 Pt 2):278–80.

57. Kawaguchi M, Hashizume K, Iwata T, Furuya H. Percutaneous radiofrequency lesioning of sensory branches of the obturator and femoral nerves for the treatment of hip joint pain. Reg Anesth Pain Med. 2001;26(6):576–81.
58. Rivera F, Mariconda C, Annaratone G. Percutaneous radiofrequency denervation in patients with contraindications for total hip arthroplasty. Orthopedics. 2012;35(3):e302–5.
59. Locher S, Burmeister H, Bohlen T, Eichenberger U, Stoupis C, Moriggl B, et al. Radiological anatomy of the obturator nerve and its articular branches: basis to develop a method of radiofrequency denervation for hip joint pain. Pain Med. 2008;9(3):291–8.
60. Wu H, Groner J. Pulsed radiofrequency treatment of articular branches of the obturator and femoral nerves for management of hip joint pain. Pain Pract. 2007;7(4):341–4.
61. Akkaya T, Unlu E, Alptekin A, Gumus HI, Umay E, Cakci A. Neurolytic phenol blockade of the obturator nerve for severe adductor spasticity. Acta Anaesthesiol Scand. 2010;54(1):79–85.
62. Tilton AH. Injectable neuromuscular blockade in the treatment of spasticity and movement disorders. J Child Neurol. 2003;18 Suppl 1:S50–66.
63. Rigaud J, Labat JJ, Riant T, Bouchot O, Robert R. Obturator nerve entrapment: diagnosis and laparoscopic treatment: technical case report. Neurosurgery. 2007;61(1):E175. discussion E.
64. Tipton JS. Obturator neuropathy. Curr Rev Musculoskelet Med. 2008;1(3–4):234–7.
65. Mittal G, Bhandarkar D, Katara A, Udwadia TE. Temporary obturator nerve paresis after spray of local anesthetic during laparoscopic extraperitoneal inguinal hernia repair. Surg Laparosc Endosc Percutan Tech. 2010;20(5):357.
66. Akata T, Murakami J, Yoshinaga A. Life-threatening haemorrhage following obturator artery injury during transurethral bladder surgery: a sequel of an unsuccessful obturator nerve block. Acta Anaesthesiol Scand. 1999;43(7):784–8.

Proximal Sciatic Nerve Entrapment

65

Annemarie E. Gallagher, Amitabh Gulati,
and Terri Dallas-Prunskis

Introduction

Leg, foot, and ankle pain may be caused by entrapment of the sciatic nerve, an etiology that is often underdiagnosed. There are many potential areas of entrapment of this nerve. The focus of this chapter will be potential proximal sciatic nerve (SN) entrapments.

Clinical Presentation (Table 65.1)

Entrapment of the proximal sciatic nerve (SN) causes pain down the leg; since the Greek term "sciatica" (pain down the leg) has been used primarily to describe lumbar radiculopathy, the pain from proximal sciatic pathology has been called "extra-spinal sciatica" or "pseudosciatica." The patient with proximal sciatic nerve entrapment will initially present with one of three clinical stages. During *Stage I*, the patient will complain of resting leg pain and paresthesias or dysesthesias in the sciatic distribution (Fig. 65.1), which is often worse at night. The patient in *Stage II* will present with leg weakness and numbness in the sciatic distribution. *Stage III* symptoms consist of complaints of constant pain, with muscle atrophy and sensory loss that will be apparent on physical examination [23]. The patient may also complain of sharp low back pain, aching buttock pain, and occasionally dull pain from the buttocks to the popliteal fossa to the foot [24] (Fig. 65.2).

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Since each lumbar nerve root innervates a different part of the foot, the patient who complains of pain or numbness of the "whole foot" is likely to have a sciatic entrapment rather than a radiculopathy.

Other common symptoms include pain with extended periods of sitting or twisting toward the unaffected side (Table 65.1). Patients will often weight-shift when sitting, to minimize pressure on the painful side [24].

Anatomy (Table 65.2)

The *sciatic nerve* (SN) is the largest branch of the sacral plexus and is the largest nerve in the human body. Originating from the ventral divisions of L4–S3 of the sacral plexus, the SN courses through the pelvis, enters the gluteal region

Table 65.1 Occupation/exercise/trauma history relevant to proximal sciatic entrapment

Trauma	Hip surgery [1, 2], hematoma, hardware migration/laceration [3]
	Hip fracture/heterotopic ossification [4]
	Penetrating injuries [5]
	Intramuscular gluteal injection [6]
	Acetabular fracture [7]
Compression	Endopelvic pathology [8]
	Cyclic "sciatica" due to endometrial nodules [9]
	Prolonged squatting position/sitting [10–12]
	Pregnancy [13]
	Intraneural tumor/cyst [14]
	Piriformis syndrome [15]
	Acute external compression [12, 16, 17]
	Vascular causes [18, 19]
	Lumbar spinal stenosis/disc herniation/ spondylolisthesis [20]
Inflammation	Sacroiliitis [21]
	Infection [22]

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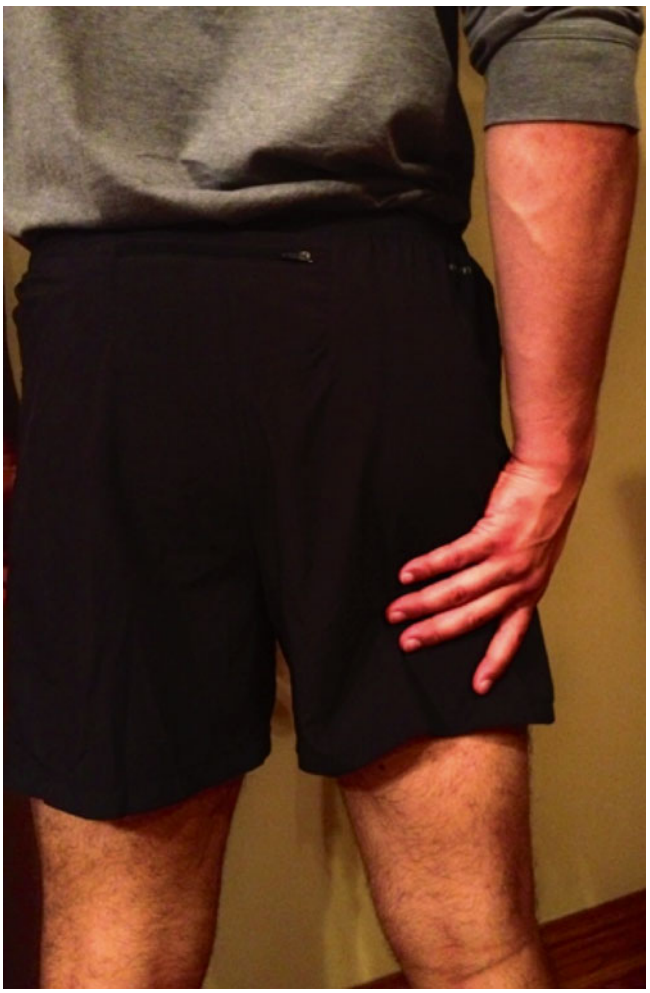

Fig. 65.1 Patient complaint of pain from proximal sciatic nerve entrapment (Image courtesy of Andrea Trescot, MD)

Fig. 65.2 Pattern of pain from proximal sciatic entrapment. *A* Low back and buttocks, *B* low back to the foot (Image courtesy of Andrea Trescot, MD

through the *greater sciatic foramen*, and then exits the greater sciatic foramen at the inferior border of the *piriformis muscle* in 79 % of patients [7] (Fig. 65.3). Anatomic variants include having the sciatic nerve pass through the piriformis muscle itself (either the tibial or peroneal divisions or both) or superior to it [26].

The sciatic nerve then travels between the *greater trochanter* and the *ischial tuberosity* (Fig. 65.4), passing deep to the gluteus maximus and posteriorly to the *quadratus femoris muscle* (Fig. 65.5). The nerve then curves around the ischial spine and descends past the origin of the hamstrings laterally (Fig. 65.6). In the proximal thigh, the sciatic nerve runs posteriorly to the hamstrings and anteriorly to the adductor magnus.

Distally, the sciatic nerve divides into the common peroneal/fibular nerve (see Chap. 67) and tibial nerves (see Chap. 73) (Fig. 65.7). Although the bifurcation is classically taught to occur just above the popliteal crease, cadaver and ultrasound studies have shown that the bifurcation occurs within 8 cm from the popliteal crease in only 75 % of the legs dissected, occurring more proximally in 25 % of the cases [27].

The sciatic nerve innervates most of the muscles of the posterior compartment of the thigh (*semitendinosus*, *semimembranosus*, and the *biceps femoris*) and is responsible for most sensorimotor functions below the knee [28] (Table 65.2). In the proximal thigh, there are three main muscle innervations – the sciatic, femoral, and obturator nerves (Fig. 65.8).

Entrapment

Sciatic neuropathy is one of the most common nerve pains of the lower extremity, mimicking "sciatica" from a herniated disc, hence the name "pseudosciatica." The most common entrapment of the SN is at the level of the piriformis muscle (Fig. 65.9). Piriformis entrapment is more common in women,

Table 65.2 Sciatic nerve anatomy

Origin	Ventral divisions of L4–S3 of the sacral plexus
General route	The sciatic nerve comprises the lateral division which eventually forms the *common fibular nerve* and the medial division which forms the *tibial nerve*, each separately encased from the outset [25]. The sciatic nerve courses through the pelvis, enters the gluteal region through the *greater sciatic foramen*, then exits at the inferior border of the *piriformis muscle*; the nerve is covered by the gluteus maximus muscle and soft tissue. The nerve then travels halfway between the bony landmarks, *greater trochanter* laterally, and *ischial tuberosity* medially and then descends into the subgluteal area. It then runs posteriorly in the midthigh, remaining dorsal to the adductor magnus and ventral to the long head of the biceps femoris
Sensory distribution	Back of the leg, back and lateral side of calf, most of the foot
Motor innervation	*Tibial division*: hamstring muscles (semimembranosus, semitendinosus, long head of the biceps femoris), adductor magnus in the thigh
	Common fibular division: short head of biceps femoris
Anatomic variability	The sciatic nerve usually travels under the piriformis muscle, except in 10–30 % of cases where the nerve passes through the piriformis muscle or above it [26]
Other relevant structures	Piriformis, sacrotuberous ligament, ischial tuberosity

perhaps because of the wider *quadriceps femoris muscle angle* ("Q angle") [26]. The peroneal division of the sciatic nerve is more frequently injured because its fibers are more superficial, have less supporting connective tissue, and are fixed at two points (the sciatic foramen and the fibular head), whereas the tibial division is fixed only at the sciatic foramen [5].

There are various potential causes for proximal sciatic nerve entrapment that include traumatic, compressive, ischemic, neoplastic, and iatrogenic causes (see Table 65.1). Athletes, particularly those participating in high impact

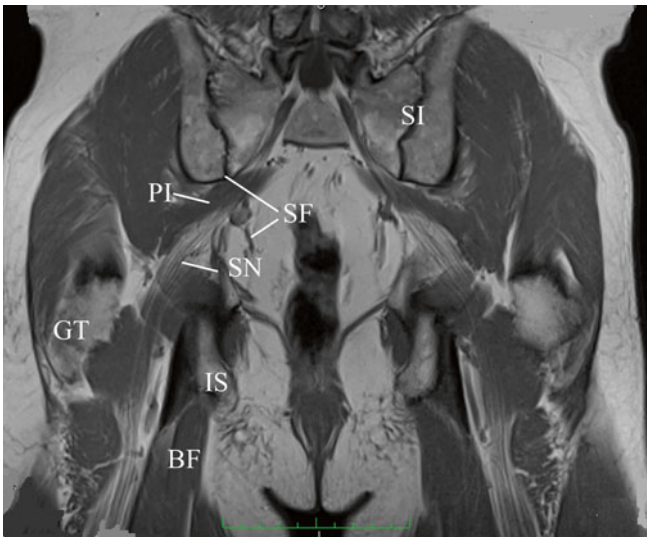

Fig. 65.4 MRI coronal image. *SI* sacroiliac joint, *IS* ischium/ischial tuberosity, *GT* greater trochanter, *PI* piriformis, *BF* biceps femoris muscle, *SF* sciatic foramen, *SN* sciatic nerve (Image created by Andrea Trescot, MD)

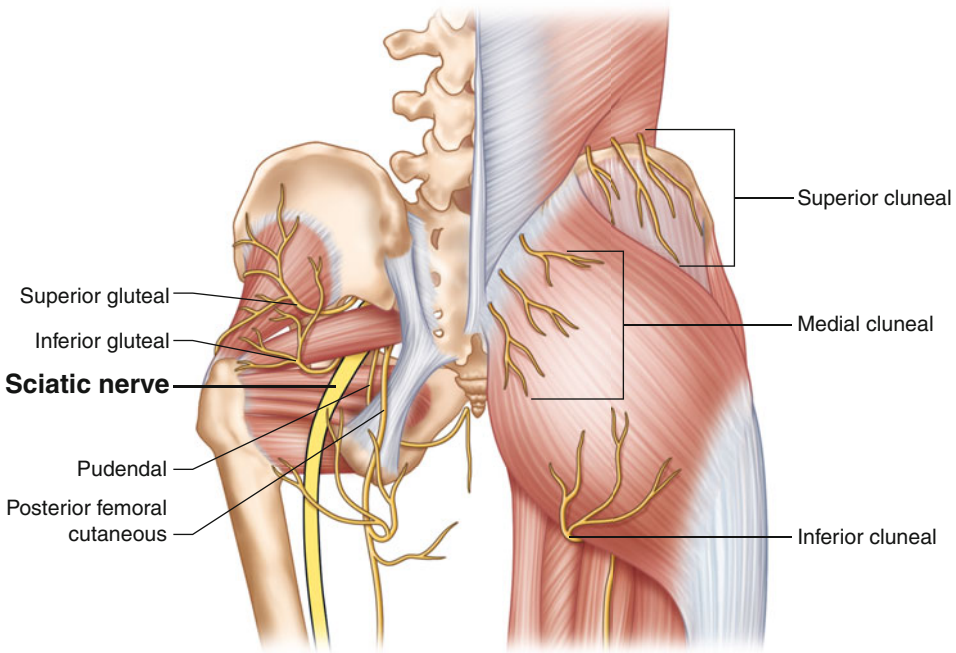

Fig. 65.3 Proximal sciatic anatomy (Image courtesy of Springer)

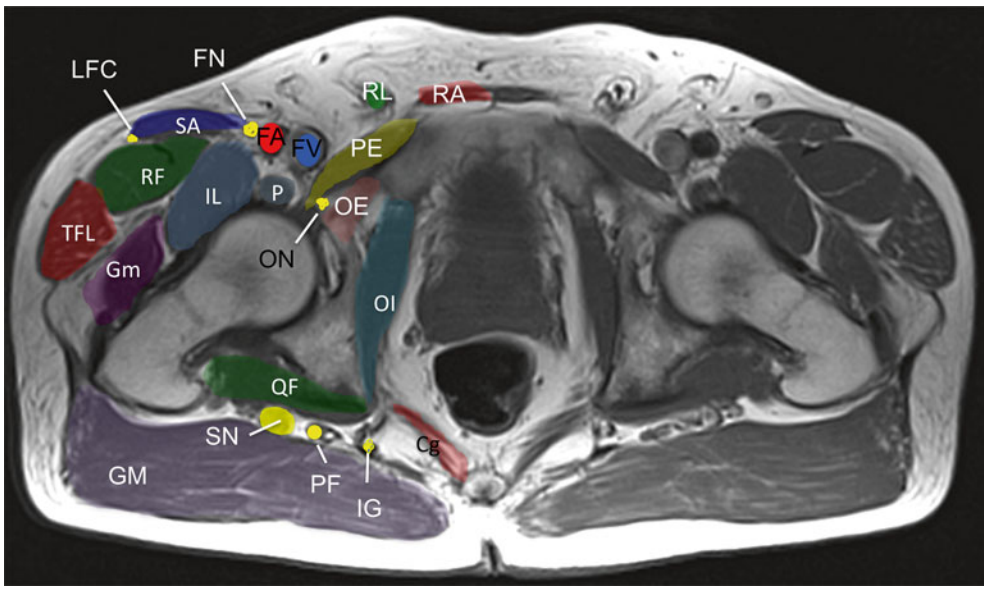

Fig. 65.5 Axial MRI of the pelvic structures. *Cg* coccygeus muscle, *FA* femoral artery, *FN* femoral nerve, *FV* femoral vein, *GM* gluteus maximus muscle, *Gm* gluteus medius, *IG* inferior gluteal nerve, *IL* iliopsoas muscle, *LFC* lateral femoral cutaneous nerve, *OI* obturator internus muscle, *P* psoas muscle, *PE* pectineus muscle, *PF* posterior femoral cutaneous nerve, *QF* quadratus femoris muscle, *RA* rectus abdominis muscle, *RF* rectus femoris muscle, *RL* round ligament, *SA* sartorius muscle, *SN* sciatic nerve, *TFN* tensor fascia lata muscle (Image courtesy of Andrea Trescot, MD)

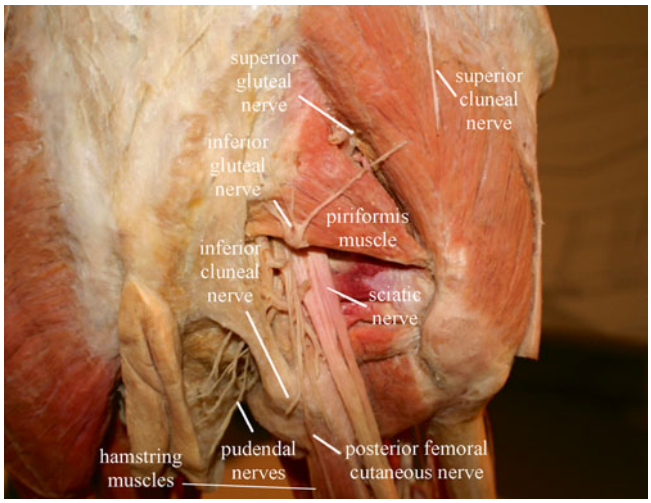

Fig. 65.6 Buttocks dissection, modified from an image from *Bodies*, *The Exhibition*, with permission (Image courtesy of Andrea Trescot, MD)

sports, can develop an *overuse syndrome* in the posterior compartment of the thigh, a partial hamstring tear, or a complete hamstring avulsion that can present as a compartment syndrome [28, 29]. With high pressures in the posterior thigh, the sciatic nerve can become significantly compressed. When examining a patient with possible sciatic nerve compression, it is important to keep in mind the possibility of an acetabular fracture and/or dislocation. In fact, a study by Letournel and Judet reported that the highest incidence of sciatic palsy was due to posterior fracture/dislocation of the hip [7, 30].

The most common iatrogenic cause of proximal sciatic nerve entrapment occurs in the retroacetabular region secondary to total hip arthroplasty (a 3.7 % incidence of nerve palsy in 108 hip replacements) [31] that may be attributed to a posterior surgical approach, limb lengthening, compression from a postoperative hematoma, laceration from a screw used for acetabular cup fixation (Fig. 65.10), or hardware migration [7, 28].

Heterotopic ossification (HO) around the SN is also a common cause of sciatic nerve entrapment in the postoperative period. HO is an extra-skeletal bone formation, most often around the hip, and occurs commonly following hip arthroplasty and also occurs in the setting of fractures, traumatic brain injury, spinal cord injury, and burns [7, 32]. Cases have been reported where mature heterotopic bone formed around the hip and extended around the sciatic nerve, resulting in pain and weakness in the sciatic distribution [7, 33].

It is important to keep in mind the other diagnoses that can result in proximal sciatic nerve entrapment, including endopelvic pathology (e.g., infiltrating endometriosis), metastatic tumors, or scarring from radiation therapy in those with a known cancer diagnosis [34, 35]. Infiltration of the sciatic nerve by tumor can occur from nearby pelvic bones, including the sacrum and the periacetabular region [24, 28]. Vascular compression of the sacral plexus may also occur from local tumor infiltration.

Anomalies of the piriformis muscle may be responsible for sciatic nerve entrapment. The piriformis can be hypertrophied, secondary to extreme lumbar lordosis or hip flexion deformities, resulting in gait abnormalities. The hypertrophied piriformis may cause constriction at the greater sciatic foramen with resultant sciatic nerve compression [28].

Physical Exam

The most specific diagnostic test for sciatic entrapment is the *straight leg raise* to produce *Lasegue's sign* (Fig. 65.11), which is considered positive if the pain in the distribution of the sciatic nerve is reproduced with between 30° and 70° passive flexion of the straight leg [36]. While this test is positive in about 90 %

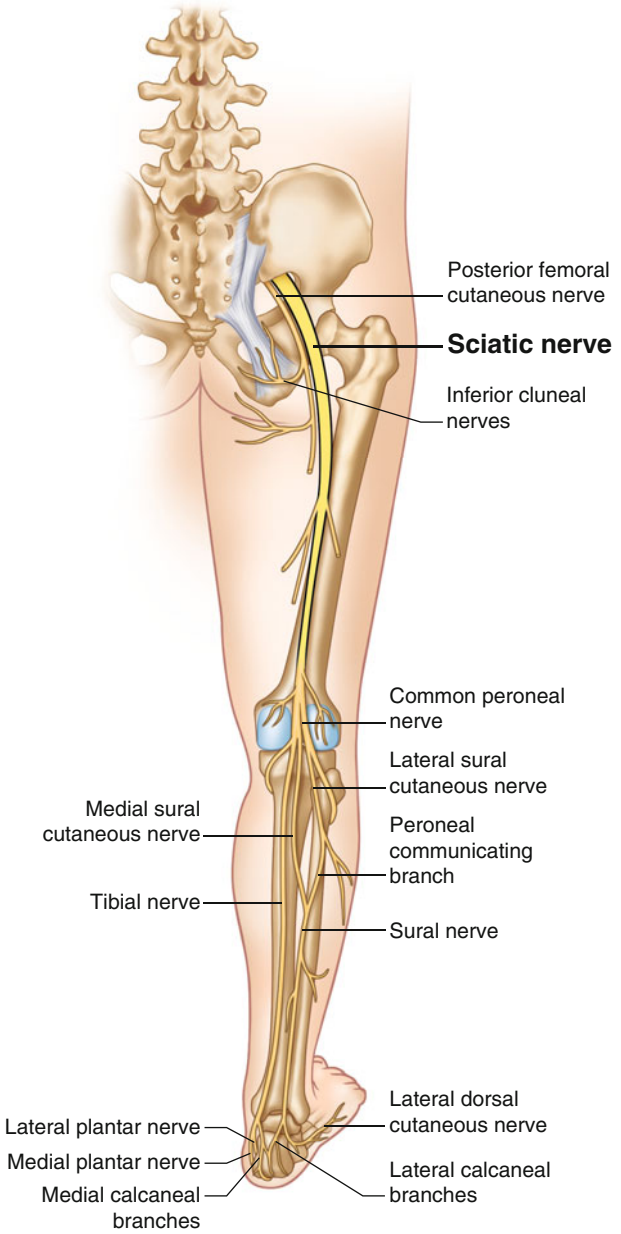

Fig. 65.7 Sciatic nerve anatomy (Image courtesy of Springer)

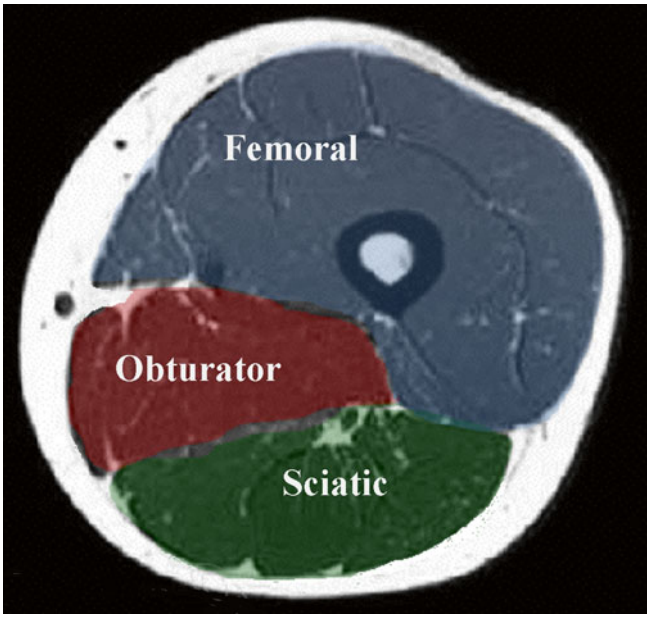

Fig. 65.8 Demarcation of the distribution of the three nerves that innervate the proximal thigh – sciatic, femoral, and obturator nerves (Image courtesy of Andrea Trescot, MD)

of people with sciatic entrapment, approximately 75 % of people with a positive test do not have sciatica [20].

A complete examination of the hip and lumbar spine should also take place, in conjunction with a neurological exam of the lower limb. When examining the hip and lumbar spine, range of motion testing, palpation of the sacroiliac joint, and dural tension signs should be tested in order to rule out other etiologies, including disc herniation, facet arthropathy, sacroiliac joint dysfunction, or piriformis syndrome (Fig. 65.10).

With a proximal sciatic entrapment, there can be tenderness at the piriformis muscle (Fig. 65.12) or distal to the piriformis. If sacroiliac (SI) joint pathology is present, tenderness will be medial to the posterior iliac crest at the posterior superior iliac spine PSIS (Fig. 65.13). If there is proximal sciatic nerve pathology (such as superior gluteal branch involvement – see Chap. 53), the patient will experience pain more laterally [24] (Fig. 65.14). Gait should also be observed for any abnormalities consistent with weakness in this particular nerve distribution [36].

Differential Diagnosis (Table 65.3)

The history and physical examination will narrow the list of differential diagnoses (Table 65.3). If a patient is complaining of leg pain, weakness, and/or sensory disturbances, particularly in a radicular distribution, then lumbar pathology must be ruled out. Hip joint pathology, as well as hamstring tendinopathy, should also be among the differential diagnoses.

If sciatic nerve entrapment remains at the top of the differential, it is important to localize the site of entrapment, as both proximal and distal nerve compressions can present with similar findings, including foot drop. Electrodiagnostic studies can help to differentiate between an entrapment proximally along the sciatic nerve itself or more distally along the peroneal or tibial divisions [38]. If the diagnostic tests show that proximal sciatic nerve entrapment is the cause of the patient's symptoms, then the etiology of the entrapment itself must be further explored, including traumatic, compressive, ischemic, neoplastic, and iatrogenic reasons, as described above. Both electrodiagnostics and magnetic resonance imaging can be useful in assessing the cause of symptoms [7, 28] (Table 65.4).

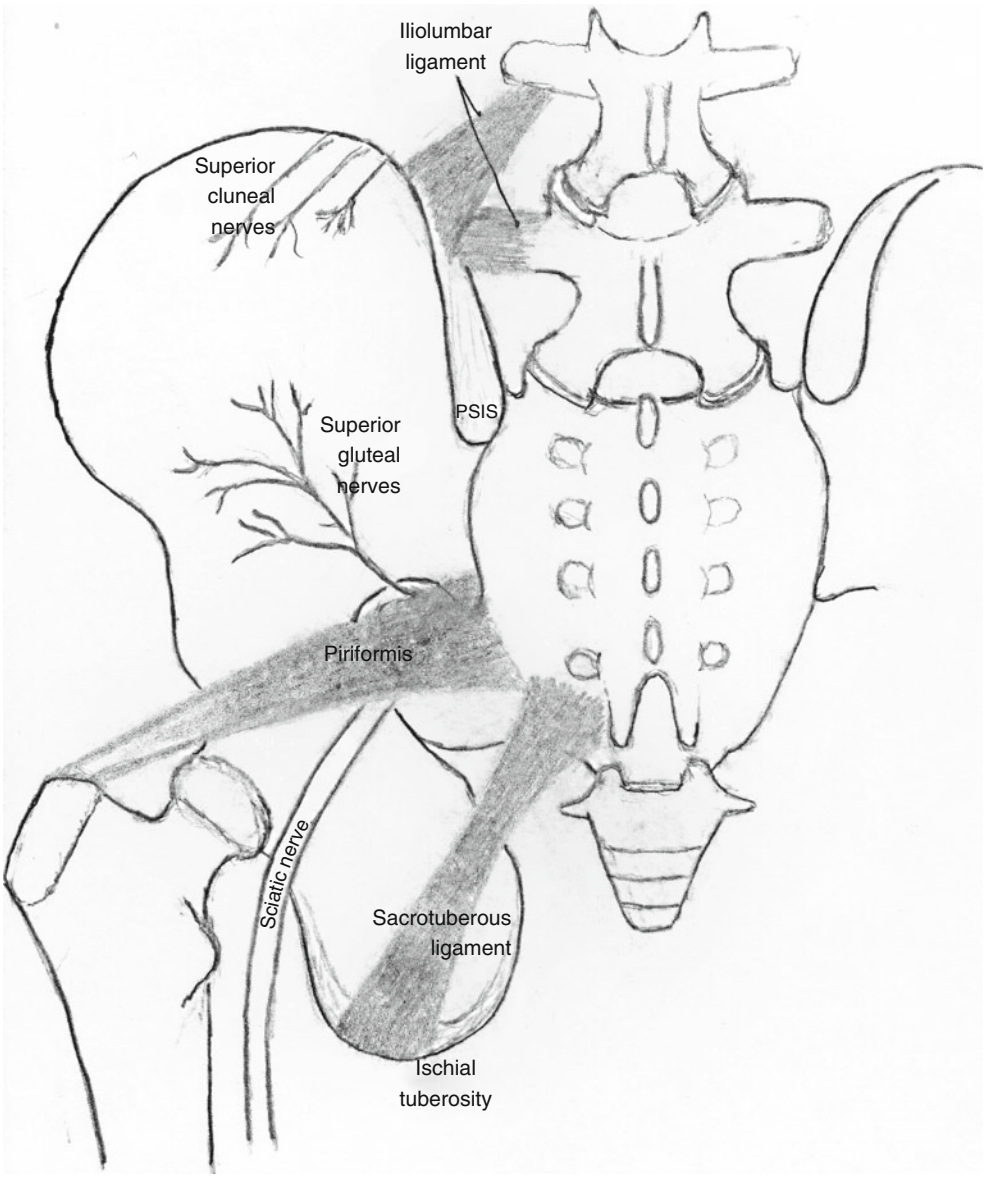

Fig. 65.9 Sites of several causes of "pseudosciatica," including piriformis entrapment of the sciatic nerve (Image drawn by Luis N. Hernandez, MD, with permission)

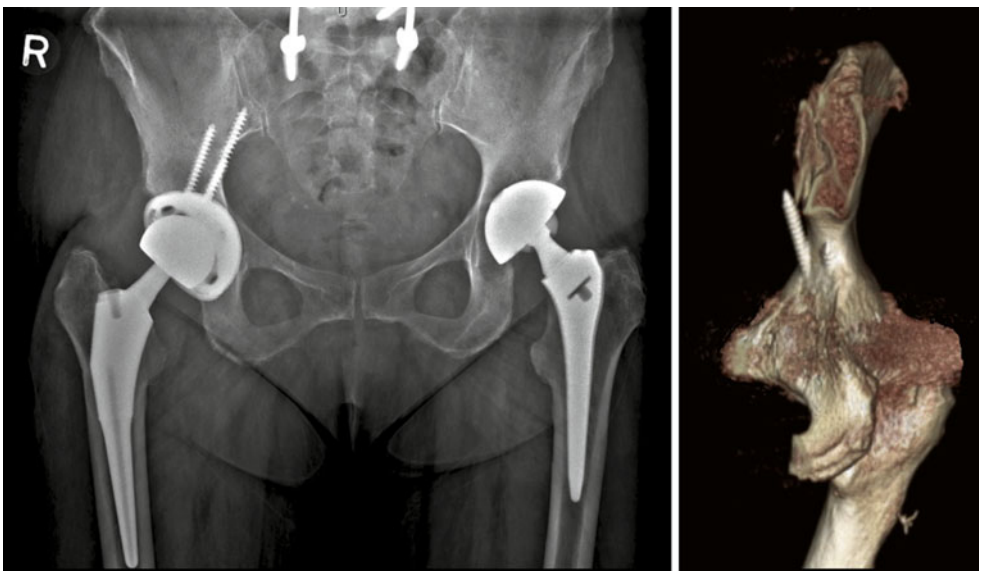

Fig. 65.10 Example of impingement from an acetabular screw (Image courtesy of Andrea Trescot, MD)

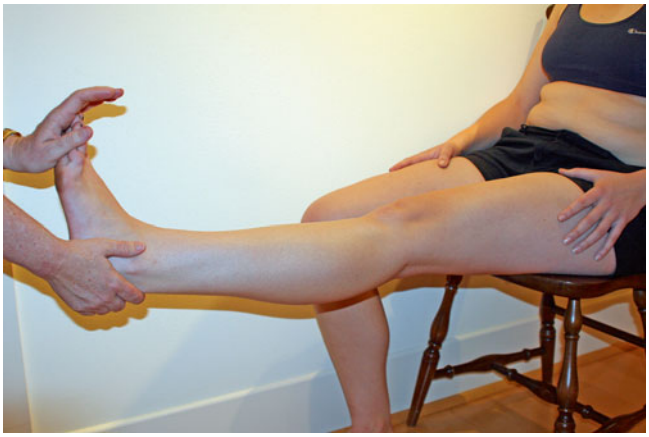

Fig. 65.11 Lasegue's sign (straight leg raise) (Image courtesy of Terri Dallas-Prunskis, MD)

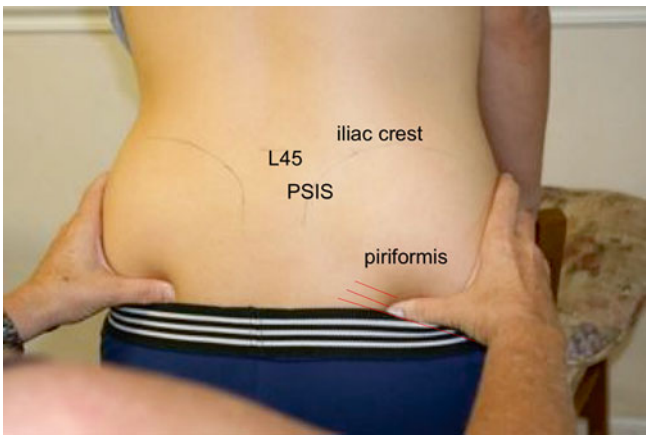

Fig. 65.12 Proximal sciatic physical exam (Image courtesy of Andrea Trescot, MD)

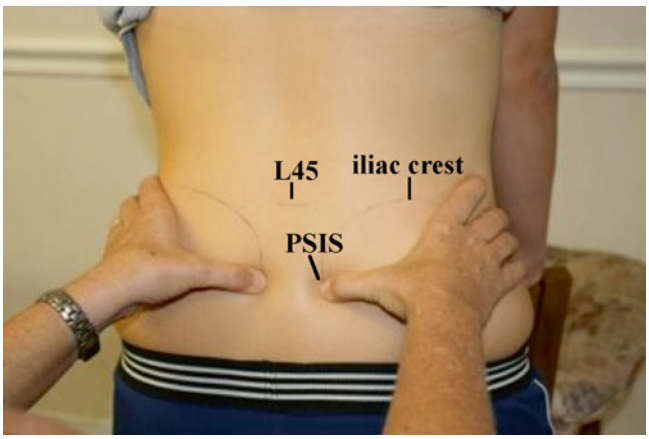

Fig. 65.13 Sacroiliac physical exam (Image courtesy of Andrea Trescot, MD)

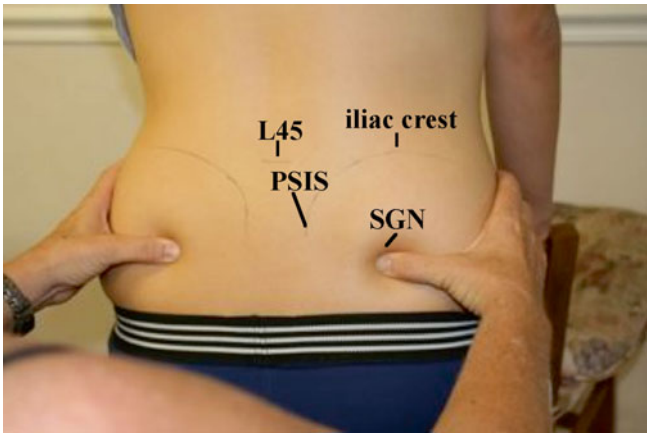

Fig. 65.14 Superior gluteal nerve physical exam (Image courtesy of Andrea Trescot, MD)

Table 65.3 Differential diagnosis of posterior leg pain

	Potential distinguishing features
LS plexopathy [37]	Greater degree of weakness in the gluteal muscle, ankle dorsal, and plantar flexion
L5 radiculopathy [38]	Low back pain with posterolateral thigh pain, gluteal atrophy
S1 radiculopathy [38]	Low back pain → posterior thigh pain, gluteal atrophy
Common peroneal (fibular) neuropathy [38]	Sensory loss in the distal 2/3 of the lateral leg
Hip joint pathology [39]	Stiffness, limited range of motion; X-ray and MRI show abnormality
Hamstring tendinopathy [40]	Sciatic nerve is under compression only with certain activities; EMG is inconclusive
Piriformis muscle syndrome	Increased pain with hip internal rotation, taut piriformis muscle
Inferior cluneal entrapment	Innervates the lateral anus and lateral region of the labium majorum
Posterior femoral cutaneous entrapment	Innervates the skin of the perineum and the back surface of the thigh and leg

Identification and Treatment of Contributing Factors

If a patient presents with signs of proximal sciatic nerve entrapment, observing the patient's gait can aid in deciphering the etiology. If the sciatic nerve is irritated at the greater sciatic foramen, the patient may alter his or her gait to alleviate the neuropathic symptoms. Ambulation will be characterized by the foot pointed outward on the affected side, due to external rotation of the hip, which decreases the stretch on the piriformis muscle (and therefore the compression on the SN) [44]. This should prompt the clinician to ask about the patient's habits, including placing a wallet in a rear pocket on

Table 65.4 Diagnostic tests for sciatic entrapment

	Potential distinguishing features
Physical exam	Weakness in hamstring with foot inversion, toe flexion, and possible knee flexion, decreased sensory to upper 1/3 lateral leg, + SLR
Diagnostic injection	Local anesthetic injection is suitable to confirm a diagnosis
Ultrasound	May show level of entrapment
MRI	Depending on the severity of nerve injury, a high-intensity signal in the nerve fibers or increased nerve dimension, deformation, or total loss of the nerve integrity may be seen [41]; affected muscles may show fatty infiltration, edema, and atrophy [42]
CT Scan	Reveals lesions that impact the sciatic nerve involving the bone (e.g., sacrum fractures), vessel abnormalities (e.g., aneurysm), or hematoma [43]
Arteriography	Not useful
X-ray	To rule out other causes
Electrodiagnostic studies	Needle EMG consistent with denervation depending on the lesion site: short and long heads of the biceps femoris, semimembranosus, or semitendinosus [38]

that side or frequently sitting on a hard surface [44]. A recognized entity known as "wallet sciatica" or "fat wallet syndrome" can cause significant sciatic nerve irritation, mimicking an actual nerve entrapment. Piriformis entrapment has been associated with a high incidence of increased lumbar lordosis; compensatory flexion of the hip tightens the hip rotators, which pulls the sciatic nerve against the ischium [45]. Gluteal injections can also cause nerve injury and contribute to sciatic entrapment and scarring [46].

High-risk occupations or jobs that require awkward positions or those that cause whole-body vibration, such as long distance truck driving, place workers at particular risk. Other factors may include hyperlordosis and lumbar vertebral fractures [47].

Sciatic nerve compression due to postoperative hematoma can result in continued symptoms [28]. While time can alleviate the hematoma-induced nerve entrapment, sometimes the hematoma must be surgically evacuated for symptom relief. Other times, the sciatic nerve can become entrapped from acetabular cup fixation or from hardware migration even years after initial hip arthroplasty [28] (Fig. 65.10). Hip revision surgery is required if the hardware is the perpetrator. Following surgical intervention, particularly at the hip joint, patients can also develop HO that can result in sciatic nerve entrapment. Surgical removal of the mature heterotopic bone is required to alleviate this etiology of nerve entrapment [7]. Entrapment of the sciatic nerve by the piriformis muscle will cause persistent pathology. Kirschner et al. described the use of myofascial release, trigger point massage, and possible trigger point injections to relieve piriformis entrapment [44].

Injection Technique

Landmark-Guided Injection

Many of the causes of proximal sciatic nerve entrapment require surgical intervention. For both diagnostic and anesthetic purposes, a proximal sciatic nerve block may be performed blindly in the gluteal region. Landmarks for the injection should be determined first.

In the *classic or posterior approach (Labat approach)*, the patient is placed in the lateral position with the extremity to be blocked flexed maximally at the hip and slightly at the knee (Fig. 65.15). The gluteal region, the greater trochanter of the affected side, the PSIS, the sacral hiatus, and the ischial tuberosity are identified. Utilizing an aseptic technique, a line is drawn between the greater trochanter and the PSIS (line labeled GT –> PSIS). A second line is drawn between the greater trochanter and the sacral hiatus (line labeled GT –> SH). A perpendicular line is next drawn that bisects the first line and should extend through the second line, which is the injection site (site X). The needle should be inserted at the marked point perpendicular to the skin in all planes and advanced until a parenthesis is noted. If a nerve stimulator is utilized, search for movement in the leg and foot [48].

With the *parasacral sciatic nerve approach*, a block is performed in the sacral plexus after its emergence from the greater sciatic foramen. This technique will block the sciatic nerve as well as the *superior gluteal nerve* (see Chap. 53) and *inferior gluteal nerve* (see Chap. 54), *posterior femoral cutaneous nerve of the thigh* (see Chap. 56), and *pudendal nerve* (see Chap. 47). The patient is positioned as for the posterior approach. The anatomic landmarks are the PSIS and the ischial tuberosity (Fig. 65.16). A point is marked 7 cm distal from the PSIS on a line connecting the PSIS and ischial tuberosity; a 22-guage, 100-mm needle is introduced perpendicular to the skin and advanced 6–8 cm. If the bone is contacted, the needle is reintroduced along a line more caudally until it misses the bone. Nerve stimulation should be utilized with each approach to confirm needle placement [49].

Fluoroscopic-Guided Injection

Under fluoroscopic guidance, a sciatic nerve block can be performed at the sciatic notch. The patient is placed in a prone position, and utilizing the same sterile precautions described above, a 25- or a 22-gauge 3.5-inch spinal needle is advanced toward the sciatic notch, parallel to the

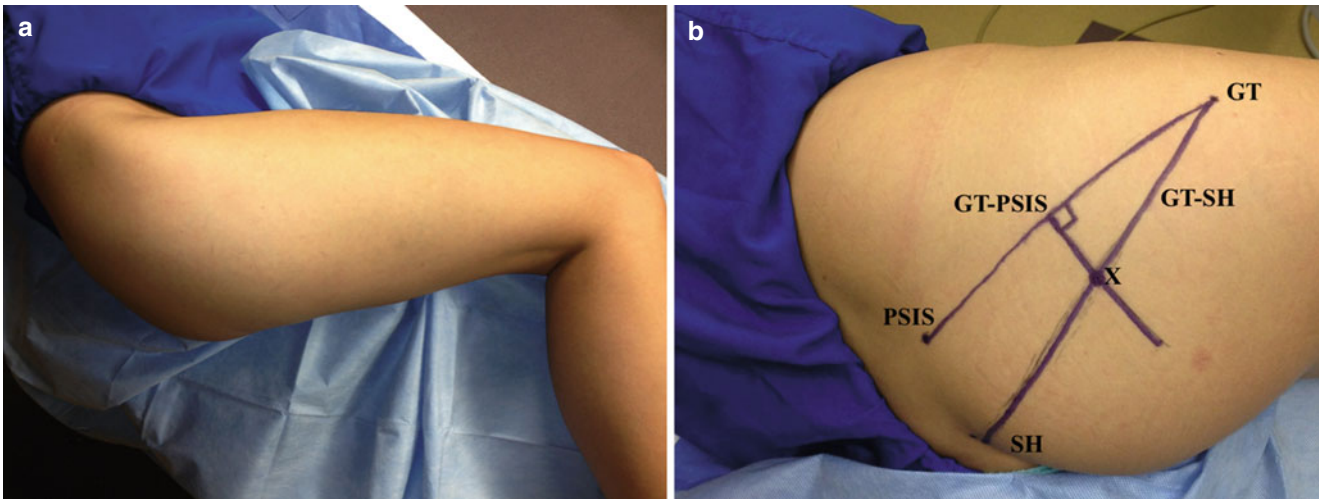

Fig. 65.15 Landmark-guided sciatic nerve injection – classic/posterior approach. (**a**) leg position; (**b**) landmarks. *PSIS* posterior superior iliac spine, *GT* greater trochanter, *SH* sacral hiatus, *X* injection site (Image courtesy of Terri Dallas-Prunskis, MD)

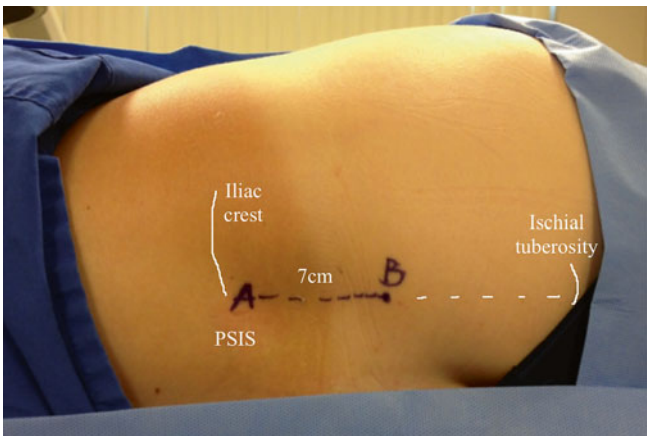

Fig. 65.16 Landmark-guided sciatic nerve injection – parasacral approach. *A* posterior superior iliac spine, *B* injection site (Image courtesy of Terri Dallas-Prunskis, MD)

fluoroscopy beam (Fig. 65.17). As the needle tip comes into close proximity with the sciatic nerve, a paresthesia may be elicited, or the procedure may be performed with peripheral nerve stimulation for confirmation of needle placement. Radiopaque contrast dye may be injected for the purpose of outlining the sciatic nerve.

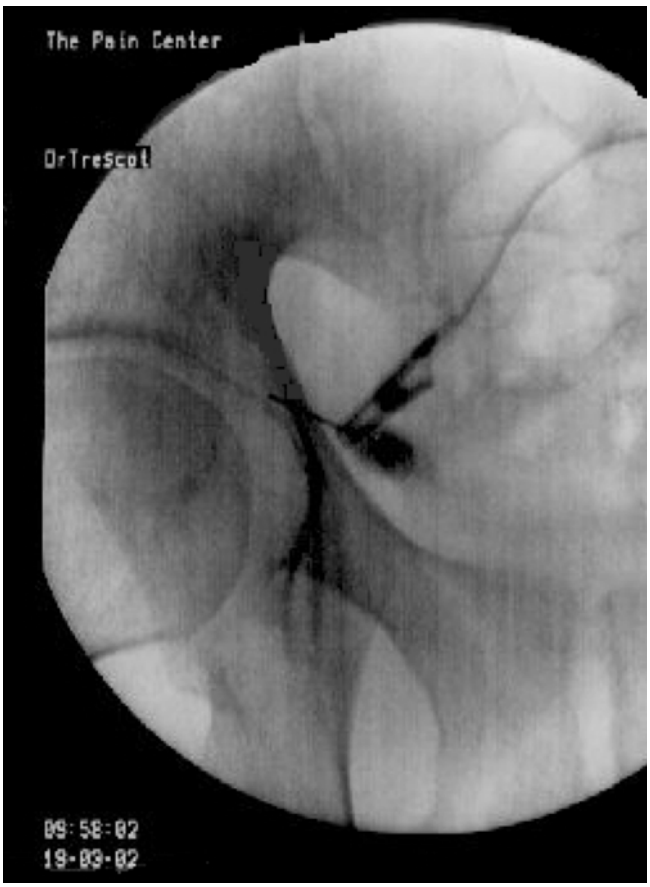

Fig. 65.17 Fluoroscopic sciatic nerve injection (Image courtesy of Andrea Trescot, MD)

Ultrasound-Guided Injection

Using ultrasound guidance, the sciatic nerve may be blocked from a gluteal approach or from a proximal thigh approach. For the gluteal approach, the technique is the same as when the injection is performed landmark guided (as above), with the added benefit of being able to visualize the path of the needle and the injectate in real time. The patient is placed in the lateral position with the extremity to be blocked flexed maximally at the hip and slightly at the knee. The gluteal region, the greater trochanter of the affected side, the PSIS, the sacral hiatus, and the ischial tuberosity are identified by palpation, and the SN is identified between the greater trochanter and the ischial spine (Fig. 65.18). Using an in-plane approach, an 8-cm 25- or 22-gauge insulated block needle is

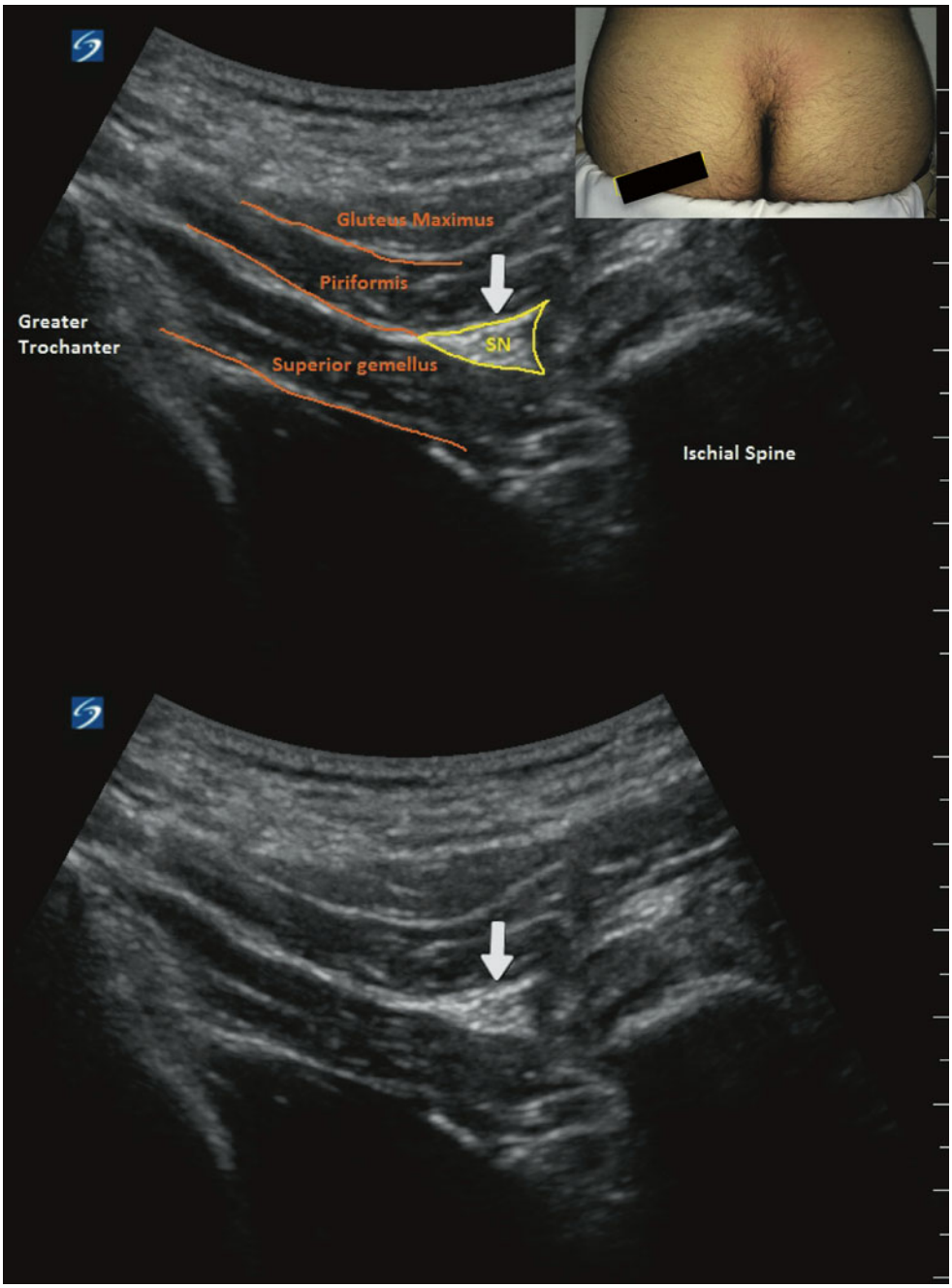

Fig. 65.18 Ultrasound identification of the sciatic nerve. *Arrow* identifies the sciatic nerve (Image courtesy of Agnes Stogicza, MD)

inserted adjacent to the lateral aspect of the ultrasound transducer, approximately 3 cm below the midpoint between the greater trochanter and the PSIS (Fig. 65.19). After local anesthetic infiltration of the skin and under sterile precautions, the needle is then advanced along the long axis of the transducer until the needle reaches the area around the sciatic nerve. After negative aspiration for blood, the local anesthetic spread can be observed [50].

Performing a sciatic nerve block from a proximal thigh approach is considered an advanced skill set, as the injection is performed from the anterior aspect of the thigh, and being able to visualize the sciatic nerve with ultrasound guidance can be challenging. Using an anterior in-plane approach, the thigh should be externally rotated in order to laterally displace the femoral neurovasculature. An 8–12-cm 22-gauge insulated block needle is utilized, depending on the thickness and depth of the patient's thigh muscles. Under sterile precautions and after local skin anesthetic, the needle is inserted adjacent to the medial aspect of the ultrasound transducer, medial to the femoral vessels. The needle is then advanced along the transducer beam and moved posteriorly from medial to lateral. Again, needle placement can be confirmed with the use of peripheral nerve stimulation.

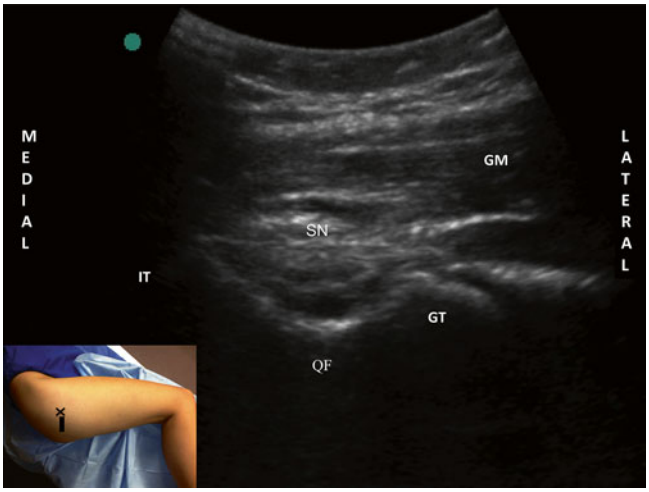

Fig. 65.19 Ultrasound injection of the sciatic nerve. *IT* ischial tuberosity, *GT* greater trochanter, *SN* sciatic nerve, *GM* gluteus maximus, *QF* quadratus femoris, *X* injection site (Image courtesy of Thiago Nouer Frederico, MD)

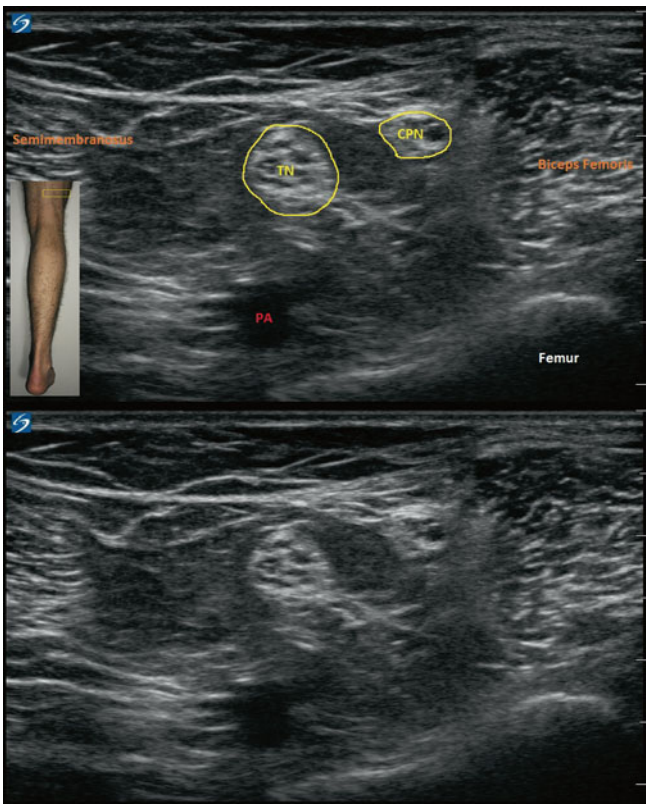

Fig. 65.20 Ultrasound evaluation of the distal sciatic nerve. *PA* popliteal artery, *TN* tibial nerve, *CPN* common peroneal nerve (Image courtesy of Agnes Stogicza, MD)

Tracing the nerve distally to the popliteal fossa, the sciatic nerve can be seen to split into the common peroneal nerve (see Chap. 67) and the tibial nerve (see Chap. 73) (Fig. 65.20).

Neurolytic Technique

The sciatic nerve is predominately a motor nerve with branches of sensory innervation. Direct ablation of the sciatic nerve would be expected to cause significant loss of motor function.

Cryoneuroablation

Because of the motor function, cryoneuroablation of the sciatic nerve itself would not be usually appropriate, except perhaps in the case of a dysfunctional limb or phantom limb pain [51].

Radiofrequency Lesioning (RF)

Because of the large, myelinated nature of the sciatic nerve, conventional radiofrequency lesioning would be inappropriate, though theoretically, pulse radiofrequency lesioning might be an option. The exception might be in the case of a dysfunctional limb or phantom limb pain.

Surgical Technique

Depending on the etiology, sometimes a surgical intervention is required for alleviation of proximal sciatic nerve entrapment. Surgical intervention is also the reported treatment for sciatic entrapment by the hamstring tendons and for the one reported case of a proximal sciatic nerve intraneural ganglion cyst [14]. In the cases of vascular malformations, Van Gompel et al. [52] described external neurolysis and limited microvascular dissection of the sciatic nerve and its terminal branches.

If the cause is an acetabular fracture, then surgical fixation can decompress the nerve [28]. If posterior thigh compartment syndrome is the etiology, fasciotomy is necessary [28, 29]. For an endopelvic pathology, laparoscopic therapy for infiltrating endometriosis may be performed to alleviate vascular entrapment of the sacral plexus [34]. Pelvic tumor excision may need to be performed if the tumor or metastasis is infiltrating the nerve. With any hip surgery, the possibility of the patient developing heterotopic ossification should be kept in mind. If this occurs, surgical excision of heterotopic bone is performed, especially if it is causing entrapment of the sciatic nerve [7]. If piriformis syndrome is diagnosed and if conservative therapies including physical therapy, anti-inflammatory medications, and piriformis injection have failed to alleviate symptoms, the patient may be a candidate for surgical release of the sciatic nerve proximally [53]. It is important

to note that the very reason for the surgical intervention may result in further sciatic nerve entrapment.

Complications

There is a significant risk of life-threatening arterial puncture, hematoma formation, and localized bleeding because of the close proximity of the inferior gluteal artery to the sciatic nerve when using the transgluteal approach. With the anterior approach, there can be blood vessel puncture of the femoral and femoris profunda artery or a neural injury to the femoral nerve or its branches [54]. Injury to the sciatic nerve can also occur during the sciatic injection itself [46].

Summary

Etiologies of sciatic nerve entrapment causing lower extremity pain are multiple, and a review of the anatomy is essential for the understanding of the biological basis of its various injuries.

References

1. Fischer SR, Christ DJ, Roehr BA. Sciatic neuropathy secondary to total hip arthroplasty wear debris. J Arthroplasty. 1999;14(6):771–4.
2. Schmalzried TP, Amstutz HC, Dorey FJ. Nerve palsy associated with total hip replacement. Risk factors and prognosis. J Bone Joint Surg Am. 1991;73(7):1074–80.
3. Weil Y, Mattan Y, Goldman V, Liebergall M. Sciatic nerve palsy due to hematoma after thrombolysis therapy for acute pulmonary embolism after total hip arthroplasty. J Arthroplasty. 2006;21(3):456–9.
4. Kemler MA, de Vries M, van der Tol A. Duration of preoperative traction associated with sciatic neuropathy after hip fracture surgery. Clin Orthop Relat Res. 2006;445:230–2.
5. Yuen EC, So YT. Sciatic neuropathy. Neurol Clin. 1999;17(3):617–31, viii.
6. Ramtahal J, Ramlakhan S, Singh K. Sciatic nerve injury following intramuscular injection: a case report and review of the literature. J Neurosci Nurs J Am Assoc Neurosci Nurs. 2006;38(4):238–40.
7. Issack PS, Helfet DL. Sciatic nerve injury associated with acetabular fractures. HSS J Musculoskelet J Hosp Spec Surg. 2009;5(1):12–8.
8. Bodack MP, Cole JC, Nagler W. Sciatic neuropathy secondary to a uterine fibroid: a case report. Am J Phys Med Rehabil. 1999;78(2):157–9.
9. Vilos GA, Vilos AW, Haebe JJ. Laparoscopic findings, management, histopathology, and outcome of 25 women with cyclic leg pain. J Am Assoc Gynecol Laparoscopists. 2002;9(2):145–51.
10. Holland NR, Schwartz-Williams L, Blotzer JW. "Toilet seat" sciatic neuropathy. Arch Neurol. 1999;56(1):116.
11. Scherer K, Skeen MB, Strine SA, Juel VC. Hanging leg syndrome: combined bilateral femoral and sciatic neuropathies. Neurology. 2006;66(7):1124–5.
12. Gozal Y, Pomeranz S. Sciatic nerve palsy as a complication after acoustic neurinoma resection in the sitting position. J Neurosurg Anesthesiol. 1994;6(1):40–2.
13. Sciatic nerve compression during pregnancy [11/3/14]. Available from: http://www.americanpregnancy.org/pregnancyhealth/sciatica.
14. Swartz KR, Wilson D, Boland M, Fee DB. Proximal sciatic nerve intraneural ganglion cyst. Case Rep Med. 2009;2009:810973.
15. Robinson DR. Pyriformis syndrome in relation to sciatic pain. Am J Surg. 1947;73(3):355–8.
16. Shields Jr RW, Root Jr KE, Wilbourn AJ. Compartment syndromes and compression neuropathies in coma. Neurology. 1986;36(10):1370–4.
17. Wilbourn AJ, Mitsumoto H. Proximal sciatic neuropathies caused by prolonged sitting. Neurology. 1988;38:400.
18. Chatillon CE, Guiot MC, Jacques L. Lipomatous, vascular, and chondromatous benign tumors of the peripheral nerves: representative cases and review of the literature. Neurosurg Focus. 2007;22(6), E18.
19. Ricci S, Georgiev M, Jawien A, Zamboni P. Sciatic nerve varices. Eur J Vasc Endovasc Surg. 2005;29(1):83–7.
20. Valat JP, Genevay S, Marty M, Rozenberg S, Koes B. Sciatica. Best Pract Res Clin Rheumol. 2010;24(2):241–52.
21. Wong M, Vijayanathan S, Kirkham B. Sacroiliitis presenting as sciatica. Rheumatology. 2005;44(10):1323–4.
22. Goh KJ, Tan CB, Tjia HT. Sciatic neuropathies – a retrospective review of electrodiagnostic features in 29 patients. Ann Acad Med Singapore. 1996;25(4):566–9.
23. Hirose CB, McGarvey WC. Peripheral nerve entrapments. Foot Ankle Clin. 2004;9(2):255–69.
24. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
25. Sunderland S. Nerves and nerve injuries. 2nd ed. London: Churchill Livingstone; 1978.
26. Boyajian-O'Neill LA, McClain RL, Coleman ML, Thomas PP. Diagnosis and management of piriformis syndrome: an osteopathic approach. JAOA. 2006;108(11):657–64.
27. Chiang EP, Dangerfield P, Asay D, Cucchiaro A, Berger JS. Ultrasound imaging of the sciatic nerve division in the popliteal fossa: a volunteer study. Open J Anesthes. 2013;3(5):288–92.
28. Martinoli C, Miguel-Perez M, Padua L, Gandolfo N, Zicca A, Tagliafico A. Imaging of neuropathies about the hip. Eur J Radiol. 2011;82(1):17–26.
29. Mallo GC, Stanat SJ, Al-Humadi M, Divaris N. Posterior thigh compartment syndrome as a result of a basketball injury. Orthopedics. 2009;32(12):923.
30. Letournel E, Judet R. Early complications of operative treatment within three weeks of injury. In: Elso RA, editor. Fractures of the Acetabulum. 2nd ed. Berlin: Springer; 1993. p. 535–40.
31. Unwin A, Scott J. Nerve palsy after hip replacement: medico-legal implications. Int Orthop. 1999;23(3):133–7.
32. Safaz I, Alaca R, Bozlar U, Yasar E. Bilateral sciatic nerve entrapment due to heterotopic ossification in a traumatic brain-injured patient. Am J Phys Med Rehabil. 2008;87(1):65–7.
33. Thakkar DH, Porter RW. Heterotopic ossification enveloping the sciatic nerve following posterior fracture-dislocation of the hip: a case report. Injury. 1981;13(3):207–9.
34. Possover M, Schneider T, Henle KP. Laparoscopic therapy for endometriosis and vascular entrapment of sacral plexus. Fertil Steril. 2011;95(2):756–8.
35. Pradat PF, Bouche P, Delanian S. Sciatic nerve moneuropathy: an unusual late effect of radiotherapy. Muscle Nerve. 2009;40(5):872–4.
36. Speed C. Low back pain. BMJ. 2004;328(7448):1119–21.
37. Katirji B. Electromyography in clinical practice – a case study approach. St. Louis: Mosby; 1998.

38. Distad BJ, Weiss MD. Clinical and electrodiagnostic features of sciatic neuropathies. Phys Med Rehabil Clin N Am. 2013;24(1):107–20.
39. Nguyen T, Sheikh K. Sciatic neuropathy: pathogenesis and pathophysiology. 2011. [10/14/14]. Available from: http://www.medmerits.com/index.php/article/sciatic_neuropathy.
40. Saikku K, Vasenius J, Saar P. Entrapment of the proximal sciatic nerve by the hamstring tendons. Acta Orthop Belg. 2010;76(3):321–4.
41. Maravilla KR, Bowen BC. Imaging of the peripheral nervous system: evaluation of peripheral neuropathy and plexopathy. AJNR Am J Neuroradiol. 1998;19(6):1011–23.
42. Chhabra A, Chalian M, Soldatos T, Andreisek G, Faridian-Aragh N, Williams E, et al. 3-T high-resolution MR neurography of sciatic neuropathy. AJR Am J Roentgenol. 2012;198(4):W357–64.
43. Ergun T, Lakadamyali H. CT and MRI in the evaluation of extraspinal sciatica. Br J Radiol. 2010;83(993):791–803.
44. Kirschner JS, Ben-Meir RS, Diamond J. Piriformis syndrome. In: Wyss JF, Patel AD, editors. Therapeutic programs for musculoskeletal disorders. 1st ed. New York: Demos; 2013. p. 363–9.
45. Kashuk K. Proximal peripheral nerve entrapment syndromes in the lower extremity. J Am Podiatry Assoc. 1977;67(8):529–44.
46. Jung Kim H, Hyun Park S. Sciatic nerve injection injury. J Int Med Res. 2014;42(4):887–97.
47. Curlee PM. Other disorders of the spine. In: Canale ST, Beatty JH, editors. Campbell's operative orthopaedics. 11th ed. Philadelphia: Mosby Elsevier; 2007.
48. Stein BE, Srikumaran U, Tan EW, Freehill MT, Wilckens JH. Lower-extremity peripheral nerve blocks in the perioperative pain management of orthopaedic patients: AAOS exhibit selection. J Bone Joint Surg Am. 2012;94(22), e167.
49. Gaertner E. Parasacral nerve block. JNYSORA. 2006;11:11–5.
50. Sites BD, Antonakakis JG. Ultrasound guidance in regional anesthesia: state of the art review through challenging clinical scenarios. Local Reg Anesth. 2009;2:1–14.
51. Moesker AA, Karl HW, Trescot AM. Treatment of phantom limb pain by cryoneurolysis of the amputated nerve. Pain Pract. 2014;14(1):52–6.
52. Van Gompel JJ, Griessenauer CJ, Scheithauer BW, Amrami KK, Spinner RJ. Vascular malformations, rare causes of sciatic neuropathy: a case series. Neurosurgery. 2010;67(4):1133–42; discussion 42.
53. Kobbe P, Zelle BA, Gruen GS. Case report: recurrent piriformis syndrome after surgical release. Clin Orthop Relat Res. 2008;466(7):1745–8.
54. Atchabahian A. Sciatic nerve block: anterior approach using neurostimulator without ultrasound guidance. JNYSORA. 2009;12(24).

Lumbar Plexus Entrapment: Lower Extremity

66

Susan R. Anderson-Jones, Tiffany Zhang, and Andrea M. Trescot

Introduction

The *lumbosacral plexus* provides the nerve supply to the legs, pelvis, lower back, and abdomen. As the lumbosacral plexus travels from the lower spine to the lateral pelvic walls, there are characteristic sites of compression and entrapment. These areas can present with confusing and overlapping clinical features. These compressions would seem to manifest clinically with predictable symptoms, specific to the nerve or nerves involved. However, more often, they present as vague and poorly localized symptoms that make it difficult to distinguish from more common causes of pain. The plexopathies can go under-recognized and underdiagnosed. They may be mistaken for more proximal lesions in the plexus such as compression from a disk within the canal or foramen. In addition, since the plexus traverses a number of structures, the generator of the pain may be misdiagnosed as primary foot, ankle, knee, hip, sciatic, or low back pain. Clues within the history and physical examination, electrodiagnostic testing, and imaging studies may provide a correct diagnosis. Lumbar and sacral plexus entrapments are described in the abdominal (Chap. 43), pelvic (Chap. 49), and lower extremity (this chapter) plexus chapters, with specific regional emphasis in each respective chapter, although overlapping information is inevitable.

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Clinical Presentation (Tables 66.1, 66.2, and 66.3)

The clinical picture of *lumbosacral plexopathy* varies and depends on the location and degree of involvement. Patients may present with significant and debilitating pain, proximal muscle weakness and wasting, sensory deficits, and loss of deep tendon reflexes. They have difficulty getting up from a chair and climbing stairs. Bowel and bladder incontinence can occur rarely and clinically mimic *cauda equina syndrome*.

In addition to nonspecific groin pain (Fig. 66.1), pelvic, or low back pain, the patients with lumbosacral plexus pathology often present with asymmetric motor (weakness) and

Table 66.1 Occupation/exercise/trauma history relevant to lumbosacral plexus entrapment

Mechanical compression (cysts, aneurysms, tumor, hematoma, etc.)	Fallopian and ovarian cysts [1]
	Intra-abdominal or pelvic aneurysm [2]
	Lymphoma or enlarged lymph node [3]
	Retroperitoneal or psoas hematoma [4–6]
Tumor compression or invasion	Malignant psoas syndrome
	Adjacent tumor (colorectal, ovarian, uterine, or cervical) [7, 8]
	Metastatic tumor (breast, sarcoma, lymphoma, multiple myeloma) [9]
Trauma (plexus traction)	Pelvic or sacral fracture [10–13]
Intraoperative compression or traction	From intraoperative patient positioning leading to stretching or compressing the plexus [14, 15]
Infection	Psoas abscess [16]
Endometriosis	Pelvic endometriosis, not common [17, 18]
Pregnancy and/or delivery	Occurring in 3rd trimester [19];
	Labor/delivery when lumbosacral trunk is compressed against less cushioned pelvic rim [19, 20]

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Table 66.2 Lumbar plexopathy clinical presentations

	Ilioinguinal/iliohypogastric neuropathy	GFN neuropathy	Femoral neuropathy	Obturator neuropathy	LFC neuropathy
Motor findings	Possible weakness of abdominal wall muscles	Weakness of the cremasteric muscle	Weakness of quadriceps and iliopsoas muscles	Weakness of hip adductor muscles	None
Sensory findings	Deficits of sensation of the lower abdomen and groin skin	Deficits of sensation of the lower abdomen and groin skin	Deficits of sensation of the anterior and medial thigh and anteromedial leg skin	Deficits of sensation of the skin of the upper medial thigh	Deficits of sensation of the skin of the anterolateral thigh

Table 66.3 Sacral plexopathy clinic presentations

	Sciatic nerve	Posterior cutaneous nerve of the thigh	Superior and inferior gluteal nerve	Pudendal nerve
Motor findings	Weakness of hamstrings; dorsal and plantar flexion of ankle and toes	None	Weakness of gluteal muscles (superior, medius, and minimus)	Weakness of anal and urethral sphincters; erectile dysfunction
Sensory changes	Posterior calf, sole, and dorsum of foot	Lower buttock and posterior thigh	None	Lower anal canal and perineal skin

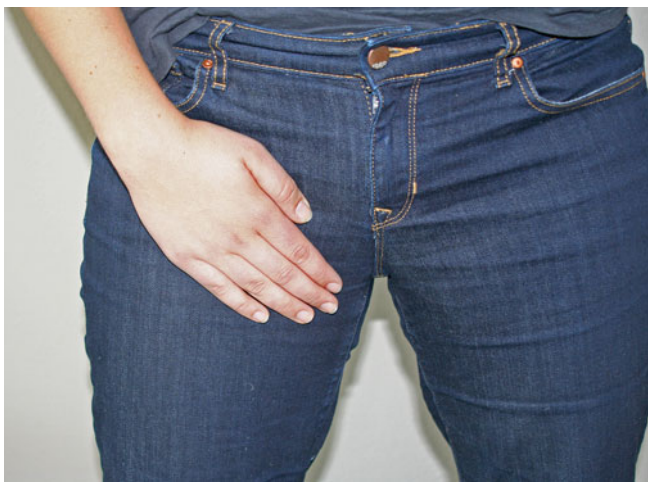

Fig. 66.1 Pattern of nonspecific upper leg pain due to lumbosacral plexus entrapment (Image courtesy of Andrea Trescot, MD)

sensory changes (numbness, dysesthesia, and/or paresthesia) and pain areas involving multiple consecutive nerve levels, which is a key element differentiating lumbar plexopathy from more proximal spinal radiculopathy. Careful examination may help to localize the lesion within the plexus. The neurological findings are often related to the levels and peripheral nerves involved (Tables 66.2 and 66.3).

Typically, patients with upper nerve root involvement present with symptoms in the femoral and obturator nerves (Fig. 66.2). *Femoral neuropathy* causes weakness of the quadriceps and iliopsoas major muscles, with or without sensory deficits over the anterior and medial thigh and anteromedial leg, whereas *obturator neuropathy* causes weakness in the hip adductors and sensory changes in the upper medial thigh [14, 21]. Sensory symptoms may include abnormal sensation (dysesthesia), decreased sensation (paresthesia), or complete loss of sensation in the area of the anterolateral thigh as a result of *lateral femoral cutaneous nerve* involvement and in the mons and labia majora due to *genitofemoral nerve* involvement. Damage to the *ilioinguinal or iliohypogastric nerves* manifests with burning pain in the upper medial thigh, lower abdomen, or pelvic area and altered sensation in the inguinal area [22]. Patients may present with debilitating pain that radiates to the lower back and buttocks that progresses posterolaterally down the leg, followed by numbness and weakness, due to sciatic, posterior femoral cutaneous, or obturator nerve entrapment (Fig. 66.3). In some patients, the clinical picture may be further complicated by foot drop, sensory changes to the top of the foot, a loss of tendon reflexes, and rarely, bowel and bladder incontinence, as well as sexual dysfunction [10, 23, 24] (Tables 66.2 and 66.3). The ilioinguinal, iliohypogastric, and genitofemoral nerves also exit through the psoas muscle, causing abdominal, groin, and pelvic pain (see Chaps. 43 and 49).

Typically, nerve entrapment presents with unilateral localization of symptoms, which indicates a localized entity, whereas bilateral symptoms indicate a systemic process such as diabetes or neurofibromatosis [23].

Onset of entrapment syndrome can be acute (e.g., hematoma, trauma), subacute, or insidious (e.g., tumor). Careful history taking may help define the underlying pathology. Clinical assessment is focused on muscle weakness and sensory disturbances as well as pain. Most lumbosacral plexopathies have an acute to subacute onset, which is helpful in identifying the process. A slow progressive course may point to a malignant cause, whereas a relapsing course may favor an inflammatory cause. Determine if the process was unilateral or bilateral at onset. Inflammatory plexopathies are unilateral and focal in onset, but become bilateral and widespread with time. At clinical presentation, the disease may be

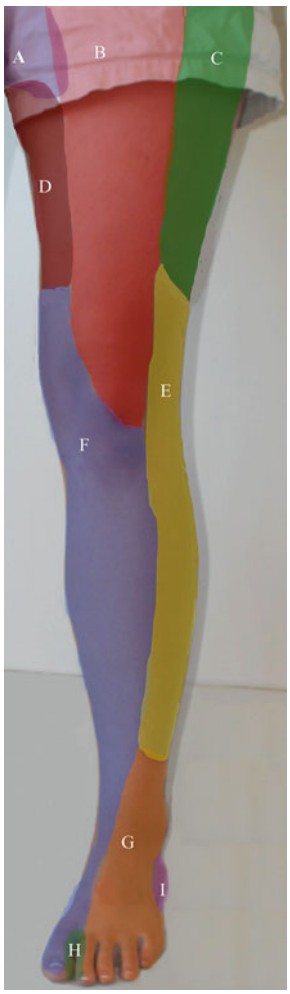

Fig. 66.2 Pattern of pain from anterior lower extremity nerve entrapments. *A* Genitofemoral nerve, *B* femoral nerve, *C* lateral femoral cutaneous nerve, *D* obturator nerve, *E* lateral sural cutaneous nerve, *F* saphenous nerve, *G* superficial peroneal nerve, *H* deep peroneal nerve, *I* sural nerve (Image courtesy of Andrea Trescot, MD)

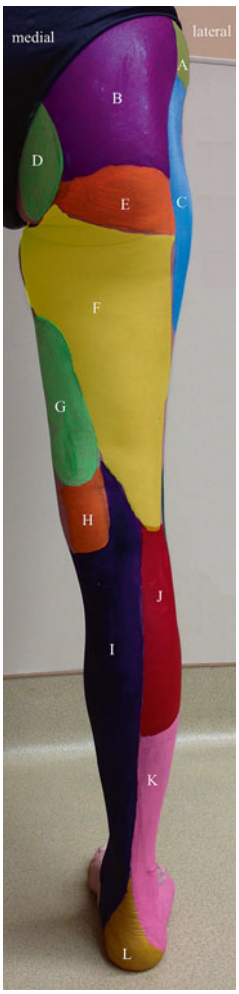

Fig. 66.3 Pattern of pain from of posterior leg nerve entrapments. *A* Lateral branch iliohypogastric nerve, *B* superior cluneal nerve, *C* lateral femoral cutaneous nerve, *D* middle cluneal/sacral nerve, *E* inferior cluneal nerve, *F* posterior femoral cutaneous nerve, *G* obturator nerve, *H* femoral nerve, *I* saphenous nerve, *J* lateral sural cutaneous nerve, *K* superficial peroneal nerve, *L* medial calcaneal nerve (Image courtesy of Terri Dallas-Prunskis, MD)

bilateral, but it should not be assumed that symptoms were bilateral at onset; it is important to clarify the time course with the medical history. Confirm that symptoms are confined to the lower limb; if upper limbs are involved, it makes a structural process less likely and raises concern for a more diffuse (possibly inflammatory) process affecting cervical, thoracic, as well as lumbosacral segments.

Associated pain and sensation changes are important clues in the lumbosacral plexopathies. If the weakness seems to be confined to the plexus distribution, the sensory loss will seem to follow a more dermatomal distribution; this may encourage imaging and further workup, because concern for a primary root level process (i.e., radiculopathy) may be heightened. Weight loss, in addition to constitutional symptoms such as fever and night sweats, may support the diagnosis of neoplastic infiltration or a paraneoplastic cause. A rash associated with the neuropathic symptoms may occur in the context of certain vasculitis. The majority of abscesses are usually secondary to local gastrointestinal, urinary, or spinal infection [7]. Abscesses affecting the psoas, gluteal, and pelvic regions are uncommon but carry a significant morbidity [16]. If symptoms occurred in anticoagulant patients, or shortly after invasive procedures, bleeding disorder or hematoma should be immediately sought after [4].

Lymphoma may cause extrinsic compression of the plexus by enlarged lymph nodes [3]. There may be direct spread of the primary tumor to the plexus or metastatic deposits in the surrounding soft and bony tissues or plexus itself. Patients with tumor plexopathy report pain as their predominant symptom [3, 8, 9]. Many different tumors have been reported to produce tumor plexopathy including colorectal, genitourinary, and lung carcinomas, as well as a range of sarcomas [25]. Benign neurofibromas and plexiform lesions are reported to be relatively common in patients

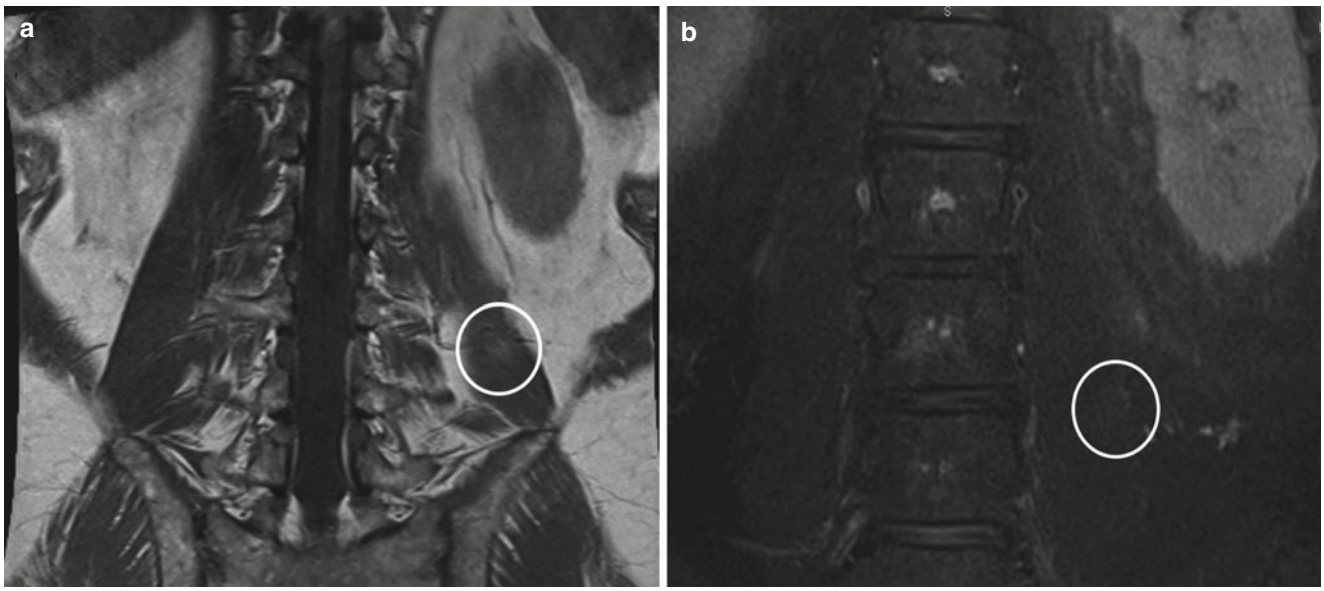

Fig. 66.4 MRI showing psoas lesion, *circled in white*. (**a**) T1 sagittal image; (**b**) T2 sagittal image with contrast (note ring enhancement of the lesion) (Image courtesy of Andrea Trescot, MD)

with *neurofibromatosis* type 1, occurring in the abdominopelvic region in up to 40 % of patients. The most common site where the lumbosacral plexus may be involved is the retroperitoneum [26].

Iliopsoas hematoma is a rare complication that occurs in patients receiving anticoagulant therapy. The clinical manifestation of iliopsoas hematoma is nonspecific. It can mimic orthopedic or neurological disorders, including paresthesia or paresis of the thigh and leg due to compression of the nerve plexus. Computed tomography (CT) is the most useful radiological method for diagnosis. If the patient is hemodynamically unstable or has active bleeding, transcatheter arterial embolization and surgical intervention may be required [5]. *Retroperitoneal hematoma* can compress the lumbar plexus diffusely within the psoas muscle, leading to weakness in both obturator and femoral nerve distributions. More often, the intrapelvic portion of the femoral nerve is compressed by relatively small hematomas within the non-expandable fascia of the iliacus muscle. These syndromes usually occur during anticoagulant therapy but can occur with *hemophilia*, leaking *aortic aneurysms*, or idiopathically [6].

Psoas muscle disease, whether related to surgery or trauma, anticoagulant therapy-related hematoma, abscess, or tumor infiltration (*neoplastic plexopathy*), can directly compress the lumbar plexus. Painful neoplastic plexopathy due to tumor infiltration of the psoas muscle is known as *malignant psoas syndrome*, most commonly following direct invasion of the psoas muscle by adjacent tumors of colorectal, ovarian, uterine, or cervical origin or in the setting of metastatic spread of disease, most commonly from breast cancer, sarcoma, lymphoma, and multiple myeloma. Direct invasion of the lumbosacral plexus from perineural tumors is less common. Malignant psoas syndrome can mimic *cauda equina syndrome*. It typically involves a proximal lumbosacral plexopathy (L1–4) and presents as a painful fixed flexion of the ipsilateral hip with pain exacerbated by attempted extension of the hip (*positive psoas test*). Radiological or pathological evidence will demonstrate malignant involvement of the ipsilateral psoas muscle (Fig. 66.4).

The sacral plexus may also be affected by infection, arthritis, or compression injuries. Infectious or arthritic diseases of the sacroiliac joints, pelvic and hip fractures, and pelvic surgery may directly injure or stretch the sacral plexus. Colorectal and cervical cancers can directly involve or compress the sacral plexus. Pelvic irradiation and aortic aneurysms are less common causes of sacral plexopathy.

Endometriosis of the lumbosacral plexus has been reported in approximately 20 cases in the literature. It presents with *catamenial sciatica* (related to menses) [17]. The lesion is usually solitary and lies on the sciatic nerve within the pelvis, just as it passes into the gluteal compartment. The focal mass is classically of high signal intensity on both T2- and T1-weighted images, suggesting acute hemorrhage [18].

Anatomy (Tables 66.4 and 66.5)

The lumbosacral plexus passes through the psoas muscle (Fig. 66.5) and may be divided functionally and anatomically into the *lumbar plexus* and the *sacral plexus*. The lumbar plexus is formed from the anterior rami of the

Table 66.4 Lumbar plexus anatomy

Origin	Anterior rami from T12 to L5
General route	Passes anterior to transverse process from L2 to L5 within the psoas muscle. Exits at lateral psoas muscle. 6 nerves: *iliohypogastric* (Chap. 40), *ilioinguinal* (Chap. 44), *genitofemoral* (Chap. 45), *lateral femoral cutaneous* (Chap. 61), *femoral* (Chap. 57), *obturator* (Chap. 64) nerves
Sensory distribution	*Iliohypogastric/ilioinguinal nerves* (from L1) – suprapubic and inguinal regions
	Genitofemoral nerve (L1 and L2) – inguinal region
	Lateral femoral cutaneous nerve (L2 and L3) – lateral thigh region
	Femoral nerve (L2–L4) – anterior and medial thigh, medial leg distal to the knee
	Obturator nerve (L2–L4) – medial leg proximal to the knee
Motor innervation	*Genitofemoral nerve* (L1 and L2) – cremaster muscle
	Femoral nerve (L2–L4) – rectus femoris, vastus medialis, vastus intermedius, and vastus lateralis muscles of the thigh
	Obturator nerve (L2–L4) – adductor thigh muscles

Table 66.5 Sacral plexus anatomy

Origin	Anterior rami of L4–L5 and S1–S4
General route	Located on the posterolateral wall of the lesser pelvis, where it is closely related to the anterior surface of the piriformis. Most branches leave the pelvis through the greater sciatic foramen. Two main nerves: *sciatic* (see Chap. 55) and *pudendal* (see Chap. 47), plus the *posterior femoral cutaneous* (see Chap. 46), *superior gluteal* (see Chap. 53), and *inferior gluteal* (see Chap. 54) nerves
Sensory distribution	*Sciatic nerve* (L4–S3) – posterior calf, sole, and dorsum of foot
	Posterior cutaneous nerve of the thigh (S1–S3) – posterior thigh and lower posterior buttock
	Pudendal nerve (S2–S4) – lower anal canal and perineal skin
Motor innervation	*Sciatic nerve* – hamstrings, dorsi and plantar flexors of ankle and toes, intrinsic foot muscles
	Superior and inferior gluteal nerves (L5–S2) – superior gluteal, gluteus medius and minimus, tensor fasciae latae muscles
	Pudendal nerve – sphincters (external anal and urethral), erectile vessels
Anatomic variability	The sacral plexus may arise higher or lower

T12–L5 nerve roots (Fig. 66.6). The sacral plexus is the union of the lumbosacral trunk and the anterior rami of the S1–S5 nerve roots [27]. The lumbar plexus passes anterior to the transverse processes from L2 to L5 (ventral rami of L1–L4) and lies within the psoas major muscle (Fig. 66.7). It exits at the lateral border of the psoas muscle (Fig. 66.8) and consists of six nerves: *iliohypogastric*, *ilioinguinal*, *genitofemoral*, *lateral femoral cutaneous*, *femoral*, and *obturator* (Fig. 66.9).

The *iliohypogastric and ilioinguinal nerves* (see Chap. 40) are primarily sensory nerves that arise from L1 and supply innervation to the skin of the suprapubic and inguinal regions. They are terminal branches that emerge from the lateral border of the psoas muscle. The *genitofemoral nerve* (see Chap. 41) arises from L1 and L2 to supply motor innervation to the cremaster muscle and additional sensory innervation to the inguinal area. The *lateral femoral cutaneous nerve* (see Chap. 61) is formed from the L2 to L3 nerve roots and is a sensory nerve. It provides sensation to the lateral aspect of the thigh. The *femoral nerve* (see Chap. 57) from L2 to L4 is the major motor nerve of the thigh. It emerges from the lateral psoas border before coursing along the groove between the iliacus and psoas muscles, lateral to the external iliac artery before reaching the thigh. The femoral nerve provides extension at the knee through innervation of the *rectus femoris*, *vastus medialis*, *vastus intermedius*, and *vastus lateralis* muscles of the thigh. It also provides cutaneous sensory innervation to much of the anterior and medial thigh, as well as the medial portion of the leg distal to the knee. The *obturator nerve* (see Chap. 64) (which comes from L2 to L4) emerges from the medial border of the psoas and provides sensory innervation to a portion of the medial leg proximal to the knee as well as motor innervation to the adductor muscles of the thigh.

The lumbosacral trunk joins with the upper sacral roots anterior to the piriformis, behind the internal iliac vessels. It passes out of the *greater sciatic foramen* deep to the *piriformis muscle* and continues as the *sciatic nerve* (see Chap. 65). The *inferior gluteal nerve* (see Chap. 54) also

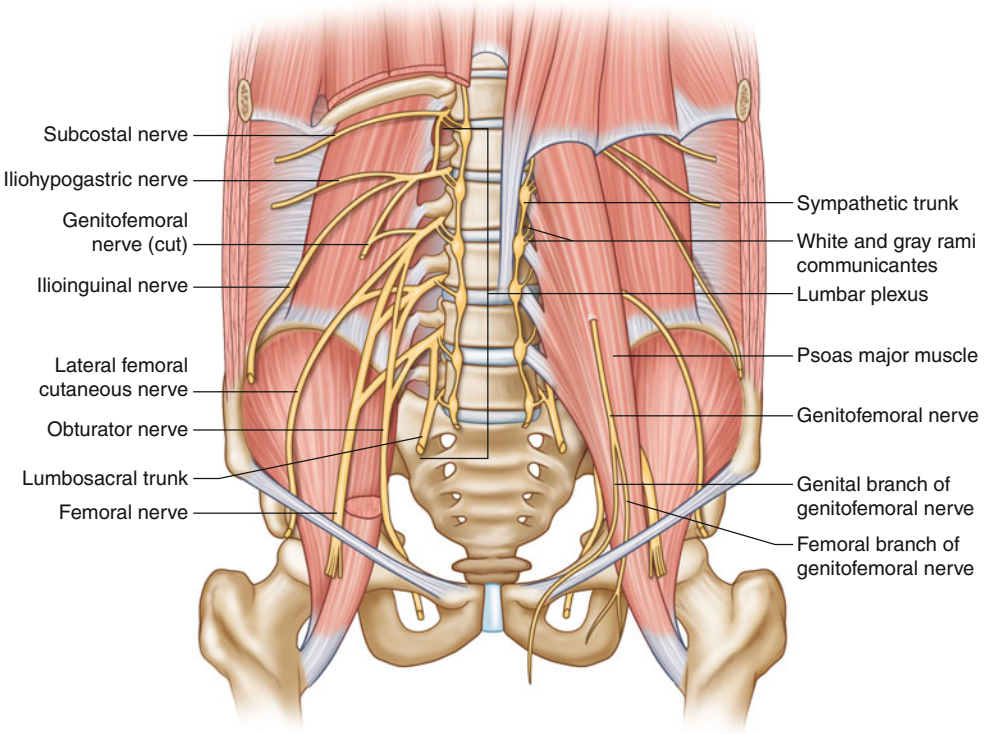

Fig. 66.5 Anatomy of the lumbosacral plexus (Image courtesy of Springer)

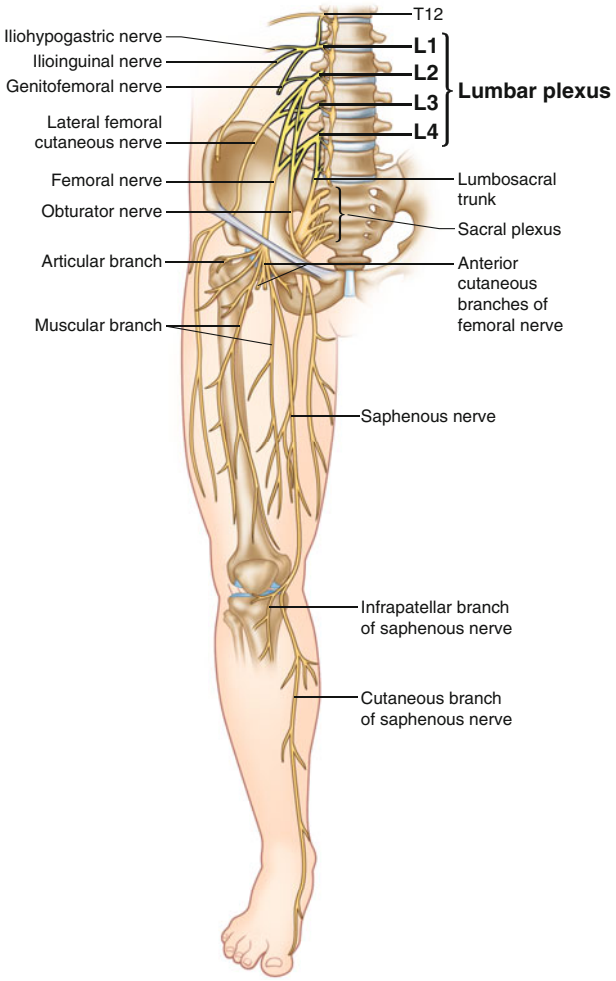

Fig. 66.6 Anatomy of the lumbosacral plexus and lower extremity (Image courtesy of Springer)

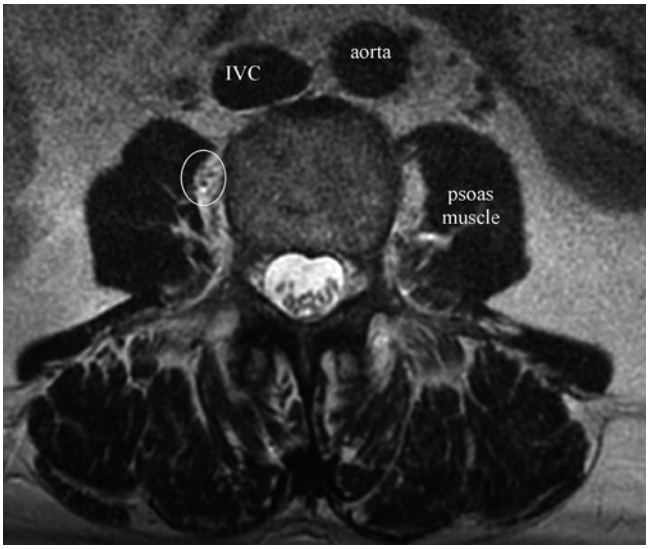

Fig. 66.7 Axial MRI image of the lumbosacral plexus (Image courtesy of Andrea Trescot, MD)

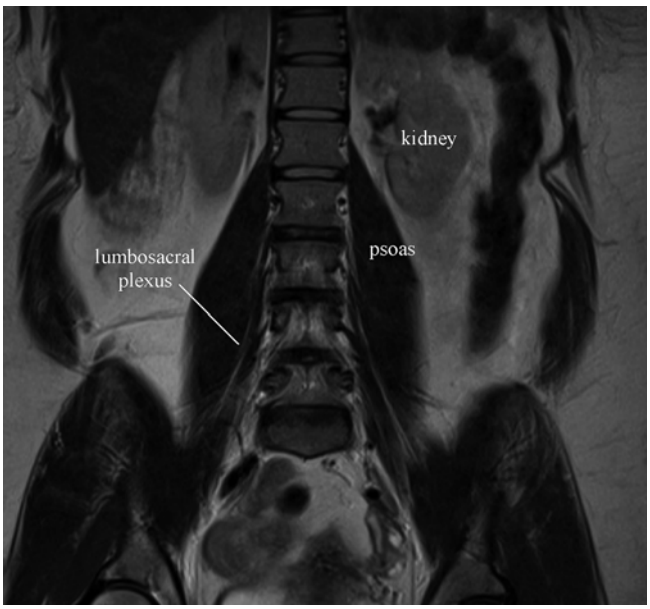

Fig. 66.8 Coronal MRI image of the lumbosacral plexus (Image courtesy of Andrea Trescot, MD)

leaves the *greater sciatic foramen* below the piriformis before entering the gluteus maximus muscle, while the *superior gluteal nerve* (see Chap. 53) emerges above the piriformis. Finally, the *pudendal nerve* (see Chap. 47) passes out of the greater sciatic foramen close to the ischial spine before reentering the pelvis through the *lesser sciatic foramen*. It continues on the lower aspect of the *obturator internus muscle* and in the lateral wall of the ischioanal fossa before terminating in the perineum [28] (Fig. 66.10).

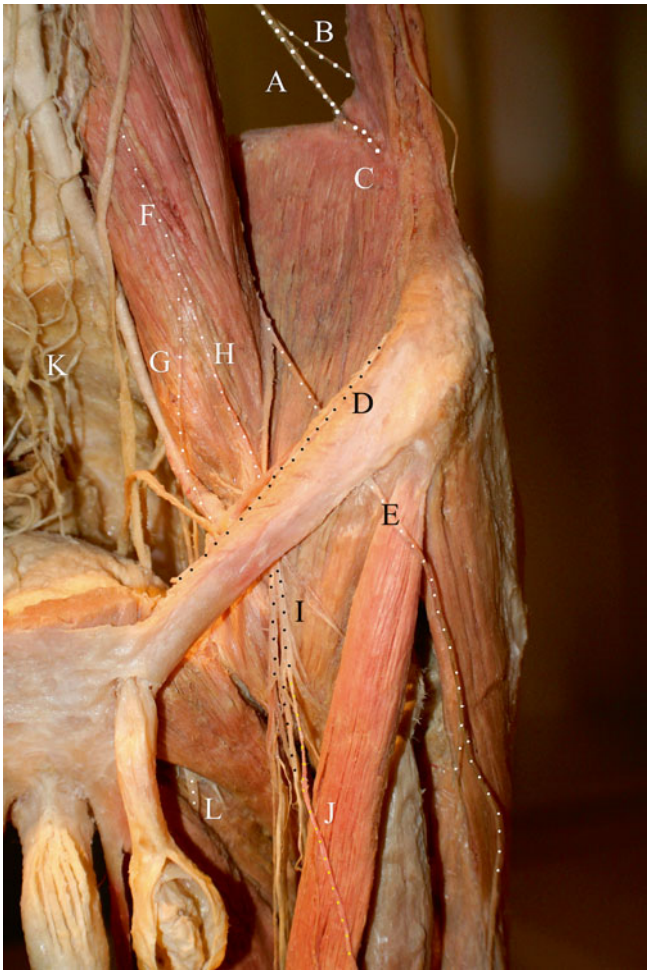

Fig. 66.9 Lumbar plexus nerves, modified from an image from *Bodies, The Exhibition*, with permission. *A* Ilioinguinal nerve, *B* iliohypogastric nerve, *C* site of ilioinguinal nerve entrapment at the external oblique, *D* ilioinguinal nerve over the inguinal ligament, *E* lateral femoral cutaneous nerve, *F* genitofemoral nerve, *G* genital branch of the genitofemoral nerve, *H* femoral branch of the genitofemoral nerve, *I* femoral nerve, *J* saphenous nerve, *K* inferior hypogastric plexus, *L* obturator nerve (Image courtesy of Andrea Trescot, MD)

Entrapment

Lumbosacral plexus entrapment may occur in various intra-abdominal or pelvic conditions that produce mechanical mass effect or may be related to trauma, surgery, or even systemic disease (Table 66.1). If pelvic trauma causes double vertical fracture-dislocations of the bony pelvic ring, lumbosacral plexus traction injury may occur, most often on the same side as the sacroiliac joint injury [12]. The lumbosacral plexus may be directly compressed at the pelvic brim, causing postpartum foot drop after protracted, cephalopelvic disproportion or midpelvic forceps delivery. The intrapelvic femoral nerve may be damaged during surgical procedures involving angulation under the inguinal ligament in the lithotomy position or by compression from surgical retractor blades in the gutter between the iliacus and psoas muscles.

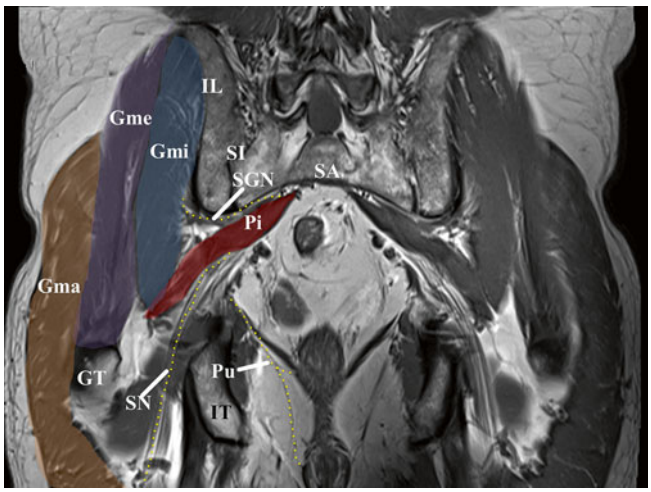

Fig. 66.10 MRI coronal image. *SA* sacrum, *SI* sacroiliac joint, *IL* ilium, *IT* ischial tuberosity, *GT* greater tuberosity, *Gmi* gluteus minimus, *Gme* gluteus medius, *Gma* gluteus maximus, *PI* piriformis, *SGN* superior gluteal nerve, *SN* sciatic nerve, *PU* pudendal nerve (Image created by Andrea Trescot, MD)

Lumbar and sacral plexopathies can be induced by extrinsic compression of a mass or diffuse infiltration by being secondarily involved in the setting of systemic or inflammatory processes. Retroperitoneal processes are more likely to affect the lumbar plexus, while pelvic disease is more likely to affect the sacral plexus.

Eighty-eight percent of posttraumatic lumbosacral plexus injuries in one study were found to have bone injuries, including pelvic fractures, acetabular fractures, sacroiliac dislocations, and sacral fractures [13]. There were both bone and vascular injuries in 30 % of the cases. Bilateral plexus injuries were seen in 5.5 % of these traumatic injuries.

Physical Exam

As opposed to injuries to peripheral nerves, where symptoms follow stereotypical innervation patterns, injuries that involve the lumbosacral plexus manifest with symptoms and skeletal muscle changes related to multiple spinal levels or multiple nerve distributions. When the symptoms are unilateral, the contralateral plexus can be used as an internal standard for comparison. However, bilateral involvement should raise the possibility of a systemic process such as diabetes mellitus or neurofibromatosis.

The signs and symptoms can be vague and nonspecific; however, this entrapment often presents with asymmetric and focal weakness, likely sensory changes, involving multiple lumbosacral nerve levels. A thorough examination of the hip girdle and leg enables classification of the injury pattern. Lumbar plexopathy tends to cause weakness of hip flexion and adduction and/or knee extension, whereas sacral plexopathy often leads to weakness of knee flexion or foot drop. Lumbar plexus entrapment may affect anteromedial thigh and leg sensation, whereas sacral plexus collectively provides sensation to the posterior thigh and leg, as well most of the foot.

The *superior gluteal nerve* (SGN) (see Chap. 53) and the *posterior femoral cutaneous nerve* (PFCN) (see Chap. 56) can be evaluated to determine level of injury. The SGN emerges from the L5 to S1 nerve roots in the proximal part of the lumbosacral plexus; abolishing activity in the gluteal muscles can impair pelvic stability, resulting in a positive *Trendelenburg test* (the inability to hold pelvis level when standing on the opposite leg) (Fig. 66.11). The PFCN forms from the sacral spinal nerves that make up the *sciatic nerve*; its intact function (normal sensation on the posterior aspect of the thigh) with weakness of the hamstrings and muscles distal to the knee indicates a proximal sciatic nerve lesion rather than a sacral injury [13].

Refer to Tables 66.2 and 66.3 for specific patterns of the lumbosacral plexus and its branches. Because contraction of the psoas muscle will increase compression of the plexus, resistance against hip flexion (Fig. 66.12) may provoke or exacerbate the symptoms. Distal tendonitis of the psoas at its attachment onto the lesser trochanter (Fig. 66.13) may also worsen with this maneuver.

Differential Diagnosis (Tables 66.6 and 66.7)

Because the causes and presentation of lumbosacral plexus entrapment can be vast and vague, the differentials are extensive. Table 66.6 lists some of common conditions that should be considered or ruled out during workup.

It should be recognized that the differential diagnosis includes many conditions invisible on imaging [30]. Patients receiving pelvic irradiation for cancer may develop insidious leg weakness [29], often bilateral, 5 or more years after the radiation; however, imaging is crucial to the differential diagnosis from cancer recurrence. Pain tends not to be a distinguishing feature of presentation from tumor infiltration. The diagnostic tests for lumbosacral plexus entrapment can be found in Table 66.7.

Electrodiagnostic Testing

For cases of lumbosacral plexopathy, electrodiagnostic studies incorporating *nerve conduction velocity* (NCV) studies and needle *electromyography* (EMG) can be helpful for localization and characterization of the underlying process. The presence of lumbosacral plexopathy can be defined when there is evidence for electrophysiologic abnormalities

Fig. 66.11 Trendelenburg's sign (Image courtesy of Andrea Trescot, MD)

in the distribution of at least two different peripheral nerves in at least two different nerve root distributions. The sparing of the paraspinal muscles on needle examination can be helpful in confirming a pure lumbosacral plexopathy. The sensory studies are likely to be most helpful for localization. Recall that in most spinal segments, the dorsal root ganglion lies lateral to and outside the intervertebral foramen. In electrodiagnostics, this feature is important because it helps assist in localization of a preganglionic process (i.e., radiculopathy) versus a postganglionic process (i.e., plexopathy or mononeuropathy). Reduced sensory nerve action potential amplitudes imply a postganglionic process and can help to exclude a radiculopathy as the main cause for clinical symptoms [31].

The femoral nerve is the only motor nerve conduction study likely to give meaningful information about the possibility of a pure lumbar plexopathy. The most common method is to stimulate the nerve high in the inguinal region and record from a quadriceps muscle. Because of the significant overlying connective tissue, needle stimulation is often necessary and may be contraindicated in some patients on anticoagulation, given the proximity to the femoral artery [32]. Using a good examination and history cannot be overstated in deciding which muscles to test.

Fig. 66.12 Physical exam of resistance to hip flexion (Image courtesy of Andrea Trescot, MD)

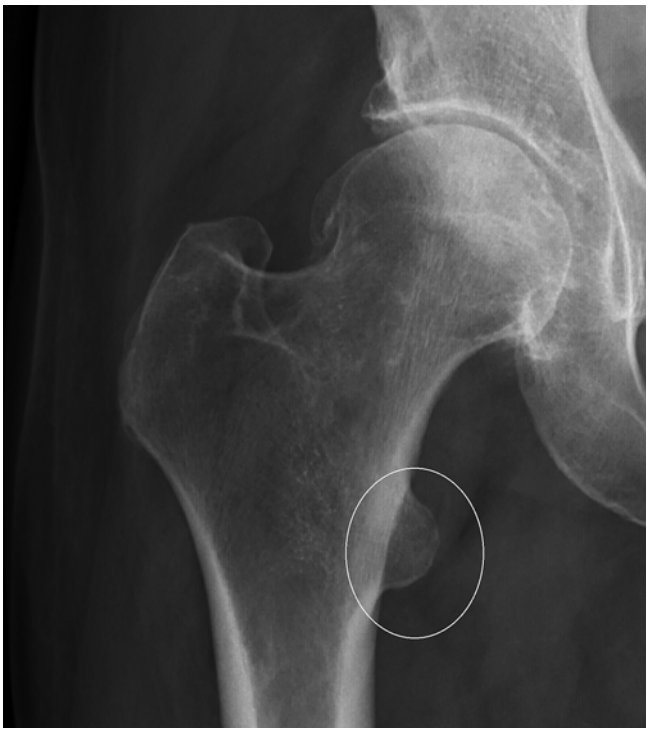

Fig. 66.13 X-ray showing lesser trochanter (Image courtesy of Andrea Trescot, MD)

Magnetic Resonance Imaging (MRI)

In the evaluation of lumbosacral plexopathy, magnetic resonance imaging (MRI) is a valuable adjunct to clinical examination and electrodiagnostic testing because it provides anatomic information that is not obtainable with other modalities and is useful for assessing lesions [21, 23, 25]. Perifascicular and perineural high signal intensity from fat makes nerves conspicuous on T1-weighted images and provides a model road map.

Table 66.6 Differential diagnosis of lower extremity pain

	Potential distinguishing features
Neuropathy after radiation therapy	Insidious bilateral leg weakness; history of radiation therapy [29]
Diabetic peripheral neuropathy	History of diabetes; other symptoms and signs of diabetes and diabetic peripheral neuropathy
Lumbosacral radiculopathy	Sensory and motor symptoms follow single nerve root; NCV/EMG may be diagnostic
Caudal equine syndrome	Urinary and/or bowel incontinence; saddle anesthesia; loss of rectal tone; often acute onset
Lumbar facet pathology	Paravertebral tenderness; lumbar spondylosis
Hematoma	Anticoagulant therapy; recent invasive procedure
Systemic inflammatory disease	Widespread and symmetrical symptoms and signs
Abscess	GI, pelvic, or abdominal infection
Hip pathology	Joint disease on X-ray
Sciatic entrapment	Piriformis spasm and tenderness

Table 66.7 Diagnostic tests for lumbosacral plexus entrapment

	Potential distinguishing features
Physical exam	Sensory and motor changes often involve overlapping contiguous spinal levels (see Tables 66.2 and 66.3)
Diagnostic injection	Psoas plexus block
Ultrasound	Less value in diagnosis due to the deep location of lumbosacral plexus
MRI	Valuable for detailed nerve, psoas muscle, and surrounding lesions; increased T2 intensity of the nerves; may enhance with contrast (infection, tumor invasion, inflammatory)
X-ray	Rule out other bony causes
Electrodiagnostic studies	EMG abnormalities in at least 2 different peripheral nerves and in at least 2 nerve roots with no paravertebral abnormalities

The plexus can be seen as fascicular structures surrounded by fat within or posterior to the muscles (Figs. 66.7 and 66.8). Terminal branches of the lumbar plexus are first detected once they exit the psoas muscle at the lateral border of the muscle (*iliohypogastric*, *ilioinguinal*, *genitofemoral*, *lateral femoral cutaneous*, and *femoral nerves*). The most useful imaging plane tends to be the axial. Disruption of the perineurial *blood-nerve barrier*, with nerve injury, can cause a change in the distribution of the endoneurial fluid and therefore increased perifascicular and endoneurial signal intensity on T2-weighted images, though it is a nonspecific response. T2-weighted *neurography* allows evaluation of changes in the perifascicular

and endoneurial signal intensity. The disruption of the perineurial blood-nerve barrier may also allow neural enhancement with *gadolinium* contrast material, if the barrier is compromised. It is not yet agreed upon the necessity for this assessment. Other valuable MRI findings of neural injuries include size and morphological changes, such as neuronal enlargement, loss of the normal fascicular appearance, or blurring of the perifascicular fat [21, 23, 25].

Identification and Treatment of Contributing Factors

The anatomical restriction of the lumbosacral plexus makes it susceptible to entrapment by variety of conditions. Any occupying lesion intrinsic or external of the psoas muscle may cause lumbar plexus entrapment [30], including retroperitoneal processes [23] and muscle invasion [33]. Its proximity to bones and major blood vessels, such as the abdominal aorta and the iliac artery and its branches, also makes it prone to compression from aneurysms of those arteries or metastatic lesions. The sacral plexus trunk passes over the sacral ala and lies over the posterior lateral wall of the pelvis; it is, therefore, easily injured from sacral and pelvic trauma or during delivery.

It has been reported that leg-length discrepancies, including from improper use of a heel lift, might irritate the psoas muscle, causing muscle spasm and leading to lumbar plexus entrapment. Proper correction of the leg length and hip level or osteopathic manipulation may prevent recurrent lumbar plexopathy [34].

Injection Techniques

The lumbar plexus block is most frequently used for surgical anesthesia of the lower extremity. It is occasionally used for the treatment of inflammatory conditions of the lumbar plexus, such as *idiopathic lumbosacral plexitis* or when the tumor has invaded the tissues innervated by the lumbar plexus or the plexus itself [33, 35]. It can be utilized as a diagnostic maneuver when performing differential neural blockade for evaluation of lower extremity pain. It may also be used palliatively for acute pain emergencies, including lower extremity trauma or fracture, acute herpes zoster, or cancer pain including tumor invasion while waiting for pharmacologic, surgical, and anticancer therapies to become effective [36].

Landmark-Guided Technique

The patient is placed in the lateral decubitus position, with the painful or operative side up and a slight forward tilt. The foot on the side to be blocked is positioned over the dependent leg so that twitches of the quadriceps muscle can be easily seen. Although several needle insertion sites have been suggested, locating the transverse process of the lumbar vertebral body with the needle tip after insertion is common to all techniques. The two surface anatomic landmarks of importance for determining the insertion point are the iliac crest and the midline spinous processes. The top of the iliac crest correlates with the body of the L4 vertebral body or the L4–5 interspace in most patients. Draw a line between the iliac crests (*Tuffier's line*) and then a vertical line marking the midline. A second vertical line is made 5 cm parasagittal to the midline on the side to be anesthetized. On this second line, a mark is made 3 cm caudal to Tuffier's line, which identifies the needle entry point [37] (Fig. 66.14). A 20- or 22-gauge, 15 cm needle is then inserted to the depth of the transverse process of the fifth lumbar vertebra. Once the transverse process is located, the needle is partially withdrawn and redirected cephalad until it slides past the transverse process. Attach a 5 cc saline-filled syringe to the needle and slowly advance it until loss of resistance is achieved. The needle tip is then within the psoas compartment. The loss of resistance typically occurs at a depth of 12 ± 2 cm. This is followed by 30 cc of local anesthetic for a surgical block or 5 cc of local anesthetic for a diagnostic injection. Keep the patient in the lateral position for 5 min following completion of the local anesthetic injection.

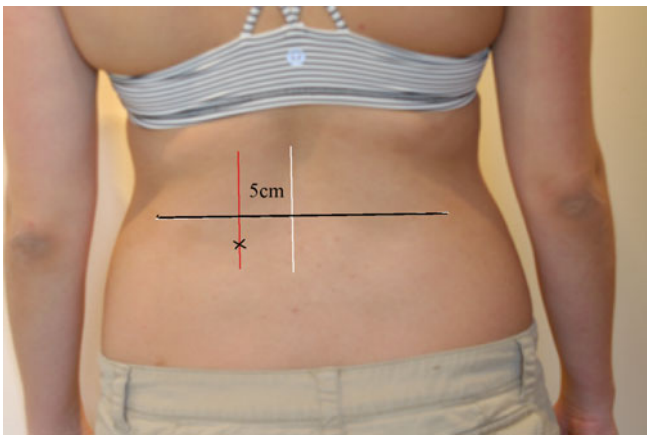

Fig. 66.14 Landmark-guided psoas injection site. *Black line* = top off iliac crests (Tuffier's line), *white line* = midline, *red line* = paravertebral line 5 cm lateral of midline, *X* = injection site (Image courtesy of Andrea Trescot, MD)

Using a Nerve Stimulator

A nerve stimulator is set to an initial current of 1.5 mA. The needle is advanced at an angle perpendicular to all skin planes. As the needle is advanced, local twitches of the

paravertebral muscles are first obtained. As the needle is further advanced, the transverse process may be encountered. Contact with the transverse process is not routinely sought but, when present, provides a consistent landmark to avoid excessive needle penetration during the lumbar plexus block [38]. The distance from the skin to the lumbar plexus ranges from 6.1 to 10.1 cm in men and 5.7–9.3 cm in women [38], with the distance correlating to gender and body mass index (BMI). The distance from the transverse process to the lumbar plexus is usually less than 2 cm, independent of BMI or gender. Contraction of the quadriceps muscle is usually obtained at a depth of 6–8 cm. The nerve stimulator current is reduced to produce stimulation of the quadriceps muscle between 0.5 and 1.0 mA, and 25–30 mL of local anesthetic is injected incrementally, with negative aspiration every 5 mL [39]. It is important to avoid too deep a needle penetration and the resultant complications that may arise, such as renal hematoma and total spinal anesthesia [40].

Ultrasound-Guided Technique

The patient is placed in the lateral decubitus position so that contractions of the quadriceps muscle are visible. Identify the iliac crests and draw a line as in the landmark-guided technique (Fig. 66.15). This time, however, the target is the paraspinal area at the level of L3/4. Ultrasound can be used to confirm the correct vertebral level and to guide the needle tip over the top of the transverse process. A low-frequency (2–5 MHz) curved array probe is used, placed in a paramedian longitudinal position. Firm pressure is required to obtain good quality images. Identify the transverse processes at the L3/4 space by moving the US probe laterally from the spinous processes in the midline, staying in the longitudinal plane. Going from the midline and moving the probe laterally, the articular processes are seen, with the adjoining superior and inferior articular processes of the facets forming a continuous "sawtooth" hyperechoic line (Fig. 66.16). As the probe is moved further laterally, the transverse processes are seen with the psoas muscle lying between them. The image is of a "trident" (Fig. 66.17), with the transverse processes causing bony shadows and the psoas muscle lying in between. A color Doppler image is then obtained to identify adjacent vasculature, to avoid inadvertent intravascular injection.

At this point, the US probe is usually 3–5 cm off the midline. The lumbar plexus is not usually directly visualized, but lies within the posterior third of the psoas muscle (i.e., the closest third of the psoas muscle seen with the US probe). The distance from the skin to the psoas muscle can be measured using the caliper function of the ultrasound machine. This gives an estimate of the depth of the lumbar plexus before needle insertion. Note that anterior to the psoas

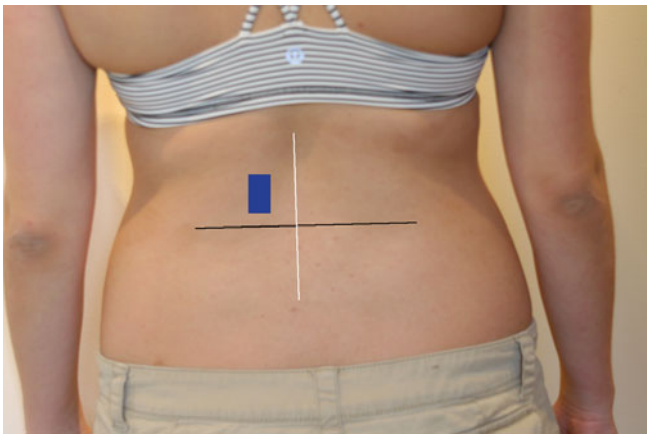

Fig. 66.15 Ultrasound-guided psoas injection site. *Black line* = top off iliac crests (Tuffier's line), *white line* = midline, *blue* = US probe (Image courtesy of Andrea Trescot, MD)

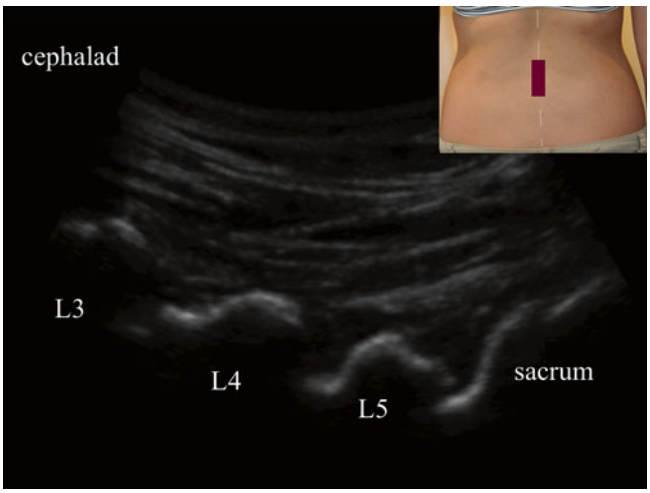

Fig. 66.16 Ultrasound image of the facets from a longitudinal approach, forming a "saw tooth" pattern (Image courtesy of Andrea Trescot, MD)

muscle (further away from the skin in this US view) lie the peritoneal cavity, the great vessels, and the kidney. Thus, care with needle tip placement should be maintained at all times.

The depth of the plexus is most often between 50 and 100 mm from the skin surface. An in-plane or out-of-plane technique may be used. If an in-plane approach is used, the usual direction for insertion is from caudad to cephalad. For the out-of-plane approach, the site for the block needle is on the medial side of the US probe (which is maintained in its longitudinal position). A 13 cm-stimulating needle may be utilized for specificity of location. The needle needs to be placed at the center of the probe, directed slightly laterally, such that in its path it comes directly under the US beam. Advancing the needle from a medial to a lateral direction is also preferred to avoid insertion into the dural cuff, which can extend laterally

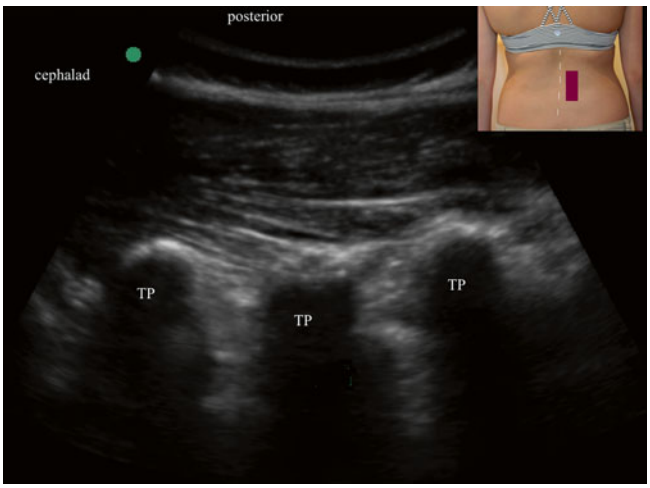

Fig. 66.17 Ultrasound image of the transverse processes from a longitudinal approach, forming a "trident" pattern (Image courtesy of Andrea Trescot, MD)

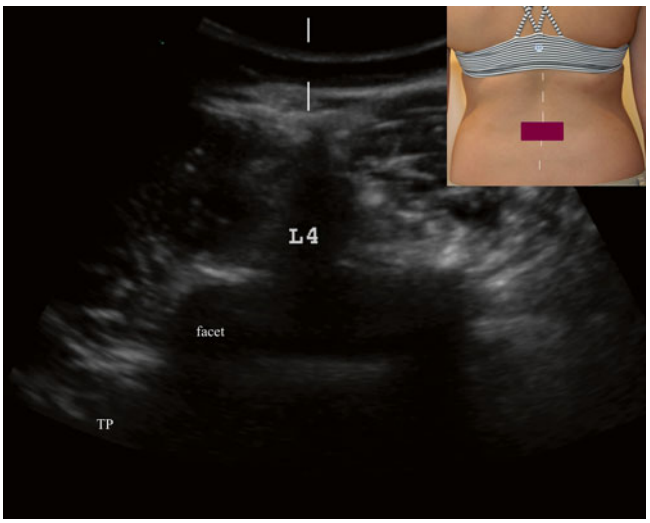

Fig. 66.18 Ultrasound image of horizontal approach to the lumbar plexus (Image courtesy of Andrea Trescot, MD)

beyond the neural foramina. Lidocaine is infiltrated into the skin and subcutaneous tissue at the point where the block needle is to be inserted. The needle is observed in real time and targeted toward the posterior third of the psoas muscle bulk. Peripheral nerve stimulation is commonly used to confirm proximity to the lumbar plexus. The target is to elicit quadriceps muscle contraction. In addition, contraction of the psoas muscle will be readily apparent under ultrasound imaging. When satisfied with needle tip position, the local anesthetic is injected incrementally (with frequent aspiration to monitor for blood or CSF), and its spread is observed, looking for fluid and tissue expansion in the psoas muscle bulk [41]. The needle is removed, and pressure is placed on the injection site to avoid hematoma formation [37].

An alternative technique uses a horizontal rather than vertical orientation. Using the same surface landmarks, the curvilinear low-frequency probe is placed parallel to the transverse processes (Figs. 66.18 and 66.19) and moved laterally. When the psoas muscle is visualized (which usually requires an ipsilateral oblique tilt of the transducer to "see under" the transverse process), the stimulating needle can be advanced in-plane to the target area (Fig. 66.20).

Fluoroscopic-Guided Technique

The patient is positioned in the prone position, and the transverse processes at L3 or L4 are identified. The psoas shadow should be visible (Fig. 66.21), and the injection site should correlate with the lateral aspect of the transverse process to avoid the nerve roots and the epidural space (Fig. 66.22). Insert a 22-gauge, 5 inch B-bevel needle using a "gun-barrel" technique until the needle is approximately at the anterior one third of the vertebral body in the lateral view. One cc of nonionic contrast is then injected, which should show the oblique flow of contract cephalad and caudad (Fig. 66.23). In the lateral view, the psoas major muscle spreads vertically over the anterior one third of the lumbar vertebral body when the contrast is injected. Note that it is always anterior to the foramina. After the correct needle placement is confirmed, 8–10 mL of a local anesthetic-steroid mixture (e.g., 0.25 % bupivacaine or 0.2 % ropivacaine) is injected into the psoas muscle on one side. Pain relief should occur approximately 30 min after injection of the local anesthetic. On examination, pain should be gone on flexion and extension of the hip [42].

The distal psoas injection can provide significant relief of both the distal tendonitis that occurs with chronic psoas spasm and the proximal spasm perpetuated by the distal pathology. The lesser trochanter is easily identified fluoroscopically (Fig. 66.13), and a 22-gauge 3 inch needle can be advanced in a "down the barrel" technique onto the bone itself (Fig. 66.24), where 1 cc of local anesthetic and deposteroid can be injected. With a gentle technique, this should not be a painful procedure, though caution must be used to avoid the femoral nerve and artery (which should be more medial). Distal psoas pathology may be a cause of persistent pain after hip replacement (Fig. 66.25), and this injection may give rapid and significant relief [43].

Inguinal Perivascular Injection or Compartment Block (Three-in-One Block)

The *inguinal perivascular block* is based on the concept of injecting local anesthetic near the femoral nerve in an amount sufficient to track proximally along fascial planes to anesthe-

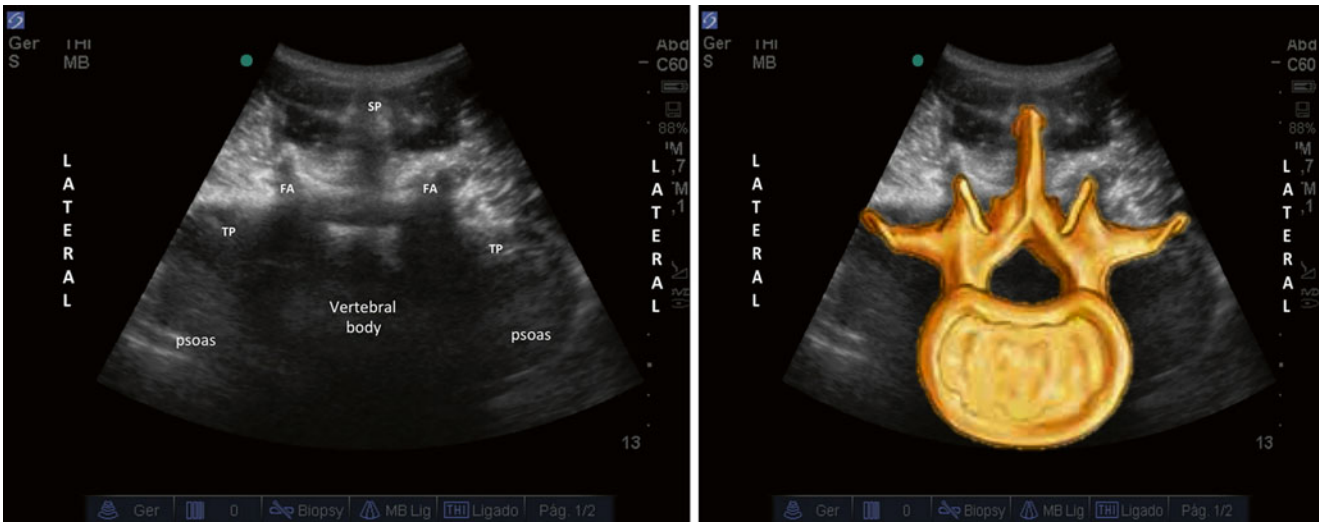

Fig. 66.19 Simulation of the ultrasound anatomy of the horizontal approach to the psoas muscle and lumbar plexus. *SP* spinous process, *FA* facet articular process, *TP* transverse process (Image courtesy of Thiago Nouer Frederico, MD)

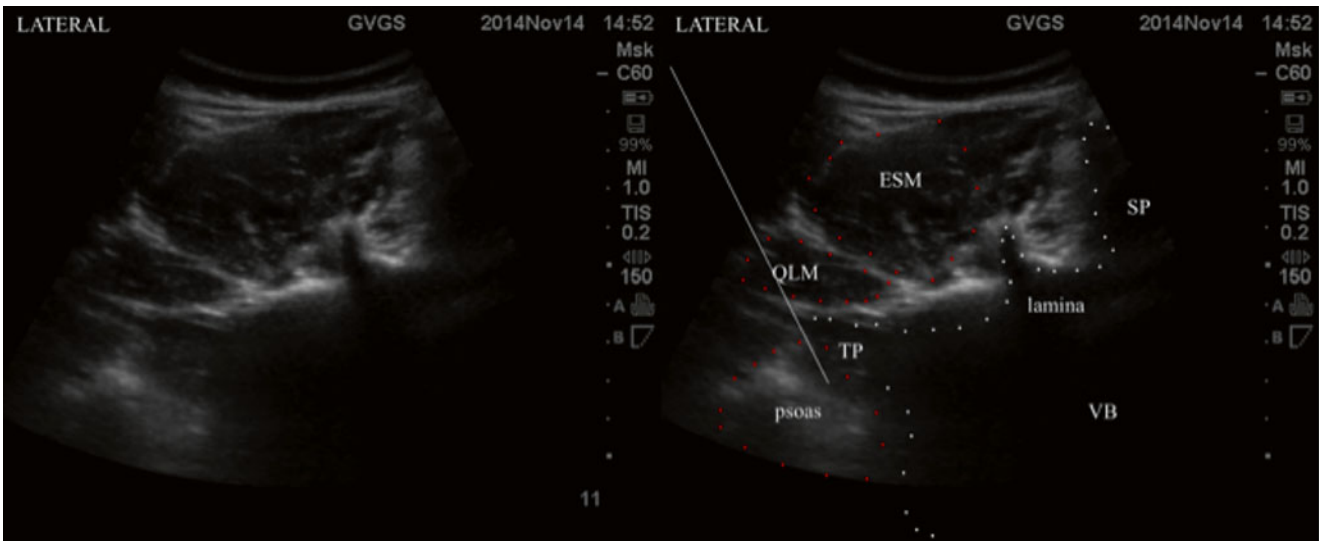

Fig. 66.20 Horizontal lateral view of the lumbar plexus (**a**), with labels and simulated needle (**b**). *SP* spinous process, *VB* vertebral body, *TP* transverse process, *ESM* erector spinous muscle, *QLM* quadratus lumborum muscle (Image courtesy of Agnes Stogicza, MD)

tize the lumbar plexus [44]. The three principal nerves of the lumbar plexus pass from the pelvis anteriorly: the lateral femoral cutaneous, the femoral, and the obturator nerves. The theory behind this block presumes that the local anesthetic will track in the fascial plane between the iliacus and psoas muscles to reach the region of the lumbar plexus roots, so that the only anatomy one needs to visualize is the extension of sheath-like fascial planes that surround the femoral nerve (Fig. 66.26). The patient should be placed supine on the operating table with the anesthesiologist standing at the patient's side in position to palpate the ipsilateral femoral artery. After local anesthetic infiltration, a short-beveled, 22-gauge 5 cm needle is inserted immediately lateral to the femoral artery, caudad to the inguinal ligament, and advanced with cephalic angulation until a femoral paresthesia is obtained. At this point, the needle is firmly fixed, and, while the distal femoral sheath is digitally compressed, the entire volume of local anesthetic is injected in divided doses.

Neurolytic Technique

Cryoneuroablation

Because the plexus is a large structure, it is not amenable to a precise technique like cryoneuroablation.

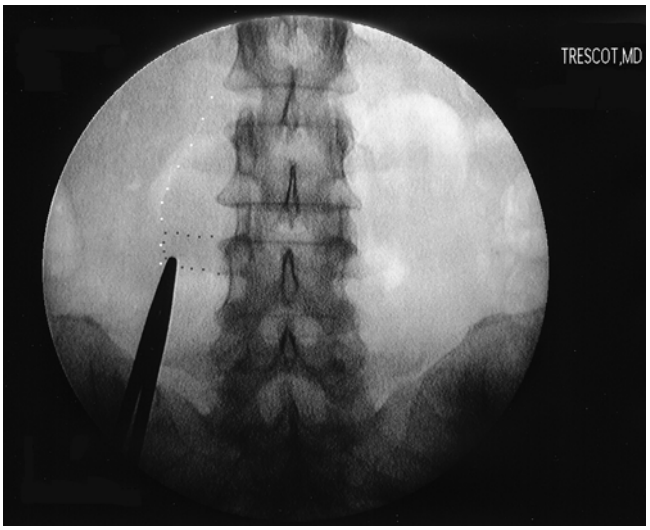

Fig. 66.21 Fluoroscopic image of the lumbar spine. Marker is on the transverse process of L4; note the distortion of the psoas shadow due to cancer mass (Image courtesy of Andrea Trescot, MD)

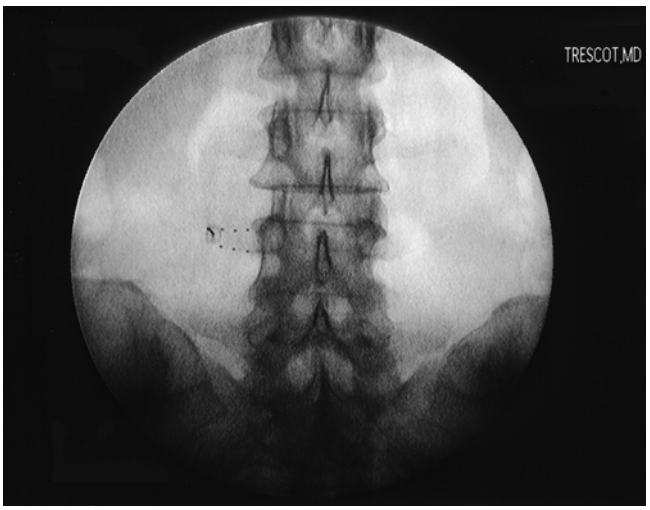

Fig. 66.22 Fluoroscopic psoas injection, needle on the transverse process of L4 (Image courtesy of Andrea Trescot, MD)

Radiofrequency Lesioning (RF)

There is no literature on the technique, but the same issues as above are involved. The lumbar plexus is not amenable to a precise technique like radiofrequency.

Alcohol/Phenol

Calava et al. [45] described the injection of neurolytic substances (6 % aqueous phenol or absolute alcohol) into the psoas sheath to provide longer-term relief for a *malignant psoas syndrome* (metastatic lipoma) for which a prior psoas compartment anesthetic block provided only temporary relief. Depending on life expectancy, a lumbar plexus catheter placement could be considered [46].

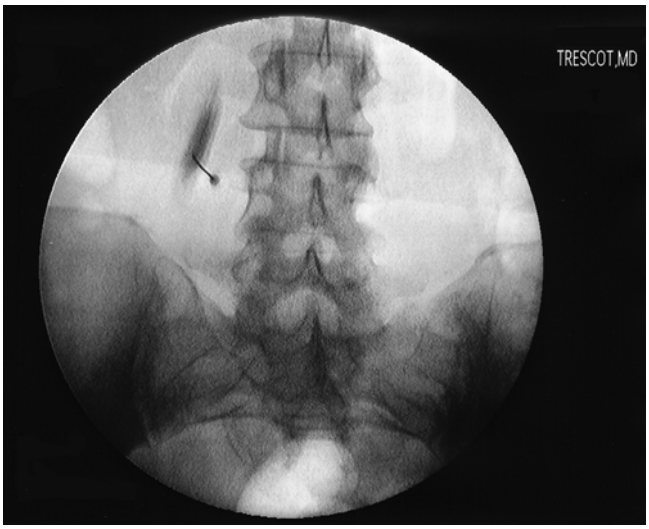

Fig. 66.23 Fluoroscopic psoas injection, contrast pattern (Image courtesy of Andrea Trescot, MD)

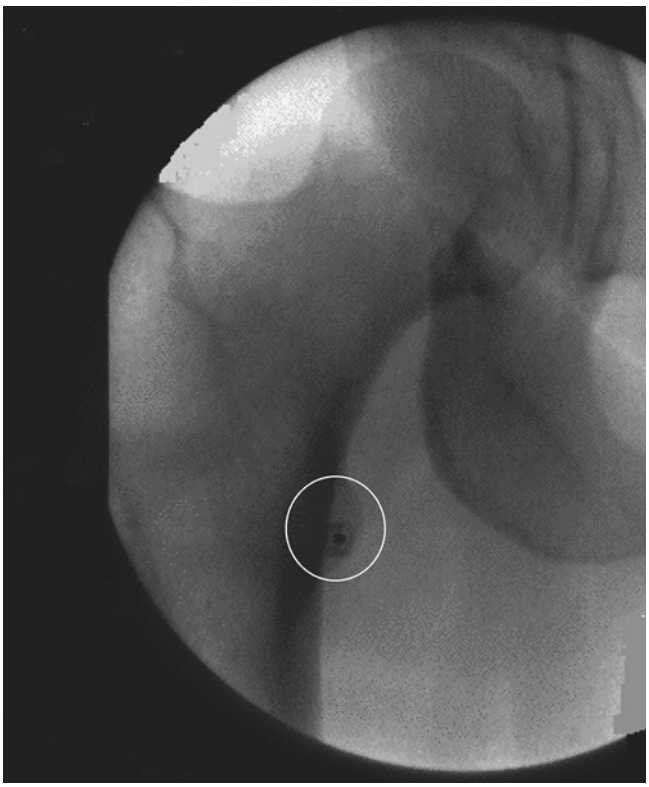

Fig. 66.24 Fluoroscopic needle placement for distal psoas injection on the lesser trochanter. Note the *white circle* at the injection site (Image courtesy of Andrea Trescot, MD)

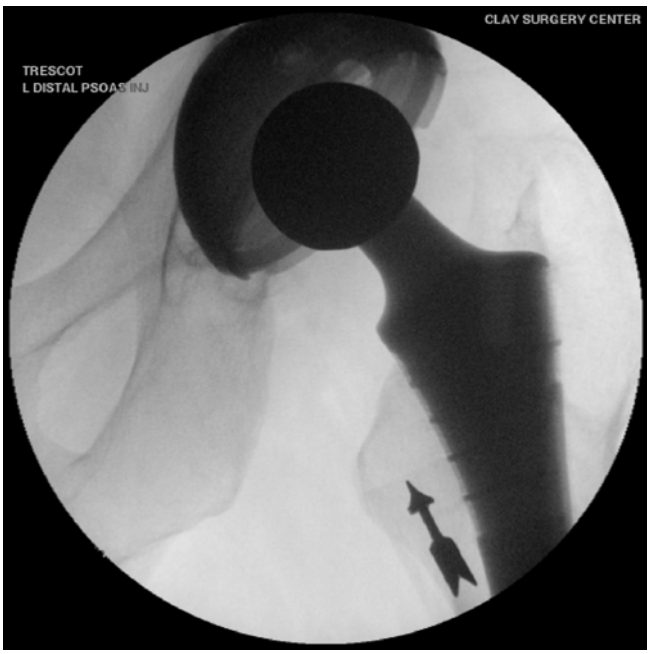

Fig. 66.25 Fluoroscopic image of the lesser trochanter after a total hip replacement. The *arrow* shows the site of injection (Image courtesy of Andrea Trescot, MD)

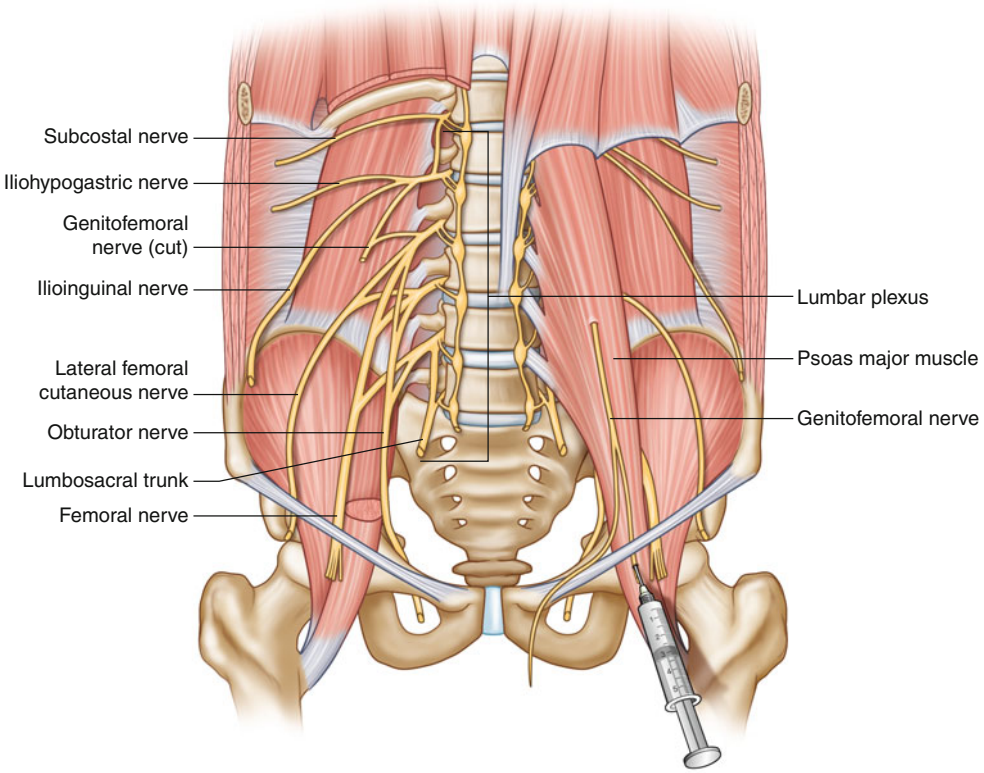

Fig. 66.26 Needle location for inguinal perivascular injection (Image courtesy of Springer)

Surgical Technique

Surgical dorsal rhizotomy may provide pain relief from lumbar plexopathy, especially in tumor patients when other conservative measures failed [15]. Surgical nerve repair and nerve grafting may lead to partial recovery of plexopathy from trauma or fractures [11].

Complications

The most serious complications associated with lumbar plexus block are related to the close proximity to the spinal cord and exiting nerve roots. The needle can cause trauma to the exiting lumbar nerve roots. If the needle is directed too dorsally and medially, there may be inadvertent subarachnoid,

subdural, and/or epidural injection. While inadvertent dural puncture is rare, unintentional dural or subdural injection can result in immediate total spinal anesthesia with associated loss of consciousness, hypotension, and apnea. This must be recognized immediately. Intravascular injections can lead to local anesthetic toxicity with loss of consciousness, hypotension, apnea, or cardiac arrest. This risk can be decreased using digital subtraction (fluoroscopy) or color mode for Doppler (ultrasound).

Summary

Lumbosacral plexus entrapments can present in a myriad of ways, including lower extremity pain, which makes diagnosis difficult if there is not a high index of suspicion. A careful history and physical exam along with a diagnostic injection can help to elucidate the cause.

References

1. Yeo PT, Grice AC. Lumbosacral nerve plexus compression by ovarian-fallopian cysts. Clin Exp Neurol. 1986;22:139–43.
2. Batchelor TJ, Vowden P. A persistent sciatic artery aneurysm with an associated internal iliac artery aneurysm. Eur J Vasc Endovasc Surg: Off J Eur Soc Vasc Surg. 2000;20(4):400–2.
3. Vinnicombe SJ, Norman AR, Nicolson V, Husband JE. Normal pelvic lymph nodes: evaluation with CT after bipedal lymphangiography. Radiology. 1995;194(2):349–55.
4. Jaeckle KA, Young DF, Foley KM. The natural history of lumbosacral plexopathy in cancer. Neurology. 1985;35(1):8–15.
5. Ozkan OF, Guner A, Cekic AB, Turan T, Kaya U, Reis E. Iliopsoas haematoma: a rare complication of warfarin therapy. J Coll Physicians Surg – Pak: JCPSP. 2012;22(10):673–4.
6. Ozcakar L, Sivri A, Aydinli M, Tavil Y. Lumbosacral plexopathy as the harbinger of a silent retroperitoneal hematoma. South Med J. 2003;96(1):109–10.
7. Paley M, Sidhu PS, Evans RA, Karani JB. Retroperitoneal collections – aetiology and radiological implications. Clin Radiol. 1997;52(4):290–4.
8. Jaeckle KA. Nerve plexus metastases. Neurol Clin. 1991;9(4):857–66.
9. Taylor BV, Kimmel DW, Krecke KN, Cascino TL. Magnetic resonance imaging in cancer-related lumbosacral plexopathy. Mayo Clin Proc. 1997;72(9):823–9.
10. Gibbons KJ, Soloniuk DS, Razack N. Neurological injury and patterns of sacral fractures. J Neurosurg. 1990;72(6):889–93.
11. Kutsy RL, Robinson LR, Routt Jr ML. Lumbosacral plexopathy in pelvic trauma. Muscle Nerve. 2000;23(11):1757–60.
12. Magu NK, Singla R, Gogna P, Amanpreet, Jain N, Aggarwal S. Lumbar plexus injury in an anterior fracture dislocation of sacroiliac joint: a case report and review of literature. Strateg Trauma Limb Reconstr. 2013;8(3):181–5.
13. Garozzo D, Zollino G, Ferraresi S. In lumbosacral plexus injuries can we identify indicators that predict spontaneous recovery or the need for surgical treatment? Results from a clinical study on 72 patients. J Brachial Plex Peripher Nerve Inj. 2014;9(1):1.
14. Irvin W, Andersen W, Taylor P, Rice L. Minimizing the risk of neurologic injury in gynecologic surgery. Obstet Gynecol. 2004;103(2):374–82.
15. Lang EM, Borges J, Carlstedt T. Surgical treatment of lumbosacral plexus injuries. J Neurosurg Spine. 2004;1(1):64–71.
16. Chevalier X, Larget-Piet B. Femoral neuropathy due to psoas hematoma revisited. Report of three cases with serious outcomes. Spine (Phila Pa 1976). 1992;17(6):724–6.
17. Hughes MS, Burd TA, Allen WC. Post-traumatic catamenial sciatica. Orthopedics. 2008;31(4):400.
18. Cottier JP, Descamps P, Sonier CB, Rosset P. Sciatic endometriosis: MR evaluation. AJNR Am J Neuroradiol. 1995;16(7):1399–401.
19. Feasby TE, Burton SR, Hahn AF. Obstetrical lumbosacral plexus injury. Muscle Nerve. 1992;15(8):937–40.
20. Katirji B, Wilbourn AJ, Scarberry SL, Preston DC. Intrapartum maternal lumbosacral plexopathy. Muscle Nerve. 2002;26(3):340–7.
21. Soldatos T, Andreisek G, Thawait GK, Guggenberger R, Williams EH, Carrino JA, et al. High-resolution 3-T MR neurography of the lumbosacral plexus. Radiographics. 2013;33(4):967–87.
22. Whiteside JL, Barber MD, Walters MD, Falcone T. Anatomy of ilioinguinal and iliohypogastric nerves in relation to trocar placement and low transverse incisions. Am J Obstet Gynecol. 2003;189(6):1574–8.
23. Petchprapa CN, Rosenberg ZS, Sconfienza LM, Cavalcanti CF, Vieira RL, Zember JS. MR imaging of entrapment neuropathies of the lower extremity. Part 1. The pelvis and hip. Radiographics. 2010;30(4):983–1000.
24. Hacker HS, King JC. Plexopathy: lumbosacral. In: Frontera WR, Silver SK, Rizzo Jr TD, editors. Essentials of physical medicine and rehabilitation: musculoskeletal disorders, pain, and rehabilitation. Philadelphia: Sanders Elsevier; 2008. p. 779–86.
25. Tonsgard JH, Kwak SM, Short MP, Dachman AH. CT imaging in adults with neurofibromatosis-1: frequent asymptomatic plexiform lesions. Neurology. 1998;50(6):1755–60.
26. Griffiths EA, Kalbassi MR, Cunliffe WJ, Browell DA. Psoas abscess as a secondary complication of gastrointestinal disease: report of five cases. Case Rep Clin Pract Rev. 2003;4(2):73–6.
27. Whitaker R, Borley N. Chapter 6. Instant anatomy. 1st ed. Oxford: Blackwell Scientific; 1994. p. 114–23.
28. Planner AC, Donaghy M, Moore NR. Causes of lumbosacral plexopathy. Clin Radiol. 2006;61(12):987–95.
29. Thomas JE, Cascino TL, Earle JD. Differential diagnosis between radiation and tumor plexopathy of the pelvis. Neurology. 1985;35(1):1–7.
30. Donaghy M. Lumbosacral plexus lesions. In: Dyk PJ, Thomas PK, editors. Peripheral neuropathy, vol. 2. 4th ed. Philadelphia: Elsevier Saunders; 2005. p. 1375–90.
31. Laughlin RS, Dyck PJ. Electrodiagnostic testing in lumbosacral plexopathies. Phys Med Rehabil Clin N Am. 2013;24(1):93–105.
32. Boon AJ, Gertken JT, Watson JC, Laughlin RS, Strommen JA, Mauermann ML, et al. Hematoma risk after needle electromyography. Muscle Nerve. 2012;45(1):9–12.
33. Hammersted JP. Plexopathy syndromes. In: Goetz CG, editor. Textbook of clinical neurology. 2nd ed. Philadelphia: Saunders; 2003. p. 271–2.
34. Rancont CM. Chronic psoas syndrome caused by the inappropriate use of a heel lift. J Am Osteopath Assoc. 2007;107(9):415–8.
35. Waldman SD. Lumbar plexus block: psoas compartment technique. In: Waldman SD, editor. Atlas of interventional pain management. 2nd ed. Philadelphia: Saunders; 2004. p. 443–8.
36. Waldman SD. Ultrasound-guided lumbar plexus nerve block. In: Waldman SD, editor. Comprehensive atlas of ultrasound-guided pain management injection techniques. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2014. p. 719–26.
37. David L. Brown. Lumbar plexus block. In: L. BD, editor. Atlas of regional anesthesia. Philadelphia: W. B. Saunders; 1992. p. 76–7.
38. Capdevila X, Macaire P, Dadure C, Choquet O, Biboulet P, Ryckwaert Y, et al. Continuous psoas compartment block for

postoperative analgesia after total hip arthroplasty: new landmarks, technical guidelines, and clinical evaluation. Anesth Analg. 2002;94(6):1606–13, table of contents.
39. Hadzic A, Vioka JD. Lumbar plexus block. In: Hadzic A, editor. Hadzic's peripheral nerve blocks and anatomy for ultrasound-guided regional anesthesia. 2nd ed. New York: McGraw-Hill; 2012. p. 217–28.
40. Enneking FK, Chan V, Greger J, Hadzic A, Lang SA, Horlocker TT. Lower-extremity peripheral nerve blockade: essentials of our current understanding. Reg Anesth Pain Med. 2005;30(1):4–35.
41. Mulchandani H, Awad I, McCaetney C. Ultrasound-guided nerve blocks of the lower limb. In: Narouze SM, editor. Atlas of ultrasound-guided procedures in interventional pain management. New York: Springer; 2010. p. 249–50.
42. Paradise LA, Raj PP, Fryer JR. Lumbar myofascial blocks. In: Raj PP, Lou L, Erdine S, Staats PS, Waldman SD, Racz GB, et al., editors. Interventional pain management image-guided procedures. 2nd ed. Philadelphia: Saunders; 2008. p. 382–90.
43. Nunley RM, Wilson JM, Gilula L, Clohisy JC, Barrack RL, Maloney WJ. Iliopsoas bursa injections can be beneficial for pain after total hip arthroplasty. Clin Orthop Relat Res. 2010;468(2):519–26.
44. Lang SA, Yip RW, Chang PC, Gerard MA. The femoral 3-in-1 block revisited. J Clin Anesth. 1993;5(4):292–6.
45. Calava JM, Patt RB, Reddy S, Varma DG, Chiang J. Psoas sheath chemical neurolysis for management of intractable leg pain from metastatic liposarcoma. Clin J Pain. 1996;12(1):69–75.
46. Douglas I, Bush D. The use of patient-controlled boluses of local anaesthetic via a psoas sheath catheter in the management of malignant pain. Pain. 1999;82(1):105–7.

Common Peroneal Nerve Entrapment

67

Natalia Murinova, Sheila C. Chiu, Daniel Krashin, and Helen W. Karl

Introduction

Entrapment of the common peroneal nerve (CPN) is one cause of CPN dysfunction (one of the most common focal neuropathies of the lower extremities), although CPN injuries are usually due to trauma [1, 2]. In one report of patients seen for paresis of the foot dorsiflexors (foot drop), CPN lesions accounted for 31 % of those originating in the peripheral nervous system; of these, 76 % were the result of trauma [3]. They are particularly common in younger men, probably because younger men are injured more frequently [3, 4], while older women have more adipose tissue, a significant source of protection for the CPN near the knee [5]. In one series of operative decompressions of the CPN, 92 % of the patients presented with weakness, 92 % had a sensory disturbance, and 84 % had pain, all of which are sources of substantial disability [6].

In 1998, the peroneal nerve was renamed the *fibular nerve*, to avoid confusion with the perineal pelvic region; but the name change has not been commonly accepted, and both terms are used [7].

Clinical Presentation (Table 67.1)

Patients with CPN compression usually present with foot drop, a significant disability, which causes a "slapping" gait, toe dragging, problems in walking and climbing stairs, and

Table 67.1 Occupation/exercise/trauma history relevant to common peroneal nerve entrapment

Trauma: accidents and surgery	Forced foot inversion (ankle sprain) [8]
	Activities that tense the peroneus longus (PL) muscle [6]
	Fibula fracture
	Knee dislocation [9]
	Total knee arthroplasty
	Arthroscopic meniscus surgery [10]
Postural compression	Prolonged squatting [1, 3, 4, 11]
	Crossing legs [1, 3, 4]
	Surgical positioning, especially lithotomy
	"Strawberry picker's" palsy [2]
	Particularly common among Asian populations [11]
	Running and bicycling [12], yoga [13, 14]
	Exercise involving inversion and pronation [15, 16]
Extrinsic compression	Intraneural ganglion from the proximal tibial-fibular joint into the articular branch [5, 17]
	Popliteal venous aneurysm [18]
	Tight short leg cast, pneumatic compression device, or tall boot
Other	Weight loss [1, 19, 20]

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frequent falling. They also often have decreased sensation, tingling, numbness, or burning on the lateral lower leg (Figs. 67.1 and 67.2), down to the top of the foot [11] (Fig. 67.3). When pain is present, it typically worsens (and may radiate proximally) with physical activities, such as walking, jogging, running, or squatting. As with many peripheral nerve entrapments, symptoms differ depending on the origin and extent of the problem (Fig. 67.4).

The most common cause of CPN injury is trauma [3, 4]. Open and blunt force injuries of the lateral knee and open or arthroscopic knee surgery can compromise CPN integrity [21]. There is an approximately 1 % incidence of CPN injury after *tibial plateau fractures* [22]. Sedel and Nizard [23] described 17 cases of traction injuries to the CPN; the initial injury for all the cases was a severe *varus deformity of the knee*. Foot drop may also be the result of a "straightforward acute *inversion sprain* of the ankle" [8, 15]. In a series of 66 patients with ankle sprains, 86 % of the patients with grade III sprains and 17 % of the patients with grade II sprains had evidence of CPN injury on needle EMG (see section "Diagnostic tests" below). Traction on the *peroneus longus muscle* (PL) likely stretches the CPN and compresses it at the fibular neck; hematoma from the injury may aggravate the situation. Night calf cramps are common [24].

Prolonged extrinsic pressure is another significant cause of CPN neuropathy. This can be discovered postoperatively in patients who had been in the lateral decubitus or lithotomy position during surgery. The CPN may be susceptible to damage in patients who have lost a significant amount of

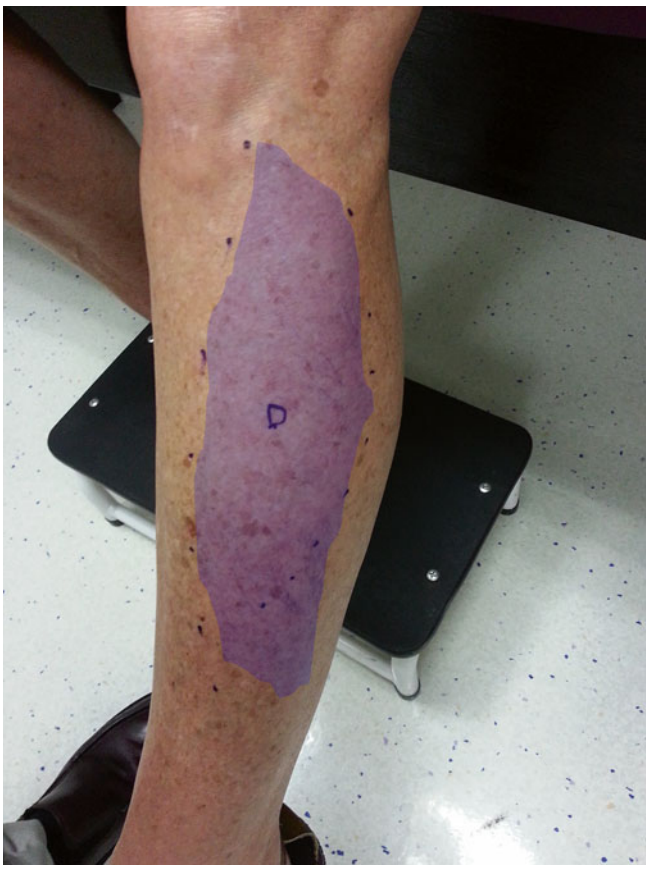

Fig. 67.2 Pattern of pain in a patient with presumed common peroneal entrapment (Image courtesy of Eric Wilson, MD)

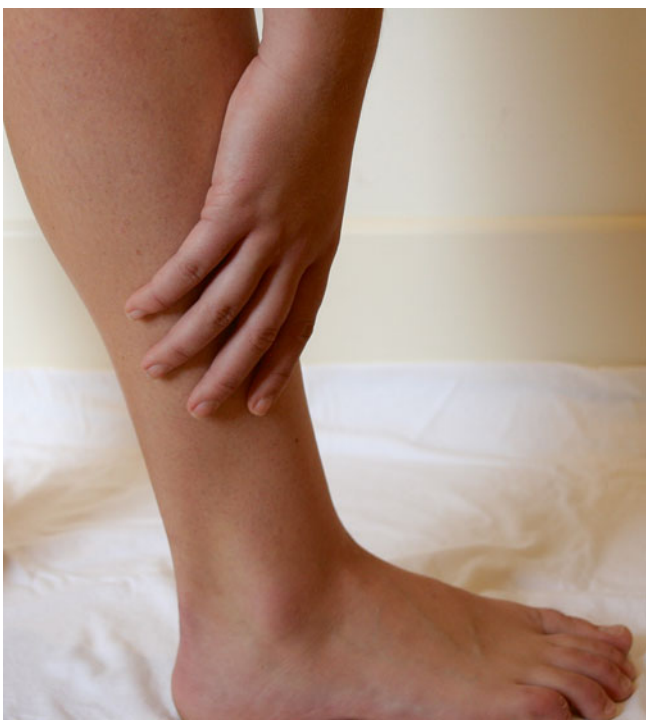

Fig. 67.1 The proximal pattern of pain from common peroneal entrapment (Image courtesy of Andrea Trescot, MD)

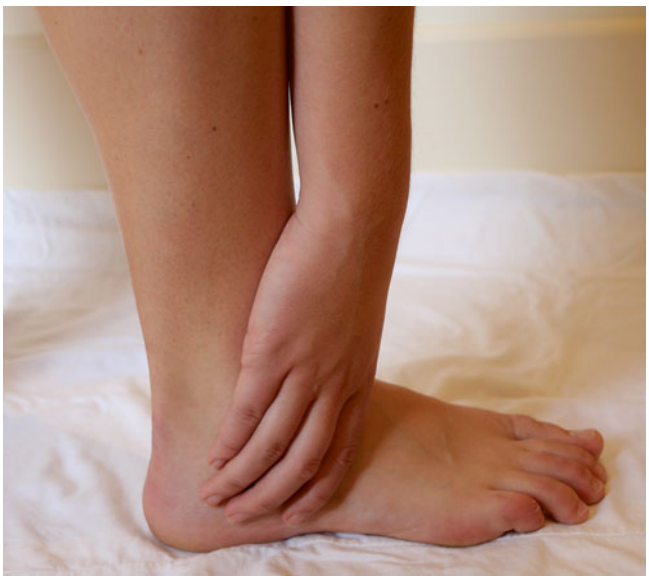

Fig. 67.3 The distal pattern of pain from common peroneal entrapment (Image courtesy of Andrea Trescot, MD)

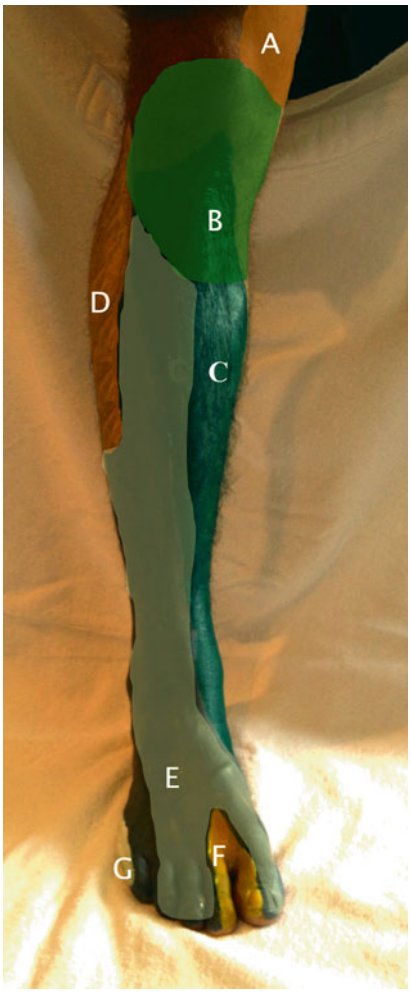

Fig. 67.4 The pattern of pain from lower extremity nerve entrapments. *A* proximal saphenous nerve, *B* infrapatellar saphenous nerve, *C* distal saphenous nerve, *D* lateral sural cutaneous nerve, *E* common peroneal nerve, *F* deep peroneal nerve, *G* sural nerve (Image courtesy of Terri Dallas-Prunskis, MD)

weight, particularly if they are confined to bed where the natural position of the leg is in external rotation with knees flexed [22]. Twenty percent of 150 cases of peroneal neuropathy were associated with weight loss and dieting [20]. CPN palsies were noted in World War II prisoners of war who lost 5–11 kg [20].

Sitting for prolonged periods with legs crossed, prolonged squatting ("yoga foot drop") [14], and pressure to the lateral knee during deep sleep all have been reported to cause CPN injury [11]. One study identified common peroneal injury after maintaining the same posture for an average of 124 min [11]. When there is evidence of CPN compression, medical conditions such as diabetes mellitus or toxin exposure that increase the vulnerability of the nerve should be considered. CPN is the most common low limb mononeuropathy in athletes, most likely due to compression by the *peroneal longus muscle* (PL) (also known as the *fibularis longus muscle*) [20].

Space-occupying lesions such as intraneural ganglion cysts [25], vascular aneurysms [18], or lipomas [26] can also compress the CPN.

Anatomy (Table 67.2)

The CPN is the smallest of the two main branches of the *sciatic nerve* (see Chap. 65), about half the size of the other branch, the *tibial nerve* (see Chap. 73) (Fig. 67.5). It provides motor and sensory function to the lower leg, dorsum of the foot, and toes. In or near the popliteal fossa, the CPN gives off three branches: the nerve to the short head of the *biceps femoris muscle* (BF); the *lateral sural cutaneous nerve* (*lateral cutaneous nerve of the calf*), which supplies sensation to the upper lateral calf (see Chap. 72); and the *sural communicating branch*, which travels posteriorly to join the *medial sural cutaneous nerve* to form the *sural nerve* (see Chap. 71) (Fig. 67.6). The CPN follows the medial edge of the BF to the lateral popliteal fossa to the fibular head (Fig. 67.7), where it changes its downward course to wind laterally around the neck of the fibula between the two heads of the PL, dividing into its terminal branches. The CPN gives off some muscular branches, and then all of the branches cross the intermuscular septum from the lateral to the anterior compartment.

Most commonly, the very proximal muscular branch (to the *tibialis anterior muscle*) pierces the septum directly, while the main nerve and remaining muscular branches traverse an osteofibrous hiatus, opening between the septum and the fibula [28]. It continues into the foot as the *superficial peroneal nerve* (SPN) (see Chap. 68) and the *deep peroneal nerve* (*DPN*) (see Chap. 69). CPN dysfunction results in weakness of foot dorsiflexion and eversion, as well as sensory changes in the lateral aspect of the leg and dorsum of the foot and toes.

There is also a variant called the *accessory peroneal nerve* (*accessory fibular nerve*) that branches from the superficial peroneal nerve (superficial fibular nerve) (see Chap. 68) underneath the *peroneus brevis muscle* (*fibularis brevis muscle*), traveling to the foot, posterior to the lateral malleolus [20]. There have also been descriptions of the CPN separating from tibial nerve proximal to the piriformis and passing between the heads of the piriformis muscles, with the tibial nerve passing inferiorly [29].

Entrapments

The CPN is most commonly entrapped at the *peroneal tunnel* (*fibular tunnel*), where the nerve winds around the fibular neck (Fig. 67.6 Site 1) [27]. The entrance to this tunnel was first described in 1973 as a "fibrous arch located on the lat-

Table 67.2 Common peroneal nerve anatomy

Origin	L4–S2
General route	The *sciatic nerve* travels behind the hip joint and then divides deep in the mid-thigh into the CPN and *tibial nerve*. The CPN lies on the proximal *gastrocnemius muscle* (G) and follows the medial border of the *biceps femoris* (BF) along the lateral edge of the *popliteal fossa*. It enters the fibro-osseous peroneal tunnel near the head of the fibula and goes between the two heads of the *peroneus longus muscle* (PL), while curling from posterior to anterior around the fibular neck. It then divides into the *deep peroneal nerve* (DPN) and *superficial peroneal nerve* (SPN)
Sensory distribution	*Lateral sural cutaneous*: lateral calf
	Sural communicating branch: posterior calf
	Anterior recurrent branch: knee joint
	From the SPN: *medial and intermediate dorsal cutaneous nerves*, top and lateral edge of the foot
	From the DPN: *lateral cutaneous nerve of the great toe* and *medial cutaneous nerve of the second toe*, between the first and second toes
Motor innervation	Short head of the BF
	From the SPN: lateral compartment muscles, including peroneus longus and brevis which evert the foot
	From the DPN: anterior compartment muscles, including tibialis anterior and toe extensors which dorsiflex the ankle
Anatomic variability	Site of division of the sciatic nerve and relationship to the piriformis muscle: some divide in the pelvis and may go through the piriformis [27]
	Relationship to the distal BF: in 77 %, the CPN was superficial to G and posterior to the short head of the BF; in 23 %, the nerve was in a fat-filled tunnel between G and the short head of the BF [5]
	Site of division into deep and superficial branches: 81 % distal to the fibular neck, 10 % proximal to the joint, 9 % between the joint and the fibular neck [10]
	Pattern of branching at the joint line: 1–5 branches [10]
	Presence of a cutaneous branch to the lateral knee: 30 % [10]
	Accessory peroneal (*fibular*) *nerve*: common variant (17–28 % of anatomic studies and 12–22 % of electrophysiologic studies) that generally arises from the superficial peroneal (fibular) branch under the PL and travels to the foot to innervate the extensor digitorum brevis [20]
Other relevant structures	*Peroneus longus muscle* (PL): activities that tense this muscle (ankle inversion, plantar flexion) compress the CPN in the *peroneal/fibular tunnel* [15]
	Peroneal tunnel: the tough proximal edge is formed by the combined aponeurosis of the *soleus* and PL muscles; its floor is the fibula, and it is generally considered to end where the DPN goes through the anterior intermuscular septum, about 3 cm distal to the CPN bifurcation [27]

eral border of the fibula about 1 to 2 cm inferior to its head…," consisting of fibers from combined aponeuroses of the soleus and PL muscles [30]. At this location, the CPN lies on the bone, protected only by the fascia and skin, and thus is vulnerable to even modest external compression. The peroneal tunnel is considered to have both superficial and deep parts [16, 27], both of which must be released for successful neurolysis [6]. More recent detailed dissections have raised questions about the functional anatomy of this area [31]; further work is needed to resolve the differences.

The main trunk of the CPN may also become trapped in a tunnel between the gastrocnemius and biceps femoris muscles (Fig. 67.6 site 2) [5]. Also, if there is a high division of the sciatic nerve and its peroneal division pierces the piriformis muscle, the CPN can be trapped there, especially if the patient has piriformis hypertrophy or scarring [27].

In 1972, Haimovici [13] described a series of 48 patients (60 limbs) with "exquisite" tenderness along the lateral aspect of the popliteal space, radiating down the lateral calf, which he attributed to the entrapment of the CPN branches (*lateral sural nerve* and *sural communicating branch*) as they pass through fascial openings. Although the pain was in a CPN pattern, there is no motor weakness.

There is also a potential entrapment of the CPN by an occasionally occurring *accessory sesamoid bone* (called a *fabella*) [32] near the attachment of the lateral gastrocnemius muscle, which is found in 8.5 % of the population [33]. On physical examination, there may be discrete tenderness in the lateral popliteal fossa, often accompanied by a 1 cm tender nodule.

An under-recognized branch of the CPN is the *recurrent auricular branch* (also known as the *anterior recurrent*

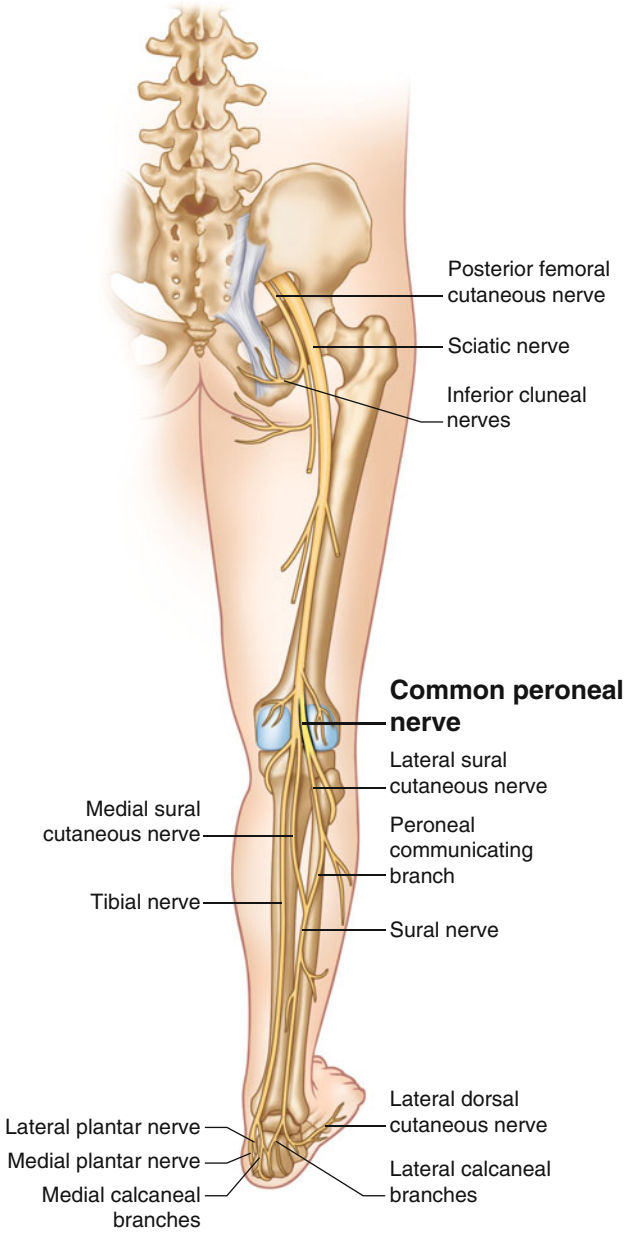

Fig. 67.5 Posterior lower extremity anatomy (Image by Springer)

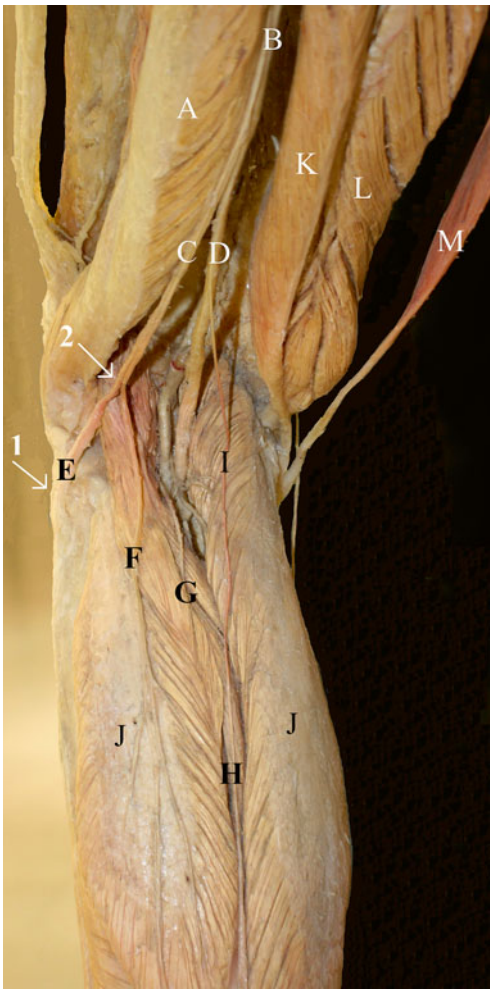

Fig. 67.6 Dissection of the posterior thigh and calf (Modified from an image from *Bodies, The Exhibition*, with permission). *A* biceps femoris muscle, *B* sciatic nerve, *C* peroneal division, *D* tibial division, *E* common peroneal nerve, *F* lateral sural cutaneous nerve, *G* sural communicating branch, *H* sural nerve, *I* medial communicating branch, *J* gastrocnemius muscle, *K* semitendinosus muscle, *L* semimembranosus muscle, *M* gracilis muscle, (*1*) fibular tunnel, (*2*) entrapment site between the biceps femoris and gastrocnemius muscles (Image courtesy of Andrea Trescot, MD)

peroneal nerve), which exits the fibular tunnel with the CPN but travels cephalad to the lateral patella [34]. This entrapment causes pain below the patella, which may be misdiagnosed as *patellar tendinopathy* (Fig. 67.7). The presence of pain localized to the lateral border of the proximal patellar tendon and the presence of increased peroneus muscle tone may help to differentiate these conditions [34].

The cutaneous branches of the CPN (the *lateral sural cutaneous* and *sural communicating nerves*) (Fig. 67.6) may become entrapped in the popliteal fossa [13]. These branches cross the popliteal fossa and become subcutaneous behind the knee joint. Patients with cutaneous branch entrapment describe an acute onset of a sensation of heaviness or pain behind the knee or on the lateral leg after prolonged sitting. In contrast to patients with CPN entrapment, they had no motor symptoms and few sensory changes. Fibrous bands constricting the CPN near the fibular head and proximal PL have been reported at operation in patients with CPN palsy [6, 35] but not in normal cadavers [27].

It is also important for the clinician to remember the potential for a "double crush" phenomenon (see Chap. 1). Ang and Foo [36] described a patient with leg pain and paresthesias who underwent spinal surgery for lateral spinal stenosis and yet had persistent leg pain postoperatively. The patient was subsequently found to have peroneal muscle

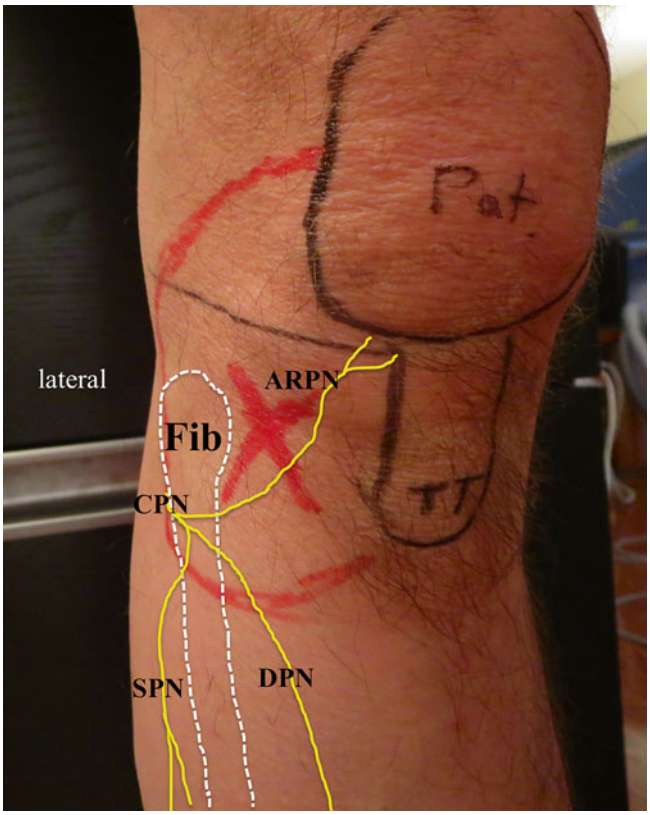

Fig. 67.7 Pain pattern of a patient with presumed anterior recurrent peroneal nerve entrapment. *Pat* patella, *TT* tibial tubercle, *Fib* fibula, *CPN* common peroneal nerve, *SPN* superficial peroneal nerve, *DPN* deep peroneal nerve, *ARPN* anterior recurrent peroneal nerve, *X* site of tenderness (Image courtesy of Peter Mouldey, MD; modified by Andrea Trescot, MD)

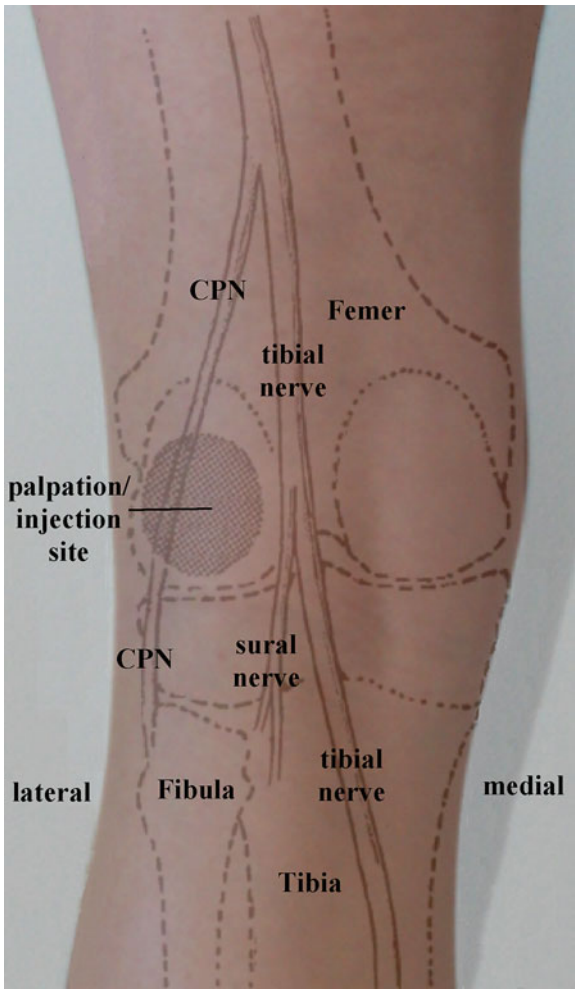

Fig. 67.8 Posterior knee, location of proximal tenderness of the common peroneal nerve. Image modified from Haimovici [13]. (Image courtesy of Andrea Trescot, MD)

herniations at two separate locations that were entrapping the CPN. That case report went on to encourage clinicians to consider distal entrapments as an additional or potentially primary diagnosis.

Physical Exam

The physical examination should begin with a general evaluation of the leg, looking for signs of trauma, surgery, or vascular insufficiency. The strength of the leg muscles and sensory examination should be compared to the unaffected side. With CPN dysfunction, the weakness of ankle dorsiflexion and foot eversion is likely ("foot drop") and is considered the hallmark finding (see http://www.youtube.com/watch?v=J7-L9MFRXD8 for a video of the gait disturbance – with permission). Patients may complain of more subtle tripping or catching their toe during ambulation [20]. Sidey described 23 patients with CPN entrapment confirmed at surgery; 18 patients either described or were found to have ankle weakness [37]. Sensory disturbances of the skin over the lateral distal lower leg and dorsum of the foot are also common but may be absent [20]. Sensation on the sole of the foot should be normal. Provocative maneuvers include *Tinel's sign* with palpation over the lateral popliteal fossa (Fig. 67.8) and superior fibula, as well as reproduction of the pain with palpation along the fibular tunnel (Video 67.1) (Fig. 67.9). These findings are potentially increased with the foot in plantar flexion and inversion, which are positions that stretch the CPN. Pain or paresthesias with either of these tests indicate probable CPN compression and the need for further investigation. There can also be a slightly more proximal site of tenderness posteriorly at the lateral edge of the popliteal fossa at the level of the knee joint line and at the level of the takeoff of the *lateral sural cutaneous nerve* and the *sural communicating nerve* [13] (Fig. 67.6). Dorsiflexion weakness (but not usually pain) may also be the presenting symptom of conditions such as amyotrophic

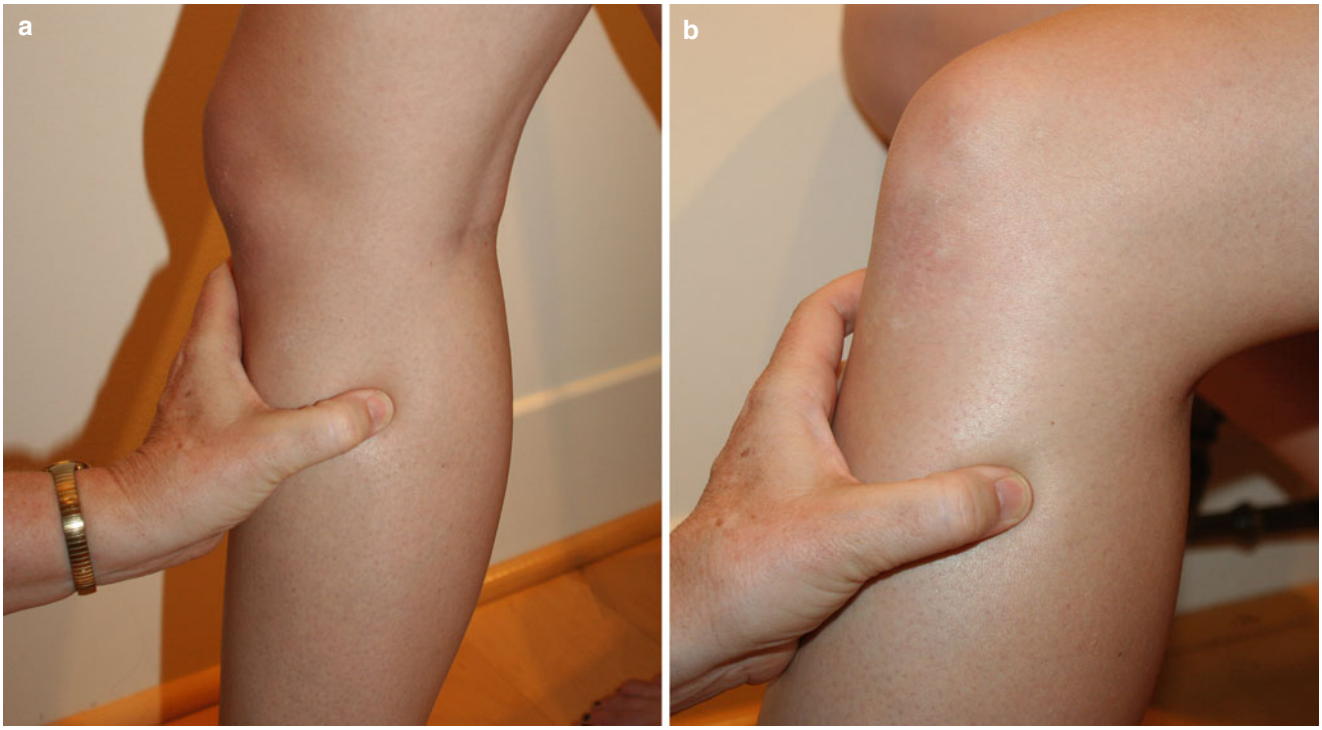

Fig. 67.9 Physical exam of the common peroneal nerve, standing (**a**) or sitting (**b**) (Image courtesy of Andrea Trescot, MD)

lateral sclerosis [19]. Signs and symptoms of CRPS may be present [38].

For the *anterior recurrent peroneal nerve* (ARPN), the physical exam will show no sensory or motor loss; resisted eversion and plantar flexion are sometimes painful, and *superior tibiofibular subluxation* or *inferior tibiofibular subluxation* can be found. There is also a tender spot located at the lateral border of the proximal insertion of the patellar tendon (Fig. 67.7). The triad of symptoms associated with the *anterior recurrent peroneal syndrome* [34] includes the "exact painful spot, peroneus muscular hypertonicity, and some degree of varied subluxation" [34]. The list of differential diagnoses is found on Table 67.3.

Diagnostic Tests (Table 67.4)

Electrodiagnostic studies are important for diagnosis and prognosis. Motor studies are usually performed at the *extensor digitorum brevis* and *tibialis anterior muscles* [20]. Interestingly, muscles supplied by the DPN (deep fibular nerve) are more likely to be affected, since the nerve fibers of the DPN are located anteriorly and are therefore more sensitive to compression [20]. Any compound muscle action potential response from *tibialis anterior muscle* or extensor digitorum brevis muscle EMG is associated with a likely positive outcome of treatment (81 % and 94 %, respectively). Even those without a response had an approximately 50 % chance of a good result from treatment [40]. Many protocols for surgical treatment of CPN dysfunction require preoperative electrodiagnostic studies [6, 16, 41].

MRI technology continues to develop, and high-intensity MRIs are now available that can visualize the larger nerves. In addition to showing direct evidence of nerve injury and signs of muscle denervation [2], the new MRI technology can be used for investigation of anatomic variation in large numbers of asymptomatic individuals [5].

Table 67.3 Differential diagnosis of foot drop and/or lateral calf pain

	Potential distinguishing features
Sciatic nerve injury	Proximal pain and weakness
Radiculopathy or plexopathy	Likely to have low back pain and/or involvement of non-CPN innervated muscles
Tibiofibular joint pathology	Inflammation at joint by MRI
Polyneuropathy [3]	Involvement of other nerves
Ankle ligament instability may be PL weakness from partial denervation [38]	Ligament laxity
"Restless legs" may be a low-grade CPN neuropathy [38]	Sleep study could help identify restless legs
Peripheral vascular disease [13]	Abnormal pulses

Table 67.4 Diagnostic tests for common peroneal nerve entrapment/injury

	Potential distinguishing features
Physical exam	Positive Tinel's sign at the fibular neck is present in 97 % of patients with CPN entrapment [16]
Diagnostic injection	May help to localize the lesion [39]
Ultrasound [26]	Complements MRI and may be able to define a mass not seen on MRI, especially when a lipoma is present in a fat-filled area
MRI [2, 5]	May identify soft tissue masses; the proximal CPN can be visible and may be increased in size when the nerve is injured [2]
Arteriography	May be useful if there is evidence of peripheral vascular disease
X-ray	Identifies bony abnormalities of the fibula such as fracture, tumor, or exostosis [39]
Electrodiagnostic studies	"A superb diagnostic and prognostic tool" [3]
	CPN sensory action potential (SNAP): decreased amplitude
	Motor conduction: tibialis anterior, extensor digitorum brevis, short head of BF

Identification and Treatment of Contributing Factors

Peroneal neuropathy has been associated with a variety of endocrine and metabolic conditions such as diabetes, alcoholism, thyrotoxicosis, or vitamin B deficiency [42], so blood work may be indicated for diagnosis and treatment.

In-shoe devices to maintain the foot in eversion may improve biomechanics and decrease symptoms of CPN dysfunction [38]. If this is insufficient, an ankle foot orthosis (AFO) may be needed to manage foot drop. Reife and Coulis described a patient with persistent leg pain after spinal surgery; physical exam was consistent with common peroneal entrapment, and the symptoms resolved after the patient was counseled to stop crossing her legs [42].

Injection Technique

Care must be taken when doing injections of the CPN. Because of its exposed location, there is a risk of post-procedure foot drop and damage to the nerve with the needle.

Landmark-Guided Injection

After an appropriate skin prep, the CPN is localized at the fibular head and stabilized, using the non-injecting hand. With the injecting hand, the needle (25–27 gauge) is advanced slowly and obliquely to the bone to avoid deposit of steroid superficially in the skin (Video 67.2) (Fig. 67.10). The use of a short-bevel needle and a peripheral nerve stimulator can increase the efficacy and safety of this injection. A small volume of local anesthetic with a deposteroid may be injected into the area of maximum tenderness as an aid to localization of the source of sensory symptoms and treatment of the pain generator [39].

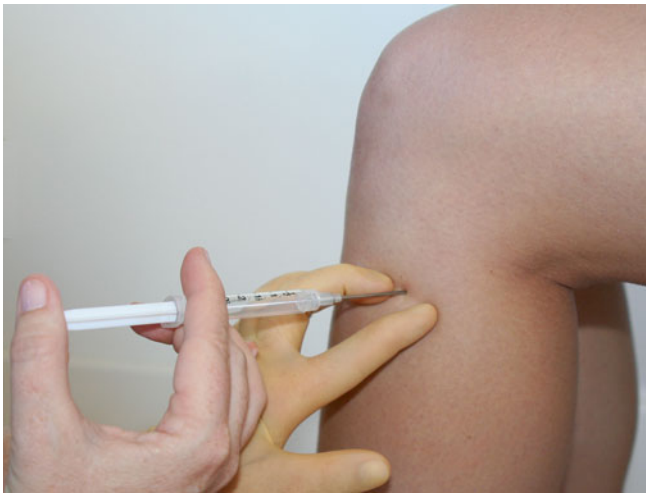

Fig. 67.10 Landmark-guided injection of the common peroneal nerve (Image courtesy of Andrea Trescot, MD)

Fluoroscopic-Guided Injection

There are no published fluoroscopic-guided techniques, though the fibular head is a good fluoroscopic landmark (Fig. 67.11).

Ultrasound-Guided Injection

The superficial location of the CPN and its close approximation to the fibular head are clear advantages to the use of ultrasound guidance (US) for injections of the CPN. The patient is placed in the lateral decubitus position, with the symptomatic knee up and slightly flexed. A linear transducer is placed over the sciatic nerve in the posterior thigh and directed distally to the popliteal fossa (Fig. 67.12), to identify the split of the sciatic nerve into the tibial and common peroneal nerves (Fig. 67.13). The probe is then used to trace the CPN as it wraps around the fibular head, allowing visualization of the CPN posterior and lateral to the fibula (Fig. 67.14). Using an in-plane technique, the needle is advanced from the posterior aspect of the probe (Fig. 67.15). When the needle is near the nerve, 5–10 cc of local anesthetic can provide a surgical block. Of concern with this

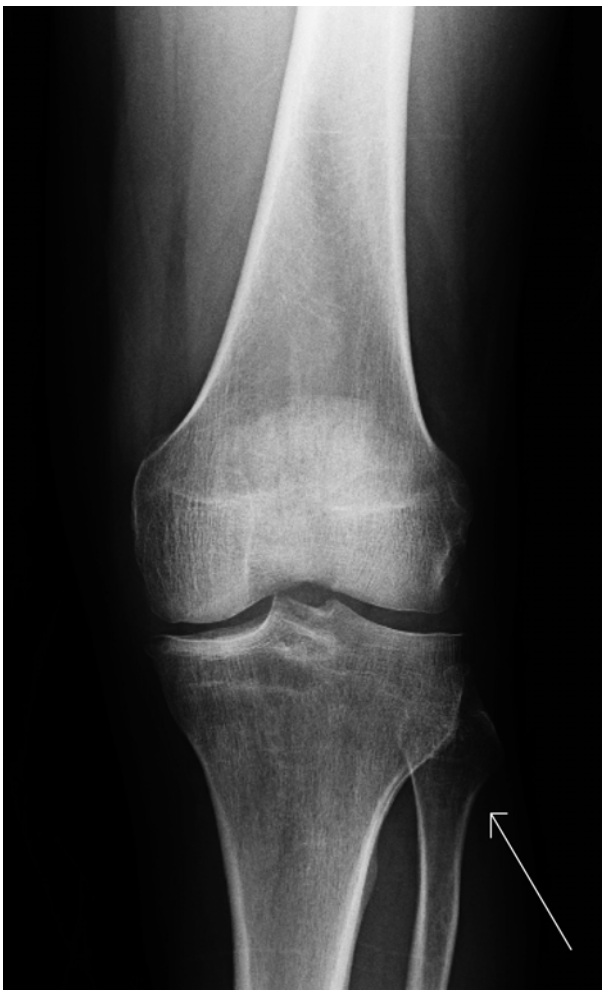

Fig. 67.11 Arrow shows the fluoroscopic landmark for a common peroneal nerve injection (Image courtesy of Andrea Trescot, MD)

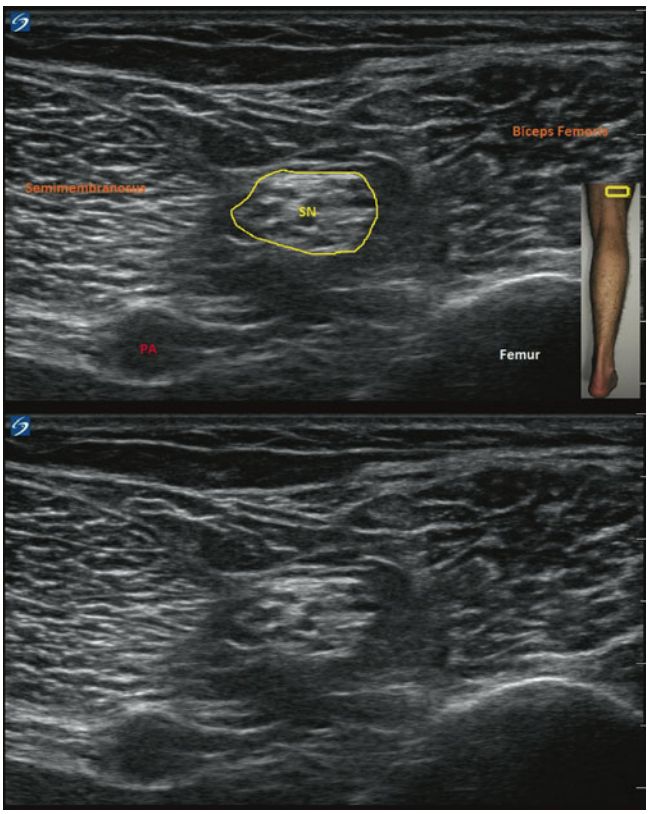

Fig. 67.12 Ultrasound identification of the sciatic nerve in the posterior thigh, between the biceps femoris and the semimembranosus muscles. *PA* popliteal artery (popliteal vein is compressed, lateral to the popliteal artery), *SN* sciatic nerve (Image courtesy of Agnes Stogicza, MD)

injection is the possibility of nerve injury by the needle or by a large volume of local anesthetic compressing the CPN against the bone in the low-volume fibular tunnel [43]. Diagnostic injections should be limited to no more than 2 cc of local anesthetic and deposteroid. Patients must be warned of the possible, even probable, foot drop associated with this injection.

Neurolytic Techniques

Cryoneuroablation

Because the CPN has such a significant motor component, it is rarely an appropriate neurolysis target. However, when there are specific conditions that require temporary neurolysis (such as neuroma treatment just distal to the fibular head or *phantom limb pain*), cryoneuroablation with the use of an AFO splint to manage the foot drop might be appropriate (personal communication, Andrea Trescot, MD), since the nerve, and therefore motor function, will return within 3 months. Although it has not been described, there is a theoretic potential for neurolysis of the anterior recurrent peroneal nerve. The cryoprobe is placed inferior to the fibular head, and stimulation (and possibly ultrasound) is used to identify the nerve (Fig. 67.16) (see Chap. 8).

Radiofrequency Lesioning

There are no reported cases of radiofrequency lesioning of the CPN, most likely because of the significant motor component of this nerve.

Neurostimulation

Because of the limitations of neurolytic techniques in this region, there is a rationale for the use of peripheral nerve stimulation (see Chap. 9). Unfortunately, generator placement has been a potential problem, usually requiring

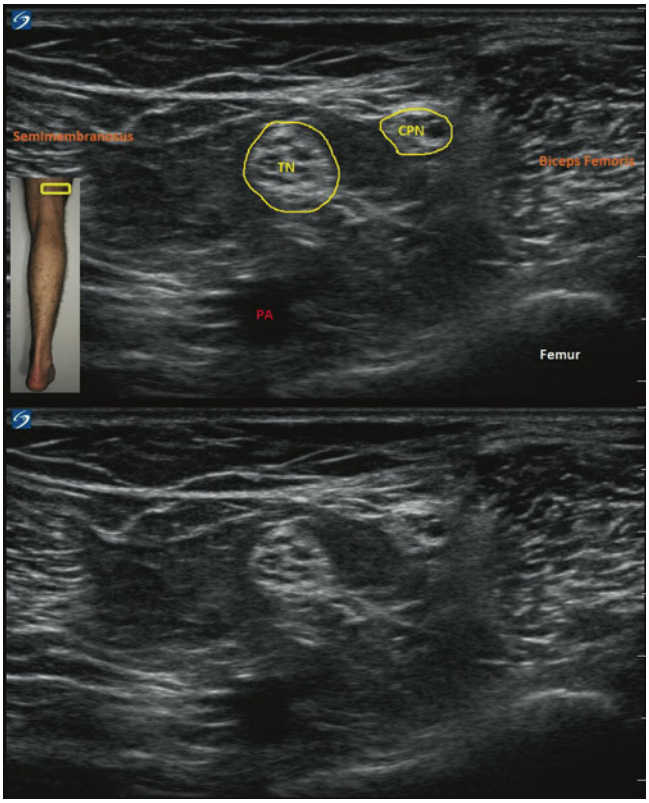

Fig. 67.13 Ultrasound identification of the sciatic nerve splitting into the common peroneal nerve and the tibial nerve. *PA* popliteal artery, *TN* tibial nerve, *CPN* common peroneal nerve (Image courtesy of Agnes Stogicza, MD)

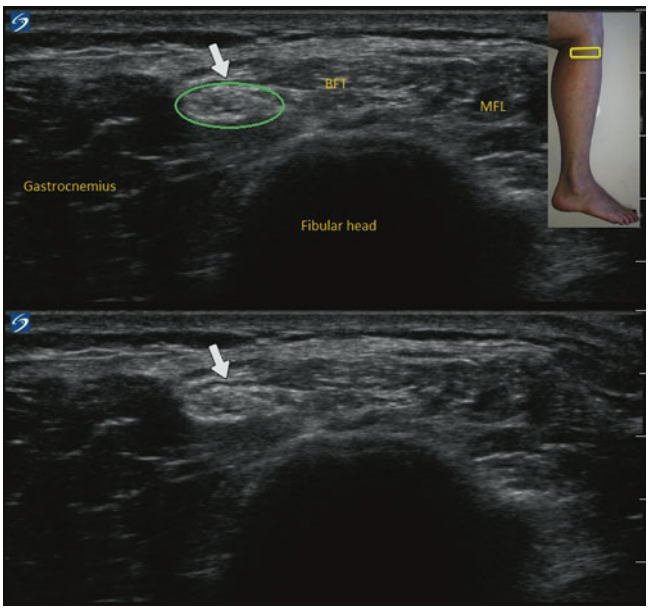

Fig. 67.14 Ultrasound identification of the common peroneal nerve at the fibular head. *MFL* musculus fibularis longus (peroneus longus muscle), *BFT* biceps femoris tendon. *Arrow* points to the common peroneal nerve (Image courtesy of Agnes Stogicza, MD)

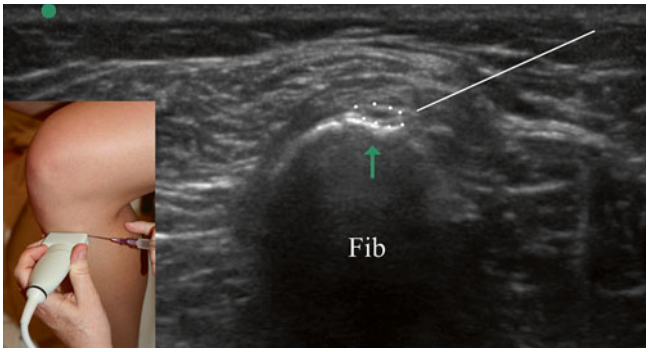

Fig. 67.15 Simulated needle placement for a common peroneal nerve injection at the fibular head using an in-plane approach. *Fib* fibula; *green arrow* identifies the nerve (Image courtesy of Andrea Trescot, MD)

placement in the groin or buttocks (Fig. 67.17). Lynch et al. described creating a pocket between the gastrocnemius and soleus muscles [44]. New technology (SpineWave®), where the receiver is incorporated into the lead itself, may solve this problem.

Surgical Techniques

Surgery, either neurolysis or grafting, is the most common and most effective treatment for CPN entrapment [39]. Many authors state that surgery is indicated if there is no return of function after 3–4 months, especially in cases of severe paresis [9, 35, 39], and that time to recovery was shorter with surgical release than with nonoperative rehabilitation [41]. As with neurolysis at other locations, it is important to release all entrapment sites; for the CPN trapped near the fibular head, this entails release of both the superficial and deep fibrous arches [6].

Sidey described 23 patients with CPN entrapment who underwent nerve release at the fibular head under local anesthetic; 20 of these patients had relief "rapidly and completely" [36].

Some CPN injuries may require nerve grafting instead of simple neurolysis. Sedel and Nizard [23] described 17 consecutive patients with traction injuries of the CPN; they were treated with grafts from sural nerves, but only 37.5 % had satisfactory results. The length of the graft required is an important factor in recovery. Forty-three percent of patients with 6–12 cm grafts had good outcomes, whereas only 25 % of those with 13–24 cm grafts did well [9]. If neurolysis and grafting are not successful, tendon transfer may improve foot drop and thereby decrease disability [9].

Although the concept of surgical release implies cutting fascial layers, El Gharbawy and colleagues [31] have postulated that the fascia surrounding the peroneal (fibular) tunnel actually serves to hold the tunnel open, and therefore care must be taken to preserve this fascia during surgical releases.

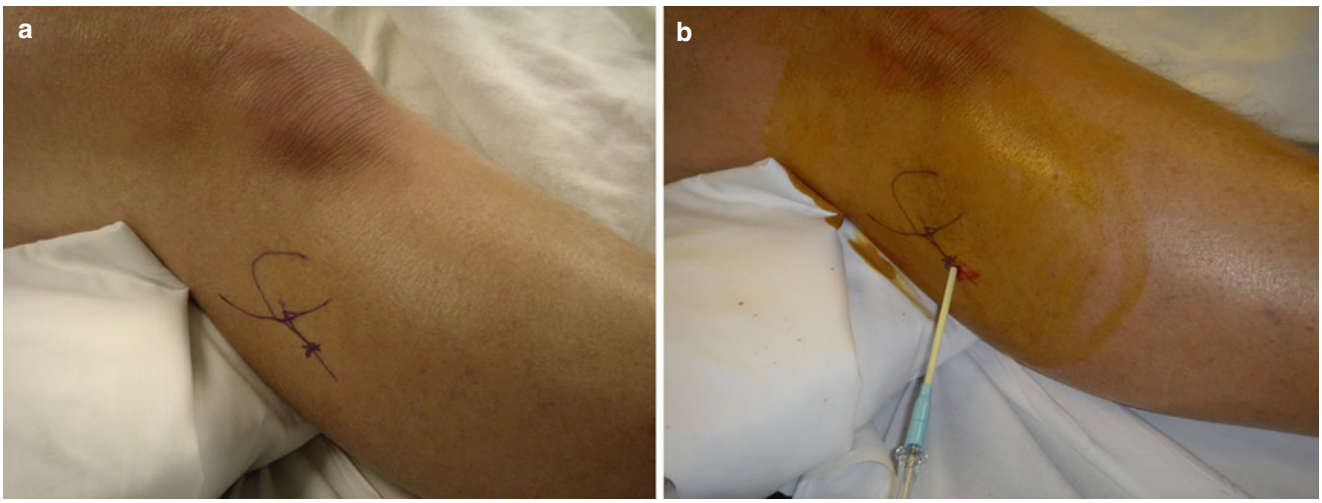

Fig. 67.16 Cryoneuroablation of the common peroneal nerve. (**a**) Surface landmarks, with the fibular head outlined and the area of maximal tenderness marked with an X, (**b**) the introducer placement for the cryoneuroablation probe (Image courtesy of Agnes Stogicza, MD)

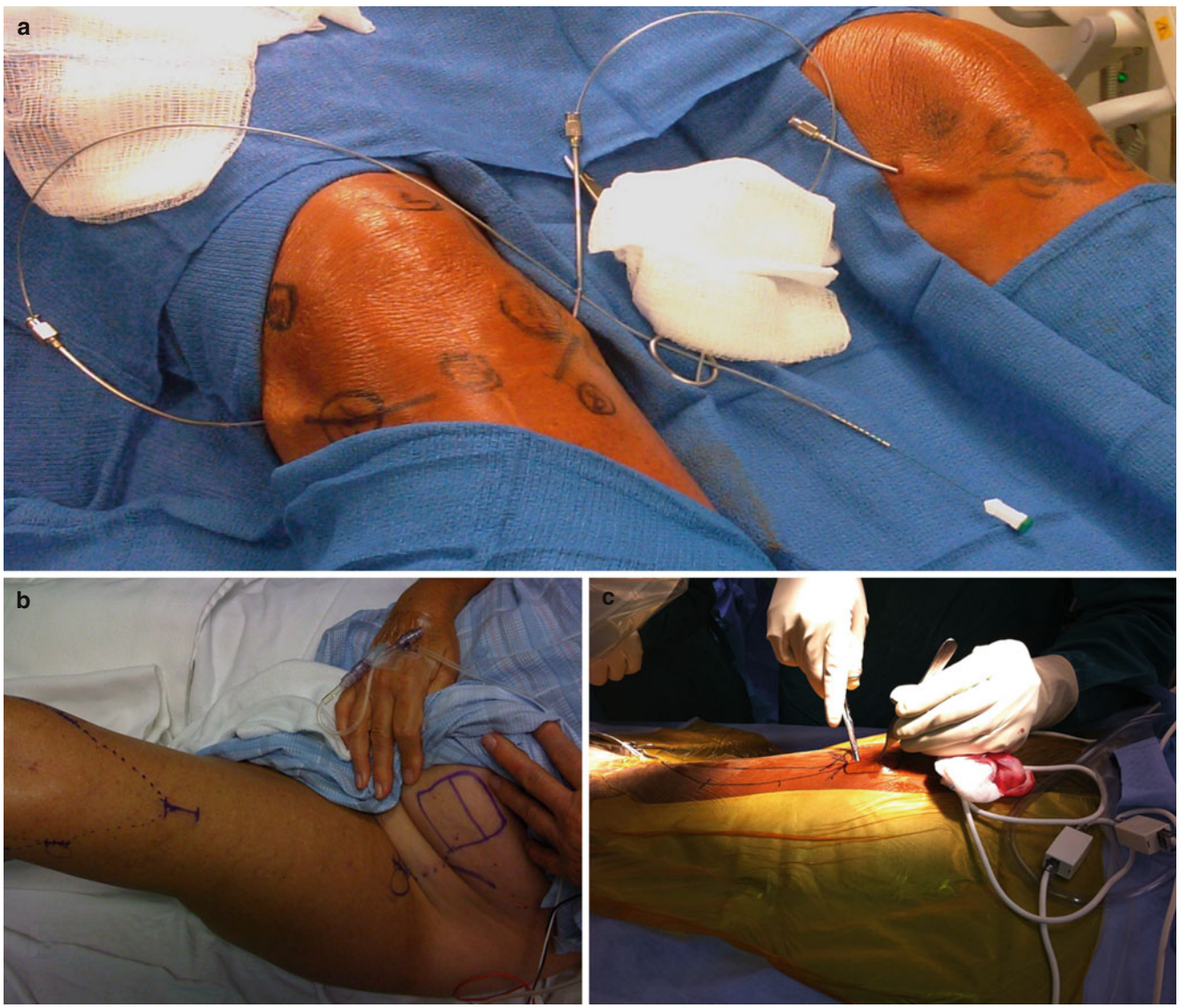

Fig. 67.17 Peripheral nerve stimulation for peroneal and saphenous neuralgia. (**a**) Percutaneous trial of bilateral peroneal and saphenous peripheral stimulator leads, (**b**) planning for lead and generator placement for peroneal peripheral stimulator, (**c**) preparing a thigh pocket for a peroneal peripheral nerve stimulator (Images courtesy of W. Porter McRoberts, MD)

Some authors consider the presence of a polyneuropathy (such as that due to diabetes or alcoholism) as a contraindication to surgery [16], though more recent work shows good results in restoring function in diabetics with CPN decompression [45].

Complications

Patients who have severe motor symptoms due to CPN compression are at risk of permanent paralysis. Even though there are reports that surgical neurolysis of compressed CPN can lead to recovery years after the onset of symptoms [6], most authors recommend much earlier intervention if nonoperative measures fail to relieve symptoms.

Summary

Common peroneal/fibular entrapment is one of the most common lower extremity entrapments; it can present in a variety of ways. Diagnostic injections require particular vigilance because of the potential of post-procedure foot drop.

References

1. Katirji MB, Wilbourn AJ. Common peroneal mononeuropathy: a clinical and electrophysiologic study of 116 lesions. Neurology. 1988;38(11):1723–8.
2. Donovan A, Rosenberg ZS, Cavalcanti CF. MR imaging of entrapment neuropathies of the lower extremity. Part 2. The knee, leg, ankle, and foot. Radiographics. 2010;30(4):1001–19.
3. Van Langenhove M, Pollefliet A, Vanderstraeten G. A retrospective electrodiagnostic evaluation of footdrop in 303 patients. Electromyogr Clin Neurophysiol. 1989;29(3):145–52.
4. Aprile I, Caliandro P, La Torre G, Tonali P, Foschini M, Mondelli M, et al. Multicenter study of peroneal mononeuropathy: clinical, neurophysiologic, and quality of life assessment. J Peripher Nerv Syst. 2005;10(3):259–68.
5. Vieira RL, Rosenberg ZS, Kiprovski K. MRI of the distal biceps femoris muscle: normal anatomy, variants, and association with common peroneal entrapment neuropathy. AJR Am J Roentgenol. 2007;189(3):549–55.
6. Humphreys DB, Novak CB, Mackinnon SE. Patient outcome after common peroneal nerve decompression. J Neurosurg. 2007;107(2):314–8.
7. Whitmore I. Terminologia anatomica: new terminology for the new anatomist. Anat Rec. 1999;257(2):50–3.
8. Brief JM, Brief R, Ergas E, Brief LP, Brief AA. Peroneal nerve injury with foot drop complicating ankle sprain – a series of four cases with review of the literature. Bull NYU Hosp Jt Dis. 2009;67(4):374–7.
9. Cho D, Saetia K, Lee S, Kline DG, Kim DH. Peroneal nerve injury associated with sports-related knee injury. Neurosurg Focus. 2011;31(5), E11.
10. Deutsch A, Wyzykowski RJ, Victoroff BN. Evaluation of the anatomy of the common peroneal nerve. Defining nerve-at-risk in arthroscopically assisted lateral meniscus repair. Am J Sports Med. 1999;27(1):10–5.
11. Yu JK, Yang JS, Kang SH, Cho YJ. Clinical characteristics of peroneal nerve palsy by posture. J Korean Neurosurg Soc. 2013;53(5):269–73.
12. Leach RE, Purnell MB, Saito A. Peroneal nerve entrapment in runners. Am J Sports Med. 1989;17(2):287–91.
13. Haimovici H. Peroneal sensory neuropathy entrapment syndrome. Arch Surg. 1972;105(4):586–90.
14. Chusid J. Yoga foot drop. JAMA J Am Med Assoc. 1971;217(6):827–8.
15. McCrory P, Bell S, Bradshaw C. Nerve entrapments of the lower leg, ankle and foot in sport. Sports Med. 2002;32(6):371–91.
16. Fabre T, Piton C, Andre D, Lasseur E, Durandeau A. Peroneal nerve entrapment. J Bone Joint Surg Am. 1998;80(1):47–53.
17. Spinner RJ, Amrami KK. Superficial peroneal intraneural ganglion cyst originating from the inferior tibiofibular joint: the latest chapter in the book. J Foot Ankle Surg. 2010;49(6):575–8.
18. Jang SH, Lee H, Han SH. Common peroneal nerve compression by a popliteal venous aneurysm. Am J Phys Med Rehabil. 2009;88(11):947–50.
19. Cruz-Martinez A, Arpa J, Palau F. Peroneal neuropathy after weight loss. J Peripher Nerv Syst. 2000;5(2):101–5.
20. Marciniak C. Fibular (peroneal) neuropathy: electrodiagnostic features and clinical correlates. Phys Med Rehabil Clin N Am. 2013;24(1):121–37.
21. Anderson AW, LaPrade RF. Common peroneal nerve neuropraxia after arthroscopic inside-out lateral meniscus repair. J Knee Surg. 2009;22(1):27–9.
22. Baima J, Krivickas L. Evaluation and treatment of peroneal neuropathy. Curr Rev Musculoskelet Med. 2008;1(2):147–53.
23. Sedel L, Nizard RS. Nerve grafting for traction injuries of the common peroneal nerve. A report of 17 cases. J Bone Joint Surg (Br). 1993;75(5):772–4.
24. Kashuk K. Proximal peripheral nerve entrapment syndromes in the lower extremity. J Am Podiatry Assoc. 1977;67(8):529–44.
25. Dong Q, Jacobson JA, Jamadar DA, Gandikota G, Brandon C, Morag Y, et al. Entrapment neuropathies in the upper and lower limbs: anatomy and MRI features. Radiol Res Pract. 2012;2012:230679.
26. Vasudevan JM, Freedman MK, Beredjiklian PK, Deluca PF, Nazarian LN. Common peroneal entrapment neuropathy secondary to a popliteal lipoma: ultrasound superior to magnetic resonance imaging for diagnosis. PM R. 2011;3(3):274–9.
27. Ryan W, Mahony N, Delaney M, O'Brien M, Murray P. Relationship of the common peroneal nerve and its branches to the head and neck of the fibula. Clin Anat. 2003;16(6):501–5.
28. Aigner F, Longato S, Gardetto A, Deibl M, Fritsch H, Piza-Katzer H. Anatomic survey of the common fibular nerve and its branching pattern with regard to the intermuscular septa of the leg. Clin Anat. 2004;17(6):503–12.
29. Sumalatha S, DS AS, Yadav JS, Mittal SK, Singh A, Kotian SR. An unorthodox innervation of the gluteus maximus muscle and other associated variations: a case report. Aust Med J. 2014;7(10):419–22.
30. Gloobe H, Chain D. Fibular fibrous arch. Anatomical considerations in fibular tunnel syndrome. Acta Anat. 1973;85(1):84–7.
31. El Gharbawy RM, Skandalakis LJ, Skandalakis JE. Protective mechanisms of the common fibular nerve in and around the fibular tunnel: a new concept. Clin Anat. 2009;22(6):738–46.
32. Boon AJ, Dib MY. Peripheral nerve entrapment and compartment syndromes of the lower leg. In: Akuthola V, Herring SA, editors. Nerve and vascular injuries in sports medicine. Philadelphia: Springer; 2009. p. 139–59.
33. Zipple JT, Hammer RL, Loubert PV. Treatment of fabella syndrome with manual therapy: a case report. J Orthop Sports Phys Ther. 2003;33(1):33–9.

34. Rousseau E. The anterior recurrent peroneal nerve entrapment syndrome: a patellar tendinopathy differential diagnosis case report. Man Ther. 2013;18(6):611–4.
35. Mont MA, Dellon AL, Chen F, Hungerford MW, Krackow KA, Hungerford DS. The operative treatment of peroneal nerve palsy. J Bone Joint Surg Am. 1996;78(6):863–9.
36. Ang CL, Foo LS. Multiple locations of nerve compression: an unusual cause of persistent lower limb paresthesia. J Foot Ankle Surg. 2014;53(6):763–7.
37. Sidey JD. Weak ankles. A study of common peroneal entrapment neuropathy. Br Med J. 1969;3(5671):623–6.
38. Kopell HP, Thompson WA. Peripheral Entrapment Neuropathies. Baltimore: Williams and Wilkins; 1976.
39. Flanigan RM, DiGiovanni BF. Peripheral nerve entrapments of the lower leg, ankle, and foot. Foot Ankle Clin. 2011;16(2):255–74.
40. Derr JJ, Micklesen PJ, Robinson LR. Predicting recovery after fibular nerve injury: which electrodiagnostic features are most useful? Am J Phys Med Rehabil. 2009;88(7):547–53.
41. Maalla R, Youssef M, Ben Lassoued N, Sebai MA, Essadam H. Peroneal nerve entrapment at the fibular head: outcomes of neurolysis. Orthop Traumatol Surg Res. 2013;99(6):719–22.
42. Reife MD, Coulis CM. Peroneal neuropathy misdiagnosed as L5 radiculopathy: a case report. Chiropr Man Ther. 2013;21(1):12.
43. Ting PH, Antonakakis JG, Scalzo DC. Ultrasound-guided common peroneal nerve block at the level of the fibular head. J Clin Anesth. 2012;24(2):145–7.
44. Lynch P, McJunkin T, Barrett S, Maloney J. A new surgical approach to peripheral nerve stimulation of the deep and superficial peroneal nerves for peroneal nerve pain (poster presentation). AAPM annual meeting. Washington, DC; 2011.
45. Aszmann OC, Kress KM, Dellon AL. Results of decompression of peripheral nerves in diabetics: a prospective, blinded study. Plast Reconstr Surg. 2000;106(4):816–22.

Superficial Peroneal Nerve Entrapment

68

Natalia Murinova, Sheila C. Chiu, Daniel Krashin, and Helen W. Karl

Introduction

Superficial peroneal nerve (SPN) entrapment was first described by Henry in 1945 as "mononeuralgia in the superficial peroneal nerve" [1]. In 1998, the *peroneal nerve* was renamed the *fibular nerve* to avoid confusion with the perineal region of the pelvis [2], but the name change has not been universally accepted, and both terms are used. The SPN has also been called the *musculocutaneous nerve of the leg* [3].

Clinical Presentation (Table 68.1)

Patients who have pain in the distal anterolateral calf, ankle, and dorsum of the foot, with or without paresthesia, should be considered to have possible SPN compression (Fig. 68.1). SPN dysfunction can result in weakness of foot eversion (with higher entrapments) as well as sensory changes in the lateral leg and dorsum of the foot and toes (Fig. 68.2). Pain typically worsens with physical activity, such as walking, running, or squatting [10, 16, 17].

SPN entrapment is an infrequent cause of anterolateral leg pain; 13 % of 98 patients referred with exercise-induced anterior leg pain thought to be chronic compartment syndrome turned out to have SPN entrapment [17]. Among 480 patients with chronic leg pain, 17 (3.5 %) were found to have entrapment of the SPN [18].

Table 68.1 Occupation/exercise/trauma history relevant to superficial peroneal (fibular) nerve entrapment

Sports	Runners [4]
	Skiing, football, soccer, basketball, ice hockey, track, and volleyball [5]
	Ballet dancers often have peroneus longus muscle hypertrophy and may have ankle instability which stretches the SPN; shoe ribbons may cross site of exit from deep fascia [6]
Extrinsic compression	Tight boots [7]
Trauma/surgery	Fasciotomy for compartment syndrome [8]
	Ankle fracture – more commonly injured by the surgical treatment than by the fracture itself [9, 10]; no evidence of SPN injury in patients who had a posterolateral surgical approach to their fracture [10]
	Fibular shaft fracture – SPN may be entrapped in the healing bone [11]
	Varicose vein surgery, including endovascular ablation [12]
	Ankle sprains – forces that pull on the SPN during sprains are particularly strong when there is an associated anterior talofibular ligament tear [13]
Other	Weight loss [14] and anorexia nervosa [15] – presumably because weight loss leads to increased exposure to compression

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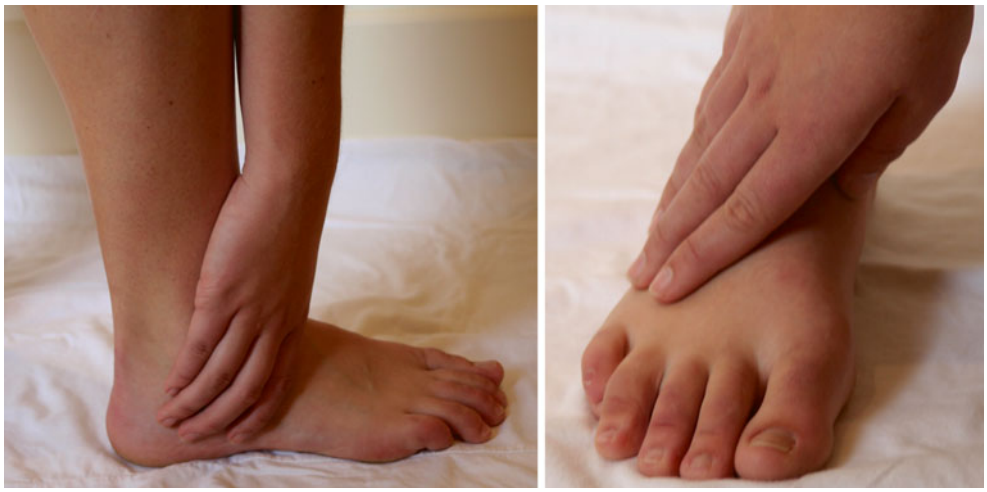

Fig. 68.1 Pain pattern of superficial peroneal (fibular) nerve entrapment (Image courtesy of Andrea Trescot, MD)

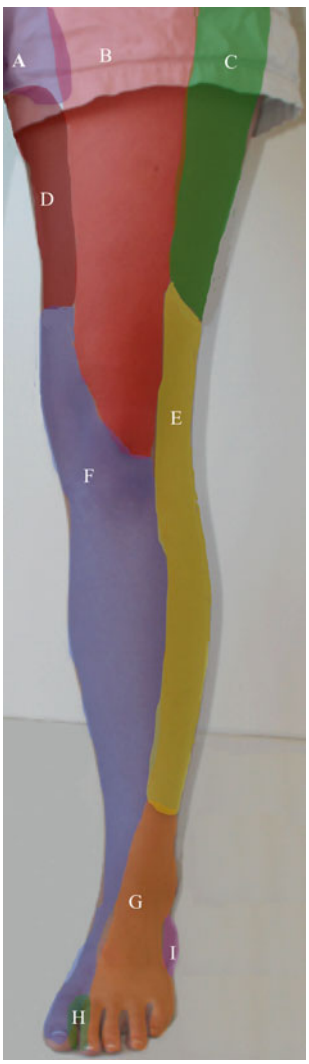

Fig. 68.2 Pattern of pain from anterior lower extremity nerve entrapments. *A* genitofemoral nerve, *B* femoral nerve, *C* lateral femoral cutaneous nerve, *D* obturator, *E* lateral sural cutaneous nerve, *F* saphenous nerve, *G* superficial peroneal nerve, *H* deep peroneal nerve, *I* sural nerve (Image courtesy of Andrea Trescot, MD)

Sports are a relatively common cause of SPN entrapment. Cho et al. evaluated 448 cases of peroneal nerve injury; 84 cases (18 %) were sports related, included skiing (42 cases), football (23 cases), soccer (eight cases), basketball (six cases), ice hockey (two cases), track (two cases), and volleyball (one case).

Symptoms include burning pain in the distribution of the nerve over the lateral calf and dorsum of the foot, at times associated with sensory abnormality [1, 16]. The pain may be aggravated by plantar flexion, a movement that stretches the SPN, which may make pressing the accelerator or brake quite difficult while driving [19].

SPN dysfunction is underdiagnosed [8]. Fifteen percent (18/120) of patients in one study of ankle fractures had symptoms of SPN neuropathy, though only two of them carried that diagnosis before the study. An additional 25 patients were less symptomatic, and SPN injury was discovered during their study follow-up. In this study then, 36 % of all ankle fracture patients had SPN injury due to their fracture; less than 2 % knew it [10].

Anatomy (Table 68.2)

The SPN is one of the terminal branches of the CPN and begins at its bifurcation near the proximal fibula [25] (Fig. 68.3). It passes deep to the *peroneus longus* muscle (PL), providing motor innervation to the PL, initially coursing between the PL and the fibula and then between the PL and *peroneus brevis* muscle (PB) to the distal leg. The SPN or its branches pierce the fascia by way of a tunnel (the *peroneal tunnel*) 3–18 cm proximal to the lateral malleolus. It then divides into its terminal branches, the *intermediate dorsal cutaneous nerve* and the *medial dorsal cutaneous nerve* [23] (Fig. 68.4). The SPN provides sensation to the distal 2/3 of the lateral leg and most of the dorsum of the foot.

Table 68.2 Superficial peroneal nerve anatomy

Origin	L4–S2 (sciatic nerve)
General route	The sciatic nerve travels behind the hip joint and then divides deep in the mid-thigh into the *common peroneal nerve* (CPN) (see Chap. 67) and *tibial nerve*. The CPN lies on the proximal *gastrocnemius muscle* (G) and follows the medial border of the *biceps femoris* (BF) along the lateral edge of the *popliteal fossa*. It enters the fibro-osseous peroneal tunnel near the head of the fibula and goes between the two heads of the *peroneus longus muscle* (PL), while curling from posterior to anterior around the fibular neck. It then divides into the *deep peroneal nerve* (DPN) (see Chap. 69) and *superficial peroneal nerve* (SPN). The SPN travels between the proximal fibula and peroneus longus muscle (PL) to the origin of the peroneus brevis muscle (PB). It becomes more superficial as it passes between the PB and extensor digitorum longus (EDL) muscles, traverses the deep (crural) fascia via the peroneal tunnel approximately 12 cm above the ankle, and divides to *intermediate and medial dorsal cutaneous nerves*. There are often multiple connections between the SPN and the sural nerve (Chap. 71) [20]
Sensory distribution	*Medial dorsal cutaneous nerve*: dorsal medial ankle, medial great toe, and second and third toes
	Intermediate dorsal cutaneous nerve: dorsal part of the ankle, the fourth toe, and parts of the third and fifth toes
Motor innervation	Lateral compartment muscles: PL and PB, which evert and plantar flex the foot
Anatomic variability	Site of division from CPN: 81 % distal to the fibular neck, 10 % proximal to the talofibular joint, 9 % between the joint and the fibular neck [21]
	Variable course of the SPN [22]: in 73 % of legs, the SPN descended in the lateral muscle compartment, in 14 %, it crossed into the anterior compartment, and in 12 %, branches were present in both the anterior and the lateral muscle compartments [23]
	Length of the peroneal tunnel: ≤3 cm is deemed normal; 42 % (10/24) of patients with known SPN entrapment had tunnels 3–11 cm long [8]
	Branching of cutaneous nerves: 72 % divided as described above; 28 % branched independently from the SPN in the calf and then showed even more variation distally [24]
Other relevant structures	Peroneus longus: hypertrophy may increase the risk of entrapment [6]

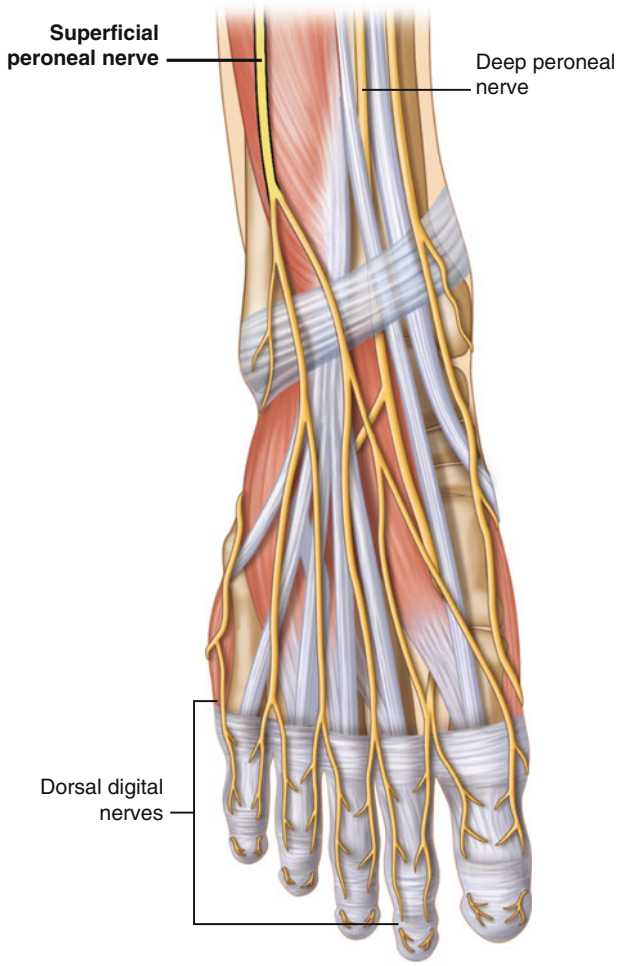

Fig. 68.3 Distal lower extremity anatomy (Image by Springer)

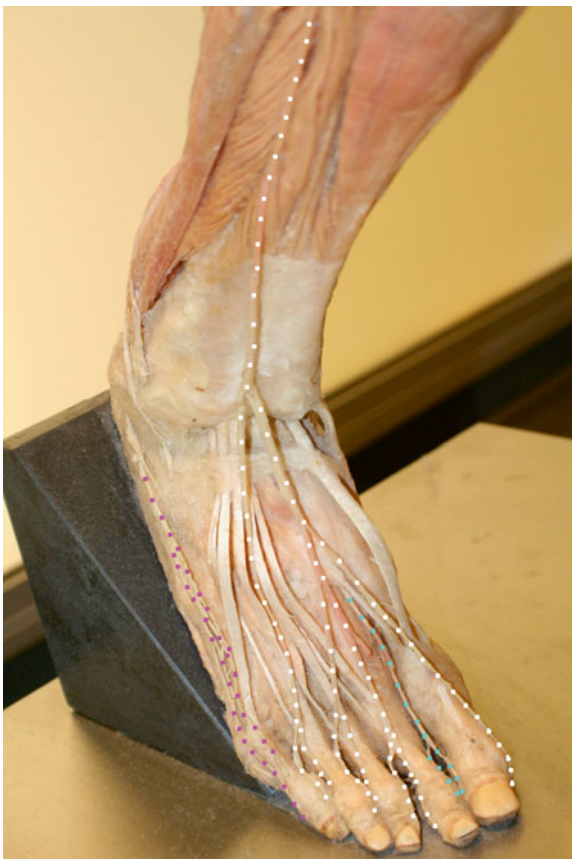

Fig. 68.4 Dorsal foot dissection, modified from an image from Bodies, The Exhibition, with permission. *White dots* superficial peroneal nerve, *green dots* deep peroneal nerve, *purple dots* sural nerve (Image courtesy of Andrea Trescot, MD)

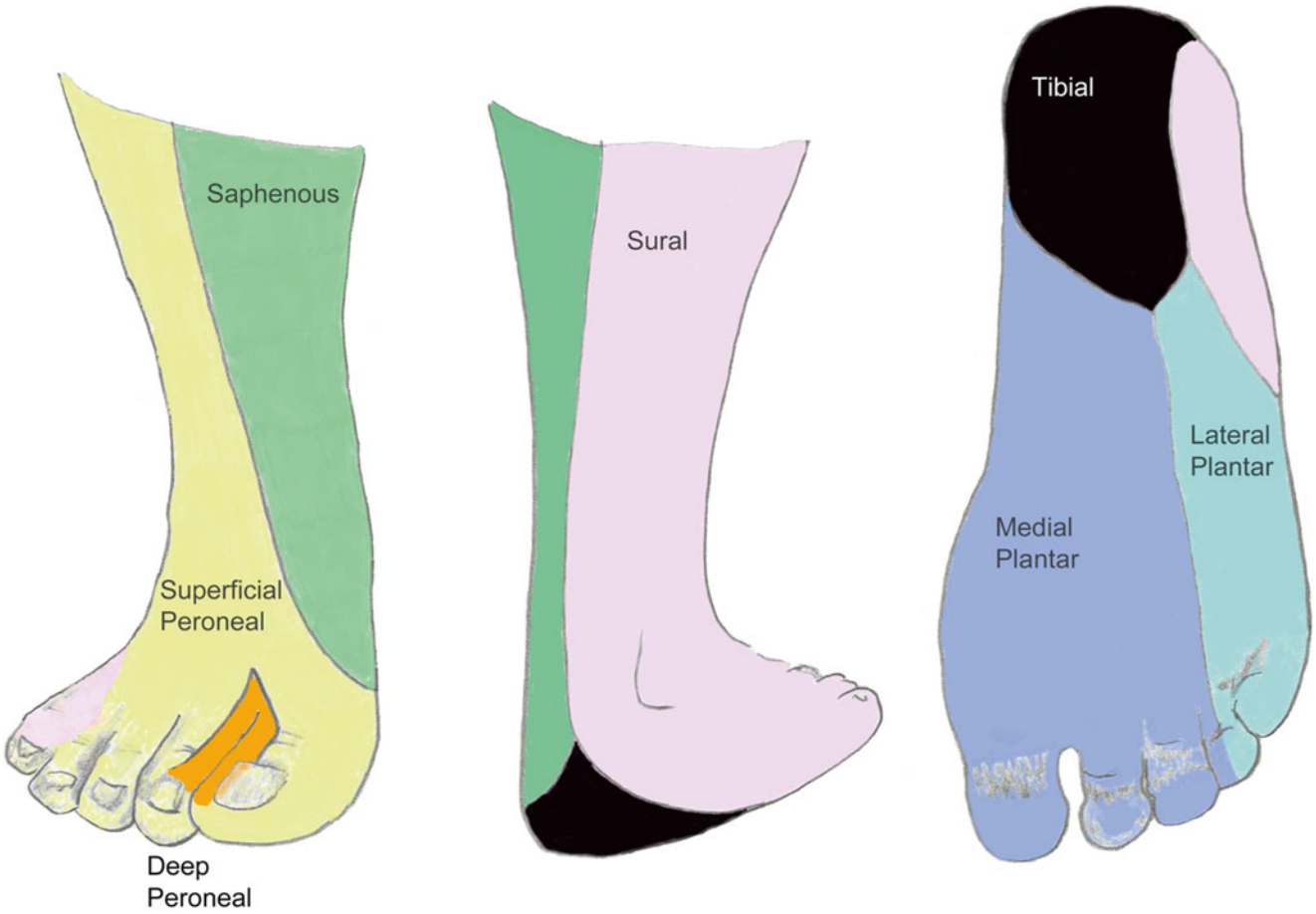

Fig. 68.5 Sensory distribution of the nerves of the foot (Image courtesy of Michael Brown, MD)

In a study of 75 lower extremities, Barrett et al. [26] found the SPN in the lateral compartment immediately next to the fascial septum in 54 of 75 (72 %) of the studied legs; however, four of the specimens (5 %) had branches in both the anterior and lateral compartments, and 17 cases (23 %) were in the anterior compartment. The authors note that these variations put the nerve at risk for trauma during fasciotomies, among other surgical techniques. Canella et al. [22] confirmed these findings, with US in live patients, noting the SPN in the anterior compartment in 26.7 % of the legs and watching it divide before piercing the crural fascia in 6.7 % of the legs.

Aktan Ikiz et al. [27] dissected 30 feet and found multiple variations in the distribution of the distal SPN, with up to seven different patterns identified.

A small twig from the *deep peroneal nerve* (Chap. 69) innervates the interspace between the great and second toes, and most of the dorsolateral foot provides sensory information via the *sural nerve* (Chap. 71) (Fig. 68.5). However, Drizenko et al. [20] showed in 55 dissections that there were communicating branches between the SPN and sural nerve in 35 cases (58 %), seen proximally at the level of the malleolus in approximately half and distal to the malleolus (in the metatarsal region) in half. The communicating branch was straight in 25 cases and curved in 11, with an average distance of the communicating branch from the crest of the lateral malleolus and the tubercle of the fifth metatarsal between 4.7 and 4.1 cm, but there was a wide range of values. They felt that stretching of the proximal communicating branch during forced inversion of the ankle and/or foot or during fractures of the calcaneus, direct injury in surgical approaches, or arthroscopy of the ankle may lead to unexplained pre- and submalleolar pain.

The SPN supplies the PL and PB muscles, which provide plantar flexion and eversion to the foot.

Entrapment

Entrapment of the SPN may occur by two mechanisms at the area where the nerve traverses the deep fascia (Fig. 68.6). The first mechanism is the presence of a long peroneal tunnel, which increases the risk of entrapment in the tunnel or at its outlet [8, 16]. The second mechanism is the existence of a defect in the fascia at that site which allows herniation of the

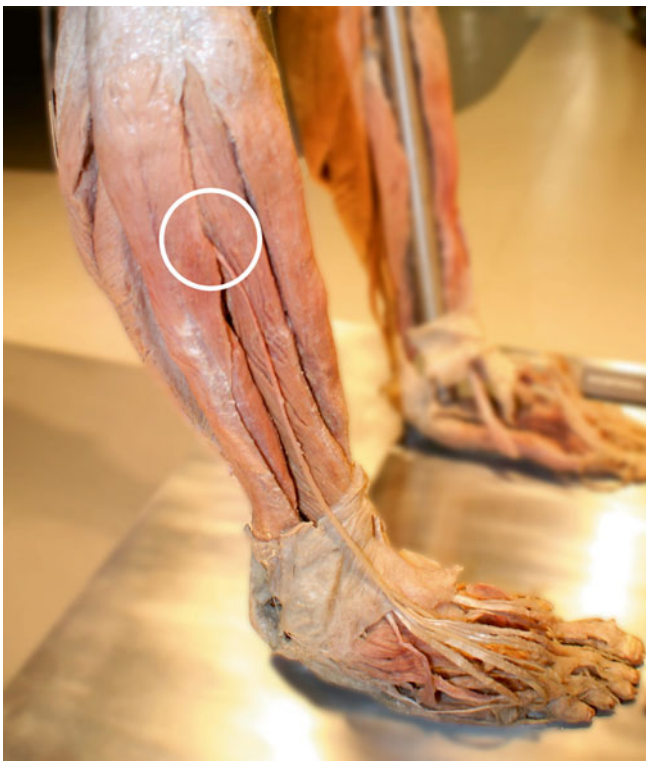

Fig. 68.6 Site of the proximal superficial peroneal nerve exit through the fascia, modified from an image from Bodies, The Exhibition, with permission (Image courtesy of Andrea Trescot, MD)

lateral compartment muscle with exercise. The herniated tissue may include the SPN, which then becomes compressed by the edge of the defect [8]. Ang and Foo described a patient who underwent a spinal surgery for lateral recess stenosis without relief of her leg pain; she was subsequently found to have entrapment of the SPN at multiple sites, which responded dramatically well to surgical release [28].

The SPN may also be compressed at the lateral calf or ankle. In this location, the nerve is superficial and thus susceptible to injury by direct trauma or by acute or chronic inversion injuries of the ankle (sprains) [13]. A significant cause of entrapment occurs with *lateral ankle sprains* (inversion injury) and plantar/flexion injuries of the foot, because of the posttraumatic edema as well as neuropraxia from the tethered nerve at the retinaculum. "Restless leg syndrome" has been associated with a low-grade neuropathy of the SPN [29].

Physical Exam

The physical examination should begin with evaluation of the low back, sciatic notch, and fibular neck. Then, look for a tender soft tissue bulge in the anterolateral leg, which increases in size with resisted dorsiflexion of the foot

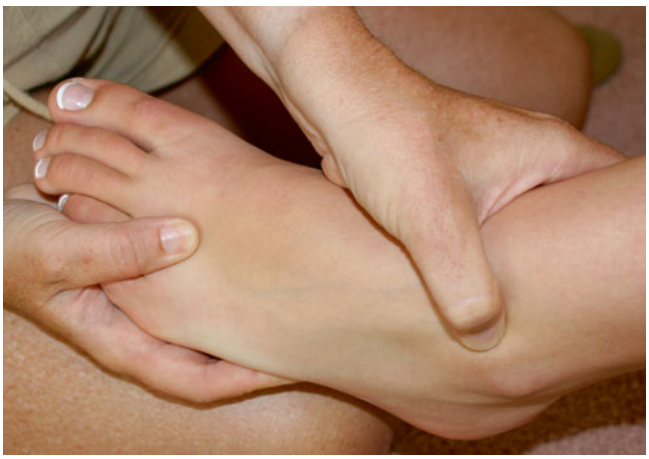

Fig. 68.7 Physical exam of the distal superficial peroneal nerve (Image courtesy of Andrea Trescot, MD)

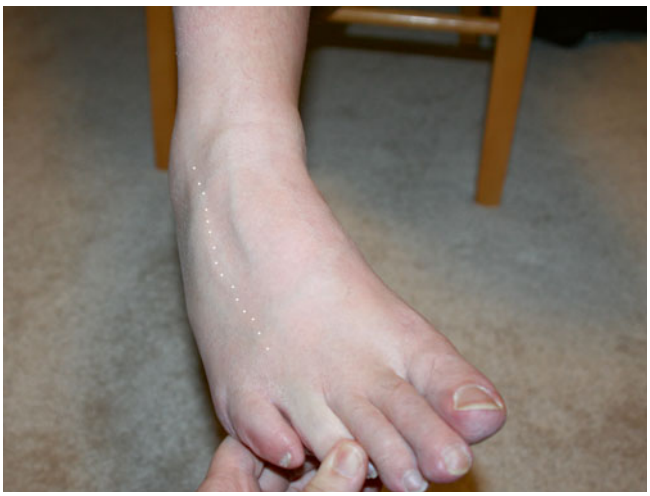

Fig. 68.8 Fourth toe sign – flexion of the fourth toe to identify the superficial peroneal nerve (Image courtesy of Andrea Trescot, MD)

(Fig. 68.7). Fascial defects at the exit of the SPN were discovered in 3/13 (23 %) of patients with SPN entrapment [17]. Percussion over the tender site of the nerve's exit through the fascia often causes shooting pain (Tinel's sign), either distally [16] or retrograde [1].

Palpation of the distal SPN is performed while holding the foot in mild plantar flexion and inversion (Video 68.1). Use the examining thumb to roll horizontally across the nerve, which is usually most tender just anterior to the lateral malleolus (Fig. 68.8), causing replication of the pain. When the fourth toe is passively plantar flexed, branches of the SPN can be seen tenting the skin of the dorsal foot (*fourth toe sign*) (Fig. 68.8). This may be useful when planning injections or procedures around the lateral ankle [30].

There are three provocative tests to diagnose SPN entrapment [8]. First, the patient actively dorsiflexes and everts the foot against resistance, while the examiner palpates the area

Table 68.3 Differential diagnosis of anterior leg or ankle pain

	Potential distinguishing features
Sciatic mononeuropathy	Proximal pain and weakness
L5 radiculopathy [1, 31]	Back pain, ankle dorsiflexion weakness [3]
Lumbosacral plexopathy	LBP and involvement of nerves
Arterial insufficiency [17]	Ankle-brachial pressure index will be low [19]
Stress fracture [17]	Positive bone scan
Ankle joint pathology [10]	X-rays, MRI
Anterior compartment syndrome [17]	Transducer to measure compartment pressures

of the peroneal tunnel. Then, the examiner passively plantar flexes and inverts the foot, first without pressure over the nerve, and then with percussion along its course [8]. Eliciting pain or paresthesias by these tests indicates SPN dysfunction and warrants further investigation [8].

Test for weakness of the peroneal muscles (foot eversion) and the presence of sensory disturbances affecting the skin of the lateral lower leg and dorsum of the foot, sparing the web space between the first and second toe. The distal sensory portion of the SPN may be affected in isolation, causing a purely sensory syndrome.

Differential Diagnosis (Table 68.3)

Common conditions that can mimic SPN entrapment include sciatic mononeuropathy, L5 radiculopathy, ankle joint pathology, and anterior compartment syndrome.

Diagnostic Tests (Table 68.4)

When the history and physical examination strongly suggest SPN entrapment, injection of a low volume of local anesthetic with or without steroid is a straightforward and cost-effective next step in diagnosis, since most patients will already have had X-rays and MRI. If further questions remain, nerve conduction velocity studies (NCV) and/or muscle pressure monitoring may be helpful.

NCV studies to confirm the location of an injury and help determine its severity and chronicity may be useful, but it is important to place the electrodes in a manner that isolates the result to the nerve of interest. A new method to measure NCV in the distal SPN in normal subjects uses two recording electrodes over the dorsal ankle and foot and a stimulation site over the anterior fibula. With this layout, stimulation of the SPN can be isolated from the deep peroneal nerve (DPN) and can be potentially helpful in diagnosing SPN neuropathy [34]. NCV studies provide more reliable information than EMG and may show decreased amplitude, increased latency, and focal slowing or conduction block. Normal NCV does not rule out the presence of SPN pathology, especially when the study is performed at rest. In one study, 46 % of patients with SPN entrapment had normal NCV at rest, but all became positive during exercise [17].

Table 68.4 Diagnostic tests for superficial peroneal nerve dysfunction

	Potential distinguishing features
Physical exam	Best done after exercise [17]
Diagnostic injection	The presence of pain relief and numbness in the SPN distribution after injection at the site of maximum tenderness is often useful [1, 31, 32]
X-ray	To exclude stress fracture, bone tumors
MRI	Near the site at which the SPN crosses the deep fascia, one may see an increase in its size or signal intensity; MRI with changes in foot position can identify muscle hernia [33]
Ultrasound	The best technique to demonstrate a muscle hernia [33], as the whole course of the SPN can be seen on US [22]
Muscle pressure monitoring [17]	Particularly useful if performed during and after exercise
Electrodiagnostic studies [34]	NCV is more useful than EMG

Monitoring muscle pressure to differentiate SPN entrapment from compartment syndrome is also particularly useful when it is done during or after exercise. In SPN entrapment, intramuscular pressure is normal (<35 mmHg muscle relaxation pressure and <30 mmHg at rest after exercise); it is considerably higher in the presence of compartment syndrome [17].

Diagnosis and Treatment of Perpetuating Factors

Prolonged extrinsic pressure is a significant cause of nerve trauma, so tight boots, tape, or ballet shoe ties that compress the nerve should be adjusted [1, 6, 7].

Serious consideration should be given to physical therapy as an early intervention when SPN entrapment at the peroneal tunnel is suspected. Exercises to stretch and strengthen the peroneal muscles may improve ankle stability, and lateral shoe wedges may help prevent further sprains [1, 35]. Techniques designed to increase the elasticity of the soft tissue in the lateral compartment combined with passive neural mobilization have been reported to relieve pain in a patient with SPN entrapment [19].

Medical conditions such as diabetic, hereditary, metabolic, and alcoholic neuropathies that increase vulnerability of the SPN to compression damage need to be treated. Spontaneous recovery is typically incomplete and occurs over 18–24 months, though addition of physical techniques can shorten this considerably [19].

The fourth toe flexion sign (see above) may be useful to identify SPN branches before any injection or procedure in this area, to avoid unexpected trauma [30].

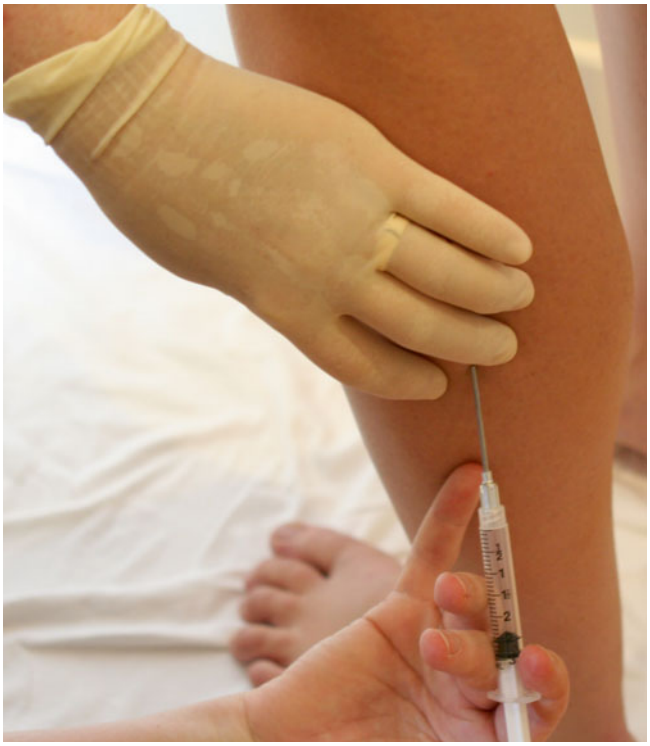

Fig. 68.9 Landmark-guided injection of the superficial peroneal nerve at the mid-calf (Image courtesy of Andrea Trescot, MD)

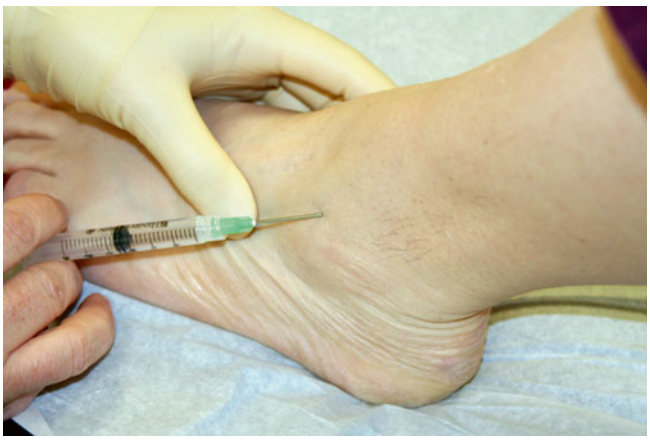

Fig. 68.10 Landmark-guided injection of the superficial peroneal nerve at the ankle (Image courtesy of Andrea Trescot, MD)

Injection Technique

Landmark-Guided Technique

Patient should be supine with their knees in slight flexion. Inject local anesthetic and steroid at the site of maximal tenderness along the anterolateral leg, usually 10–15 cm proximal to the lateral malleolus (Fig. 68.9). The more distal entrapment at the lateral malleolus can be injected just anterior the malleolus (Video 68.2) (Fig. 68.10), taking care to avoid too superficial an injection, which might cause skin atrophy from the steroid.

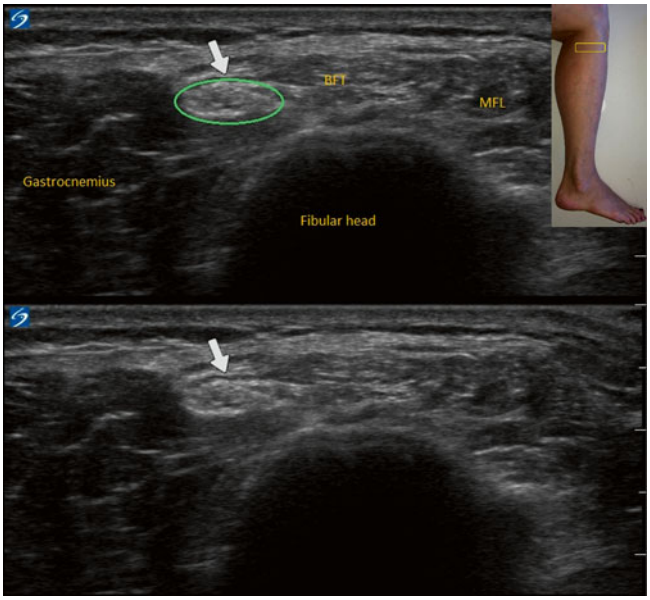

Fig. 68.11 Ultrasound image of the common peroneal nerve (*arrow*) at the fibular head before the split into the deep and superficial peroneal nerves. *MFL* fibularis longus muscle, *BFT* biceps femoris tendon (image courtesy of Agnes Stogicza, MD)

Ultrasound-Guided Technique

The anatomy of the common peroneal nerve is discussed in detail in Chap. 67 (Fig. 68.11). The substantial anatomic variability of the SPN and its branches as it becomes more distal [23, 24] make a powerful argument in favor of ultrasound (US) guidance for injections [22, 32, 36]. The nerve is relatively easy to see with a linear transducer as it becomes more superficial. A small vein consistently accompanies the SPN throughout its length, and fatty tissue surrounds it through much of its course, especially where it traverses the fascia [22] (Fig. 68.12). The nerve can be followed distally to the lateral malleolus (Fig. 68.13) and then onto the dorsum of the foot. Canella et al. [22] were able to trace the SPN with ultrasound all the way to the dorsum of the foot, but the level at which the SPN emerges between the PL and the *extensor digitorum longus muscles* (EDL) and where it pierces the crural fascia to become subcutaneous was found to be highly variable.

Ultrasound-Guided Catheter Technique

A peripheral nerve catheter and continuous infusion is a possible treatment for pain after SPN injury. A linear transducer was employed to visualize the nerve, using a posterolateral approach

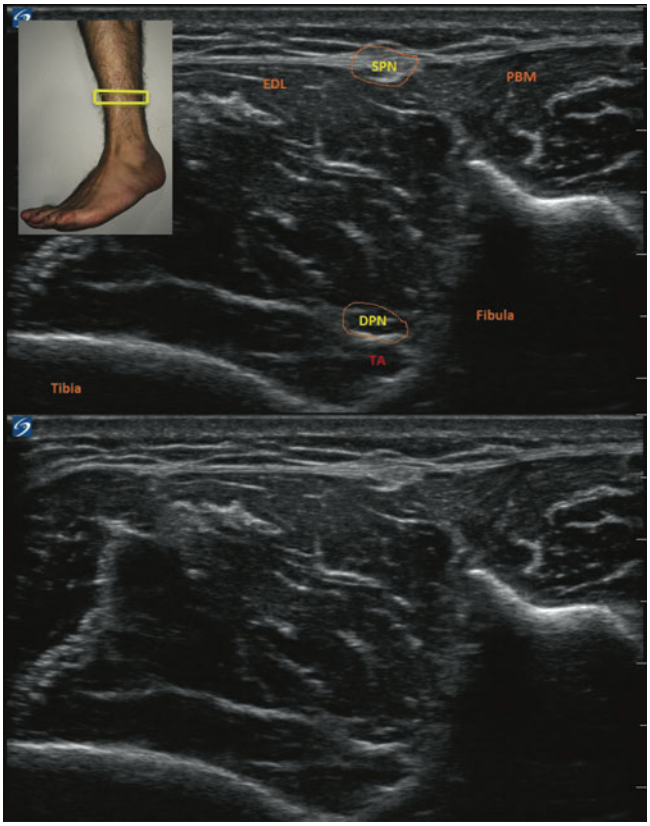

Fig. 68.12 Ultrasound imaging of the common peroneal nerve, showing the split between the deep and superficial peroneal nerves at the shin. *TA* tibial artery, *DPN* deep peroneal nerve, *SPN* superficial peroneal nerve, *EDL* extensor digitorum longus muscle, *PBM* peroneal brevis muscle (Image courtesy of Agnes Stogicza, MD)

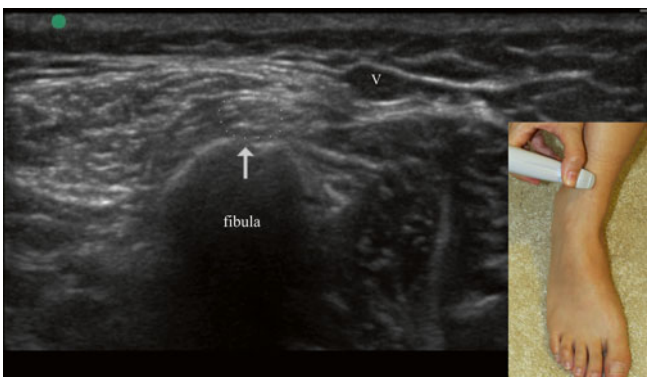

Fig. 68.13 Ultrasound imaging of the distal superficial peroneal nerve at the flexor retinaculum. *V* = vein (Image courtesy of Andrea Trescot, MD)

about 12 cm distal to the knee. Low amplitude peripheral nerve stimulation was applied to reproduce the patient's pain, and the catheter was inserted under US guidance [37].

Fluoroscopy-Guided Injections

There are no reported fluoroscopy techniques.

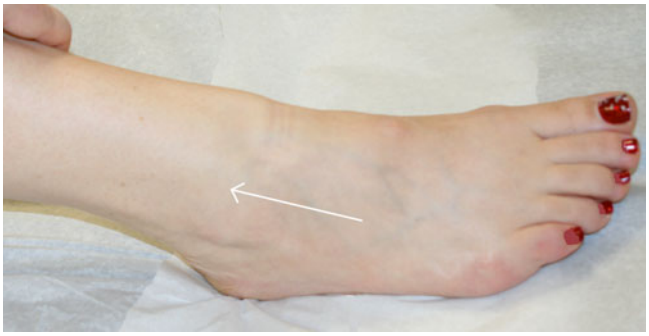

Fig. 68.14 Simulated probe location for cryoneuroablation of the superficial peroneal nerve (Image courtesy of Andrea Trescot, MD)

Neurolytic Technique

Cryoneuroablation

Trescot described a technique of cryoneuroablation of the superficial peroneal nerve [38]. A 12-gauge intravenous catheter is used as the introducer for the 2.0-mm cryoprobe, placing the probe parallel to the nerve (like starting an IV) (Fig. 68.14), using the built-in nerve stimulator, and then employing two to three 2-min freeze cycles. Special care must be taken to prevent frostbite injury to the skin. Approaching the nerve from a caudad to cephalad direction and keeping the probe positioned at an acute angle will help keep the ice ball below skin level and thereby decrease the frostbite risk.

Radiofrequency Lesioning (RF)

There are no reports of radiofrequency techniques for the superficial peroneal nerve, though pulsed radiofrequency has been used for similar nerves.

Chemodenervation

There are no reported phenol or alcohol techniques for this nerve.

Botulinum Toxin

If SPN entrapment is the result of a muscle hernia, injection of 20U of botulinum toxin into the herniated muscle using US and EMG guidance may provide relief [32].

Neurostimulation

Because of the limitations of neurolytic techniques in this region, there is a rationale for the use of peripheral nerve stimulation (see Chap. 9). Unfortunately, generator placement has

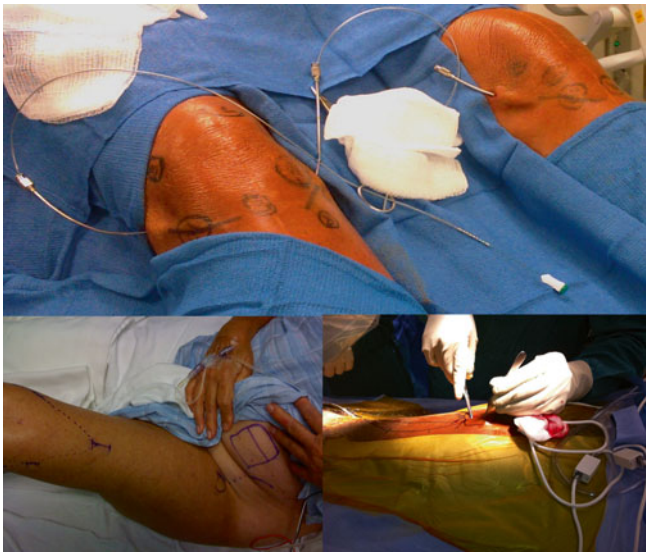

Fig. 68.15 Peripheral nerve stimulation for peroneal and saphenous neuralgia. (**a**) Percutaneous trial of bilateral peroneal and saphenous peripheral stimulator leads, (**b**) planning for lead and generator placement for peroneal peripheral stimulator, (**c**) preparing a thigh pocket for a peroneal peripheral nerve stimulator (Images courtesy of W. Porter McRoberts, MD)

been a potential problem, usually requiring generator placement in the groin or buttocks (Fig. 68.15). Lynch et al. described creating a pocket between the gastrocnemius and soleus muscles [39]. New technology (SpineWave®), where the receiver is incorporated into the lead itself, may solve this problem.

Surgical Technique

Surgery to decompress the peroneal tunnel may be needed if there is no response to nonoperative measures [18, 40]. Styr and Mosberg [18] described 17 patients who underwent surgical treatment of SPN (14 with decompression of the superficial peroneal tunnel and three with local fasciotomy); 14 patients (80 %) noted good pain relief. More recently, Cho et al. [5] looked at 84 cases of peroneal injuries related to sports; good functional outcomes from graft repair with a graft length <6 cm (70 %) and neurolysis (85 %) in low-intensity peroneal nerve injuries were seen. However, recovery from graft repair of graft lengths between 13 and 24 cm was seen in only 25 % of patients.

Complications

Neurologic injury following peripheral nerve block can be attributed to needle trauma, neuronal ischemia, or patient positioning. Because the skin can be thin (especially at the lateral malleolus), there is a risk of skin atrophy (from steroids) or "frostbite" (from cryoneuroablation).

Summary

SPN entrapment can be a perplexing and under-recognized cause of lower extremity pain, especially after lateral ankle trauma.

References

1. Kopell HP, Thompson WAL. Peripheral entrapment neuropathies. 2nd ed. Malabar, FL: R. E. Krieger Pub. Co; 1976.
2. Whitmore I. Terminologia anatomica: new terminology for the new anatomist. Anat Rec. 1999;257(2):50–3.
3. Sridhara CR, Izzo KL. Terminal sensory branches of the superficial peroneal nerve: an entrapment syndrome. Arch Phys Med Rehabil. 1985;66(11):789–91.
4. Schon LC, Baxter DE. Neuropathies of the foot and ankle in athletes. Clin Sports Med. 1990;9(2):489–509.
5. Cho D, Saetia K, Lee S, Kline DG, Kim DH. Peroneal nerve injury associated with sports-related knee injury. Neurosurg Focus. 2011;31(5), E11.
6. Kennedy JG, Baxter DE. Nerve disorders in dancers. Clin Sports Med. 2008;27(2):329–34.
7. Lindenbaum BL. Ski boot compression syndrome. Clin Orthop Relat Res. 1979;140:109–10.
8. Styf J. Entrapment of the superficial peroneal nerve. Diagnosis and results of decompression. J Bone Joint Surg (Br). 1989;71(1):131–5.
9. Baima J, Krivickas L. Evaluation and treatment of peroneal neuropathy. Curr Rev Musculoskelet Med. 2008;1(2):147–53.
10. Redfern DJ, Sauvé PS, Sakellariou A. Investigation of incidence of superficial peroneal nerve injury following ankle fracture. Foot Ankle Int. 2003;24(10):771–4.
11. Mino DE, Hughes EC. Bony entrapment of the superficial peroneal nerve. Clin Orthop Relat Res. 1984;185:203–6.
12. Shahid KR, Dellon AL, Amrami KK, Spinner RJ. Sciatic and peroneal nerve injuries after endovascular ablation of lower extremity varicosities: case reports and review of the literature. Ann Plast Surg. 2015;74(1):64–8.
13. O'Neill PJ, Parks BG, Walsh R, Simmons LM, Miller SD. Excursion and strain of the superficial peroneal nerve during inversion ankle sprain. J Bone Joint Surg Am. 2007;89(5):979–86.
14. Cruz-Martinez A, Arpa J, Palau F. Peroneal neuropathy after weight loss. J Peripher Nerv Syst. 2000;5(2):101–5.
15. Sevinç TT, Kalaci A, Doğramaci Y, Yanat AN. Bilateral superficial peroneal nerve entrapment secondary to anorexia nervosa: a case report. J Brachial Plex Peripher Nerve Inj. 2008;3:12.
16. Lowdon IM. Superficial peroneal nerve entrapment: a case report. J Bone Joint Surg (Br). 1985;67(1):58–9.
17. Styf J. Diagnosis of exercise-induced pain in the anterior aspect of the lower leg. Am J Sports Med. 1988;16(2):165–9.
18. Styf J, Morberg P. The superficial peroneal tunnel syndrome. Results of treatment by decompression. J Bone Joint Surg (Br). 1997;79(5):801–3.
19. Anandkumar S. Physical therapy management of entrapment of the superficial peroneal nerve in the lower leg: a case report. Physiother Theory Pract. 2012;28(7):552–61.
20. Drizenko A, Demondion X, Luyckx F, Mestdagh H, Cassagnaud X. The communicating branches between the sural and superficial

peroneal nerves in the foot: a review of 55 cases. Surg Radiol Anat. 2004;26(6):447–52.
21. Deutsch A, Wyzykowski RJ, Victoroff BN. Evaluation of the anatomy of the common peroneal nerve. Defining nerve-at-risk in arthroscopically assisted lateral meniscus repair. Am J Sports Med. 1999;27(1):10–5.
22. Canella C, Demondion X, Guillin R, Boutry N, Peltier J, Cotten A. Anatomic study of the superficial peroneal nerve using sonography. AJR Am J Roentgenol. 2009;193(1):174–9.
23. Adkison DP, Bosse MJ, Gaccione DR, Gabriel KR. Anatomical variations in the course of the superficial peroneal nerve. J Bone Joint Surg Am. 1991;73(1):112–4.
24. Blair JM, Botte MJ. Surgical anatomy of the superficial peroneal nerve in the ankle and foot. Clin Orthop Relat Res. 1994;305:229–38.
25. Pacha D, Carrera A, Llusa M, Permanyer E, Molona O, Morro R. Clinical anatomy of the superficial peroneal nerve in the leg. Eur J Anat. 2003;7(S1):15–20.
26. Barrett SL, Dellon AL, Rosson GD, Walters L. Superficial peroneal nerve (superficial fibularis nerve): the clinical implications of anatomic variability. J Foot Ankle Surg. 2006;45(3):174–6.
27. Aktan Ikiz ZA, Ucerler H. The distribution of the superficial peroneal nerve on the dorsum of the foot and its clinical importance in flap surgery. Foot Ankle Int. 2006;27(6):438–44.
28. Ang CL, Foo LS. Multiple locations of nerve compression: an unusual cause of persistent lower limb paresthesia. J Foot Ankle Surg. 2014;53(6):763–7.
29. Kashuk K. Proximal peripheral nerve entrapment syndromes in the lower extremity. J Am Podiatry Assoc. 1977;67(8):529–44.
30. Stephens MM, Kelly PM. Fourth toe flexion sign: a new clinical sign for identification of the superficial peroneal nerve. Foot Ankle Int. 2000;21(10):860–3.
31. Banerjee T, Koons DD. Superficial peroneal nerve entrapment. Report of two cases. J Neurosurg. 1981;55(6):991–2.
32. Yoo MJ, Kim D, Lee J, Oh-Park M. Injection of botulinum toxin as a treatment for superficial peroneal nerve entrapment caused by muscle hernia: a case report. Arch Phys Med Rehabil. 2007; 88:E70.
33. Donovan A, Rosenberg ZS, Cavalcanti CF. MR imaging of entrapment neuropathies of the lower extremity. Part 2. The knee, leg, ankle, and foot. Radiographics. 2010;30(4):1001–19.
34. Takahashi N, Takahashi O, Takahashi M, Akaboshi K, Nagata M, Ebata H, et al. A new method of superficial peroneal nerve conduction studies. Electromyogr Clin Neurophysiol. 2003;43(8): 507–10.
35. Flanigan RM, DiGiovanni BF. Peripheral nerve entrapments of the lower leg, ankle, and foot. Foot Ankle Clin. 2011;16(2):255–74.
36. Snaith R, Dolan J. Ultrasound-guided superficial peroneal nerve block for foot surgery. AJR Am J Roentgenol. 2010;194(6):W538; author reply W42.
37. Jaffe JD, Henshaw DS, Nagle PC. Ultrasound-guided continuous superficial peroneal nerve block below the knee for the treatment of nerve injury. Pain Pract. 2013;13(7):572–5.
38. Trescot AM. Cryoanalgesia in interventional pain management. Pain Phys. 2003;6(3):345–60.
39. Lynch P, McJunkin T, Barrett S, Maloney J. A new surgical approach to peripheral nerve stimulation of the deep and superficial peroneal nerves for peroneal nerve pain (poster presentation). AAPM annual meeting. Washington, DC; 2011.
40. Yang LJ, Gala VC, McGillicuddy JE. Superficial peroneal nerve syndrome: an unusual nerve entrapment. Case report. J Neurosurg. 2006;104(5):820–3.

Deep Peroneal Nerve Entrapment

69

Beth S. Pearce, Michael N. Brown, and Helen W. Karl

Introduction

Deep peroneal nerve (DPN) (also known as *deep fibular nerve*) entrapment was originally described by Gruber in 1877 and then by Kopell and Thompson in 1963 [1]. Marinacci later termed one site of DPN compression the *anterior tarsal tunnel* (ATT) and proposed an electrodiagnostic technique to assist in diagnosis [2]. The terminology can be confusing; some earlier authors do not distinguish between the DPN and its branches [the *medial branch of the deep peroneal nerve* (*MBDPN*) is particularly likely to be termed "DPN"] and also refer to entrapment of one of the branches as "*ATT syndrome*" or "*partial ATT syndrome*" [3]. The peroneal nerve was renamed the *fibular nerve* in 1998, in an effort to avoid confusion with the perineal pelvic region, but the name change has not been commonly accepted, and both terms are used [4].

The relationship between recurrent ankle sprain and DPN entrapment is of particular interest because the former is such a common injury and because post-sprain pain (*sinus tarsi syndrome*) and joint instability are so frequent. Injections of local anesthetic into the *sinus tarsi* (see below) reduce these symptoms [5, 6], but surgical denervation may be needed [7]. Patients with unstable ankles may wear tight supportive footwear in an attempt to stabilize the joint, only to develop ATT syndrome as a result [3].

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Clinical Presentation (Table 69.1)

DPN neuropathy is commonly the result of acute or repetitive insults at multiple points in the leg, ankle, and foot, which can lead to perineural adhesions and decreased mobility of the nerve (Table 69.1) [9]. The pain of DPN entrapment depends on the level of the injury. Symptoms tend to increase over time and include generalized, often vague sensations on the top of the foot (Fig. 69.1) and between the first two toes (Fig. 69.2) that tend to progress to increasing pain and disability. Shoes, if tolerated at all, will exacerbate the complaints, and an antalgic gait pattern may be observed. Diabetics and women seem to be particularly vulnerable to this injury, which often results from tightly fitting shoes [8, 13]. A detailed history of symptom onset, trauma, and recent shoe or activity changes can be helpful for diagnosis. In addition to symptom exacerbation by activity and plantar flexion of the foot, pain sufficient to wake the patient from

Table 69.1 Occupation/exercise/trauma history relevant to deep peroneal nerve entrapment

Sports	Runners and sports that involve running [8]
	Tight lacing exacerbated by extreme plantar flexion
	Kicking sports such as soccer
	Sit ups with feet hooked under a bar
	Dancers [9–11]
Mechanical	Osteoarthritis [9] with osteophyte compression
	High-heeled shoes [2, 10, 12]
	Shoe or cast that compresses the top of the foot [9, 13]
	Pes cavus [8, 9] – the high longitudinal arch makes the talonavicular and cuneonavicular joints more prominent and stretches the inferior extensor retinaculum
Trauma/surgery	Direct blow to the top of the foot with or without associated fracture [1, 2, 8, 9]
	Ankle sprain [2, 5, 14]
	Ankle arthroscopy

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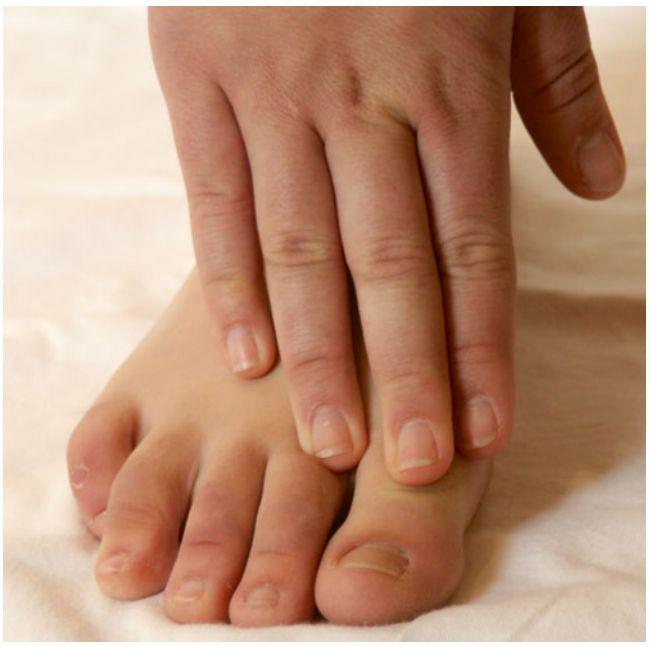

Fig. 69.1 Patient description of deep peroneal entrapment pain (Image courtesy of Andrea Trescot, MD)

sleep is not uncommon and is probably due to a sleep position that stretches the DPN or one of its branches [8].

Anatomy (Table 69.2)

The DPN is one of the major divisions of the common peroneal nerve (CPN), also known as the common fibular nerve (see Chap. 67), which is a branch of the sciatic nerve (Fig. 69.3). Near the head of the fibula, the CPN enters an osteofibrous hiatus or opening within the intermuscular septum, from the lateral to the anterior compartment and the fibula [16], splitting into the *superficial peroneal nerve* (SPN) (see Chap. 68), the DPN, and *anterior recurrent peroneal nerve* (ARPN) (Fig. 69.4). As the names imply, the SPN stays relatively superficial along the lateral portion of the calf, the DPN dives more deeply and anteriorly, and the ARPN travels cephalad to the knee. In the lower leg, the DPN sends motor branches to all of the anterior compartment muscles, which include the *tibialis anterior* (TA), *extensor digitorum longus* (EDL), *peroneus tertius*, and *extensor hallucis longus* (EHL). The

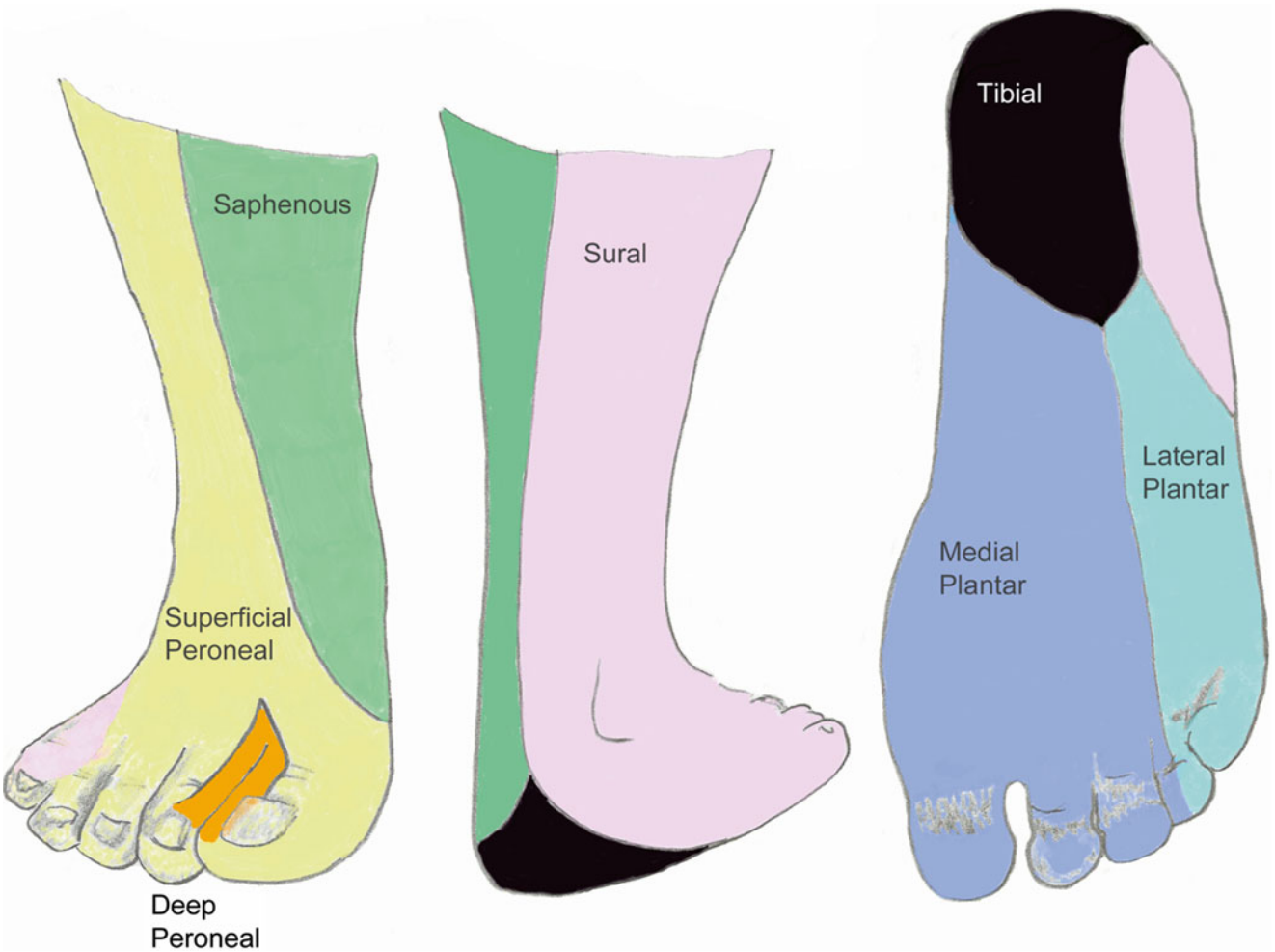

Fig. 69.2 Pattern of pain from peroneal nerve entrapment (Image courtesy of Michael Brown, MD)

Table 69.2 Deep peroneal nerve anatomy

Origin	L4 – S2
General route	Originates at the common peroneal nerve (CPN) (see Chap. 67) down to the proximal fibula, where it divides into the deep peroneal nerve (DPN) and the superficial peroneal nerve (SPN) (see Chap. 68). The DPN gives off some branches for anterior compartment muscles, crosses the anterior intermuscular septum, and travels with the anterior tibial vessels along the interosseous membrane, and then deep to the proximal and distal extensor retinacula to the foot. About 1 cm proximal to the ankle, it divides further into the medial branch of the deep peroneal nerve (MBDPN) and larger lateral branch of the deep peroneal nerve (LBDPN) branches. In the foot, the LBDPM gives off joint and muscular branches, while the MBDPN accompanies the artery along the dorsum of the foot and divides into two cutaneous digital nerves
Sensory distribution	MBDPN: first metatarsophalangeal joint; branches (lateral cutaneous nerve of the great toe and *medial cutaneous nerve of the second toe*) supply the area between the first and second toes
	LBDPN: *recurrent capsular branch*, branches to the sinus tarsi and to the tarsal and metatarsophalangeal (MTP) joints of the middle three toes
Motor innervation	DPN: anterior compartment muscles, including tibialis anterior (TA) and toe extensors (*extensor digitorum longus* (EDL), *extensor hallucis longus* (EHL), and *peroneus tertius*), which dorsiflex the ankle and toes
	MBDPN: first and second *dorsal interossei muscles*
	LBDPN: intrinsic toe extensors, *extensor digitorum brevis* (EDB) and *extensor hallucis brevis* (EHB) muscles
Anatomic variability	Site of division of CPN into DPN and SPN: 81 % distal to the fibular neck, 10 % proximal to the talofibular joint, 9 % between the joint and the fibular neck [15]
	Number of muscular branches given off in the lateral compartment: 1–6, most commonly three (35 %) [16]
	Site of division into MBDPN and LBDPN: 1.1 ± 1.2 cm from the ankle mortise, 10/22 (45 %) divided distal to the inferior retinaculum [14]. Division above the head of the talus occurred in 92 % (23/25) of cadavers but in only 50 % (4/8) of patients with documented ATTS. High division allows the two branches to pass on either side of the talar head [9]
	Relationship to the dorsalis pedis artery: four types were described. In 31 % (11/36), the nerve and artery cross each other at multiple levels, making entrapment by the artery particularly likely [17]. This relationship is also important when the artery is a landmark for block placement [18]
	Branches of the LBDPN [14]:
	Capsular: to the capsule of the ankle joint and anterior tibiofibular ligament
	Sinus tarsi: 2.7 ± 1.2 cm from the ankle mortise
	Ganglion: present in 5 % (1/22)
	EDB: 4 ± 0.8 cm from the ankle mortise; largest branch
	Subcutaneous: present in 18 % (4/22) to skin over lateral midfoot
	Terminal: interosseous nerves to the third, fourth, and fifth tarsometatarsal joints and metatarsal bones
	Presence of an accessory DPN (a branch of the SPN) to EDB occurs in about 25 % of people [8, 19]
Other relevant structures	*Anterior intermuscular septum* [16]
	Anterior tarsal tunnel (*ATT*): a flattened oval deep to the inferior band of the inferior extensor retinaculum and bounded by the bone on the other three sides (medial and lateral malleoli, talonavicular joint). Contains the DPN or its branches, anterior tibial artery and vein, and four tendons (TA, EHL, peroneus tertius, EDL)
	Sinus tarsi: a cavity between the lateral talus and calcaneus which allows access to the talocalcaneal ligament [14, 20]

main trunk of the DPN accompanies the *anterior tibial artery* on the interosseous membrane and travels beneath the tendon of the EHL in the distal third of the leg and then under the *extensor retinaculum* at the anterior ankle (Fig. 69.5).

The DPN usually divides about 1.5 cm proximal to the ankle joint into a *lateral branch of the deep peroneal nerve* (LBDPN) and a *medial branch of the deep peroneal nerve* (MBDPN) (Fig. 69.6). The MBDPN is primarily sensory but does supply motor innervation to the first and second interosseous muscles. It runs with the *dorsalis pedis artery* on the top of the foot (Fig. 69.7) and then under the *extensor hallucis brevis* (EHB) tendon to lie on the first interosseous muscle, between the first and second metatarsal bones. The MBDPN supplies the first metatarsophalangeal (MTP) joint and first dorsal interosseous muscle and ends in the skin between the first and second toes (Fig. 69.8) [12]. Distally, in up to 29 % in one study, the MBDPN communicates with the *medial dorsal cutaneous nerve*, a branch of the *superficial peroneal nerve* (SPN) [21].

As the LBDPN passes across the foot, it gathers sensory fibers from the ankle joint (important for ankle stability) and from the *sinus tarsi* (also known as the *tarsal sinus*), a cylindrical cavity between the talus and calcaneus on the lateral aspect of the foot. It usually provides motor supply to the EDB, though additional innervation of this muscle may be provided by the *accessory peroneal nerve*, a branch of the SPN. The LBDPN terminates as *interosseous nerves*, which

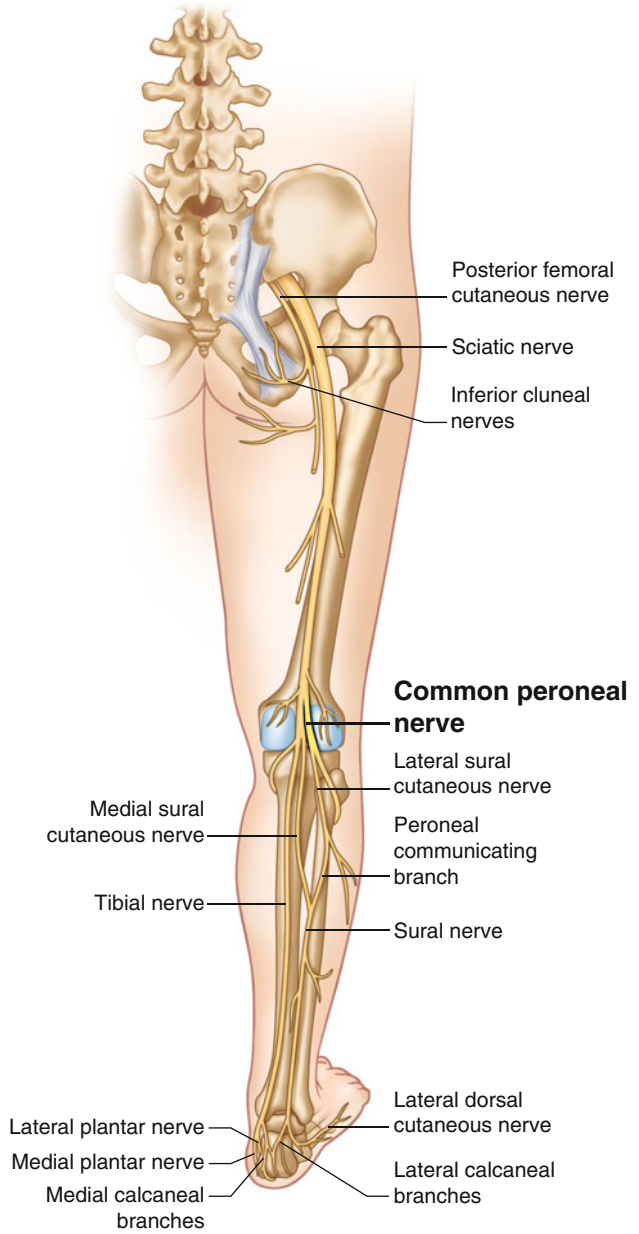

Fig. 69.3 Anatomy of the lower extremity (Image by Springer)

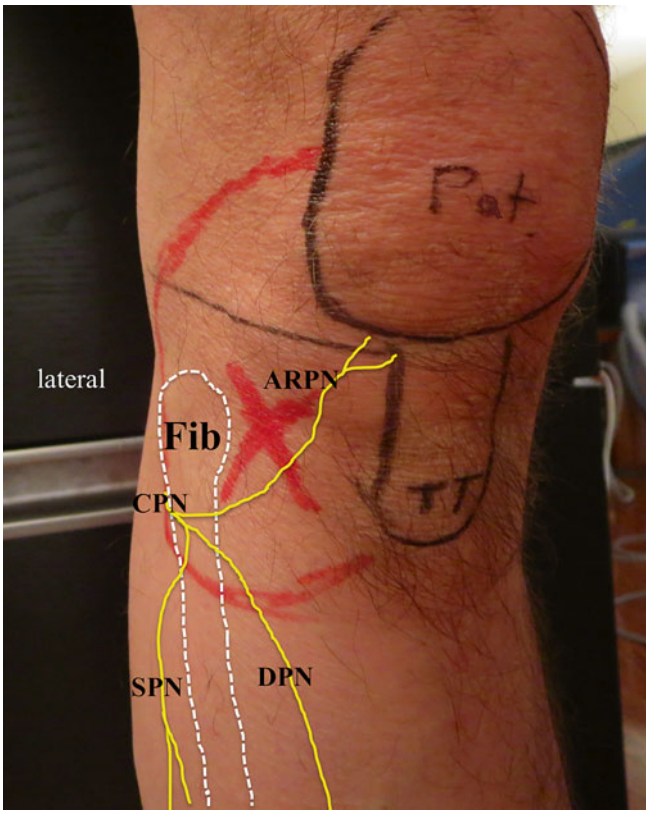

Fig. 69.4 Pain pattern of a patient with presumed anterior recurrent peroneal nerve entrapment, showing the branches of the common peroneal nerve. *Pat* patella, *TT* tibial tubercle, *Fib* fibula, *CPN* common peroneal nerve, *SPN* superficial peroneal nerve, *DPN* deep peroneal nerve, *ARPN* anterior recurrent peroneal nerve, *X* site of tenderness (Image courtesy of Peter Mouldey, MD; modified by Andrea Trescot, MD)

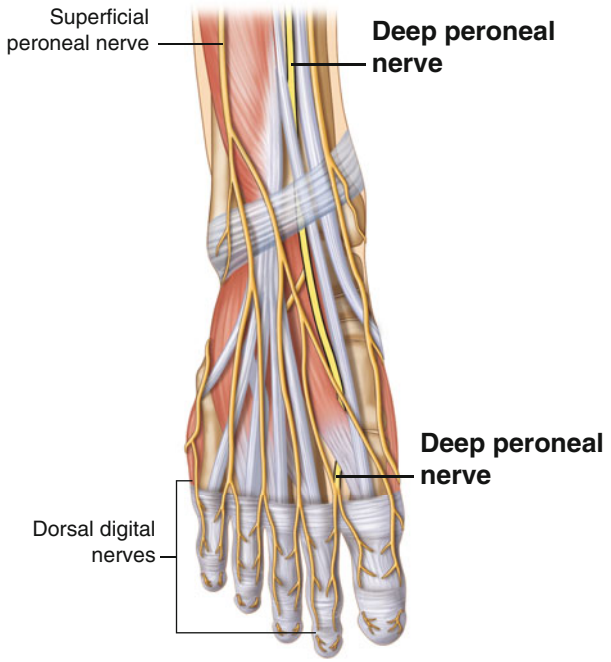

Fig. 69.5 Anatomy of the anterior ankle (Image courtesy of Springer)

innervate the *lateral tarsometatarsal joints* (TMT) and lesser digits [14].

Entrapment

The DPN may become entrapped anywhere along its course, but certain sites are particularly likely, often where it is lying on the periosteum, only thinly covered by subcutaneous tissue [1, 12]. Potential entrapment sites are discussed from distal to proximal, to simplify the accumulation of symptoms and physical findings as additional portions of

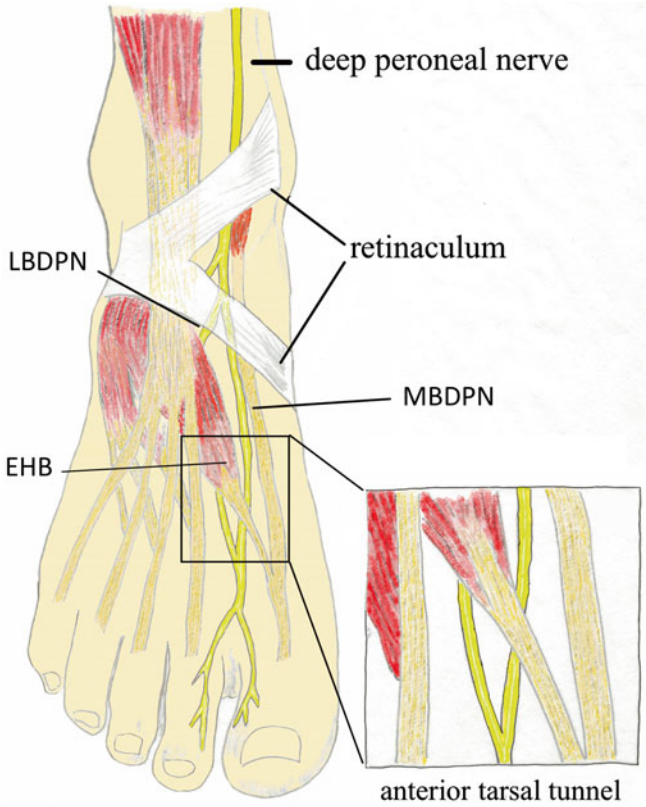

Fig. 69.6 Anatomy of the deep peroneal nerve. *LBDPN* lateral branch of the deep peroneal nerve, *MBDPN* medial branch of the deep peroneal nerve, *EHB* extensor hallucis brevis (Image courtesy of Michael Brown, MD)

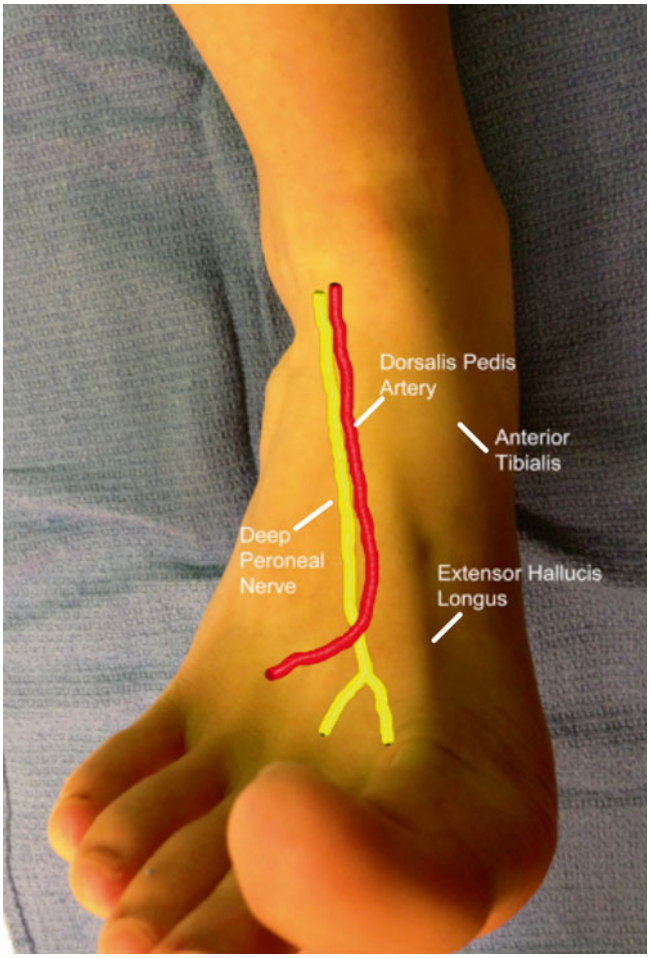

Fig. 69.7 Relationship between the dorsalis pedis artery and the deep peroneal nerve (Image courtesy of Michael Brown, MD)

the DPN are involved. A cadaver study has shown that plantar flexion of the foot with simultaneous dorsiflexion of the toes (as if wearing high-heeled shoes) maximally stretches the DPN over the talonavicular joint. Any process that constrains the motion of the nerve can magnify its dysfunction [10].

The MBDPN may be compressed at the dorsum of the foot [1, 12, 22, 23]. At the level of the first and second TMT joints, it is found in a tight tunnel beneath the EHB tendon and the deep fascia and becomes entrapped as the tendon crosses the nerve (Fig. 69.6). Contusions, soft-tissue swelling, tight footwear or a high longitudinal arch, and osteophytes can cause acute or chronic nerve compression at this site. Sports-related injuries to the DPN have been described in skiers due to tightly fitting boots [24], dancers in pointe position, as well as in soccer players due to multiple blows from the ball on the top of the foot.

Patients with MBDPN entrapment complain of dull pain in the great toe, often worse after activity. There may also be pain at ball of the foot, poorly localized and occasionally burning in nature [8]. Patients tend to walk so as to minimize weight bearing and pressure on the anterior foot. Digital pressure in the area between the first and second metatarsal heads, especially with simultaneous pressure on the metatarsal heads, commonly increases the pain (Fig. 69.9).

LBDPN function can be compromised at two sites not far from each other. Early in its course, the LBDPN crosses the lateral part of the talonavicular joint. When the foot is plantar flexed and inverted, a position that decreases nerve mobility, the prominent head of the talus can impinge on the nerve. It then passes deep to the extensor digitorum brevis (EDB) fascia, which forms a band that is closely applied to the periosteum. This band is also tightened with foot flexion [14].

Symptoms of LBDPN entrapment are more proximal, lateral, and diffuse than those described above. It is usually depicted as a deep ache in the dorsolateral foot that radiates to the TMT joints [14].

At the ankle (also called the *astragular joint* [1]), the DPN or its branches run beneath the inferior extensor retinaculum, accompanied by four tendons, an artery, and a vein. Entrapment at this location is often called the *anterior tarsal tunnel syndrome* (*ATT syndrome*) (Fig. 69.6), underlining its similarity to the *posterior tarsal tunnel*

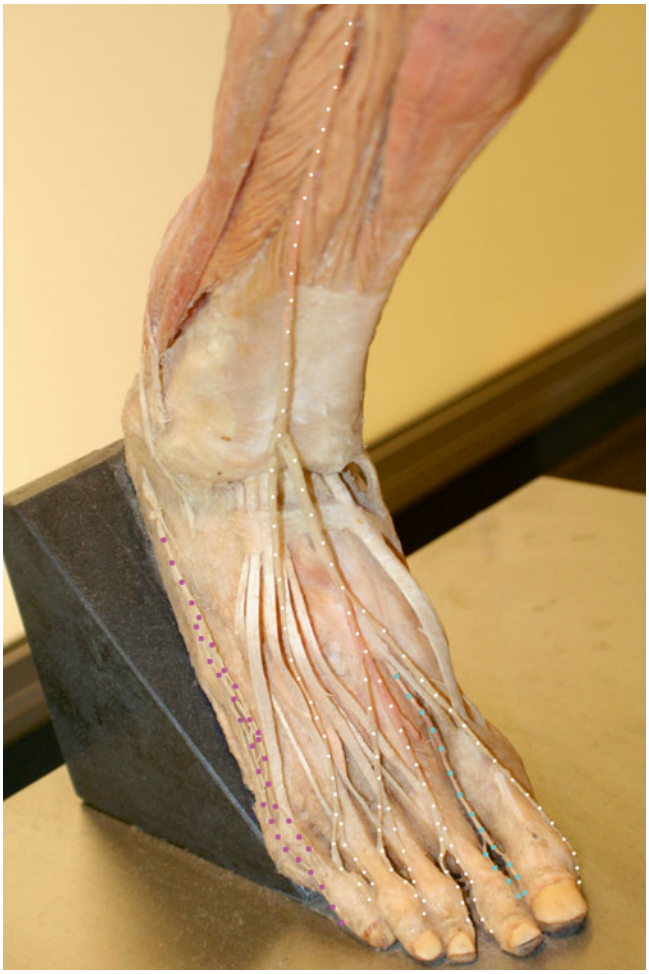

Fig. 69.8 Dorsal foot dissection, modified from an image from Bodies, The Exhibition, with permission. *White dots* superficial peroneal nerve, *green dots* deep peroneal nerve, *purple dots* sural nerve (Image courtesy of Andrea Trescot, MD)

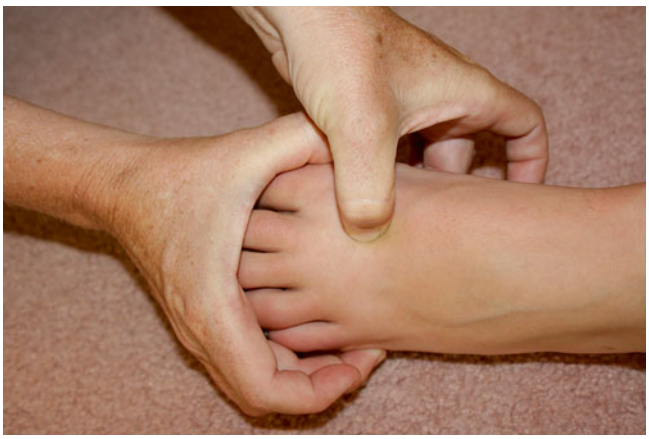

Fig. 69.9 Squeeze test to replicate deep peroneal nerve entrapment (Image courtesy of Andrea Trescot, MD)

Table 69.3 Differentiating the level of DPN entrapment

Level of DPN entrapment	Symptoms	Site of Tinel's sign
LBDPN [14]	Dorsolateral foot pain radiating to the lateral tarsometatarsal joints	Anterolateral talar head
MBDPN under the EHB [8]	Sensory symptoms in the first web space	Over the first and second metatarsocuneiform joint
ATT	Depends on the level of branching	At the lateral side of the EHL tendon, resulting in pain that often radiates to the first and second toes [9]
Anterior intermuscular septum [16]	Foot drop	

syndrome (Chap. 73), which results in entrapment of the *posterior tibial nerve* [10]. When the DPN travels under the retinaculum along the ankle, it makes a nearly 90° angle to enter the ATT. When the entrapment occurs here, both sensory and motor branches can be affected [13]. The nerve can be compressed by the extensor retinaculum, tendon pathology, talonavicular dysfunction, and bony prominences in the ankle or hindfoot. Positional strain, such as prolonged plantar flexion from high heels or mechanical compression by a shoe or cast, may also contribute.

Patients with entrapment at this level commonly report a sense of tightness in the ankle, as well as burning, aching, and sometimes weakness of the ankle or foot. Pain along the dorsum of the foot can extend proximally into the ankle and leg, as well as distally to the first interspace [13].

Most proximally, the passages through the *anterior intermuscular septum* may become narrowed, particularly if there is an increase in the pressures in either the lateral or anterior compartment [16]. Since the first motor branch to the *tibialis anterior muscle* is closely intertwined with the septum, foot drop is the most likely result of DPN entrapment at this level (see Table 69.3).

Physical Exam

Entrapment of the deep peroneal nerve can be replicated and diagnosed by the "squeeze test" – compressing the metatarsal heads together while squeezing the deep peroneal nerve from above and below (Video 69.1) (Fig. 69.9). Percuss or

deeply palpate the anterolateral talar head, with the foot in plantar flexion and inversion. If the LBDPN is entrapped, pain will radiate to the lateral tarsometatarsal joints [14]. EDB weakness is likely with ATT syndrome or LBDPN compression. Have the patient dorsiflex the ankle and extend the toes while the examiner palpates the EDB [12]. Pain, as well as decreased touch, vibration, and two-point discrimination in the web space between the first two toes, is often seen with MBDPN dysfunction [8]. Comparison of these findings with the contralateral side will often clarify the situation. Physical findings frequently include bony enlargement of the midfoot, specifically at the first and second TMT and/or navicular-cuneiform joint.

Differential Diagnosis (Table 69.4)

The presence of an accessory DPN (a branch of the SPN) to the EDB in about 25 % of people is a source of potential additional diagnostic confusion. Those who have an accessory DPN will be able to dorsiflex their fourth and fifth toes, even in the presence of a complete DPN lesion, and EMG evoked from the accessory DPN of their EDB will be normal [8, 19].

Morton's neuromas (digital neuromas, intermetatarsal space neuromas) cause symptoms similar to those of MBDPN entrapment but are located between the second and third or between the third and fourth metatarsal bones [25].

Table 69.4 Differential diagnosis of anterolateral ankle or foot pain

	Potential distinguishing features
Superficial peroneal nerve entrapment (SPN) [12]	Lateral calf pain radiating to the lateral foot and toes, sparing the first web space (Fig. 69.9); Tinel's sign over calf, where the SPN pierces the fascia (Chap. 68)
Intermetatarsal (Morton's) neuroma [25]	More commonly second or third intermetatarsal space (Chap. 70)
L5 radiculopathy [9]	Dermatomal pattern of sensory changes and decreased function of muscles not innervated by the DPN; may coexist with DPN entrapment [26]
Arterial vascular insufficiency [2]	Decreased dorsalis pedis pulse
Tendonitis	Tenderness at the tendon attachments; pain on resisted movement
Hallux valgus (bunion) pain	Tenderness and swelling over the first MTP joint
Compartment syndrome [27]	Decreased pain with exercise; increased compartment pressures

Diagnostic Tests (Table 69.5)

Ultrasonography can be useful to observe space-occupying lesions, varicosities, or tendinopathy impinging on the nerve. MRI can provide additional information about the nerves (Fig. 69.10) and the adjacent soft tissues (Fig. 69.11), while X-ray and CT are more useful in evaluating bony structures [29]. Electrodiagnostic studies should be performed at the EDB and, if no response is elicited there, then at the TA muscle [30], which is the most likely muscle to show abnormalities by needle EMG. As a note of caution, the SPN conduction studies may be normal in a CPN neuropathy despite severe abnormalities in the DPN, since there seems to be a particular vulnerability of the DPN to compression or stretch [30].

Identification and Treatment of Contributing Factors

Entrapment in the absence of trauma or mass lesion often occurs when the DPN or its branches are stretched over the midfoot and subjected to repeated dorsiflexion, plantar flexion, and mechanical irritation. Degenerative changes of the talonavicular, naviculocuneiform, and TMT joints increase the likelihood of entrapment. Removal of recur-

Table 69.5 Diagnostic tests for deep peroneal nerve entrapment

	Potential distinguishing features
Physical exam	Site of maximum tenderness and Tinel's sign
Provocative tests	Inversion and plantar flexion of the foot [12, 14] with toe dorsiflexion will increase pain [10]
Diagnostic injection	Pain relief after injection of a low volume of local anesthetic can help determine the level of nerve injury [1, 8, 11, 12]
Ultrasound	Observe space-occupying lesions, varicosities, or tendinopathy impinging on the DPN
MRI	The DPN and its branches can be seen throughout most of its length, particularly in patients with more fatty tissue [28]; identify adjacent soft-tissue pathology [29]
CT	Can identify adjacent bone pathology [29]
X-ray	Standing radiographs
Electrodiagnostic studies	EMG may show changes in EDB function; however, the variability of branching sites (into the MBDPN and LBDPN) and the presence of a normal EMG in a patient with demonstrated LBDPN pathology make this test less than ideal [8, 14]

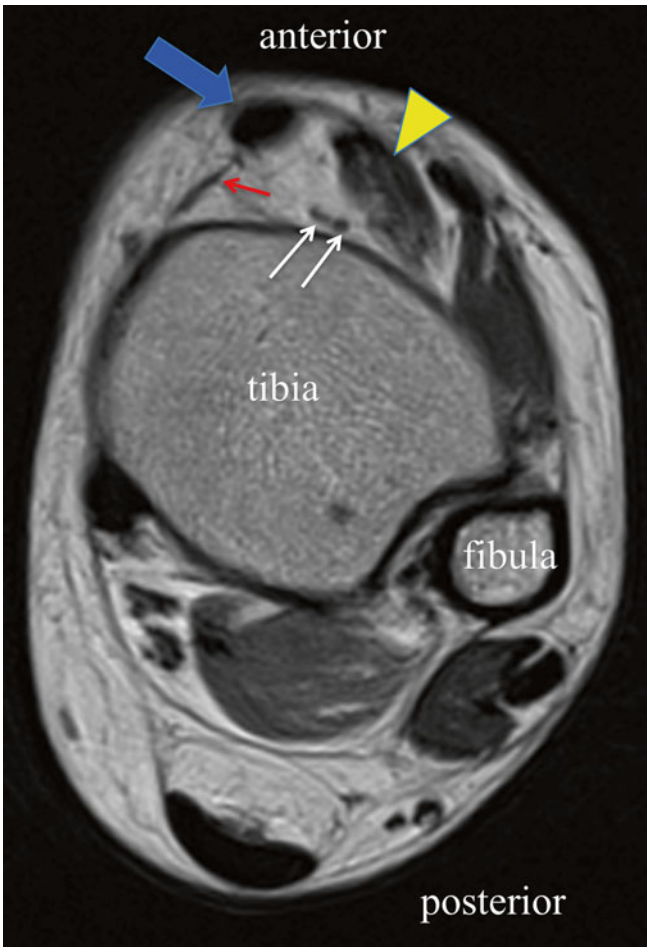

Fig. 69.10 MRI T1 Proton-density axial view showing anterior ankle structure. *Blue arrow* extensor hallucis longus muscle, *yellow arrowhead* anterior tibial tendon, *red arrow* inferior extensor retinaculum, *white arrows* dorsalis pedis artery, as well as lateral and medial distal deep peroneal nerve (Image courtesy of Andrea Trescot, MD)

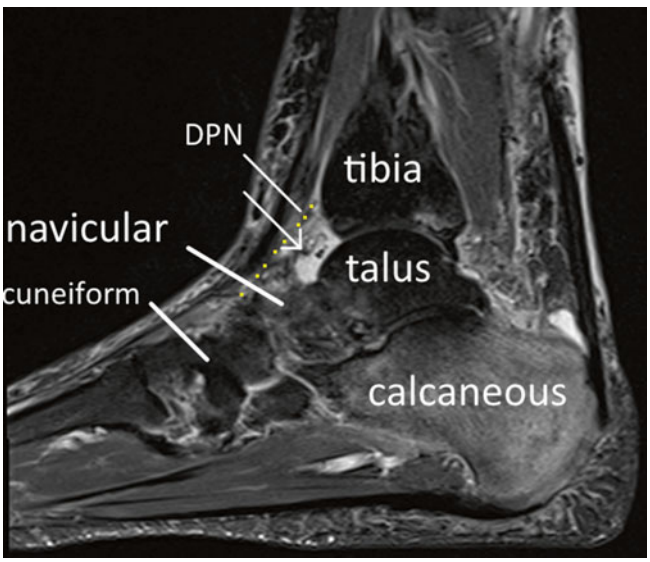

Fig. 69.11 MRI T2 sagittal view of ankle; *arrow* shows anterior ganglion cyst (Image courtesy of Andrea Trescot, MD)

ring compression by shoes, laces, or straps across the dorsum of the foot, as well as relieving postural strain by avoiding high-heeled shoes, may help alleviate symptoms. Avoiding kicking sports and the addition of shoe wedges to normalize foot position may decrease nerve stretch [10, 12]. DPN injury and entrapment may occur following spiral fibular fractures.

Injection Techniques

Landmark-Guided Technique

At the ankle The DPN is immediately lateral to the tendon of the EHL, between the EHL and EDL. Inject subcutaneous local anesthetic between the EHL tendon and the dorsalis pedis pulse, 2–3 cm distal to the joint line (Fig. 69.12). Advance a 27-gauge, 1.5 inch needle just lateral to the artery and inject 1.5–2 mL of local anesthetic followed by about 20 mg of a deposteroid. Other, more constant landmarks include the tendons of tibialis anterior and EHL at the level of the proximal edge of the medial malleolus or the indentation between EHL and EDL [18, 31].

At the toe (MBDPN) Palpate the most proximal part of the first interspace at the level of the first TMT joint. This landmark is easy to feel and can be accentuated by asking the patient to dorsiflex the foot or toes. Raise a small wheal at this location and direct the remainder of the anesthetic proximally along the line of the dorsalis pedis pulse (Video 69.2) (Fig. 69.13).

LBDPN at the foot Inject at the point of maximum tenderness, usually just distal to the anterolateral part of the head of the talus [14].

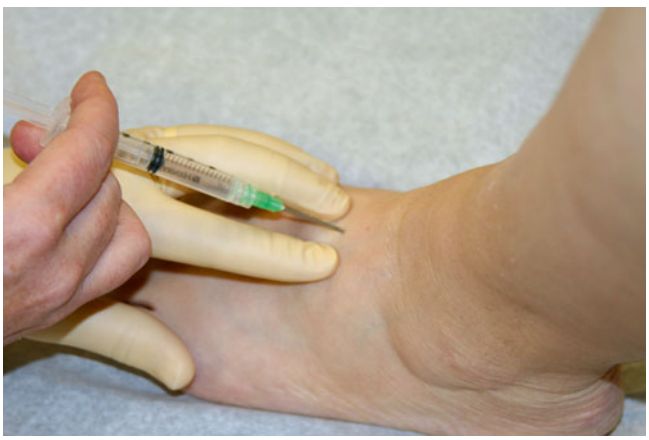

Fig. 69.12 Landmark-guided injection of the deep peroneal nerve at the ankle (Image courtesy of Andrea Trescot, MD)

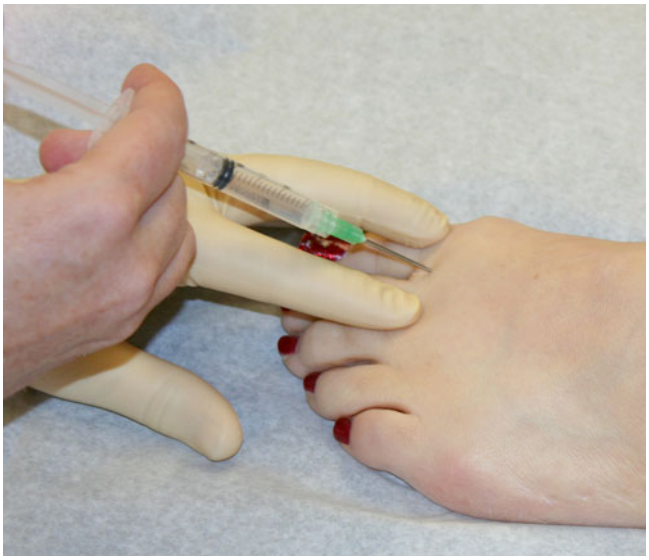

Fig. 69.13 Landmark-guided injection of the deep peroneal nerve at the first interspace (Image courtesy of Andrea Trescot, MD)

Ultrasound-Guided (US) Technique

Ultrasound can be useful to identify the anatomy in this region. Starting at the fibular head, the common peroneal nerve can be seen to split into the SPN and DPN at the mid-calf (Fig. 69.14), and the DPN can be traced onto the foot (Fig. 69.15) at the dome of the ankle (the tibial plafond). US-guided DPN injections can be done either in plane (Fig. 69.16) or out of plane (Fig. 69.17) as shown. These injections are superficial, so it is helpful to provide skin anesthesia first with a 30-gauge, 0.5 inch needle and 1 % buffered lidocaine, then insert the larger needle (27 gauge, 1.5 inch) slightly under the skin, place the ultrasound transducer over the needle, and then advance. Visualization of the needle will be excellent because the nerve is superficial.

At the ankle The DPN is immediately lateral to the tendon of the EHL, between the EHL and EDL. Identify the nerve at that location and then run the transducer cephalad and caudad from that site to confirm that you are visualizing the deep peroneal nerve. The patient is supine with the foot supported and ankle dorsiflexed. The anterior tibial artery, often accompanied by two veins, is an easily palpable starting point. Place the transducer (7.5–15 MHz) transversely over the anterior tibia, just proximal to the ankle joint, and locate the artery [32]. The DPN was lateral to the artery in 61 % of subjects at this level [17]. Inject 1 cc of local anesthetic with or without corticosteroid. In one series, using ultrasound did not improve the block [18].

MBDPN at the foot More distally, the relationship between the DPN and the artery changes and is less constant. It was found to be lateral to the artery in 36 % of the feet studied and

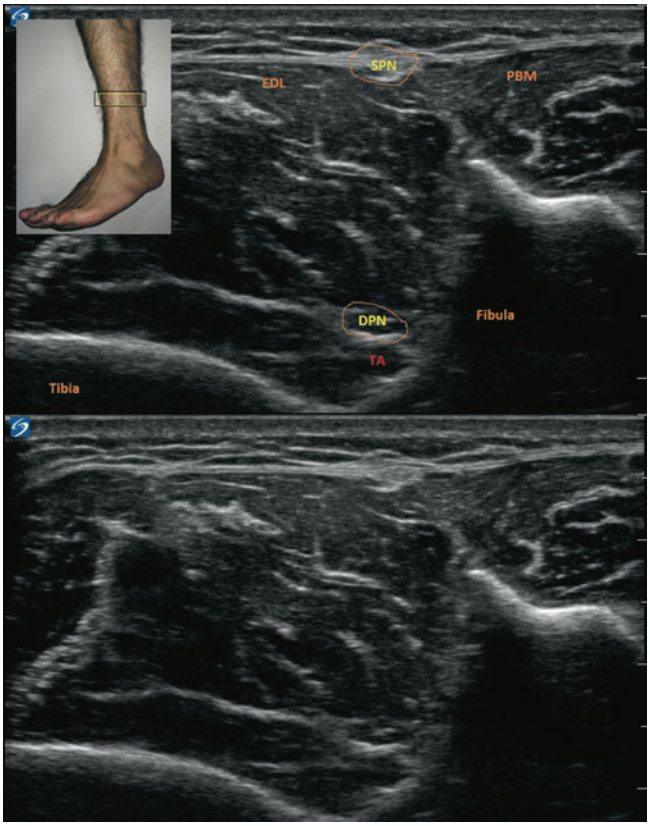

Fig. 69.14 Ultrasound evaluation of the superficial and deep peroneal nerves at the mid-calf. *TA* tibial artery, *DPN* deep peroneal nerve, *SPN* superficial peroneal nerve, *EDL* extensor digitorum longus muscle, *PBM* peroneus brevis muscle (Image courtesy of Agnes Stogicza, MD)

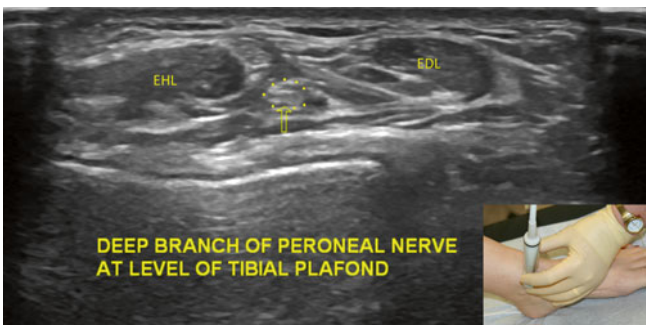

Fig. 69.15 Ultrasound image of the deep peroneal nerve at the tibial plafond. *EHL* extensor hallucis longus, *EDL* extensor digitorum longus (Image courtesy of Michael Brown, MD)

medial to the artery in 25 % [17] (Fig. 69.18). The injection can be carried out from the side using an in-plane approach or over the dorsum of the foot in an out-of-plane view.

MBDPN at the toe At its most distal aspect, the MBDPN travels between the first and second metatarsal heads. The probe is placed transversely across the interspace, and the nerve is approached out of plane; small volumes (no greater than 1 cc) of local anesthetic and deposteroid are injected.

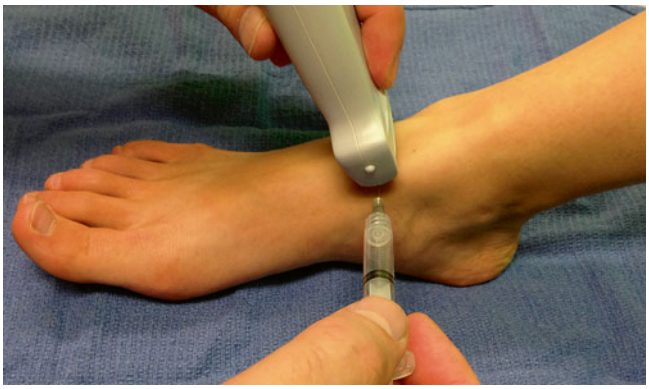

Fig. 69.16 In-plane ultrasound technique for deep peroneal nerve injection (Image courtesy of Michael Brown, MD)

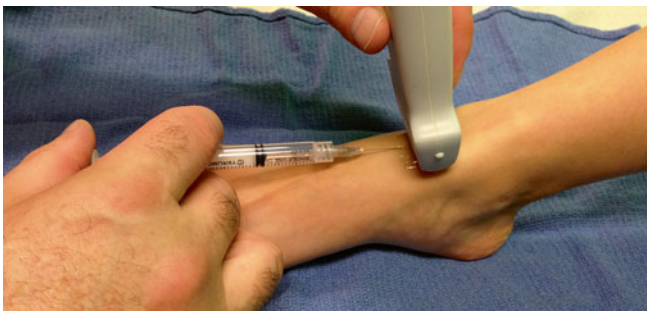

Fig. 69.17 Out-of-plane ultrasound technique for deep peroneal nerve injection at plafond (Image courtesy of Michael Brown, MD)

Fluoroscopy-Guided Technique

These blocks are superficial; there is no benefit from the added radiation exposure to the patient and clinician.

Neurolytic Techniques

Cryoneuroablation

A successful diagnostic injection must precede cryoneuroablation. Because the DPN can be easily seen with ultrasound, a cryoprobe can be placed quite precisely (Fig. 69.19). The appropriate introducer (12 gauge for nerves with no previous history of trauma or surgery and 14 gauge for postoperative neuromas) is eased through the skin parallel to the nerve (dorsal or plantar) and directed toward the apex of the metatarsal bones. Stimulation should replicate the patient's usual pattern of pain.

Cryoneuroablation of neuromas that have not been surgically treated is easier because there is room for the large (2.0 mm) probe, which increases the success rate. Unfortunately, many patients are only sent for evaluation after surgical treatment has failed. The cryoprobe must be placed proximal to the surgical trauma to be effective, and

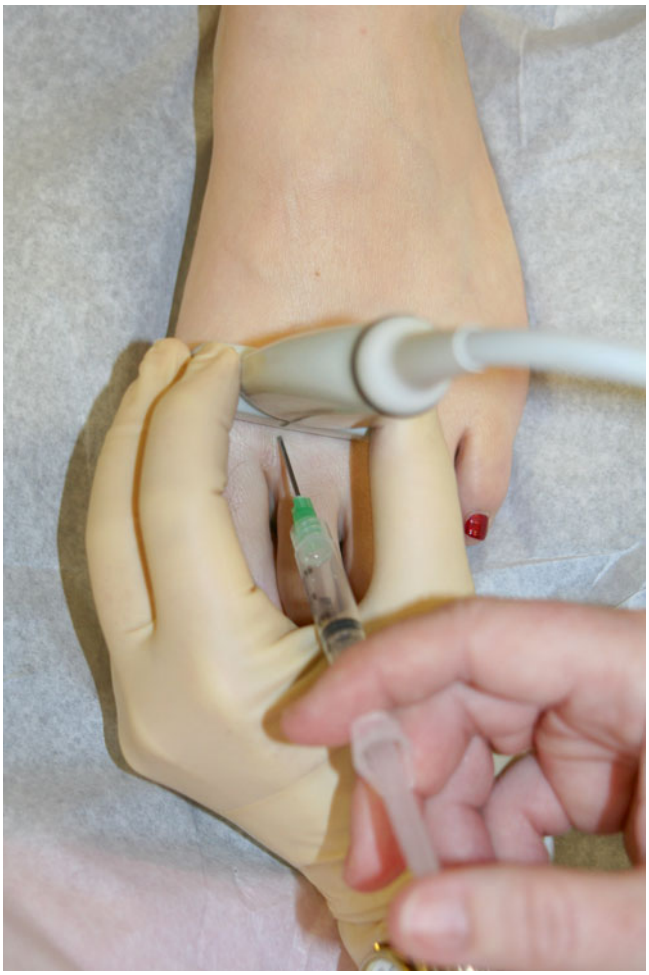

Fig. 69.18 Deep peroneal nerve injection at the first metatarsal interspace (Image courtesy of Andrea Trescot, MD)

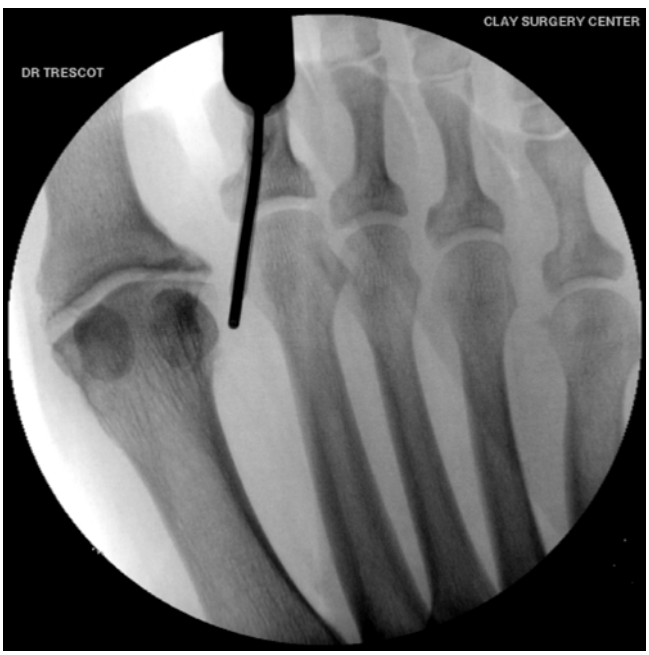

Fig. 69.19 Cryoneuroablation of the deep peroneal nerve (Image courtesy of Andrea Trescot, MD)

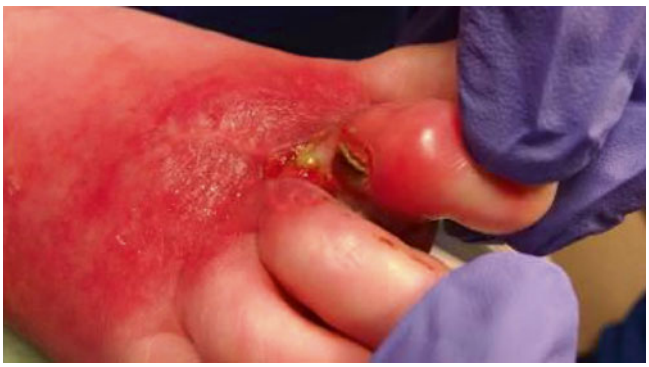

Fig. 69.20 Right foot necrosis and cellulitis 3 days after alcohol neurolysis (Image courtesy of Emily Walker, MD and Christopher Burnett, MD)

there is not much room in the proximal metatarsal triangle apex, necessitating the use of the smaller (1.4 mm) probe.

Due to the superficial course of the DPN, care must be taken to avoid skin trauma, and digital ischemia is a concern when epinephrine-containing solutions are used. Resting the non-introducing hand at the plantar surface will detect if the probe is too close to the skin of the sole of the foot. Cryoneurolysis of an intermetatarsal space neuroma has been reported to provide at least 6 months of relief [33].

Phenol and Alcohol

Both have a place in clinical practice, as they provide good relief. However, there is a possibility of local tissue damage, necrosis, and ulceration (Fig. 69.20).

Surgical Technique

A preoperative diagnostic block is the best way to confirm the level of entrapment. When injections fail, patients need complete decompression with removal of offending bony or soft tissues [9]. In one study, the release of MBDPN entrapment had excellent results in 60 % of the patients studied and good in 20 %; 20 % of the patients were not improved [8]. Unfortunately, postsurgical scarring may recreate entrapment, so early mobilization is important for good recovery [8].

Complications

Neurologic injury following peripheral nerve block can be attributed to needle trauma, neuronal ischemia, or patient positioning. Because the skin can be thin (especially at the dorsum of the foot), there is risk of skin atrophy (from steroids) or "frostbite" (from cryoneuroablation).

Summary

DPN entrapment is a very under-recognized cause of foot and toe pain, triggered by sports and tight shoes. By using the diagnostic tools of physical exam, diagnostic injections, and neurodiagnostics, the appropriate diagnosis of deep peroneal nerve pathology can be made and appropriate treatment initiated.

References

1. Kopell HP, Thompson WA. Peripheral Entrapment Neuropathies. Baltimore: Williams and Wilkins; 1976.
2. Marinacci AA. Medial and anterior tarsal tunnel syndrome. Electromyography. 1968;8(2):123–34.
3. Krause KH, Witt T, Ross A. The anterior tarsal tunnel syndrome. J Neurol. 1977;217(1):67–74.
4. Whitmore I. Terminologia anatomica: new terminology for the new anatomist. Anat Rec. 1999;257(2):50–3.
5. Rab M, Ebmer J, Dellon AL. Innervation of the sinus tarsi and implications for treating anterolateral ankle pain. Ann Plast Surg. 2001;47(5):500–4.
6. Khin-Myo-Hla IT, Sakane M, Hayashi K. Effect of anesthesia of the sinus tarsi on peroneal reaction time in patients with functional instability of the ankle. Foot Ankle Int. 1999;20(9):554–9.
7. Dellon AL, Barrett SL. Sinus tarsi denervation: clinical results. J Am Podiatr Med Assoc. 2005;95(2):108–13.
8. Dellon AL. Deep peroneal nerve entrapment on the dorsum of the foot. Foot Ankle. 1990;11(2):73–80.
9. Liu Z, Zhou J, Zhao L. Anterior tarsal tunnel syndrome. J Bone Joint Surg (Br). 1991;73(3):470–3.
10. Borges LF, Hallett M, Selkoe DJ, Welch K. The anterior tarsal tunnel syndrome. Report of two cases. J Neurosurg. 1981;54(1):89–92.
11. Kennedy JG, Baxter DE. Nerve disorders in dancers. Clin Sports Med. 2008;27(2):329–34.
12. Adelman KA, Wilson G, Wolf JA. Anterior tarsal tunnel syndrome. J Foot Surg. 1988;27(4):299–302.
13. Gessini L, Jandolo B, Pietrangeli A. The anterior tarsal syndrome. Report of four cases. J Bone Joint Surg Am. 1984;66(5):786–7.
14. Kennedy JG, Brunner JB, Bohne WH, Hodgkins CW, Baxter DB. Clinical importance of the lateral branch of the deep peroneal nerve. Clin Orthop Relat Res. 2007;459:222–8.
15. Deutsch A, Wyzykowski RJ, Victoroff BN. Evaluation of the anatomy of the common peroneal nerve. Defining nerve-at-risk in arthroscopically assisted lateral meniscus repair. Am J Sports Med. 1999;27(1):10–5.
16. Aigner F, Longato S, Gardetto A, Deibl M, Fritsch H, Piza-Katzer H. Anatomic survey of the common fibular nerve and its branching pattern with regard to the intermuscular septa of the leg. Clin Anat. 2004;17(6):503–12.
17. Ikiz ZA, Uçerler H, Uygur M. The clinical importance of the relationship between the deep peroneal nerve and the dorsalis pedis artery on the dorsum of the foot. Plast Reconstr Surg. 2007; 120(3):690–6.
18. Antonakakis JG, Scalzo DC, Jorgenson AS, Figg KK, Ting P, Zuo Z, et al. Ultrasound does not improve the success rate of a deep peroneal nerve block at the ankle. Reg Anesth Pain Med. 2010;35(2):217–21.
19. Gutmann L. Atypical deep peroneal neuropathy in presence of accessory deep peroneal nerve. J Neurol Neurosurg Psychiatry. 1970;33(4):453–6.
20. Rab M, Ebmer, Dellon AL. Anatomic variability of the ilioinguinal and genitofemoral nerve: implications for the treatment of groin pain. Plast Reconstr Surg. 2001;108(6):1618–23.

21. Lawrence SJ, Botte MJ. The deep peroneal nerve in the foot and ankle: an anatomic study. Foot Ankle Int. 1995;16(11):724–8.
22. Kanbe K, Kubota H, Shirakura K, Hasegawa A, Udagawa E. Entrapment neuropathy of the deep peroneal nerve associated with the extensor hallucis brevis. J Foot Ankle Surg. 1995;34(6):560–2.
23. Parker RG. Dorsal foot pain due to compression of the deep peroneal nerve by exostosis of the metatarsocuneiform joint. J Am Podiatr Med Assoc. 2005;95(5):455–8.
24. Lindenbaum BL. Ski boot compression syndrome. Clin Orthop Relat Res. 1979;140:109–10.
25. Franson J, Baravarian B. Intermetatarsal compression neuritis. Clin Podiatr Med Surg. 2006;23(3):569–78.
26. Upton AR, McComas AJ. The double crush in nerve entrapment syndromes. Lancet. 1973;2(7825):359–62.
27. Englund J. Chronic compartment syndrome: tips on recognizing and treating. J Fam Pract. 2005;54(11):955–60.
28. Donovan A, Rosenberg ZS, Cavalcanti CF. MR imaging of entrapment neuropathies of the lower extremity. Part 2. The knee, leg, ankle, and foot. Radiographics. 2010;30(4):1001–19.
29. Blitz NM, Amrami KK, Spinner RJ. Magnetic resonance imaging of a deep peroneal intraneural ganglion cyst originating from the second metatarsophalangeal joint: a pattern of propagation supporting the unified articular (synovial) theory for the formation of intraneural ganglia. J Foot Ankle Surg. 2009;48(1):80–4.
30. Marciniak C. Fibular (peroneal) neuropathy: electrodiagnostic features and clinical correlates. Phys Med Rehabil Clin N Am. 2013;24(1):121–37.
31. Schabort D, Boon JM, Becker PJ, Meiring JH. Easily identifiable bony landmarks as an aid in targeted regional ankle blockade. Clin Anat. 2005;18(7):518–26.
32. Sites BD, Antonakakis JG. Ultrasound guidance in regional anesthesia: state of the art review through challenging clinical scenarios. Local Reg Anesth. 2009;2:1–14.
33. Hodor L, Barkal K, Hatch-Fox LD. Cryogenic denervation of the intermetatarsal space neuroma. J Foot Ankle Surg. 1997;36(4):311–4.

Interdigital Nerve Entrapment

70

Beth S. Pearce, Michael N. Brown,
and Thais Khouri Vanetti

Introduction

Interdigital nerve (IDN) entrapment is a painful entrapment syndrome of the forefoot. The condition includes a litany of painful symptoms most often described by pain and radiating numbness and burning to the toes. Commonly termed *Morton's neuroma* [1], it was established as a disease entity in the mid-1800s [2, 3]. Other names include *intermetatarsal compression neuritis* [4] and *Joplin's neuritis* [1]. It may coexist with, or be secondary to, other pathology in up to 80 % of cases [5]. This condition is not a true neuroma or tumor as the name implies. The pathology is more correctly understood as a compression of the *common digital nerve*. It is most often found within the third intermetatarsal space, but it can occur in any of the adjacent intermetatarsal spaces. It may present either unilaterally or bilaterally and can affect more than one interspace concomitantly [6]. The *deep peroneal nerve* (see Chap. 69) is functionally a digital nerve, though it comes from the peroneal and not the plantar nerves (see Chaps. 74 and 75). The terminal branches of the plantar nerve(s) are subject to chronic impingement by the surrounding structures of the forefoot and resultant neuralgia [7] or can be affected by acute trauma. Symptoms are characteristic of other sensory nerve disorders and range from mild intermittent numbness to intolerable pain with ambulatory dysfunction.

Clinical Presentation (Table 70.1)

According to Schon and Baxter [8], the most common nerve entrapment seen in athletes is IDN entrapment. Patients will complain of forefoot pain, often of an acute onset, without noticed trauma (Fig. 70.1). There may be a history of new shoes or prolonged walking, and pain often is noticed when first putting weight on the foot in the morning. Older women who have worn high heels for many years are the most common sufferers, and women are ten times more likely to be affected than men, with an average age of onset at about 50 years [8]. The first few steps in the morning can be excruciating, though the pain may get better after a few minutes.

The most common clinical presentation is usually burning and sharp pain that radiates to the toes [1, 8]. Patients may describe a "thick sock" or "rolled up sock" or "rock" feeling on the ball of the foot even when barefoot. Patients may complain of a sharp, stabbing, burning feeling with painful numbness into the toes, sometimes spreading more proximally, which worsens as the day progresses. Patients may

Table 70.1 Occupation/exercise/trauma history relevant to interdigital nerve entrapment

Trauma, chronic or acute	Runners [8]
	Dancers [8, 9]
	High heels [8]
	Tight or pointed-toed shoes [4]
Entrapment	Deep transverse metatarsal ligament rigidity [4]
	Abductor hallucis muscle hypertrophy
	Cavus foot [4]
Neuropathy	Diabetes

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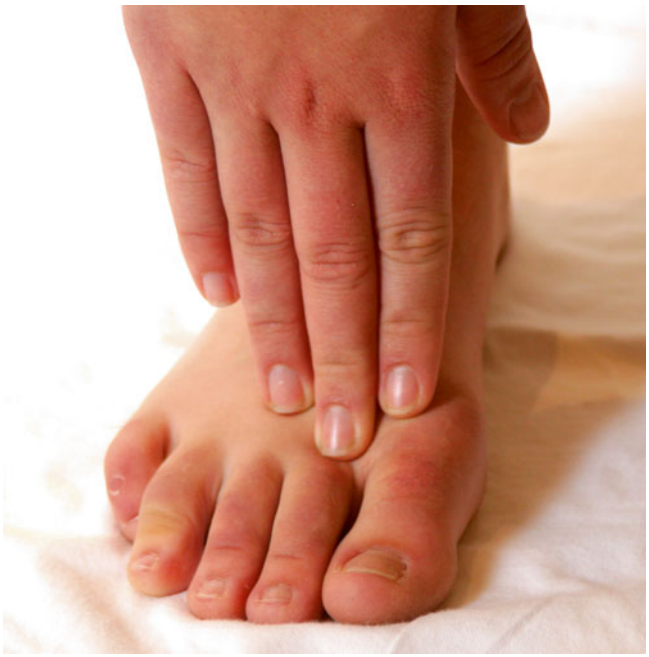

Fig. 70.1 Patient identification of digital nerve pain (Image courtesy of Andrea Trescot, MD)

Table 70.2 Digital nerve anatomy

Origin	L4–S4 ventral rami form the *sciatic nerve*
General route	The sciatic nerve bifurcates in the distal thigh into the common *peroneal* (*fibular*) *nerve* (see Chap. 67) and the *tibial nerve* (see Chap. 73)
	The tibial nerve divides into the lateral plantar nerve (LPN) (see Chap. 74) and medial plantar nerve (MPN) (see Chap. 75) in or near the tarsal tunnel (see Chap. 73), where it heads for the toes in the middle layer of the soft tissues on the sole of the foot
	Both plantar nerves fan out into multiple branches, most frequently with a branch from each reconnecting in the third intermetatarsal space
	Branches to each toe go under the deep transverse metatarsal ligament and become proper plantar digital nerves
Sensory distribution	To the skin over the plantar surfaces of the forefoot and 2nd to 5th toes
Motor innervation	The medial and lateral plantar nerves innervate all the intrinsic muscles on the plantar side of the foot
Anatomic variability	Minimal, if not postsurgical
Other relevant structures	Deep transverse metatarsal ligament

also note intermittent nighttime cramping in the foot that can awaken them from sleep.

The intermetatarsal web space between the third and fourth metatarsal bones is the classic location for the IDN entrapment, and one study showed that 81 of 100 cases were at this site [10], although IDN entrapment at the intermetatarsal space between the second and third metatarsal bones is also common [4].

The pain is aggravated by activity, and sometimes the physical exam is only positive after exercise. Rest, ice, elevation, shoe removal, foot massage, and NSAID's may reduce the pain, but symptoms will return with increased activity [1, 8]. Plain radiographs and bone scans will be negative for fracture.

Anatomy (Table 70.2)

The tibial nerve, after dividing from the sciatic nerve (L4, L5, S1–3), descends into the posterior compartment of the lower leg (Fig. 70.2). Behind the medial malleolus, in the *tarsal tunnel* (see Chap. 73), the *posterior tibial nerve* divides into its terminal three branches, the *medial calcaneal nerve* (see Chap. 77), and the *medial and lateral plantar nerves* (see Chaps. 74 and 75).

The medial planter nerve (MPN) and lateral plantar nerve (LPN) then divide into four *common plantar digital nerves*, numbered from medial to lateral. Branches 1–3 are from the MPN, while the fourth is from the LPN (Fig. 70.3). They pass deep to the *transverse intermetatarsal ligament* into a relatively small space between the metatarsal heads [11]. The third intermetatarsal space receives nerve contributions from both the MPN and the LPN, and since the resultant nerve is larger where the two nerves join, it may explain the more common entrapment at this site (Fig. 70.4) [4]. Repetitive mechanical stresses are associated with perineural fibrosis and neural edema, axonal degeneration, and local vascular proliferation, which is the most commonly accepted cause of Morton's neuroma; other possibilities include ischemia and compression of the nerve by the metatarsal head [7] or enlarged intermetatarsal bursas [12]. Franson and Baravarian suggested that the third metatarsal head is attached to the stable cuneiform bone, while the fourth metatarsal is attached to the more mobile cuboid, which might result in more movement (and therefore friction) at this level [4].

Entrapment

The common plantar nerves can become compressed against the distal edge of the deep transverse metatarsal ligament [8]. With cavus positioning, there is increased tension on the plantar fascia and the deep transverse intermetatarsal ligament, making it more rigid. This is particularly likely in situations when the toes are extended as they are when wearing high-heeled shoes or dancing on the toes [8]. Entrapment

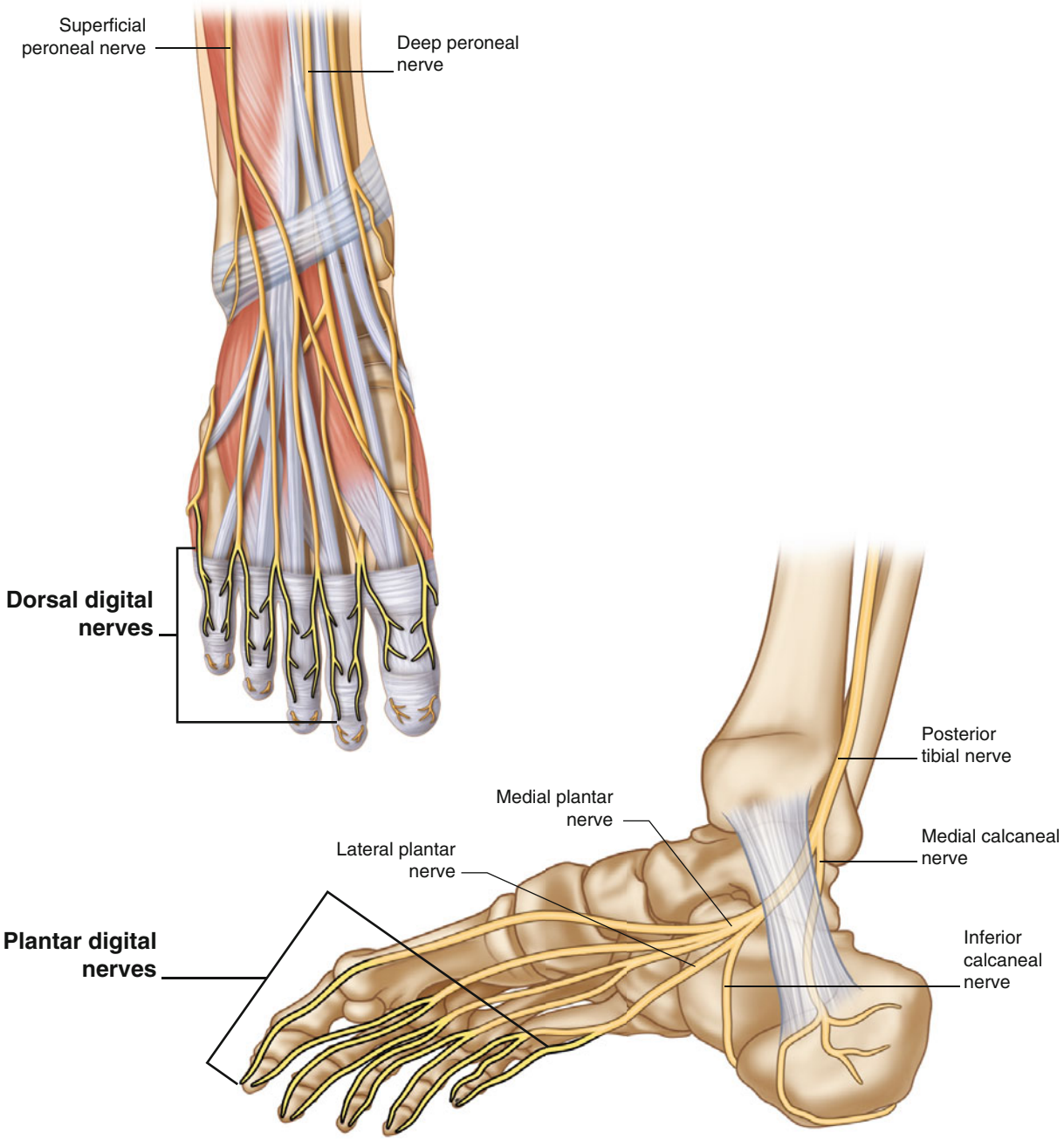

Fig. 70.2 Lower leg anatomy (Image courtesy of Springer)

may also be caused or exacerbated by abductor hallucis muscle hypertrophy [13].

Physical Exam

Visual inspections of the foot often do not provide any signs, but clinical presentation may include a splaying or divergence of the digits [14].

Occasionally, sensory disruption of the interspace can be found. Maximal tenderness is located at the interspace, but it must be differentiated from the adjacent metatarsal head/metatarsal phalangeal (MTP) joint. *Valleix's sign* (tenderness over the course of a nerve) and/or *Tinel's sign* (paresthesias with percussion of a nerve) may be demonstrated in some but not all cases. A *vertical squeeze test* (Fig. 70.5a, b) can be clinically useful to diagnose digital entrapment; adding a horizontal squeeze to mimic the "tight shoes" condition (Video 70.1) (Fig. 70.5c) may increase the sensitivity of the

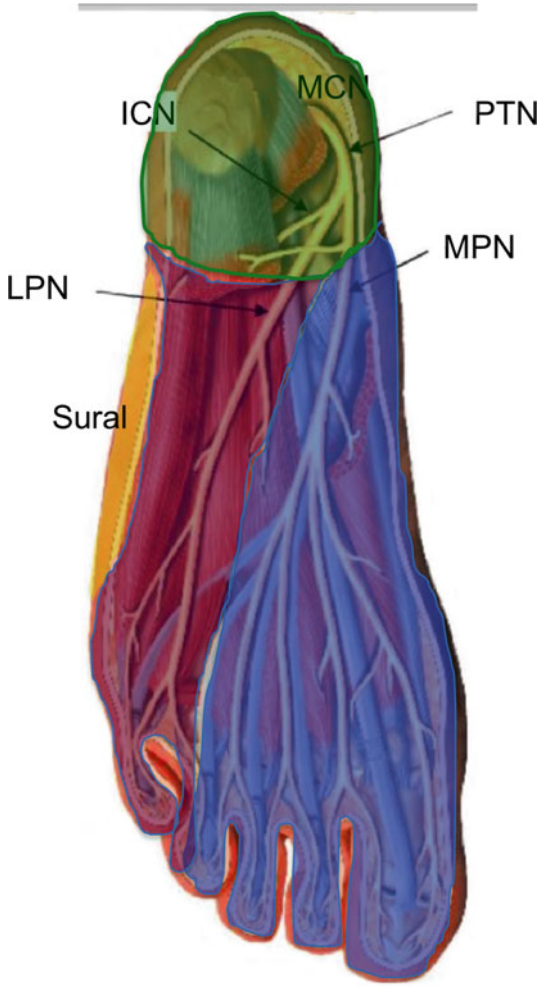

Fig. 70.3 Plantar foot anatomy. *MCN* medial calcaneal nerve, *PTN* posterior tibial nerve, *MPN* medial plantar nerve, *ICN* inferior calcaneal nerve, *LPN* lateral plantar nerve (Image courtesy of Michael Brown, MD)

exam. Place the index and thumb on the dorsal and plantar aspect of the painful intermetatarsal space; then, compress the forefoot with the opposite hand by squeezing together the metatarsal heads and feeling a *Mulder's click* – a clunk-like sound that can be palpated, likely due to subluxation of the neuroma between the metatarsal heads [15]. The *digital nerve stretch test*, when positive, is a highly sensitive indicator of intermetatarsal nerve entrapment [16, 17] (Fig. 70.6). Palpate all web spaces; a more proximal nerve lesion may be painful at multiple levels [8].

Dynamic examination of Morton's neuroma can also be performed with ultrasonography. An ultrasound linear transducer is placed transversely over the metatarsal heads, and the metatarsals are squeezed as if eliciting a "Mulder's click" while simultaneously visualizing a displacement of a focal hypoechoic neuroma in the plantar direction (toward the ultrasound transducer) (Fig. 70.7). Because of the horizontal compression on the sides of the interspace, a biconcave compression occurs causing the appearance of a shape similar to a ginkgo leaf and thus called the *ginkgo leaf sign* [18] (Fig. 70.8). The displacement of the Morton's neuroma can also be visualized by placing the ultrasound transducer on the plantar aspect of the foot, across the metatarsal heads, and applying pressure with the finger between the digits. A displacement of a hypoechoic neuroma will be seen on ultrasonography, similar to what one sees during metatarsal squeeze. The ultrasonography pictures of Fig. 70.7 demonstrate normal digital nerves on the bottom image and a displaced neuroma with metatarsal squeeze on the top image, demonstrating a "ginkgo leaf sign." It is helpful to evaluate numerous normal feet under ultrasound and elicit this sign to learn the difference in normal tissue displaced during this maneuver and what the neuroma looks like on exam.

Differential Diagnosis (Table 70.3)

Other than neuritis, alternative etiologies for a similar pattern of pain in the forefoot include stress fractures [19], neoplastic growths (i.e., rheumatoid nodules) [20], instability or overload of the adjacent MTP joint, *Freiberg's infraction* (osteochondrosis of the metatarsal heads), *metatarsalgia*, neuropathy, fibromyalgia, and other chronic pain syndromes [21]. As with all pain assessment, a careful clinical history and correlation of signs and symptoms is the key to an accurate diagnosis and a successful outcome.

Diagnostic Tests (Table 70.4)

Diagnostic testing for a Morton's neuroma can include plain radiography to exclude other pathology, ultrasound evaluation, and magnetic resonance imaging (MRI) [22]. Ultrasound also has been recommended for diagnostic evaluation of the interspaces [23]. A neuroma will appear as an ovoid, noncompressible mass with a hypoechoic signal [24, 25] (Fig. 70.7). This mass will be parallel to the long axis of the metatarsals and is best observed on the coronal view [26, 27]. When enlarged enough to be seen via MRI, the lesion can be found as a soft tissue mass with intermediate signal on both T1- and T2-weighted images between the metatarsal heads [22, 28] (Fig. 70.9).

Identification and Treatment of Contributing Factors

Faulty biomechanics can predispose entrapment neuralgias of the foot, such as uneven or functional leg length discrepancies or flatfoot, cavus, and hammertoe deformities. Gastrocnemius tightness results in a premature lifting of the

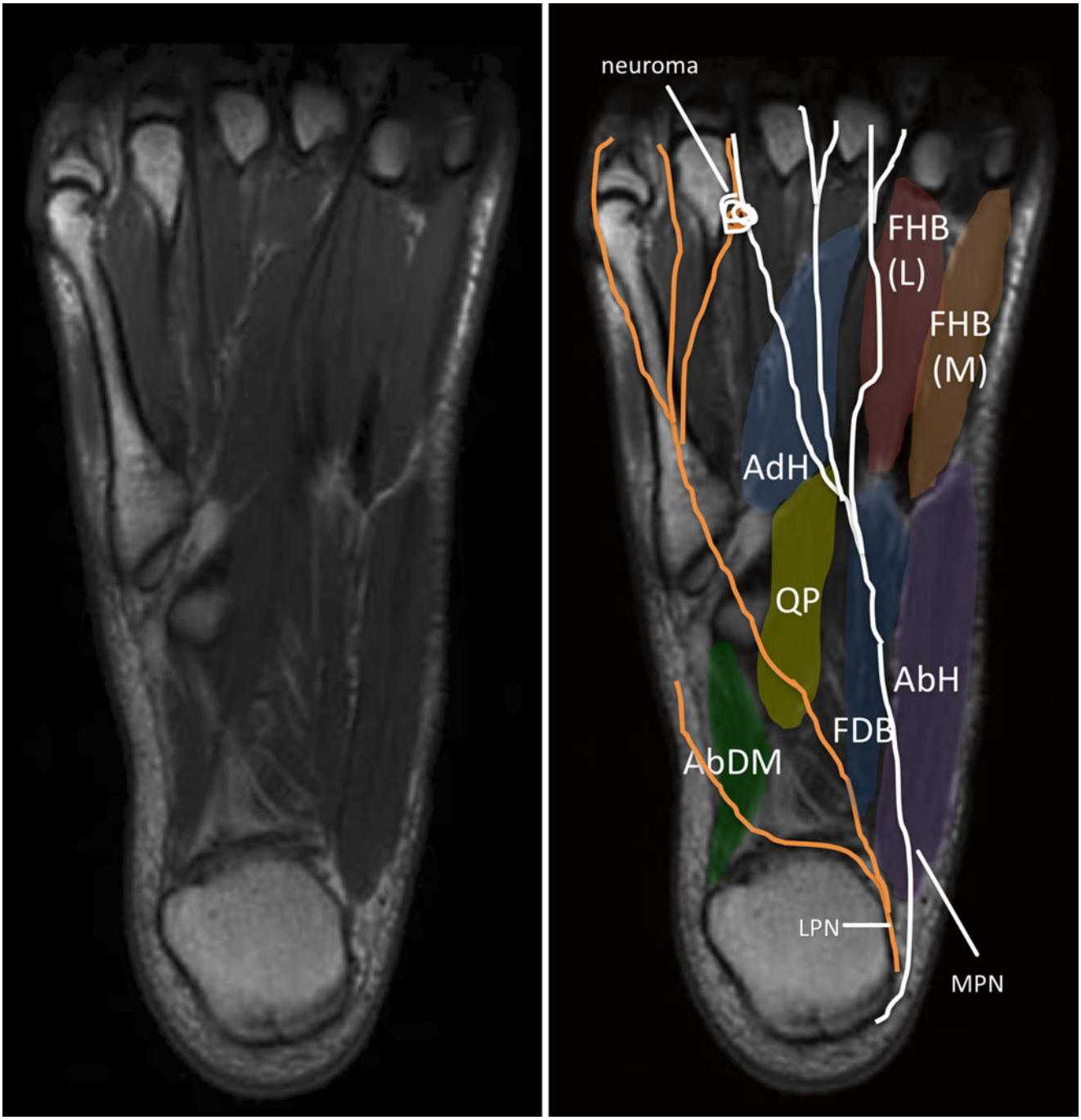

Fig. 70.4 MRI axial image showing foot muscles and nerves. *AdH* adductor hallucis muscle, *AbDM* abductor digiti minimi muscle, *AbH* abductor hallucis muscle, *FDB* flexor digitorum brevis muscle, *FHB* (*L*) flexor hallucis brevis (lateral head), *FHB* (*M*) flexor hallucis brevis (medial head), *QP* quadratus plantae, *MPN* medial plantar nerve, *LPN* lateral plantar nerve (Image courtesy of Andrea Trescot, MD)

heel during the gait cycle, which may amplify forces centered beneath the metatarsal heads during the propulsive period, traumatizing the nerves [29]. The pathology occurs when the metatarsal heads and/or the surrounding structures compress the interdigital nerve during toe-off, where the weight is transferred to the ball of the foot. High heels keeping the foot in chronic dorsiflexion against the supporting surface, narrow and tight-fitting shoes, overstress of the forefoot (e.g., such as with runners), foot deformities such as flatfoot or hallux valgus, and local trauma can increase the likelihood of local inflammation and fibrosis, leading to nerve damage from mechanical impingement.

Shoe changes to lower and wider varieties, shoe inserts such as foot orthoses or metatarsal pads elevating the

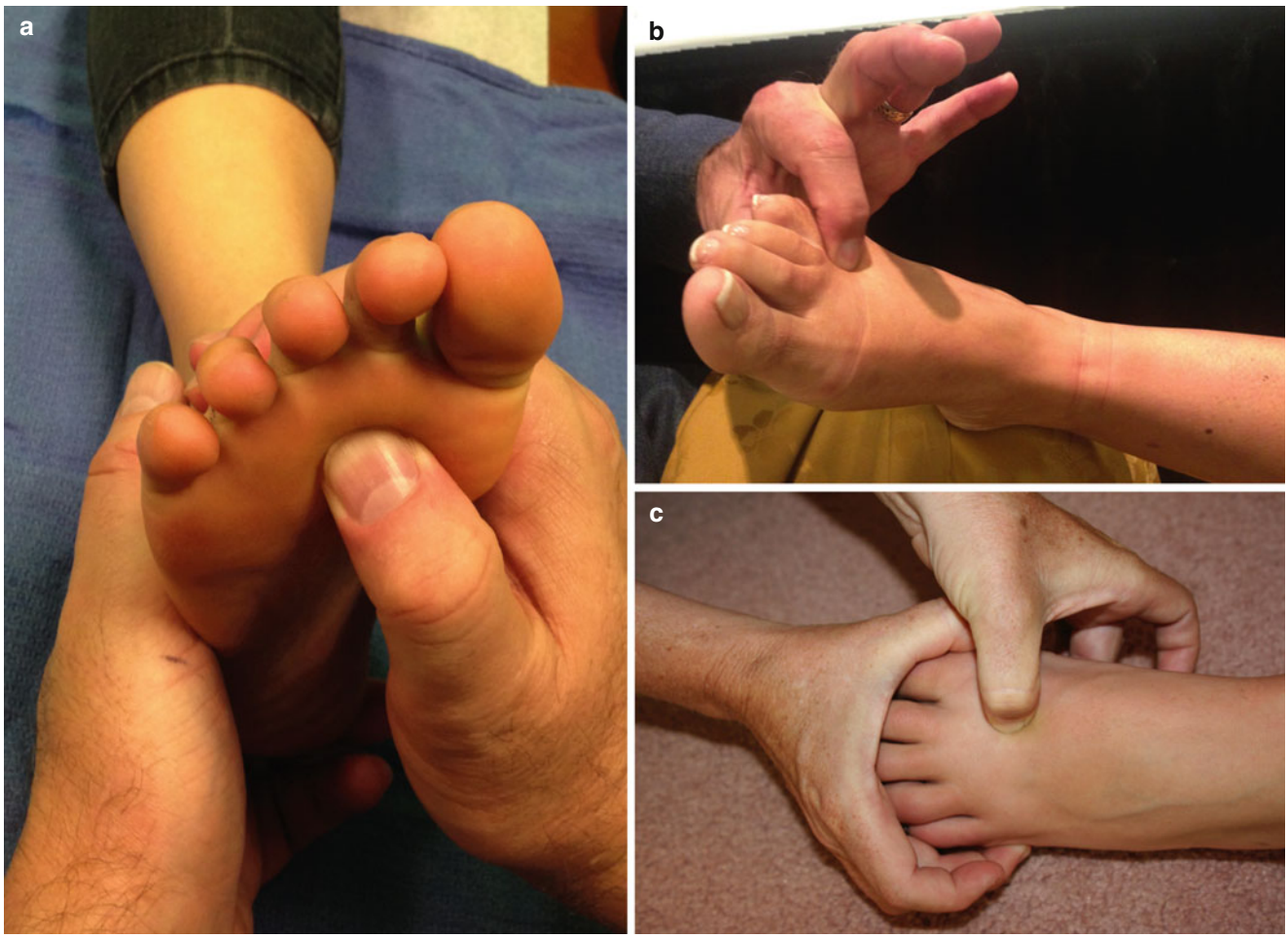

Fig. 70.5 Composite images of squeeze test to reproduce digital nerve entrapment (Images courtesy of Michael Brown, MD, Gabor Racz, MD, and Andrea Trescot, MD)

metatarsal heads, and increasing the clearance of the nerve within the anatomic structures can decrease the entrapment. Gastrocnemius stretching and accommodation of leg length can reduce anatomic stress on the forefoot, as can making adjustments in activity, such as cross-training.

Injection Techniques

Landmark-Guided Technique

Method #1: Dorsal Approach

The patient is seated on the examination table with their legs extended and the ankle dorsiflexed. Neuroma injections are performed with direction of the needle toward the site of patient's maximal tenderness within the intermetatarsal space, from distal to proximal (Video 70.2) (Fig. 70.10a) or proximal to distal (Fig. 70.10b), with the tip of the needle directed plantarly. Advance a 27-gauge, 1.5 in. needle to the nerve, and inject 1.5–2 cc of anesthetic with 20–40 mg of depo-steroid [30, 31]. Unfortunately, the site of maximum symptomatology may not always correlate with the location of the neuroma. Note that the entrapment can occur anywhere along the interspace and can be surprisingly proximal. Dr. Gabor Racz (personal communication) recommends the use of as much as 10 cc volume to lyse adhesions in the interdigital space.

Method #2: Anterior Approach

The anterior approach provides a means to direct a needle more to the inferior aspect of the interdigital space, under the metatarsal or between the metatarsal heads. The patient lies supine, and the procedure is performed after sterile skin prep and under sterile technique, with the needle directed at a trajectory between the toes at approximately 45°, toward the metatarsal heads (Fig. 70.11). Once the needle is in the inferior portion of the interdigital space, local anesthetic and corticosteroid injection can be injected.

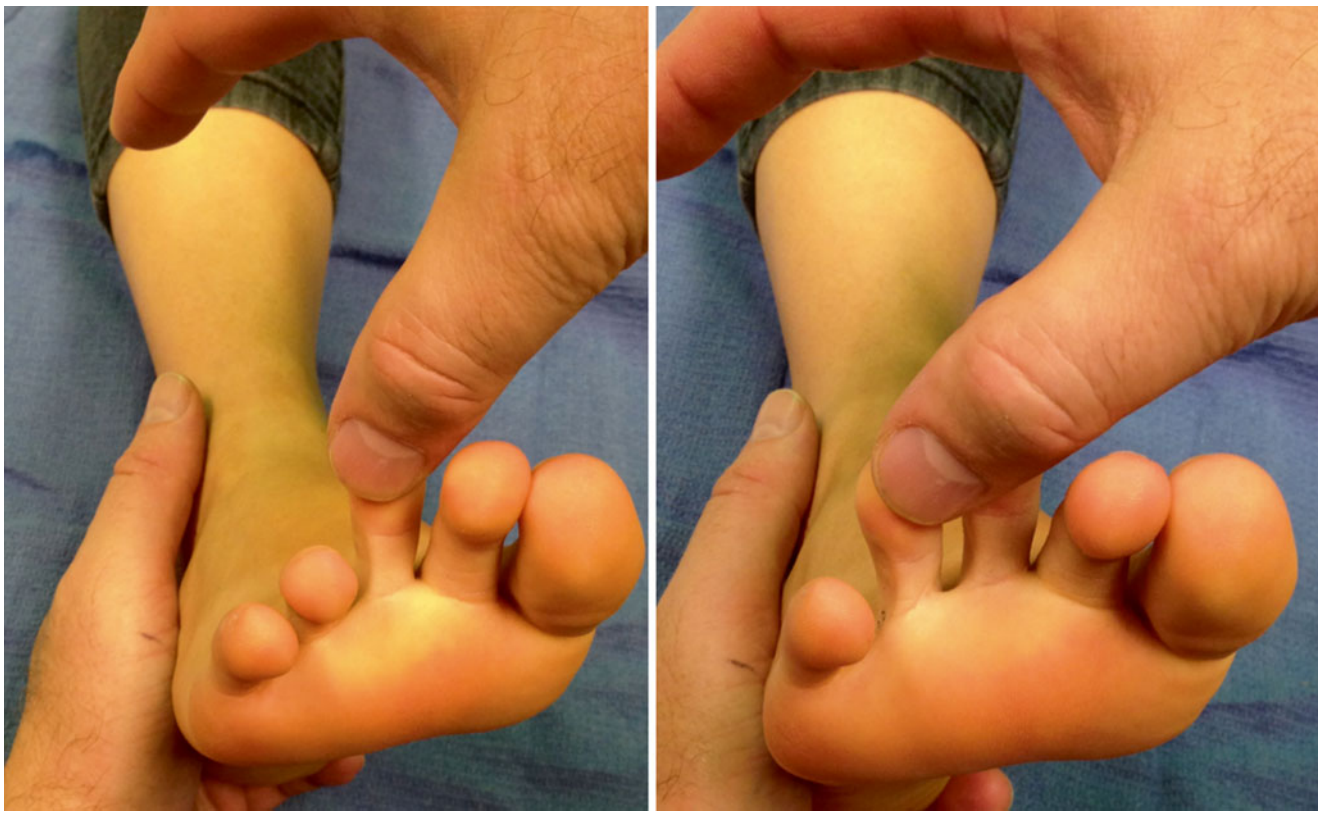

Fig. 70.6 Digital nerve stretch test to reproduce digital nerve entrapment (Image courtesy of Michael Brown, MD)

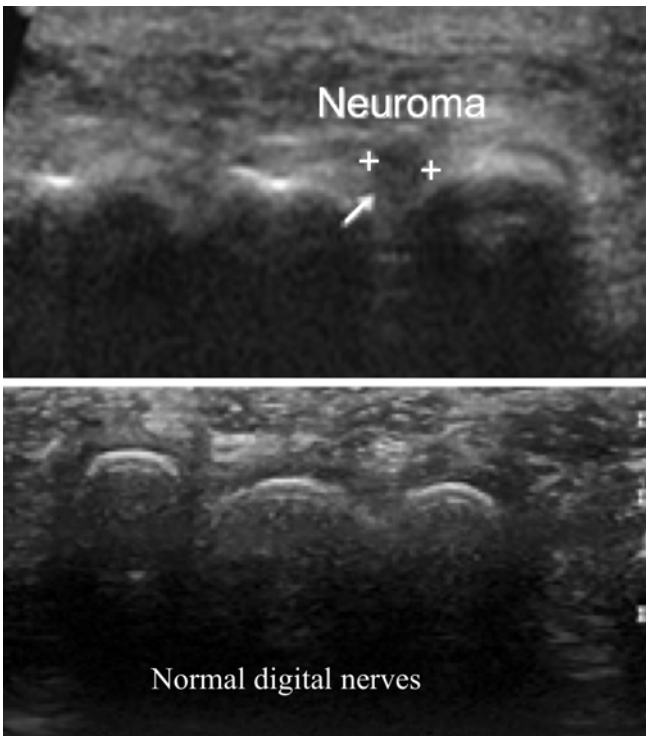

Fig. 70.7 Ultrasound evaluation of digital neuroma; *lower image* shows normal digital nerves, while *upper image* shows displacement of the neuroma with lateral squeeze (Image courtesy of Michael Brown, MD)

Fig. 70.8 Gingko shape, describing a biconcave compression of the digital interspace (Image courtesy of Michael Brown, MD)

Table 70.3 Differential diagnosis of interdigital neuroma

	Potential distinguishing features
Tarsal tunnel syndrome (TTS) [4]	See Chap. 73
Metatarsal stress fracture	Stress fracture will be positive on bone scan
AVN [9]	Doppler or arteriogram will show abnormal blood flow
Bursitis or tendonitis [4]	Tenderness along the dorsal or plantar surface of the metatarsal bone, not the interdigital space
Hammertoes [4]	Hammer malformation of toes
Plantar plate tear [4]	May be confusing because this injury also responds to diagnostic local anesthetic injection
Proximal nerve pathology [9]	No temporary relief from interdigital injection
Metatarsophalangeal joint capsulitis [9]	Pull and then flex toe; if this causes pain, the problem is likely to be capsulitis
Freiberg's infraction	Collapse and flattening of the metatarsal head, seen on X-ray

Table 70.4 Diagnostic tests for interdigital neuritis/neuroma

	Potential distinguishing features
Physical exam	Primary modality [9, 16]
Diagnostic injection	Diagnostic as well as potentially therapeutic
Ultrasound	May [4] or may not be useful, though likely will increase with operator experience and improved hardware; may be used to assess abductor hallucis hypertrophy [13]
MRI	May be useful; likely will be more so with improvements in technique
Arteriography	Not useful
X-ray	To exclude bony pathology
Electrodiagnostic studies	May be useful [1]
Other	Pressure-specified sensory testing [4]

Ultrasound-Guided Technique

Kennedy suggested that results were better with US- than landmark-guided injections [9].

Method #1: Ultrasound-Guided Dorsal Approach

The patient is positioned supine with the foot supported and ankle dorsiflexed. Place the 7.5–15 MHz linear probe longitudinally over the plantar aspect of the foot, in the long axis of the metatarsal (Fig. 70.12a). An alternative is to place the transducer transversely for out-of-plane injection; scanning can be performed in both the coronal and sagittal planes (Fig. 70.12b). As you scan up and down from proximal to distal, the distal portion of the forefoot

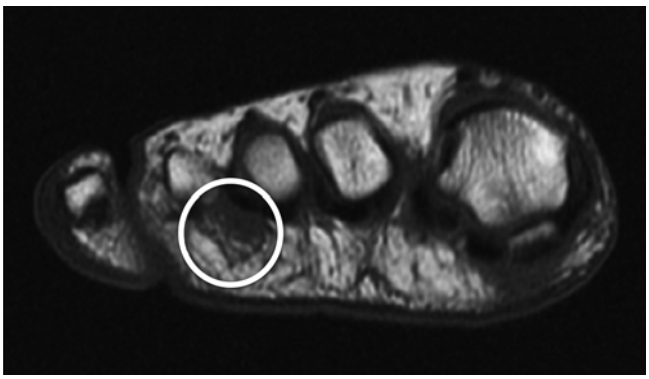

Fig. 70.9 MRI image of digital neuroma (*white circle*) (Image courtesy of Andrea Trescot, MD)

neuromas are clearly visualized as incompressible hypoechoic nodules replacing the normally hyperechoic web space fat. Confirm the neuroma by performing a *Mulder's sign* (see above) with displacement of the neuroma seen. One can also use a palpation pressure in the interdigital space, with the finger directed to the ultrasound transducer that is placed on the plantar aspect of the foot. The neuroma will appear as a focal hypoechoic structure that typically displaces in a plantar direction when eliciting the Mulder's sign or with a palpatory pressure over the dorsal surface. Guidance of the tip of the 1.5 in., 27-gauge needle into the mass will allow for optimal outcomes [32]. The disadvantage of this approach is the depth of the interdigital nerve, which lies more to the plantar region of the interdigital space.

Method #2: Anterior Plantar Approach

There are many times when the clinician may want to do an intralesional injection (injection within the neuroma). If good visualization can be made with the ultrasound transducer placed on the dorsal aspect of the metatarsal arch, this approach may provide a better means for direct visualization of the needle placement into the neuroma (Fig. 70.13a). This may include intralesional alcohol, ozone, hypertonic saline (3 %), or many other injectates.

Placement of the 7.5–15 MHz transducer begins in the transverse position. The authors recommend a "toe count," which is performed by identifying the first ray and its associated sesamoid bones and then counting laterally. Each interdigital space and metatarsal can then be accounted for as confirmation of ultrasound transducer positioning. The neuroma can be visualized by eliciting the metatarsal squeeze. Alternatively, the US probe can be placed on the plantar surface. The transducer is maintained in a transverse position, where the needle can be directed to the neuroma out-of-plane (Fig. 70.13b). The transducer can be rotated into a longitudinal position, and the needle can be

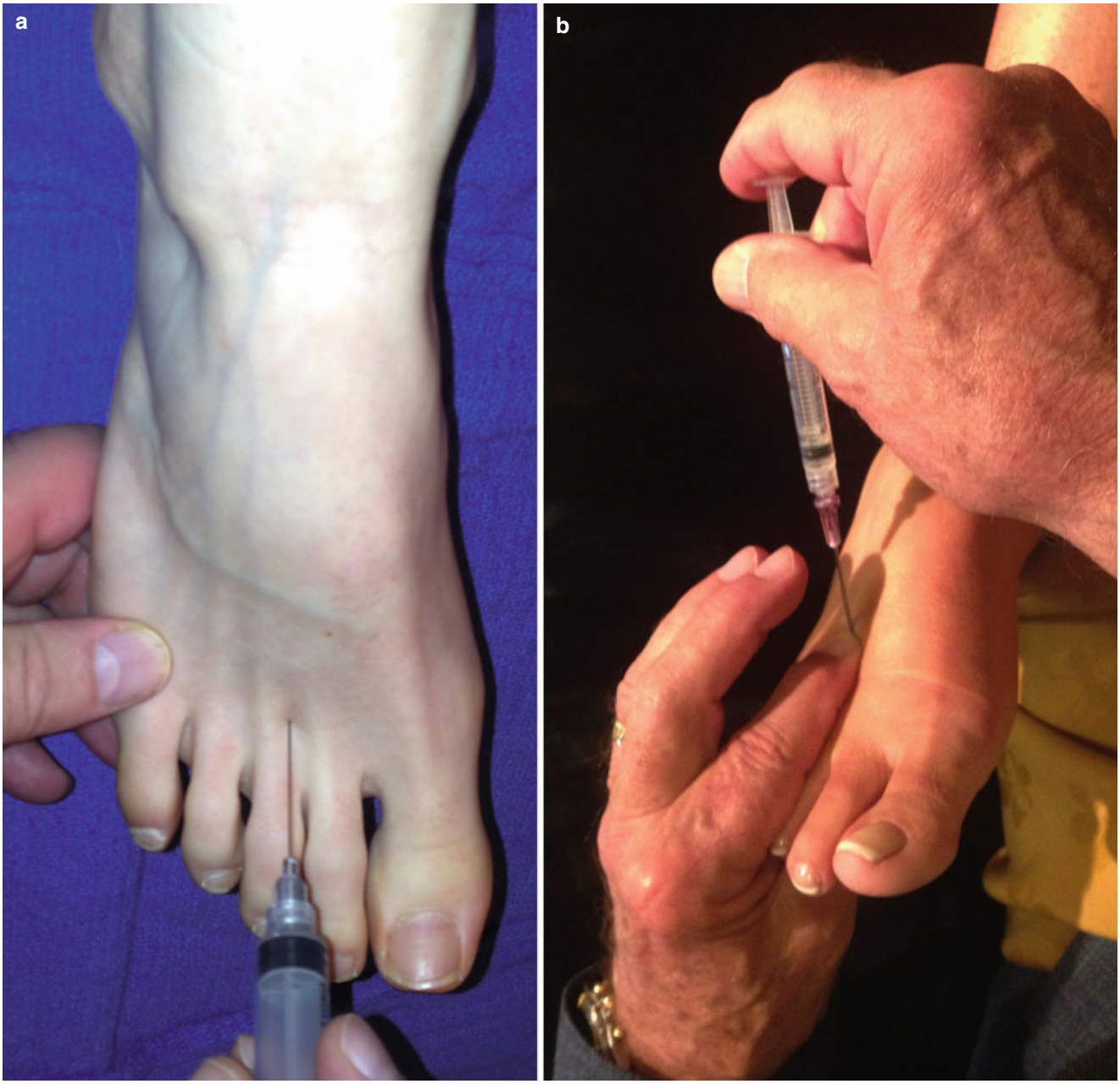

Fig. 70.10 Dorsal approach for landmark-guided digital injection. Image (**a**) shows a distal-to-proximal approach; image (**b**) shows a proximal-to-distal approach (Images courtesy of Michael Brown, MD and Gabor Racz, MD)

directed into the neuroma utilizing a longitudinal in-plane approach. Load a 3 cc syringe filled with local anesthetic, so that small aliquots of anesthetic can be injected throughout the trajectory of the needle toward the neuroma. This provides a way of monitoring the needle position (by watching for the soft tissue infiltration of the local anesthetic), as well as anesthetizing the tissue as the needle passes through. The injection can be rendered mostly painless by using this method. When the needle is in the correct position, the syringe is changed for one containing local anesthetic and depo-steroid.

Fluoroscopy-Guided Technique

These injections are superficial, with usually little benefit gained by fluoroscopy, along with the added radiation exposure to the patient and clinician. However, in the case of altered anatomy, such as with prior reconstructive surgery (e.g., bunionectomy) or prior neuroma excision (which requires more proximal injections or cryoneuroablation at the apex of the metatarsal/tarsal joint) [33], fluoroscopy may offer guidance between the osseous structures (Fig. 70.14).

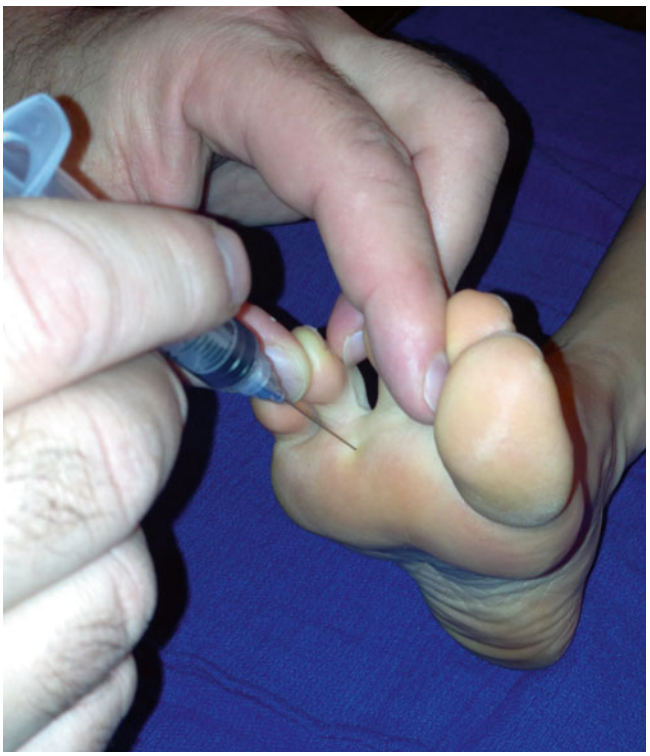

Fig. 70.11 Anterior approach for landmark-guided digital injection (Image courtesy of Michael Brown, MD)

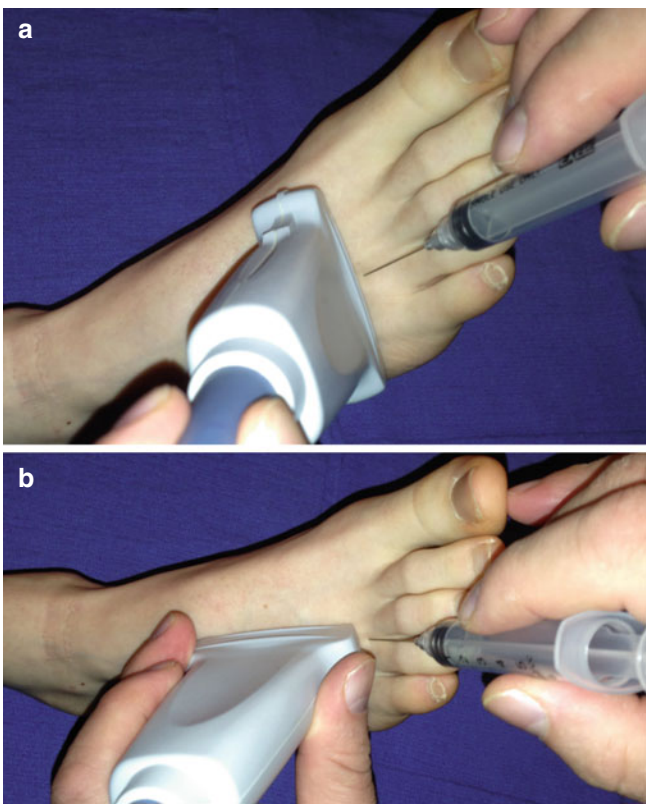

Fig. 70.12 Ultrasound-guided dorsal approach to the digital nerve. Image (**a**) shows horizontal placement of the US probe with an out-of-plane needle approach; image (**b**) shows vertical placement of the US probe with an in-plane needle approach (Image courtesy of Michael Brown, MD)

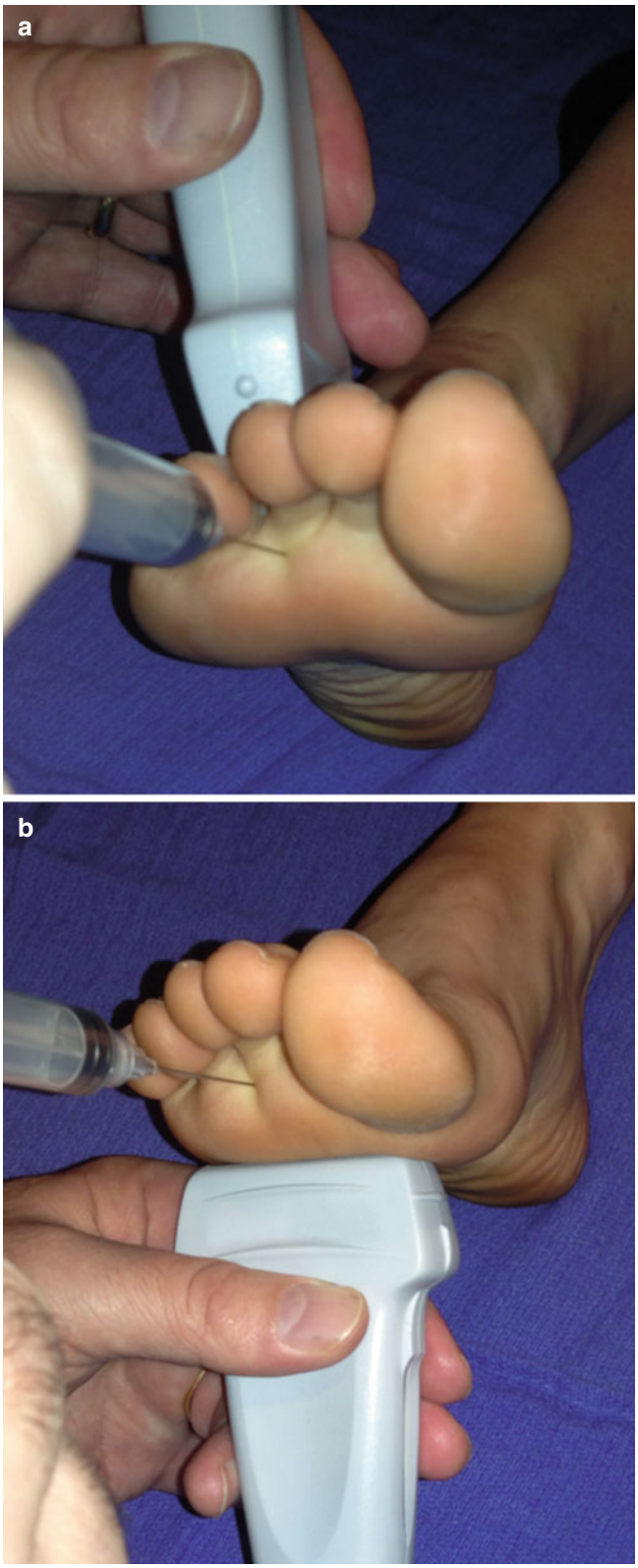

Fig. 70.13 Ultrasound-guided plantar approach to the digital nerve. Image (**a**) shows in-plane needle approach with the US probe on the dorsal surface of the foot; image (**b**) shows an out-of-plane approach with the probe on the plantar surface of the foot (Image courtesy of Michael Brown, MD)

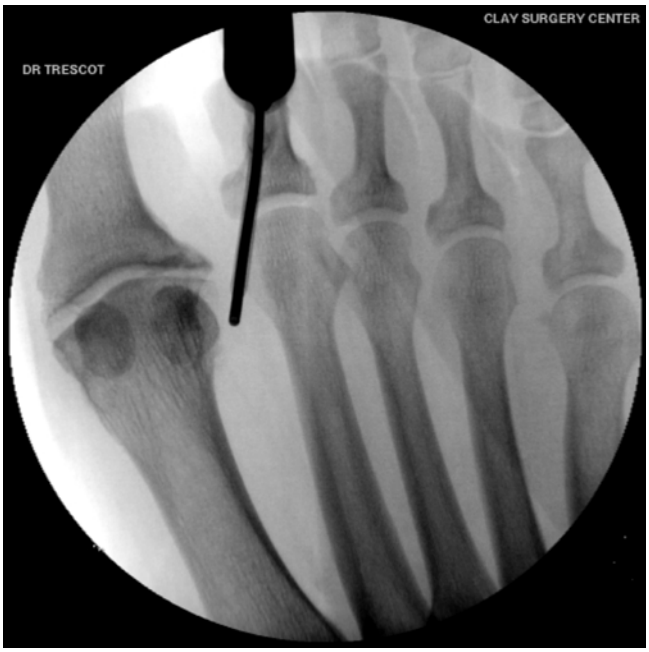

Fig. 70.14 Cryoneuroablation of the deep peroneal nerve under fluoroscopy (Image courtesy of Andrea Trescot, MD)

Neurolytic Technique

Cryoneuroablation

Cryoneuroablation is a minimally invasive effective procedure with successful outcomes that preserve the anatomy without creating stump neuromas [33, 38, 39] (Fig. 70.13). The appropriate introducer (12 gauge for "virgin" nerves and the 14 gauge for postoperative neuromas) is introduced through the dorsal foot skin, perpendicular to the nerve and directed toward the apex of the metatarsal bones. Care must be used when injecting the saline with epinephrine, because of the risk of digital ischemia. Placing the non-introducing hand at the plantar surface will detect if the probe is too close to the skin of the sole of the foot. Stimulation should replicate the patient's usual pattern of pain. Care should be taken to avoid skin injury due to the superficial nature of the nerve location (Fig. 70.15).

Cryoneurolysis of an intermetatarsal space neuroma has been reported to provide at least 6 months of relief [39].

Radio-frequency Lesioning (RF)

Percutaneous thermal destructive techniques have lacked the reproducible precision of being certain that the neuroma was completely treated. The thermal trauma caused by radio-frequency lesioning can result in post-procedural disability consistent with open surgery.

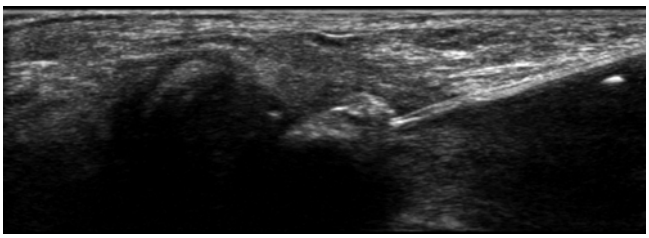

Fig. 70.15 US image of cryoneuroablation of the digital nerve, showing the cryo probe and ice ball. (Image courtesy of Agnes Stogicza, MD)

Neurolytic Injections

Dilute alcohol injections (three to seven injections of 4 % alcohol administered at 5–10 day intervals) has been associated with an approximate 85 % success rate [10] and, when combined with sonography, has offered results comparable to surgical resection [34]. Another injection modality involves injecting the nerve with vitamin B12 (cyanocobalamin); this has been discussed in the literature, but the effects observed may have been due to the preserving agent, benzyl alcohol [35]. Phenol also has been reported as a safe and effective injection modality [36].

Percutaneous injections of caustic substances, such as phenol and alcohol, have been tried with variable rates of success, and they are often accompanied by unacceptably high complication rates. The search still goes on for a technique to deal effectively with Morton's neuroma on an outpatient basis with a low rate of recurrent neuroma, rapid return to work, low morbidity, and high degrees of patient satisfaction [37].

Surgical Techniques

Decompression of the nerve via release of the deep intermetatarsal ligament through an open or endoscopic nerve release technique has been shown to offer effective outcomes [40, 41]. Excision may be chosen when prior intervention and decompression fails to resolve symptoms. Various surgical approaches have been described; the most common is a dorsal approach over the involved intermetatarsal space and excision of the affected portion of the nerve. Plantar incisions are generally reserved for revisions but have been described as an initial approach as well [1, 42].

The surgical excision of the neuroma is frequently associated with recurrent neuroma formation proximal to the original surgery site due to abnormal nerve sprouts growing from the severed ends of the nerve. Even when recurrent neuroma formation does not take place, persistent sensory dysesthesias may occur following successful neurectomy, complicating the ongoing management of these patients. With open surgery and the attendant dissection and pain of nerve transection, return to work, even when the surgery is performed

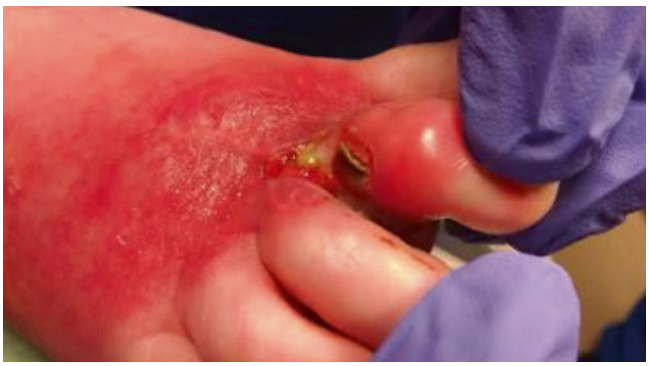

Fig. 70.16 Right foot necrosis and cellulitis 3 days after alcohol neurolysis (Image courtesy of Emily Walker, MD and Christopher Burnett, MD)

on an outpatient basis, as it nearly always is, has not been immediate; periods of post-procedural disability from several days to a few weeks are not uncommon [37].

In a study in 2008 of 120 patients who underwent open neuroma excision, 50 % of the patients noted "good" relief, and 10 % had "fair" relief, but 40 % reported "poor" relief [43]. Endoscopic resection has been reported to have better outcomes than the open procedure. Barrett et al. [44] described a 95 % "good-to-excellent" outcome for endoscopic surgeries at the third metatarsal space, compared to 85 % improvement at the second metatarsal space.

Complications

Any injection may cause the usual complications of bleeding, infection, and nerve damage. Other complications are loss of plantar fat pad, de-stabilization of the adjacent MTPJ, and potential subluxation. Discoloration of the skin could be secondary to steroid injection. Walker et al. [45] described toe necrosis and cellulitis that required hospitalization after the injection of 1.5 cc of 98 % dehydrated alcohol (Fig. 70.16).

Summary

Neuropathic pain of the interdigital nerve should be considered when the patient has sharp and burning pain in the forefoot. In most cases, a careful history and physical exam can establish the cause of the pain, along with the ultrasound and MRI diagnostic tests and diagnostic injections.

References

1. McCrory P, Bell S, Bradshaw C. Nerve entrapments of the lower leg, ankle and foot in sport. Sports Med. 2002;32(6):371–91.
2. Morton T. A peculiar and painful affection of the fourth metatarsophalangeal articulation. Am J Med Sci. 1876;71:37–45.
3. Durlacher L. Treatise on corns, bunions, diseases of nails, and the general management of the feet. London: Simpkin and Marshall; 1845. p. 52.
4. Franson J, Baravarian B. Intermetatarsal compression neuritis. Clin Podiatr Med Surg. 2006;23(3):569–78.
5. Viladot A. Morton's neuroma. Int Orthop. 1992;3:294–6.
6. Thomas J, Blitch E, Chaney D, Dinucci K, Eickmeier K, Rubin L, et al. Diagnosis and treatment of forefoot disorders. Section 3. Morton's intermetatarsal neuroma. J Foot Ankle Surg. 2009; 48(2):251–6.
7. Kim J, Choi J, Park J, Wang J, Lee I. An anatomical study of Morton's interdigital neuroma: the relationship between the occurring site and the DTML. FAI. 2007;28(9):1007–10.
8. Schon LC, Baxter DE. Neuropathies of the foot and ankle in athletes. Clin Sports Med. 1990;9(2):489–509.
9. Prisk VR, O'Loughlin PF, Kennedy JG. Forefoot injuries in dancers. Clin Sports Med. 2008;27(2):305–20.
10. Dockery GL. The treatment of intermetatarsal neuromas with 4% alcohol sclerosing injections. J Foot Ankle Surg. 1999; 38(6):403–8.
11. de Asla R, Deland J. Foot and ankle. 2nd ed. In: Thordarson DB, Tornetta P, Einhorn TA, editors. Orthopaedic Surgery Essentials: Foot and Ankle. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2004. p. 18–23.
12. Zanetti M, Strehle J, Kundert H, Zollinger H, Hodler J. Morton neuroma: effect of MR imaging findings on diagnostic thinking and therapeutic decisions. Radiology. 1999;213(2):583–8.
13. Ghosh SK, Raheja S, Tuli A. Potential sites of compression of tibial nerve branches in foot: a cadaveric and imaging study. Clin Anat. 2013;26(6):768–79.
14. Sullivan J. Neuroma diagnosis by means X-ray evaluation. J Foot Ankle Surg. 1967;6:45–6.
15. Mulder J. The causative mechanism in morton's metatarsalgia. J Bone Joint Surg (Br). 1951;33:94–5.
16. Cloke D, Greiss M. The digital nerve stretch test: a sensitive indicator of Morton's neuroma and neuritis. Foot Ankle Surg. 2006; 12(4):201–3.
17. Hassouna H, Singh D. Morton's metatarsalgia: pathogenesis, aetiology and current management. Acta Orthop Belg. 2005; 71:646–55.
18. Park H, Kim S, Rho M, Hong H, Lee S. Sonographic appearances of Morton's neuroma: differences from other interdigital soft tissue masses. Ultrasound Med Biol. 2011;37(8):1209. 1/2/04.
19. Zanetti M, Weishaupt D. MR imaging of the forefoot: morton neuroma and differential diagnoses. Semin Musculoskelet Radiol. 2005;9:175–86.
20. Hofbauer P. Rheumatoid nodule in Morton's neuroma. A case report. J Am Podiatry Assoc. 1974;64(6):424–6.
21. Iagnocco A, Coari G, Palombi G, Valesini G. Sonography in the study of metatarsalgia. J Rheumatol. 2001;28:1338–40.
22. Alexander I, Johnson K, Parr J. Morton's neuroma: a review of recent concepts. Orthopedics. 1987;10:103–6.
23. Kaminsky S, Griffin L, Milsap J, Page D. Is ultrasonography a reliable way to confirm the diagnosis of Morton's neuroma? Orthopedics. 1997;20:37–9.
24. Beggs I. Sonographic appearances of nerve tumors. J Clin Ultrasound. 1999;27:363–8.
25. Pollak R, Bellacosa R, Dornbluth N, Strash W, Devall J. Sonographic analysis of Morton's neuroma. J Foot Surg. 1992; 31:534–7.
26. Kankanala G, Jain A. The operational characteristics of ultrasonography for the diagnosis of plantar intermetatarsal neuroma. J Foot Ankle Surg. 2007;46:213–7.
27. Redd R, Peters V, Emery S, Branch H, Rifkin M. Morton neuroma: sonographic evaluation. Radiology. 1989;171:415–7.
28. Mendicino SS, Rockett MS. Morton's neuroma: update on diagnosis and imaging. Clin Podiatr Med Surg. 1997;14:303–11.

29. DiGiovani C, Kuo R, Tejwani N, et al. Isolated gastrocnemius tightness. J Bone Joint Surg. 2002;84A:962–71.
30. Rasmussen M, Kitaoka H, Patzer G. Nonoperative treatment of plantar interdigital neuroma with a single corticosteroid injection. Clin Orthop Relat Res. 1996;326:188–93.
31. Greenfield J, Rea JJ, Ilfeld S. Morton's interdigital neuroma: indications for treatment by local injections versus surgery. Clin Orthop Relat Res. 1984;185:142–4.
32. Hassouna H, Singh D, Taylor H, Johnson S. Ultrasound guided steroid injection in the treatment of interdigital neuralgia. Acta Orthop Belg. 2007;73(2):224–9.
33. Trescot AM. Cryoanalgesia in interventional pain management. Pain Phys. 2003;6(3):345–60.
34. Hughes R, Ali K, Jones H, Kendall S, Connell D. Treatment of Morton's neuroma with alcohol injection under sonographic guidance: follow-up of 101 cases. Am J Roentgenol. 2007;188(6):1535–9.
35. Steinberg M. The use of vitamin B-12 in Morton's neuralgia. J Am Podiatr Med Assoc. 1955;45:566–7.
36. Magnan B, Marangon A, Frigo A, Bartolozzi P. Local phenol injection in the treatment of interdigital neuritis of the foot (Morton's neuroma). Chir Organi Mov. 2005;90:371–7.
37. Noback CR. Podiatric treatment of Morton's neuroma with cryoneurolytic technique. Jacksonv Med J. 2002;3(2):10–2.
38. Caporusso E, Fallat L, Savoy-Moore R. Cryogenic neuroablation for the treatment of lower extremity neuromas. J Foot Ankle Surg. 2002;41:286–90.
39. Hodor L, Barkal K, Hatch-Fox L. Cryogenic denervation of the intermetatarsal space neuroma. J Foot Ankle Surg. 1997;36(4):311–4.
40. Vito G, Talarico L. A modified technique for Morton's neuroma. Decompression with relocation. J Am Podiatr Med Assoc. 2003;93:190–4.
41. Colgrove R, Huang E, Barth A, Greene M. Interdigital neuroma: intermuscular neuroma transposition compared with resection. Foot Ankle Int. 2000;21:206–11.
42. Wu K. Morton's interdigital neuroma: a clinical review of its etiology, treatment, and results. J Foot Ankle Surg. 1996;35:112–9.
43. Womack JW, Richardson DR, Murphy GA, Richardson EG, Ishikawa SN. Long-term evaluation of interdigital neuroma treated by surgical excision. Foot Ankle Int. 2008;29:574–7.
44. Barrett SL, Rabat E, Buitrago M, Rascon VP, Applegate D. Endoscopic decompression of intermetatarsal nerve (EDIN) for the treatment of Morton's entrapment—multicenter retrospective review. Open J Orthop. 2012;2:19–24.
45. Walker E, Wright G, Burnett C. Alcohol neurolysis of digital nerves: is it safe? Texas Pain Foundation 7th Annual Scientific Meeting. San Antonio; 2015.

Sural Nerve Entrapment

71

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Introduction

This chapter reviews the anatomy of the *sural nerve* (SN), its course and the common locations for potential entrapment, some clinical presentation scenarios, differential diagnoses, injection techniques, and other surgical management modalities. Sural nerve entrapment was first described in 1974 [1]. The SN (also known as the *short saphenous nerve*) is a sensory nerve that can be entrapment in different locations, causing pain in the calf as well as the lateral ankle and foot. When injured by trauma or compression, neuropathic pain, burning, cutaneous allodynia, and, on occasion, a CRPS-like picture may occur. Both the presentation and the treatment are dependent on the location of the entrapment, and identification of a sural entrapment neuropathy requires a high index of suspicion and a thorough knowledge of the clinical presentations and anatomic entrapment syndromes. This is also a nerve that is frequently biopsied, studied as part of nerve conduction velocity evaluations, and harvested for nerve grafts, so knowledge of the anatomic location is important. Although it has been considered a "purely sensory" nerve, motor fibers have been identified in 4.5 % of specimens [2].

Clinical Presentation (Table 71.1)

Patients with distal SN entrapment usually present with pain at the posterior and lateral aspect of the ankle and foot, often associated with paresthesias over the lateral ankle and the dorsum and lateral aspect of the foot [1] (Fig. 71.1). Patients describe burning pain or numbness down the lateral leg, distal to the knee, along the lateral border of the foot, extending to the base of the fifth toe (Fig. 71.2) and the bottom of the foot (Fig. 71.3). The pain is not usually worse with walking but can increase at night and with exercise, and it often appears or worsens at night [8]. In addition, the pain is worse with palpation, foot eversion, and prolonged standing. There may be a history of frequent, minor ankle inversion injuries [1]. The SN travels just lateral to the *Achilles tendon*. Therefore, injury or rupture of the Achilles tendon with associated hematoma and scar, fusiform enlargement of the Achilles tendon from tendinopathy, and a suture entrapping the nerve during Achilles tendon repair are other potential sources of SN injury [3, 9]. The SN is also potentially subject to compression neuropathy secondary to repeated microtrauma, compression, fifth metatarsal fracture, calcaneal or cuboid fracture, or space-occupying lesions [10–12]. It may also be triggered by, or mistaken for, *thrombophlebitis* [5].

Table 71.1 Occupation/exercise/trauma history relevant to sural nerve entrapment

Trauma	Frequent minor ankle inversion injuries [1]
	Fifth metatarsal fracture [3]
	Calcaneal or cuboid fracture [4]
Inflammation	Thrombophlebitis [5]
Compression [3]	Tight-fitting combat boot [5]
	Prolonged standing [6]
	Space-occupying lesion [7]

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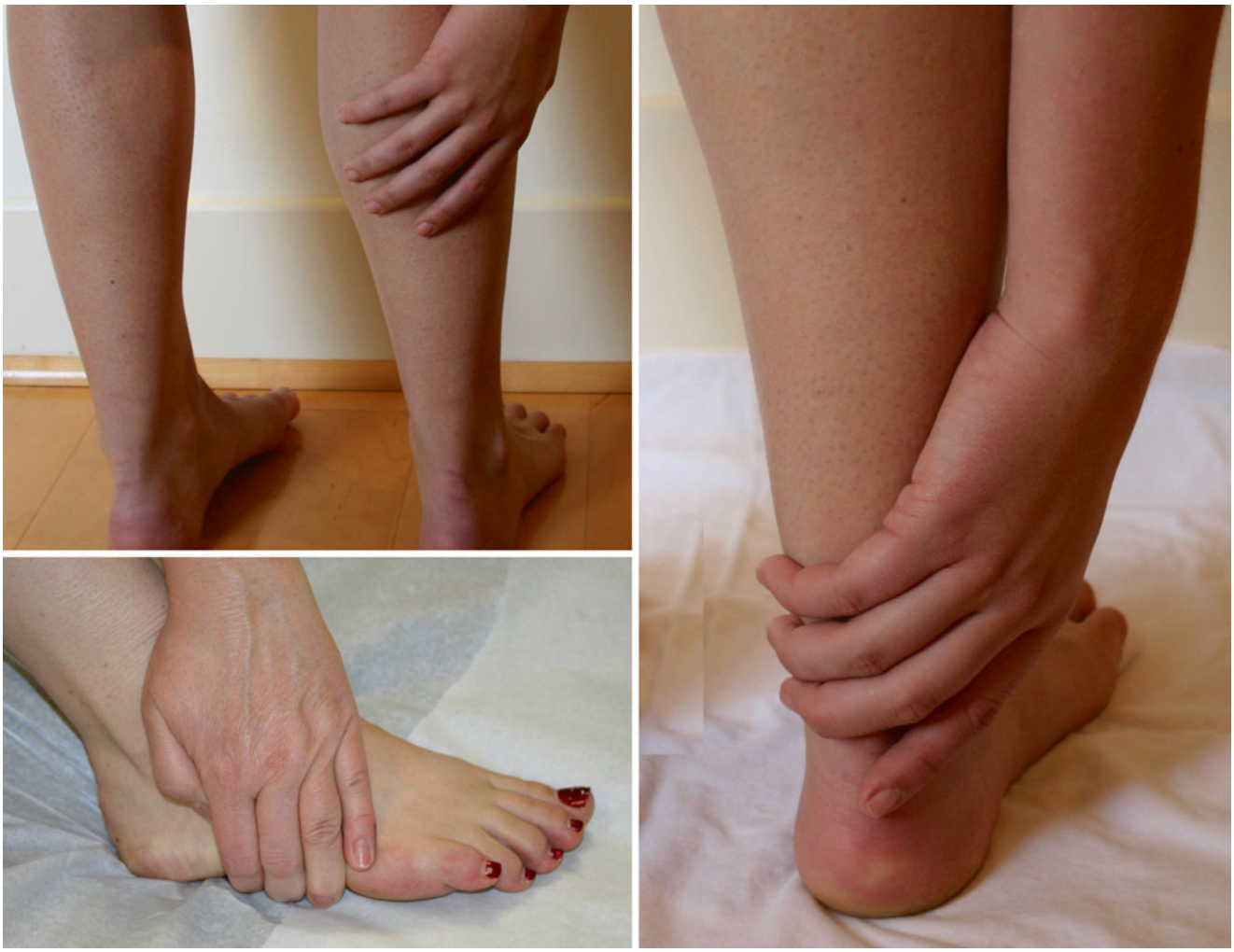

Fig. 71.1 Patient pain complaint from sural nerve entrapment (Image courtesy of Andrea Trescot, MD)

Anatomy (Table 71.2)

In the upper popliteal fossa, the *sciatic nerve* (Chap. 65) divides into its two main branches – the *tibial nerve* (Chap. 73) and the *common peroneal nerve* (*common fibular nerve* (*CPN*)) (Chap. 67) (Fig. 71.4). The tibial nerve continues down the *popliteal fossa* lateral to the popliteal vein and artery. Before traveling beneath the *gastrocnemius*, the tibial nerve gives off a small cutaneous branch, the *medial sural cutaneous nerve* (*MSCN*), which courses laterally over the head of the gastrocnemius and penetrates deep fascia but courses superficially halfway down the calf (Fig. 71.5). The CPN, after dividing from the sciatic nerve, travels parallel to the distal *biceps femoris* tendon toward the fibular head. Before the nerve dives deep between the *lateral soleus muscle* and the *fibula* to divide into the *superficial peroneal* (*fibular*) *nerve* (*SPN*) and the *deep peroneal* (*fibular*) *nerve* (*DPN*) (Chaps. 68 and 69), it also gives a small cutaneous branch called the *lateral sural cutaneous nerve* (*LSCN*) (Chap. 72).

Although it can be quite variable, the SN usually arises from the MSCN (a branch of the tibial nerve) and the peroneal communicating nerve (PCN), which can come either directly from the CPN or from the LSCN. These four components (MSCN, LSCN, PCN, and SN) have been called the "*sural nerve complex.*"

The sural nerve travels between to the two heads of the gastrocnemius muscle (Fig. 71.6) in the distal third of the leg [1, 9, 13–15], beside the *small (lesser) saphenous vein*, and pierces the deep fascia at the middle third of the posterior leg. It then exits this "gastrocnemius groove" near the musculotendinous junction of the Achilles tendon and the gastrocnemius muscle [14]. This *fibrous arcade* is wide, thick, and unyielding and may fit tightly around the nerve, causing chronic friction and irritation, which can lead to sural neuropathy [14].

At this location, the SN is often joined by the *peroneal communicating nerve* (*PCN*), which is a branch from the common peroneal nerve or from the *lateral sural cutaneous*

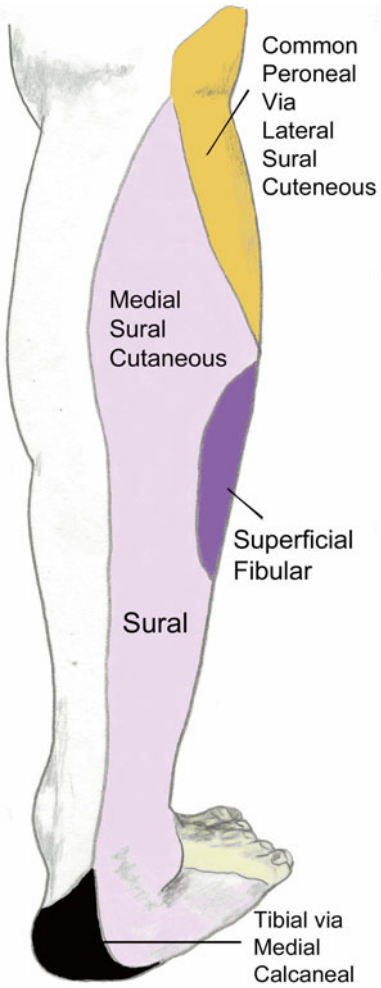

Fig. 71.2 Pattern of lower leg pain from sural nerve entrapment (Image courtesy of Michael Brown, MD)

nerve (LSCN) (see Chap. 72). The SN then continues down the leg on the posterior-lateral side; just above and posterior to the lateral malleolus and superficial to the *extensor retinaculum*, the SN gives off the *lateral calcaneal nerve* (Fig. 71.7), which supplies the sensory innervation to the Achilles tendon and the lateral aspect of the heel. The nerve typically is located about 1–1.5 cm behind the lateral malleolus and just anterolateral to the short saphenous vein on the surface of the fascia covering the muscles and tendons. It then runs deep to the *peroneal* (*fibularis*) *tendon sheath*, inferior to the lateral malleolus, and reaches the lateral tuberosity of the fifth metatarsal [11]. At the level of the base of the fifth metatarsal, the SN bifurcates into the lateral and medial terminal branches, supplying sensation to the lateral aspect of the foot and the lateral aspect of the fifth digit [16–18].

The SN has a number of anatomical variants (Fig. 71.8); the anatomy is complex and variable, with confusing terminology [19]. Kavyashree et al. [20] dissected 50 lower limbs and described three main types of sural anatomy. In *Type A*, the sural nerve was formed by the union of the MSCN and the PCN (72 %); in *Type B*, the sural nerve was just the continuation of the MSCN with the PCN absent (28 %); and in *Type C*, the sural nerve was formed by just the PCN (but not seen in this study). Only 60 % of the cadavers had the same type of sural nerve anatomy on both sides.

Mestdagh et al. [16] dissected 37 cadaver lower limbs and found that the sural nerve arose on average 66 mm (2–200 mm) below the bifurcation of the sciatic trunk. The *medial cutaneous nerve of the leg* came from the posterior aspect of the tibial nerve in 31 cases, the posterior-lateral aspect in 2 cases, and posterior-medially in 3 cases, while it was absent in 1 case. They also found the *lateral cutaneous nerve* in 30 cases and its perforating branch in 25 cases (with 3 cases coming directly from the peroneal nerve); its origin was an average of 57 mm (5–180 mm) from the sciatic bifurcation. Only 25 of the 37 legs examined had the "classic" arrangement. George and Nayak [2] reported a dissection where the sural nerve pierced the gastrocnemius muscle instead of traveling between the heads (and likely provided motor innervation to the gastrocnemius muscle); the sural communicating nerve was noted to be much larger than the lateral sural cutaneous nerve or the sural nerve itself.

Not uncommonly, there is a communicating nerve that connects the sural and tibial nerves, ventral to the calcaneal tendon. Sekiya et al. [21] dissected 52 legs and found this connecting branch in 7 specimens (13.5 %). They were able to identify three separate types of connections: Type Y (a medial branch from the sural nerve and a branch from the tibial nerve joined in a Y shape and became one terminal branch), Type U (both branches formed a loop between the sural and tibial nerves), and Type N (the communicating branch ran obliquely and medially to reach the tibial nerve distally). Since this communicating branch innervates some of the plantar muscles (and thus is likely not a pure sensory nerve), the authors suggested that biopsies and harvest for nerve grafting be performed at the more distal part of the sural nerve, rather than proximal to this divergence.

Entrapment

The sural nerve travels its anatomical course superficially through the distal leg and ankle, making it susceptible to local trauma as well as the risk of iatrogenic injury during surgical interventions at the ankle [13]. The nerve can become entrapped anywhere along its course in the lower extremity [1]. Entrapment involving the sural nerve typically occurs at the musculotendinous junction of the gastrocnemius muscle and the Achilles tendon within the calf, as the nerve travels through a fibrous arcade (which has been termed the "superficial sural aponeurosis") [8] (Fig. 71.9a), at the ankle

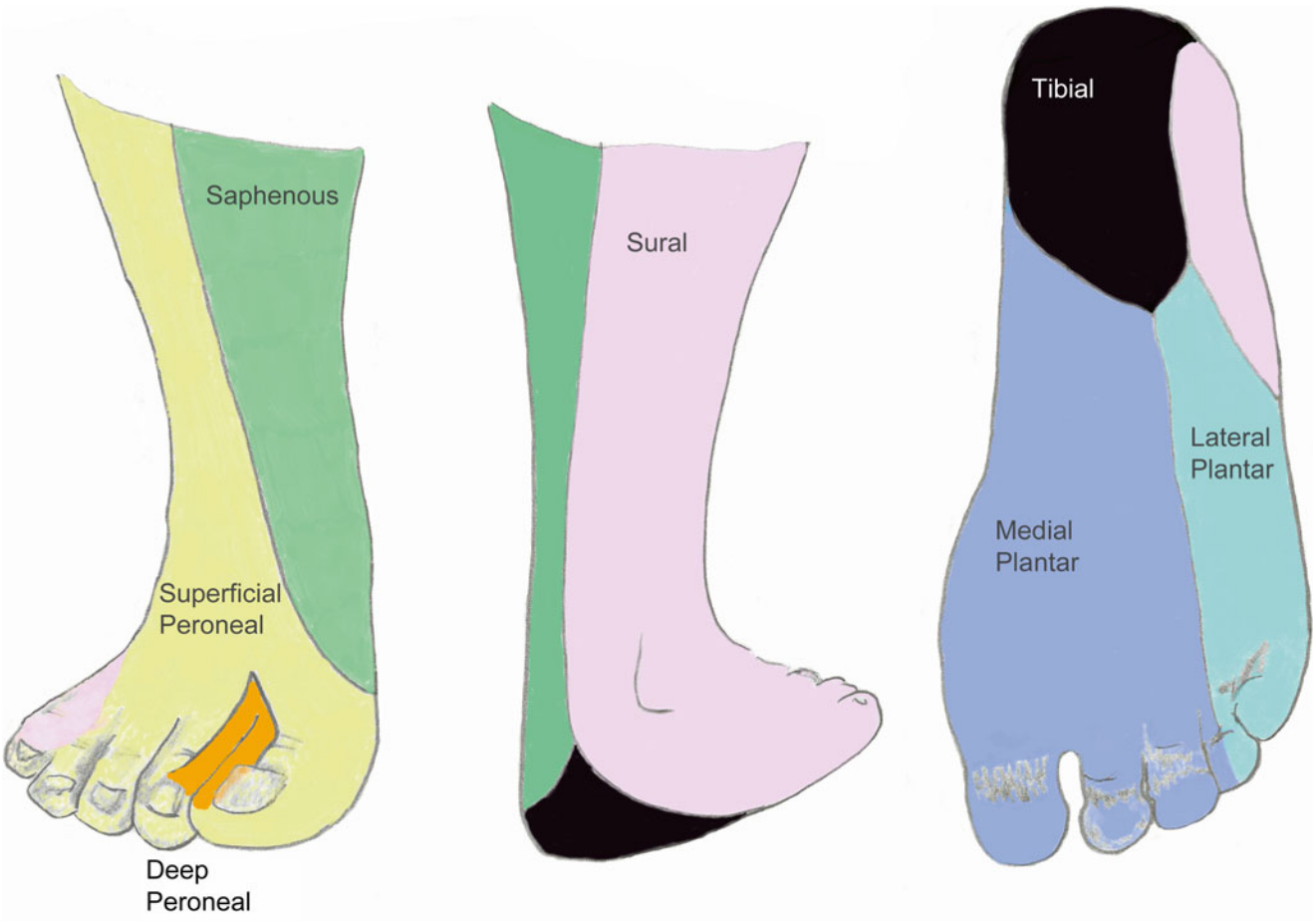

Fig. 71.3 Pattern of plantar foot pain from sural nerve entrapment (Image courtesy of Michael Brown, MD)

Table 71.2 Sural nerve anatomy

Origin	Sciatic nerve divides into tibial and common peroneal/fibular nerves; branches off of each nerve to make up the sural nerve
General route	Tibial, peroneal, and sural nerve travel deep to the gastrocnemius
Sensory distribution	The lateral and posterior distal third of the leg and lateral aspect of the foot and fifth digit
Motor innervation	None
Anatomic variability	There can be multiple variations of connections between the lateral sural cutaneous nerve, the medial sural cutaneous nerve, and a variety of connecting branches of the tibial and peroneal nerves
Other relevant structures	Achilles tendon, lateral malleolus

(Fig. 71.9b), or in the lateral foot near the base of the fifth metatarsal (Fig. 71.9c) [3, 13, 22].

Entrapment occurs primarily due to compression (such as when the feet are crossed for extended periods of time at the heel) or due to acute trauma and related scarring. Footwear, tight laces, compression with elastic bandage and socks, straps, boots (combat or occupational), and many other sources of repeated microtrauma can affect nerve function. The sural nerve can also be injured when stripping varicose veins or with thermal ablation of the small saphenous vein, which often runs in close proximity to the nerve [23]. The nerve can be entrapped by space occupying masses such as tumors or cysts [1]. As described in the *Clinical Presentation* section, the sural nerve travels lateral to the Achilles tendon, and therefore Achilles pathology can cause sural nerve injury [3, 14, 15].

Additionally, the sural nerve can be subject to distraction and injury during ankle sprains along the course of the nerve, leading to neurapraxia injury [3, 9, 22, 24]. Paraskevas et al. [8] were able to show that traction on the SN occurred during forcible plantar flexion and inversion of the foot. Sural neuropathies are relatively common in the context of generalized peripheral neuropathies, but isolated sural neuropathy is rare. Sural nerve injury or irritation in the acute phase may be secondary to displaced bone fragment,

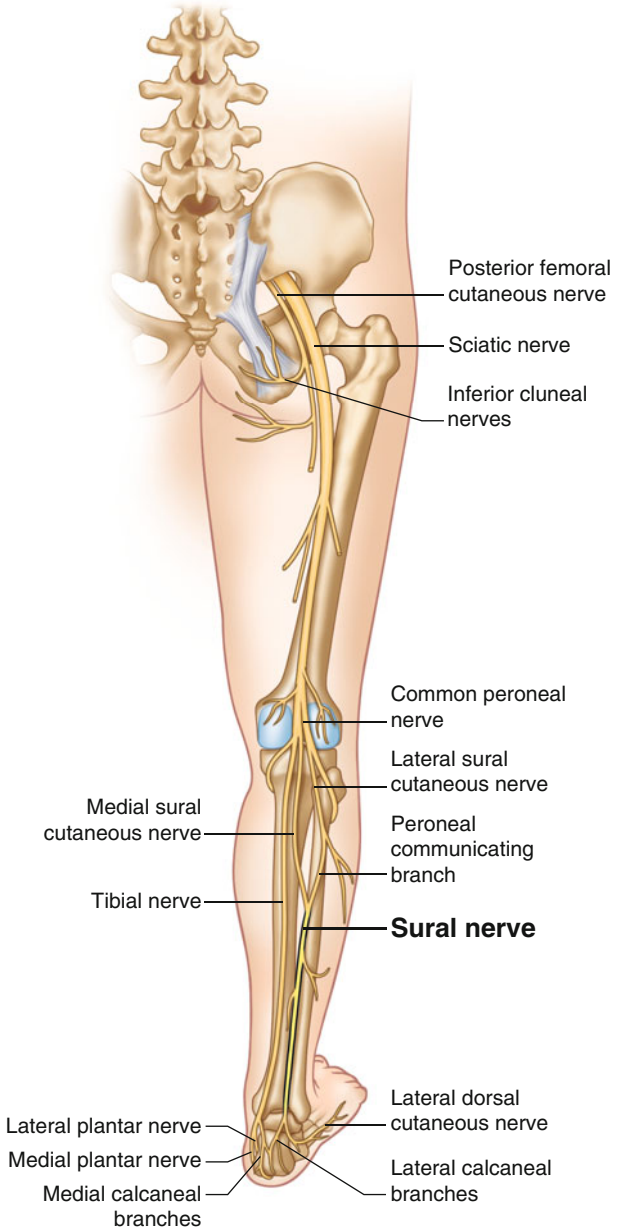

Fig. 71.4 Anatomy of the distal sciatic nerve (Image courtesy of Springer)

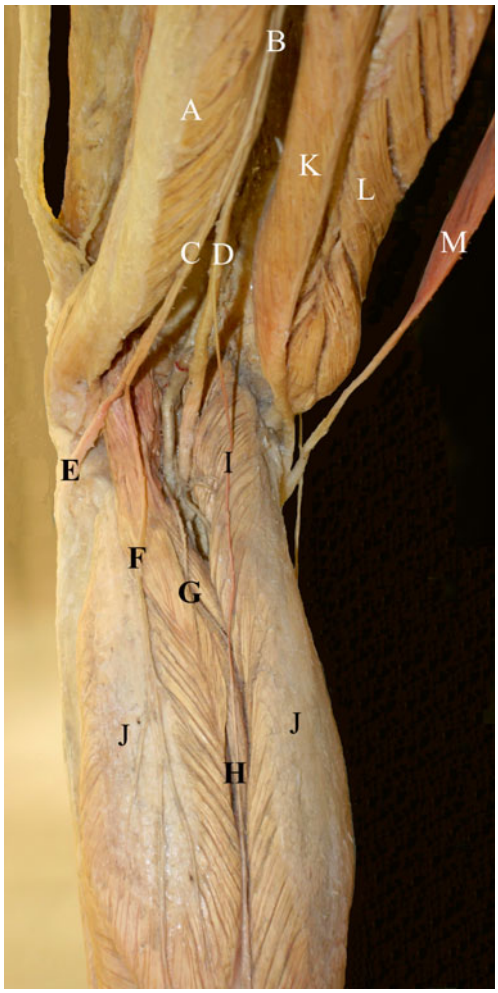

Fig. 71.5 Dissection of the posterior thigh and calf, modified from an image from *Bodies*, *The Exhibition*, with permission. *A* biceps femoris muscle, *B* sciatic nerve, *C* peroneal division, *D* tibial division, *E* common peroneal nerve, *F* lateral sural cutaneous nerve, *G* sural communicating branch, *H* sural nerve, *I* medial sural nerve, *J* gastrocnemius muscle, *K* semitendinosus muscle, *L* semimembranosus muscle, *M* gracilis muscle (Image courtesy of Andrea Trescot, MD)

hematoma, or soft tissue edema that can cause nerve compression or irritation.

Symptoms of entrapment at the fibrous arcade near the musculotendinous junction of the Achilles tendon and calf muscles can cause pain that is distally exacerbated with physical exertion but may also cause referred pain proximally into the region of the lateral gastrocnemius [4, 6, 7]. Patients with sural entrapment may present with pain and abnormal sensations such as numbness, tingling, burning, or even prolonged soreness overlying the skin of the foot and lateral ankle.

Physical Exam

The physical exam shows tenderness and often swelling in the calf or behind and below the lateral malleolus. Passive inversion of the ankle can cause pain in the sural nerve distribution, especially if the nerve is tethered along its course. Plantar flexion and inversion of foot reproduces symptoms [18]. There can be hyposensitivity or hypersensitivity along the lateral aspect of the foot and ankle.

Tinel's test is typically not helpful with sural nerve neuropathies. However, local pressure may aggravate sensory symptoms (*Hoffman-Tinel's sign*) and may indicate the site of the lesion [20]. For proximal sural nerve entrapments, there will be tenderness between the heads of the gastrocnemius muscle (Video 71.1) (Fig. 71.10), while for

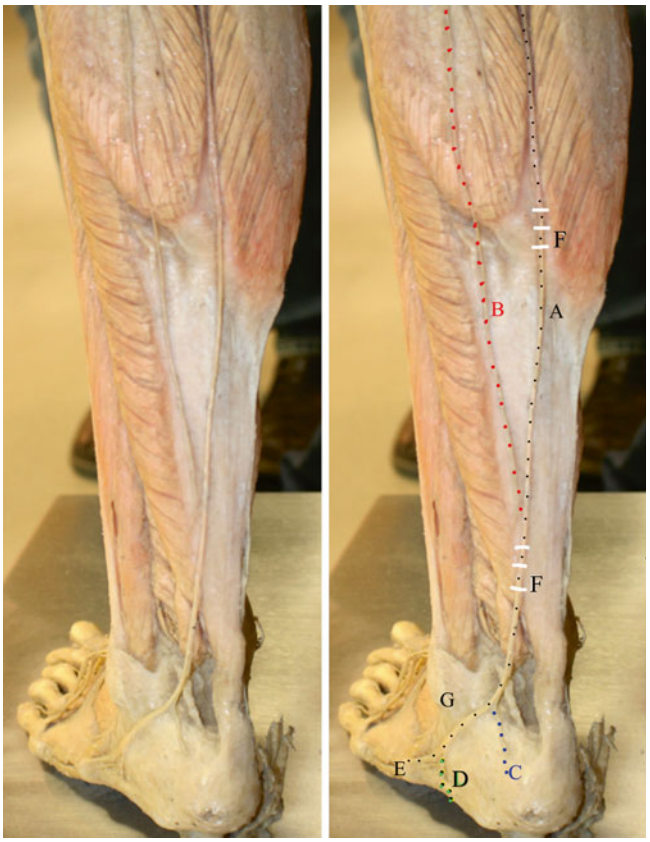

Fig. 71.6 Dissection of the posterior calf and heel, modified from an image from *Bodies*, *The Exhibition*, with permission. *A* (*black*) sural nerve, *B* (*red*) lateral sural cutaneous nerve, *C* (*blue*) posterior calcaneal nerve, *D* (*green*) lateral calcaneal nerve, *E* lateral tuberosity of the fifth metatarsal, *F* (*white*) fibrous arcade, *G* lateral malleolus (Image courtesy of Andrea Trescot, MD)

the more distal entrapments, the tenderness will be lateral to the Achilles tendon (Fig. 71.11) or along the lateral aspect of the foot.

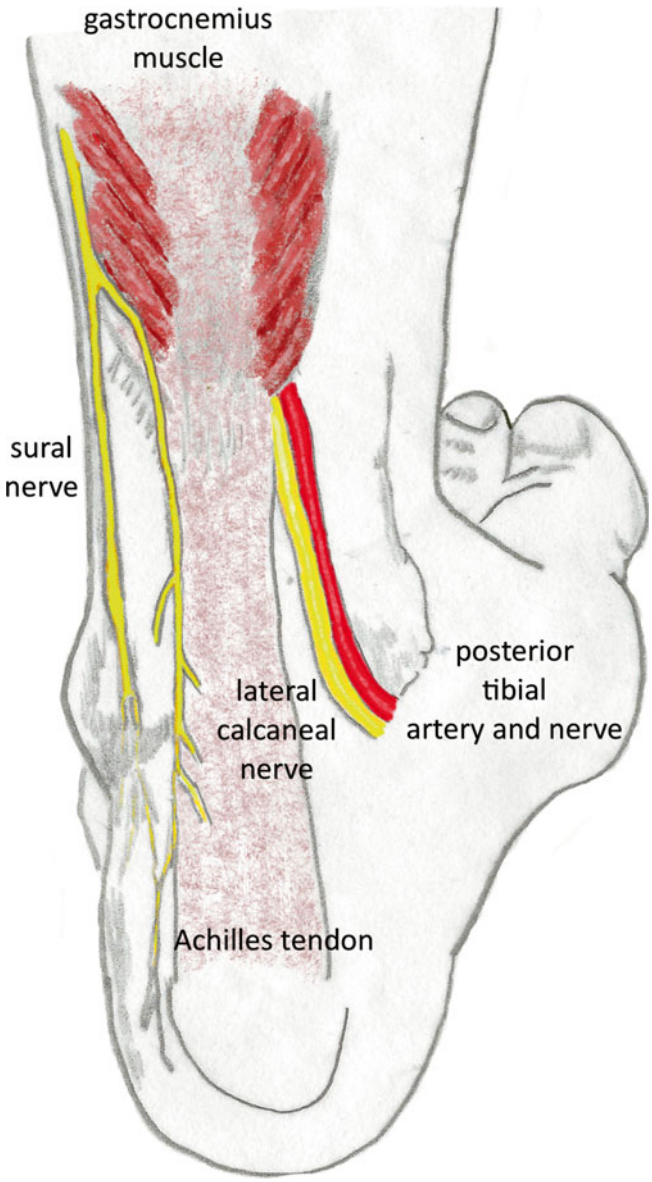

Fig. 71.7 Innervation of the Achilles tendon by the lateral calcaneal nerve (Image courtesy of Michael Brown, MD)

Differential Diagnosis (Table 71.3)

There are many causes of pain down the back of the leg and calf (Fig. 71.12). The diagnosis of sural entrapment is usually based on clinical exam [8]. Ultrasound along the path of the nerve (Fig. 71.13) can help to identify entrapment and pathology of the nerve, as well as provide a road map for diagnostic injections. Electrodiagnostic studies can assist in diagnosing sural neuropathy. Nerve conduction velocity testing, as well as an "orthodromic inching," can help to localize the site of pathology [28]. The studies, however, can be problematic. Because the nerve course is quite superficial, it is subject to recurrent compression and trauma. This can create sural nerve conduction study abnormalities, especially in the elderly, and therefore the findings need to be correlated clinically. An increase in distal latency and a decrease in amplitude of the sensory action potential of the SN could indicate long-termed SN entrapment neuropathy [8]. Schuchmann suggested a standardized approach to the nerve conduction studies of the sural nerve [29]. Sural nerve abnormalities can also be related to foot structure and function, ganglion cyst, varicosities in the leg, bone and joint abnormalities, tumors, tenosynovitis, hypertrophic muscles, and more. For this reason, ultrasonography can often be a very helpful tool in identifying the potential source of sural nerve compression. Magnetic resonance imaging can also be helpful [30, 31] (Fig. 71.14). The diagnostic tests can be found in Table 71.4.

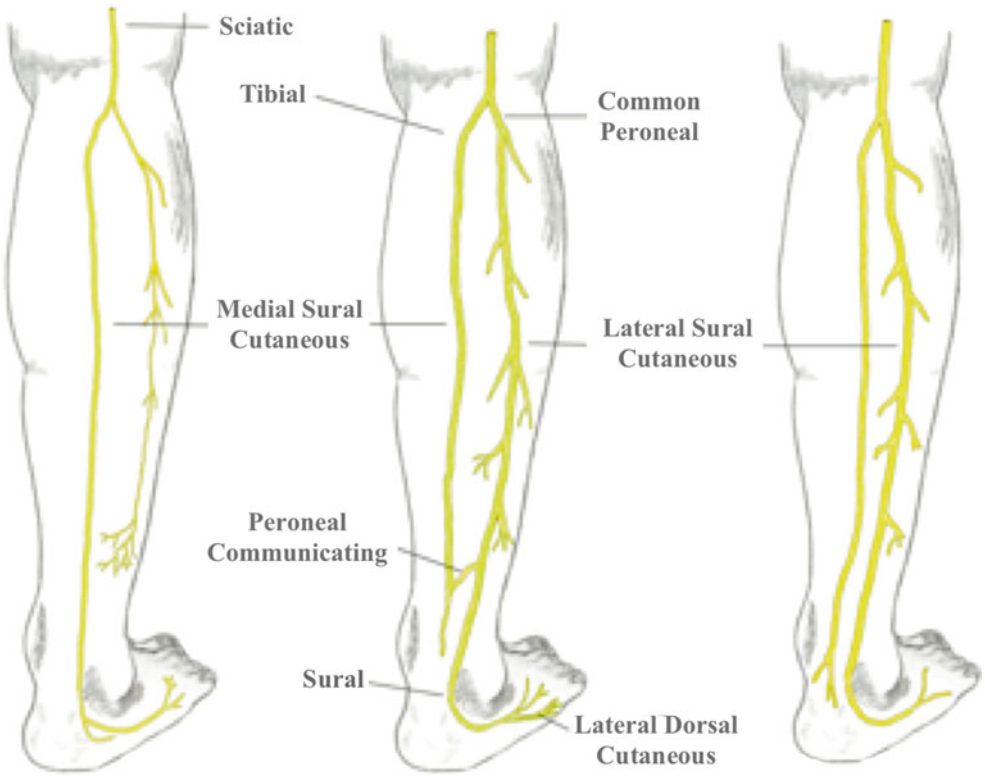

Fig. 71.8 Variations of the anatomy of the sural nerve (Image courtesy of Michael Brown, MD, inspired by Huelke [34])

Identification and Treatment of Contributing Factors

Traumas such as distal fibula and ankle fracture, contusion, and ankle sprain are possible causes of sural nerve pain. In addition, trauma to the nerve may be related to venous stripping, long-distance running, ankle laceration, or external compression of occupational or combat boots.

Although acute causes are often much easier to sort out, one needs to consider the contribution of microtrauma and recurrent compression related to Baker's cyst, constricting straps, bands, tight socks, tight shoelaces, shoe wear, high-heel shoes, and ski boots. Other possible causes may include vasculitis, ganglion cyst, sural nerve biopsy, adhesions after soft tissue injury, neuromas, fractures of the fifth metatarsal, and diabetes, as well as sitting with (or on) crossed ankles [9, 13, 22, 24].

Treatment of contributing factors is critical strategy for clinical management. The most common culprits of sural nerve entrapment are Achilles tendon lengthening surgery, Achilles tendon surgical repair after a torn tendon, ankle fracture, surgery for a broken ankle, flatfoot surgery, and fifth metatarsal fracture surgery. Ricci et al. [23] described that sural nerve ultrasound imaging allows identification of the sites where the nerve and saphenous vein come in close contact. They called these sites "risk points" because of the risk of damage to the nerve during vein procedures (7.5 % of vein stripping and 2 % of endovascular vein ablations). They also recommended US evaluation of the sural nerve location prior to Achilles tendon repairs.

Sometimes the neuritis can be improved with simple treatments like icing and anti-inflammatories. It is important, however, to make sure to stop the irritation to the nerve. Get rid of any shoes that might be irritating the nerve, such as some ski boots and cycling shoes. Physical therapy may help mobilize and break up the scar tissue.

Injection Technique

Landmark-Guided Injection

Knowledge of the anatomy and course of the sural nerve is typically all that is necessary for consideration of a landmark-guided sural nerve block. It is important to remember that a therapeutic block or a block for an anesthetic procedure is different than a diagnostic block required for confirmation of sural neuropathy prior to a neurolytic procedure. For therapeutic or sensory blockade, the anesthetic injection can be performed anywhere at or below the fibrous arcade. Below this level, the nerve lies along the lateral aspect of the Achilles tendon.

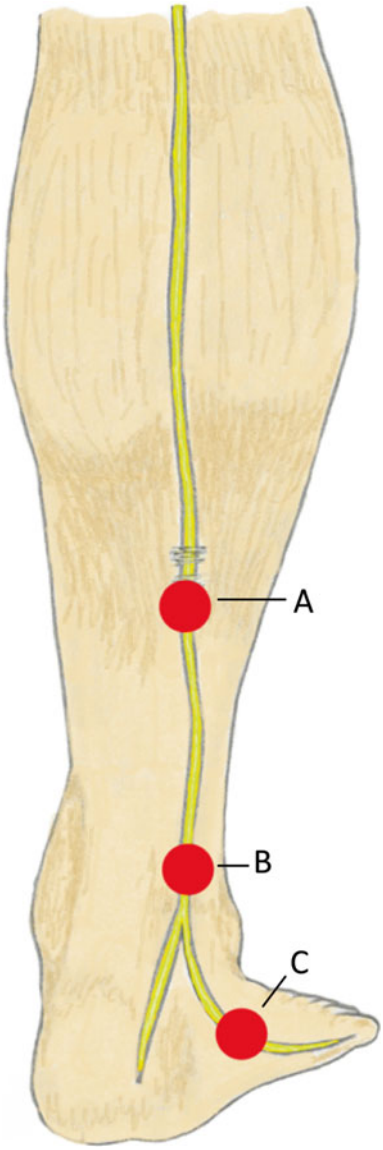

Fig. 71.9 Sites of entrapment of the sural nerve. *A* junction of the gastrocnemius muscle and Achilles tendon, *B* lateral ankle, *C* lateral foot (Image courtesy of Michael Brown, MD)

There are several sites for landmark-guided sural nerve injections:

- Between the heads of the gastrocnemius muscle
- Ten to 15 cm above the lateral malleolus
- Just above the lateral malleolus
- One centimeter posterior to the lateral malleolus

The patient is placed in a side-lying position with the involved side up, though for an injection at the gastrocnemius muscle, a prone position may be easier. Prior to sterile skin prep, identify the heads of the gastrocnemius, posterior malleolus, and the lateral border of the Achilles tendon. For the proximal sural nerve injection, straddle the nerve on either side of the gastrocnemius, and advance a 25-gauge, 2-inch needle parallel to the nerve (Fig. 71.15) (Video 71.2). A peripheral nerve stimulator helps a great deal to identify the nerve, allowing small volumes of injectate (1–2 cc) to be used.

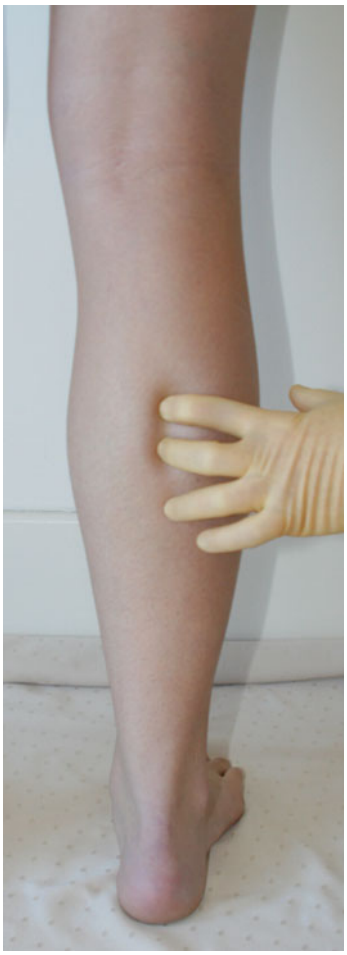

Fig. 71.10 Palpation of the sural nerve between the heads of the gastrocnemius muscle (Image courtesy of Andrea Trescot, MD)

For the more distal landmark-guided injections, mark the skin in the region between the lateral border of the Achilles and the posterior malleolus (Fig. 71.16). Caution should be taken to avoid injection within the Achilles tendon. A 27-gauge, 1–0.5-inch needle or 30-gauge, 1-inch needle is advanced through the skin, taking a posterior to anterior approach, angling superficially toward the lateral malleolus (Video 71.3). Inject approximately 2 cc of injectate after advancing slightly toward the lateral malleolus. Confirm successful anesthesia of the sural nerve block by sensory examination. The use of a peripheral nerve stimulator can increase the success of the injection, avoiding nerve injury and decreasing the amount of local anesthetic needed.

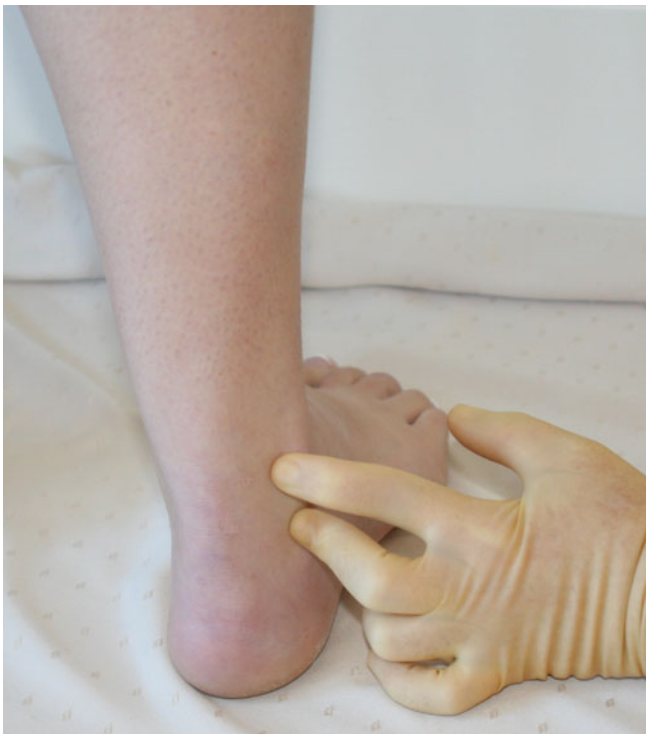

Fig. 71.11 Palpation of the sural nerve at the ankle (Image courtesy of Andrea Trescot, MD)

Table 71.3 Differential diagnosis of lateral leg and foot pain

	Potential distinguishing features
S1 radiculopathy	Motor weakness, proximal leg pain, EMG findings
Plantar fasciitis	Tenderness at the attachment of the flexor retinaculum on the calcaneus
Achilles tendonitis	Tenderness more posteriorly at the Achilles tendon attachment
Retrocalcaneal bursitis	Tenderness on palpation at medial and/or lateral Achilles attachment, US evaluation showing bursa
Osteomyelitis	Fever, elevated WBC, increased ESR, + culture
Ganglion cyst [25]	MRI or ultrasound showing cystic structure
Interdigital neuroma [26]	See Chap. 70
Infection (osteomyelitis) [27]	Bone scan or MRI showing increased uptake and edema
Varicosities	US evaluation
Tenosynovitis	Tenderness along the tendon sheath

Fluoroscopic-Guided Injection

These injections are superficial, with no specific bony landmarks, so little if any benefit is gained by using fluoroscopy, with added radiation exposure to the patient and clinician.

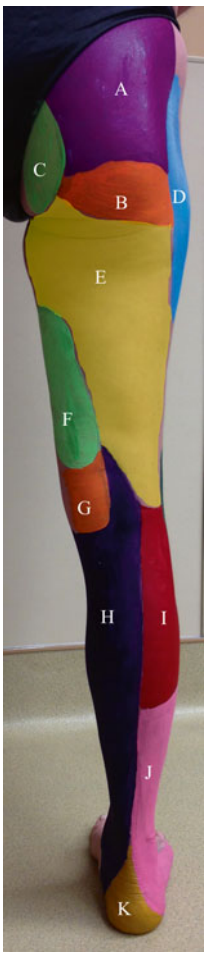

Fig. 71.12 Pain pattern from nerves of posterior leg. *A* superior cluneal nerve, *B* inferior cluneal nerve, *C* medial cluneal nerve, *D* lateral femoral cutaneous nerve, *E* posterior femoral cutaneous nerve, *F* femoral nerve, *G* obturator nerve, *H* saphenous nerve, *I* lateral sural cutaneous nerve, *J* sural nerve, *K* posterior tibial nerve (Image courtesy of Terri Dallas-Prunskis, MD)

Ultrasound-Guided Injection

The ultrasound-guided sural nerve injection can be carried out anywhere along the path of the sural nerve (Fig. 71.17). Typically, the injection is performed either at the fibrous arcade near the musculotendinous junction of the gastrocnemius muscle approximately 10–15 cm above the lateral malleolus (Fig. 71.17a) or just above or behind the lateral malleolus (Fig. 71.17b). Since the lateral malleolus provides a bony prominence that interferes with easy placement of the linear ultrasound transducer, the injection is typically carried out above it. If an ultrasound-guided injection is done near the malleolus, additional gel will need to be utilized to aid in obtaining the ultrasound image for the procedure; because of the irregularity of the bone in this region, extra gel helps to maintain the probe/skin interface.

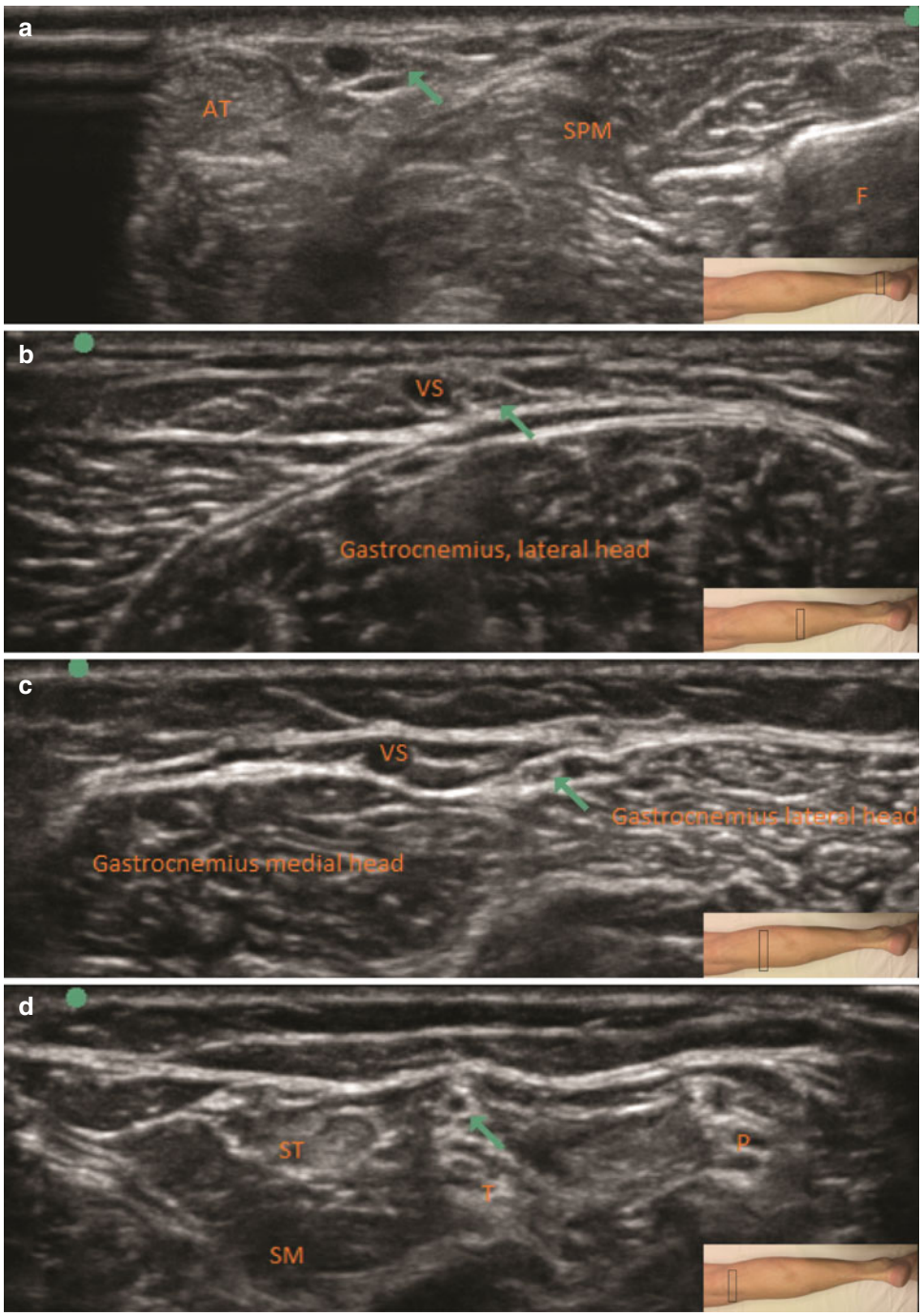

Fig. 71.13 Ultrasound identification of the sural nerve (*green arrow*): (**a**) at the level of the ankle, (**b**) at the level mid-calf, just lateral to the small saphenous vein, (**c**) just inferior to the popliteal fossa, (**d**) popliteal fossa where the medial sural nerve joins the tibial nerve. *AT* Achilles tendon, *F* fibula, *SPM* short peroneal muscle, *VS* small saphenous vein, *T* tibial nerve, *ST* semitendinosus muscle, *SM* semimembranosus muscles (Image courtesy of Agnes Stogicza, MD)

If desired, once the sural nerve is identified, it is usually possible to move the probe proximally to identify the point of separation of the two complements of the sural nerve: the MSCN, located inside the triangle of connective tissue below the small saphenous vein joining the tibial nerve, and the LSCN, which joins the common peroneal nerve (see Chap. 72).

To inject the sural nerve at the gastrocnemius, the patient is placed in a prone position and the high frequency probe is positioned horizontally to identify the sural nerve between the heads of the gastrocnemius at the musculotendinous junction (Fig. 71.18). Out-of-plane injections may be easier at this site, especially with the use of a peripheral nerve stimulator.

For the injection at the lateral malleolus, the patient is placed in a side-lying position. The high-frequency linear transducer is placed transversely over the lateral malleolus until the small saphenous vein is identified. Placing a tourniquet proximally will help engorge the vein and assist in identifying the small saphenous vein. Only gentle pressure on the transducer should be used in order to prevent collapsing the vein, which is used to identify the small saphenous nerve lateral to the Achilles tendon (Fig. 71.19). The nerve will typically be oval shaped

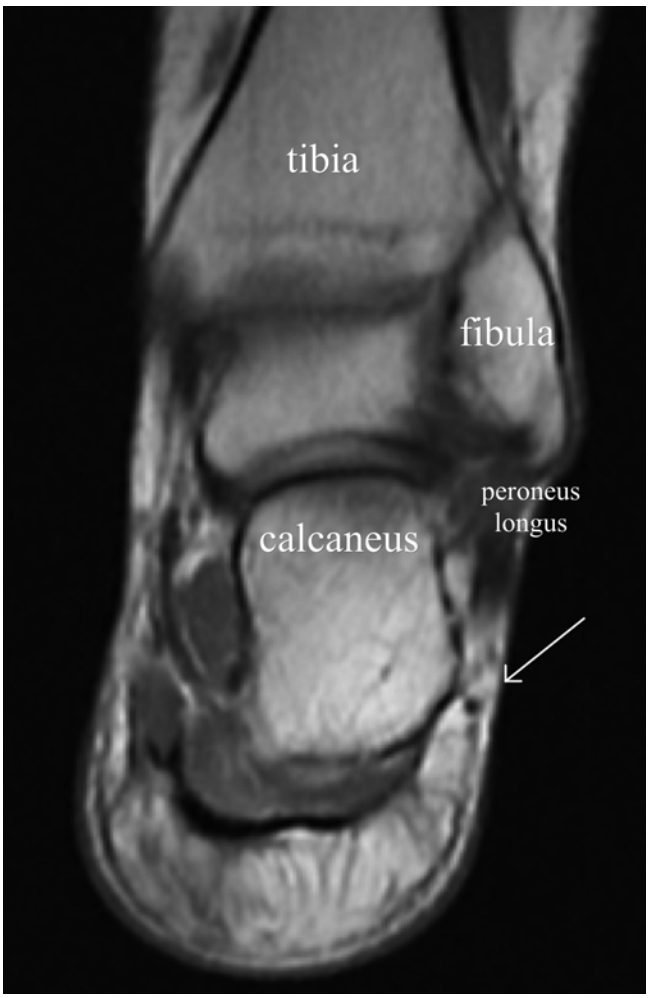

Fig. 71.14 Coronal T1 ankle MRI; *arrow* identifies sural nerve at the peroneus longus tendon attachment (Image courtesy of Andrea Trescot, MD)

Table 71.4 Diagnostic tests for sural neuropathy

	Potential distinguishing features
Physical exam	Primary modality
Diagnostic injection	Diagnostic as well as potentially therapeutic
Ultrasound	Helpful to identify potential sources of compression
MRI	May be helpful to identify extrinsic compression
Arteriography	Not useful
X-ray	To exclude bony pathology
Electrodiagnostic studies	May be useful, but need to compare to opposite leg

and can be better visualized by tilting the transducer or moving the transducer up and down the limb for short distance, which causes the soft tissues to appear "out of focus," while the nerve will seem to maintain its shape and echogenicity.

Another option is to use an acoustic "standoff" pad under the ultrasound transducer, which can enhance resolution of

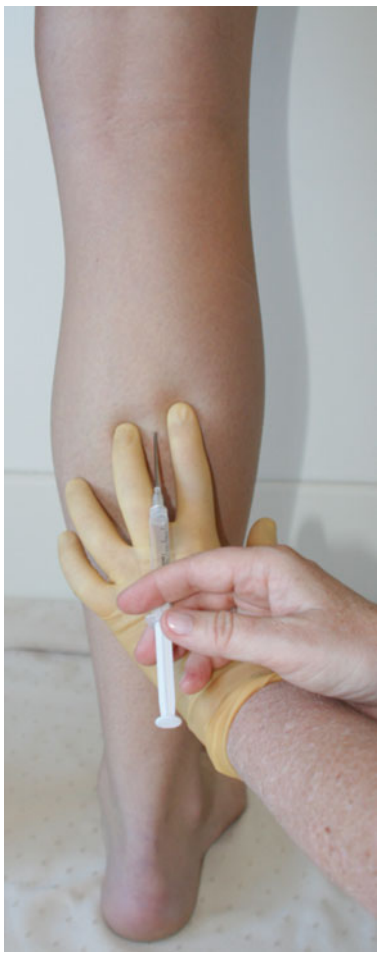

Fig. 71.15 Landmark-guided injection of the sural nerve at the gastrocnemius muscle (Image courtesy of Andrea Trescot, MD)

superficial structures. The injection can be performed utilizing the in-plane approach (Fig. 71.19) or the out-of-plane approach (Fig. 71.20).

If the nerve is not well visualized, the targeted injection can be performed around the vein and will still provide adequate anesthesia. An ultrasound-guided technique described by Redborg et al. [17] using the lesser saphenous vein as a reference point at the lateral aspect of the lateral malleolus resulted in an effective sural nerve block.

Neurolytic Techniques

Cryoneuroablation

There are occasions when cryoneuroablation of the sural nerve may be indicated. If considered, cryoneuroablation should be done just proximal to the site of compression. A careful physical examination and evaluation of the results of a diagnostic block need to be considered prior to the procedure. Before any cryoneuroablation procedure, confirmation should

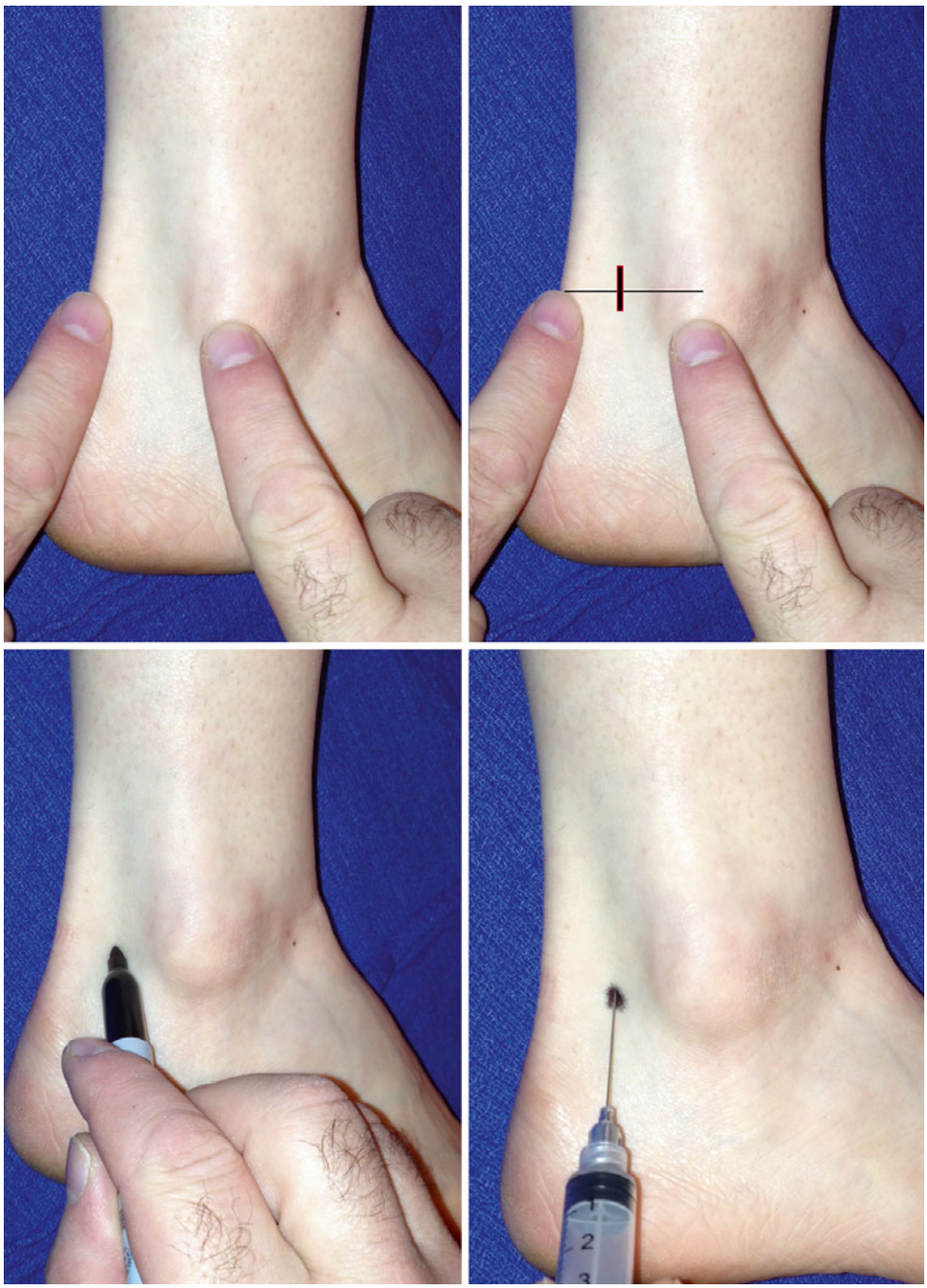

Fig. 71.16 Landmark-guided injection of the sural nerve at the ankle (Image courtesy of Michael Brown, MD)

be carried out with no more than 0.5 cc of local anesthetic injected with a peripheral nerve stimulator or under the direct visualization of ultrasound. The probe is advanced parallel to the nerve, and the built-in nerve stimulator (as well as possibly ultrasound guidance) is used to place the probe as close as possible to the nerve. Rhames et al. [32] described the use of cryoneuroablation to treat chronic sural neuroma after multiple lower extremity surgeries and skin grafts (Fig. 71.21).

Radiofrequency Lesioning

Conventional radiofrequency lesioning is not indicated for sural entrapment, but pulsed radiofrequency lesioning has been reported [33]. The author described using US to localize the nerve, which was then lesioned using a pulse count of 240 pulses, a temperature of 43 °C, a rate of 2 pulses per second, and a pulse width of 20 Ms.

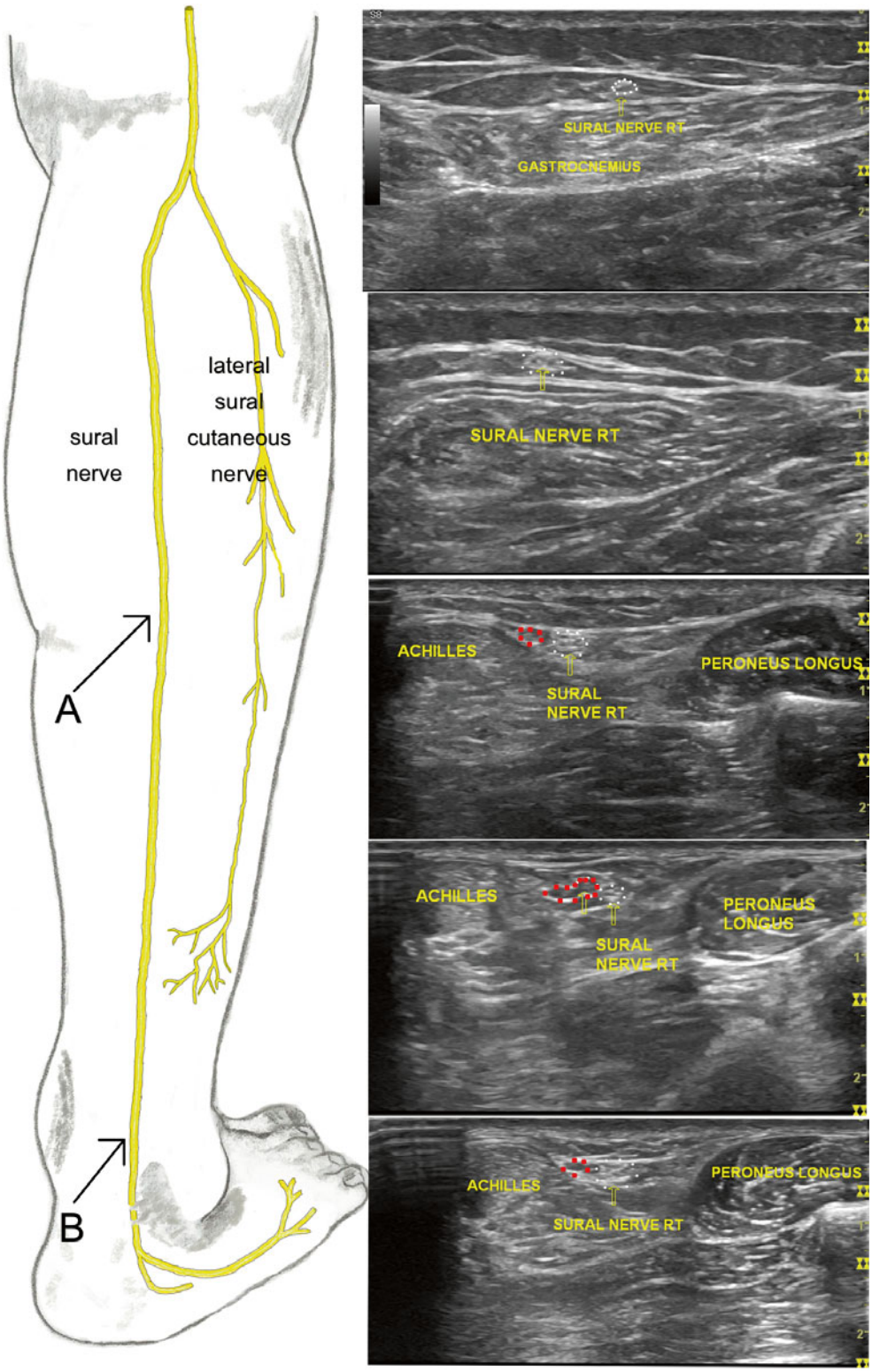

Fig. 71.17 Ultrasound identification of the sural nerve for injection. *A* the proximal injection site, *B* the distal injection site. *Red dots* outline the small saphenous vein (Image courtesy of Michael Brown, MD)

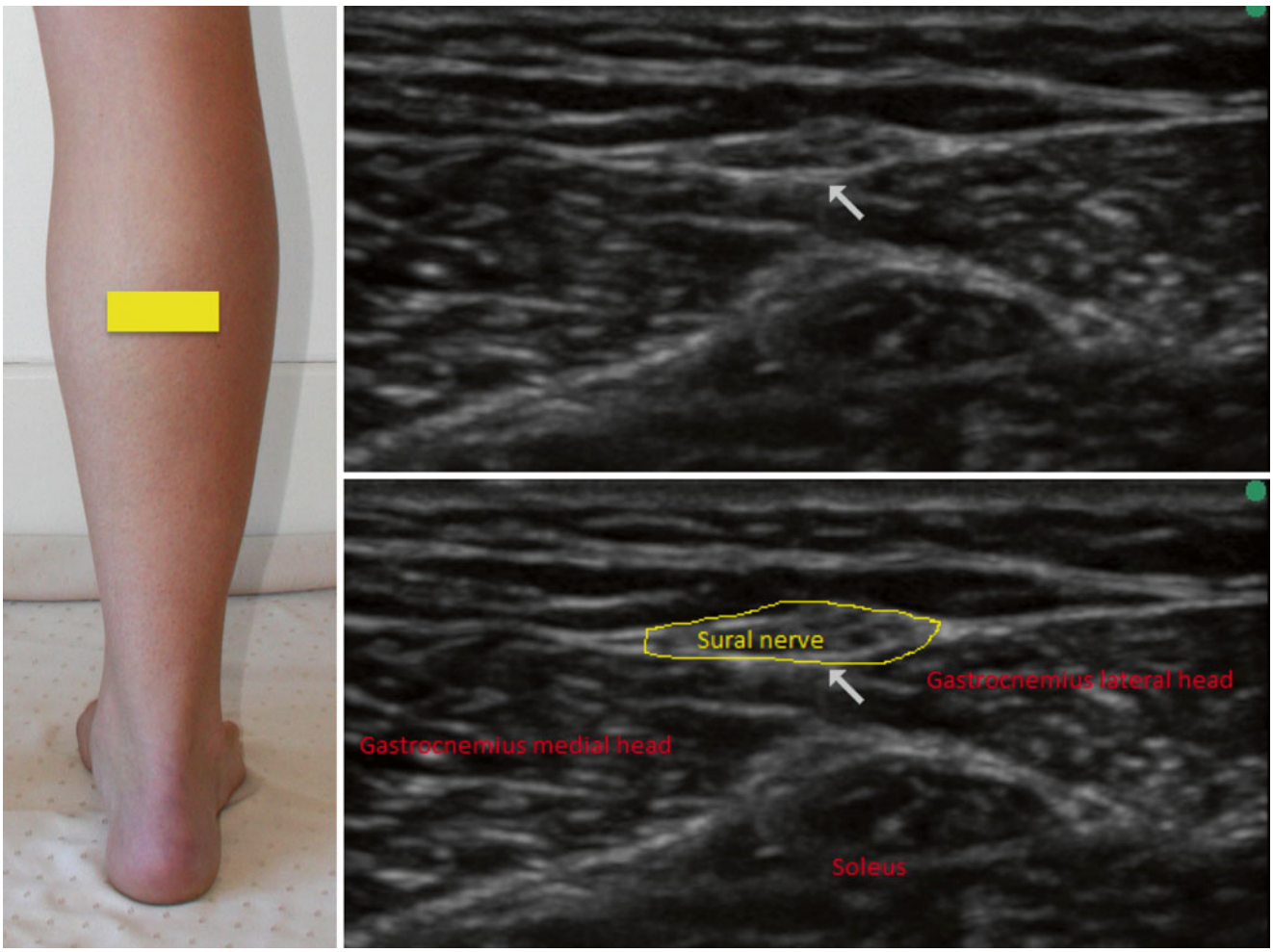

Fig. 71.18 Ultrasound image of the sural nerve between the heads of the gastrocnemius muscle (Image courtesy of Agnes Stogicza, MD)

Phenol/Alcohol Denervation

There have been no reports of phenol or alcohol denervation of the sural nerve.

Surgery

Space-occupying lesions will need surgical excision. Sometimes, surgery is required to release fascial entrapments or free scar tissue from around the nerve, but it should always be a last resort. Pringle et al. [1] described some of the first surgical releases of the sural nerve. They felt that the presence of a ganglion near the nerve was a helpful sign in the diagnosis. They also noted that one of their patients had a concomitant *tarsal tunnel* entrapment (see Chap. 73). The SN is frequently targeted for nerve biopsies and as a donor nerve for nerve transplants.

Complications

Any injection may cause the usual complications of bleeding, infection, and nerve damage. Sensory dysesthesias and neuropathic pain can occur with incomplete lesioning of the sural nerve. Consideration for repeat procedure may need to be made if such a complication is encountered.

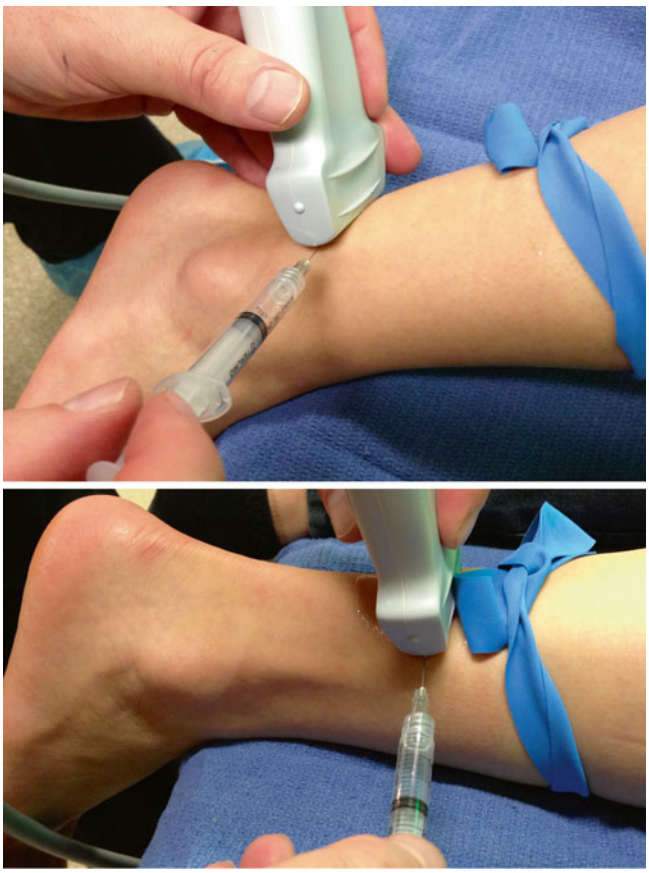

Fig. 71.19 Ultrasound-guided injection of the sural nerve at the ankle. Note the tourniquet and the in-plane injection technique (Image courtesy of Michael Brown, MD)

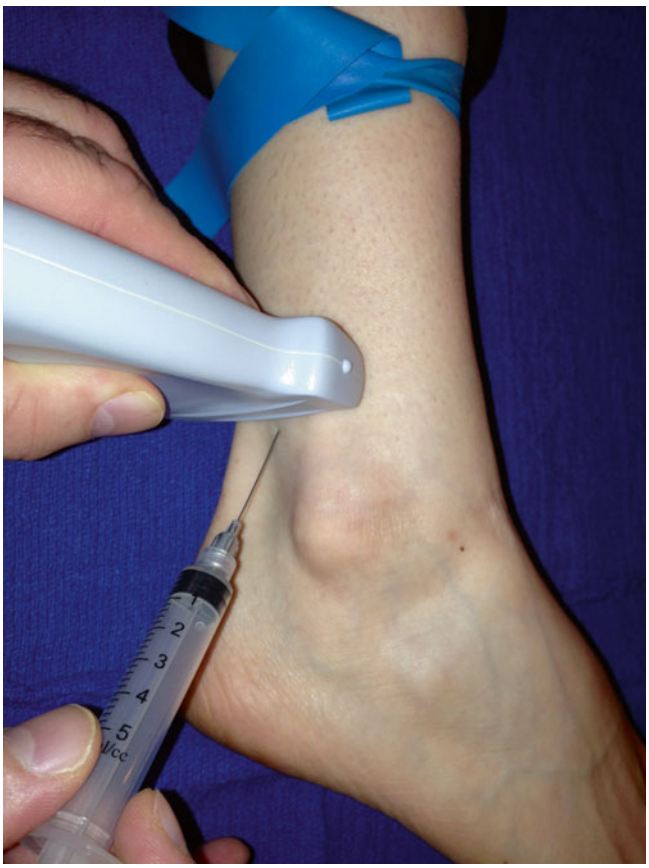

Fig. 71.20 Ultrasound-guided injection of the sural nerve at the ankle. Note the tourniquet and the out-of-plane injection technique (Image courtesy of Michael Brown, MD and Agnes Stogicza, MD)

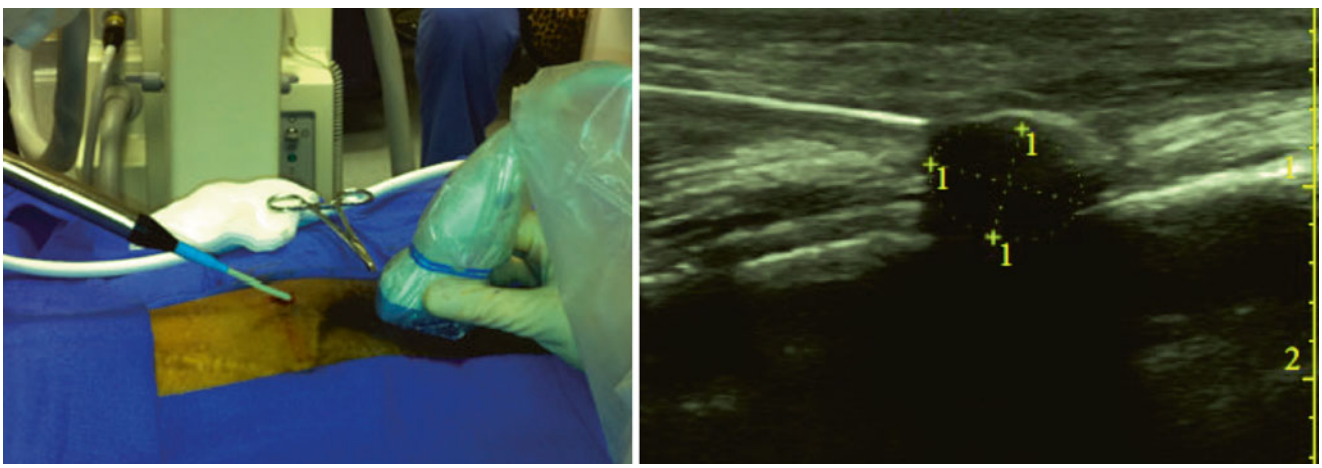

Fig. 71.21 Ultrasound-directed cryoneuroablation of the sural nerve (From Rhame et al. [32]. Copyright © 2011 Ellen E. Rhame et al., reprinted with permission)

Summary

Sural neuritis is a painful foot condition arising from irritation of the sural nerve. In most cases, a careful history and physical exam can establish the cause of the pain, along with the ultrasound, electrodiagnostic studies and MRI tests, and diagnostic blocks.

References

1. Pringle RM, Protheroe K, Mukherjee SK. Entrapment neuropathy of the sural nerve. J Bone Joint Surg Br. 1974;56B(3):465–8.
2. George BM, Nayak S. Sural nerve entrapment in gastrocnemius muscle – a case report. Neuroanatomy. 2007;8:41–2.
3. Birbilis TH, Ludwig HC, Markakis E. Neuropathy of the sural nerve caused by external pressure. Acta Neurochir (Wien). 2000;142(8):951–2.
4. Schon LC. Nerve entrapment, neuropathy, and nerve dysfunction in athletes. Orthop Clin N Am. 1994;25(1):47–59.
5. Schuchmann JA. Isolated sural neuropathy: report of two cases. Arch Phys Med Rehabil. 1980;61(7):329–31.
6. Bora Jr FW, Osterman AL. Compression neuropathy. Clin Orthop Relat Res. 1982;163:20–32.
7. Schon LC, Baxter DE. Neuropathies of the foot and ankle in athletes. Clin Sports Med. 1990;9(2):489–509.
8. Paraskevas GK, Natsis K, Tzika M, Ioannidis O. Fascial entrapment of the sural nerve and its clinical relevance. Anat Cell Biol. 2014;47(2):144–7.
9. Refaeian M, King JC, Dumitru D. Isolated sural neuropathy presenting as lateral ankle pain. Am J Phys Med Rehabil. 2001; 80(7):543–6.
10. Bora F, Osterman A. Compression neuropathy. Clin Orthop. 1982;163:20–32.
11. Schon L. Nerve entrapment, neuropathy, and nerve dysfunction in athletes. Clin Orthop. 1994;25:47–59.
12. Schon L, Baxter D. Neuropathy in the foot and ankle in athletes. Clin Sports Med. 1990;9:489–508.
13. Stickler DE, Morley KN, Massey EW. Sural neuropathy: etiologies and predisposing factors. Muscle Nerve. 2006;34(4):482–4.
14. Fabre T, Montero C, Gaujard E, Gervais-Dellion F, Durandeau A. Chronic calf pain in athletes due to sural nerve entrapment. A report of 18 cases. Am J Sports Med. 2000;28(5):679–82.
15. Pasternack WA, Lipp RM. Idiopathic sural neuroma. A case report. J Am Podiatr Med Assoc. 1992;82(8):424–7.
16. Mestdagh H, Drizenko A, Maynou C, Demondion X, Monier R. Origin and make up of the human sural nerve. Surg Radiol Anat. 2001;23(5):307–12.
17. Redborg KE, Antonakakis JG, Beach ML, Chinn CD, Sites BD. Ultrasound improves the success rate of a tibial nerve block at the ankle. Reg Anesth Pain Med. 2009;34(3):256–60.
18. Donovan A, Rosenberg ZS. MRI of ankle and lateral hindfoot impingement syndromes. AJR Am J Roentgenol. 2010;195(3): 595–604.
19. Riedl O, Frey M. Anatomy of the sural nerve: cadaver study and literature review. Plast Reconstr Surg. 2013;131(4):802–10.
20. Kavyashree AN, Subhash LP, Asha KR, Bindu Rani MK. Anatomical variations in formation of sural nerve in adult Indian cadavers. J Clin Diagn Res. 2013;7(9):1838–41.
21. Sekiya S, Tokita K, Banneheka SK, Suzuki R, Miyawaki M, Chiba S, et al. Reexamination of the communicating branch between the sural and tibial nerves. Kaibogaku Zasshi J Anat. 2006;81(3): 83–8.
22. Arimura K, Arimura Y, Ng AR, Ohkatsu Y. Inching method in compression neuropathy of the sural nerve distal to the ankle. Electromyogr Clin Neurophysiol. 2005;45(4):229–32.
23. Ricci S, Moro L, Antonelli IR. Ultrasound imaging of the sural nerve: ultrasound anatomy and rationale for investigation. Eur J Vasc Endovasc Surg Off J Eur Soc Vasc Surg. 2010;39(5):636–41.
24. Reisin R, Pardal A, Ruggieri V, Gold L. Sural neuropathy due to external pressure: report of three cases. Neurology. 1994;44(12): 2408–9.
25. Fujita I, Matsumoto K, Minami T, Kizaki T, Akisue T, Yamamoto T. Tarsal tunnel syndrome caused by epineural ganglion of the posterior tibial nerve: report of 2 cases and review of the literature. J Foot Ankle Surg. 2004;43(3):185–90.
26. Lam S. Tarsal tunnel syndrome. J Bone Joint Surg Br. 1967; 49(1):87–92.
27. Baghla DP, Shariff S, Dega R. Calcaneal osteomyelitis presenting with acute tarsal tunnel syndrome: a case report. J Med Case Rep. 2010;4:66.
28. Kim Y, Kim DH, Kim NH, Kim BH, Park BK. Dorsal sural neuropathy: electrophysiological, ultrasonographic, and magnetic resonance imaging findings. Muscle Nerve. 2012;46(4):597–600.
29. Schuchmann JA. Sural nerve condition: a standardized technique. Arch Phys Med Rehabil. 1977;58:166–8.
30. Donovan A, Rosenberg ZS, Cavalcanti CF. MR imaging of entrapment neuropathies of the lower extremity. Part 2. The knee, leg, ankle, and foot. Radiographics. 2010;30(4):1001–19.
31. Martinoli C, Court-Payen M, Michaud J, Padua L, Altafini L, Marchetti A, et al. Imaging of neuropathies about the ankle and foot. Semin Musculoskelet Radiol. 2010;14(3):344–56.
32. Rhame EE, Debonet AF, Simopoulos TT. Ultrasonographic guidance and characterization of cryoanalgesic lesions in treating a case of refractory sural neuroma. Case Rep Anesthesiol. 2011;2011:691478.
33. Todorov L. Pulsed radiofrequency of the sural nerve for the treatment of chronic ankle pain. Pain Physician. 2011;14(3):301–4.
34. Huelke DF. A study of the formation of the sural nerve in adult man. Am J Phys Anthropol. 1957;15(1):137–47.

Lateral Sural Cutaneous Nerve Entrapment

72

Heath McAnally

Introduction

The cutaneous innervation of the upper lateral aspect of the lower limb below the knee is supplied by a nerve traditionally referred to as the *lateral sural cutaneous nerve* (LSCN). It is part of the *sural nerve complex*, a highly variable network of sensory nerves derived from both the tibial nerve and the *common fibular nerve* (CFN, known classically as the *common peroneal nerve*). Given its variable origin and course, it is no wonder there exist several synonyms for it in the literature. It is also referred to as the *lateral cutaneous nerve of the calf* (LCNC) [1], *lateral sural nerve,* and *peroneal communicating nerve,* although the latter is typically used to describe an entirely different nerve arising from the CFN or less commonly from the LSCN itself.

The LSCN, as its name implies, serves the lateral aspect of the proximal lower leg. Occasionally, when it contributes to the sural nerve either directly or via a communicating branch, it may be assumed to serve the entire lateral aspect of the lower leg in the sural distribution. The LSCN typically separates from the CFN proximally and deep to the superficial course of the CFN as it travels over the head of the fibula. As such, it is far less vulnerable to injury than the CFN itself, which is frequently injured by seemingly innocuous reasons such as leg crossing or lithotomy stirrups.

Entrapment of this nerve may occur after trauma or surgery and may cause pain in the lateral lower extremity ranging from the lateral aspect of the knee down to the lateral foot. Pain from entrapment of this nerve must be differentiated from that arising from other branches of the CFN (see Chap. 67) or rarely from the *posterior femoral cutaneous nerve* (see Chap. 62).

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Clinical Presentation (Table 72.1)

Entrapment of the LSCN is rarely described, likely due to its relatively short and protected course through muscle, without close approximation to unyielding structures such as bones or retinacula. When the nerve is injured, however, it may present as pain (usually of a neuropathic "burning" or "pins-and-needles" nature) in the lateral aspect of the proximal lower leg or even the lateral aspect of the knee and calf (Fig. 72.1). There can be tremendous overlap of the nerves in this region, and nerve patterns are variable (Fig. 72.2).

Anatomy (Table 72.2)

The sciatic nerve divides just above the popliteal fossa into the tibial nerve and the CFN; the CFN travels around the fibular head, dividing into deep and superficial branches (see Chaps. 67, 68, and 69). Proximal to the fibular head, the CFN gives off two cutaneous branches, the *sural cutaneous nerve* and the LSCN. The term "LSCN" has also been used to describe the common trunk of the branch off the CFN, which then splits into the sural cutaneous nerve and what has been called the *lateral cutaneous nerve of the calf* (LCNC) [1], leading to even more confusion of the terminology.

The LSCN, along with the rest of the sural nerve complex, is variable in its origin, course, and distribution (Fig. 72.3). Riedl and Frey's excellent and comprehensive review of the sural nerve complex, comprised of data from over 2500 sural nerves described in the literature, notes that:

Table 72.1 Occupation/exercise/trauma history relevant to lateral sural cutaneous nerve entrapment

Compression	Peri-popliteal cystic bursitis [2]
	Lithotomy straps
	Prolonged crossed legs
Trauma	Lateral lower leg injury/surgery
Entrapment	Prolonged positions with knee flexed [1]

A.M. Trescot (ed.), *Peripheral Nerve Entrapments: Clinical Diagnosis and Management*, DOI 10.1007/978-3-319-27482-9_72

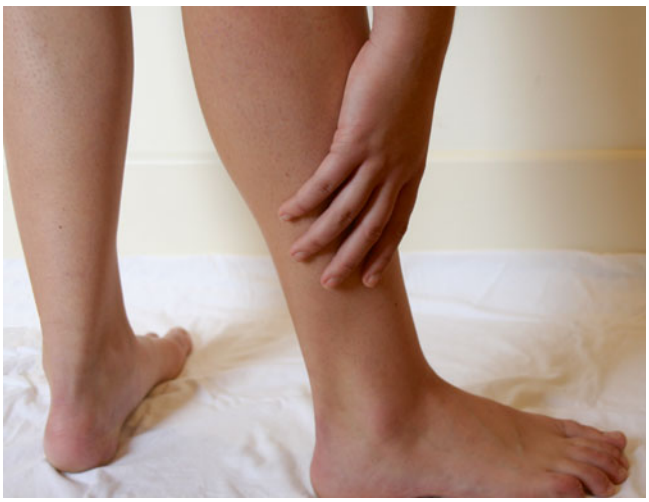

Fig. 72.1 Patient pain complaint from lateral sural cutaneous nerve entrapment (Image courtesy of Andrea Trescot, MD)

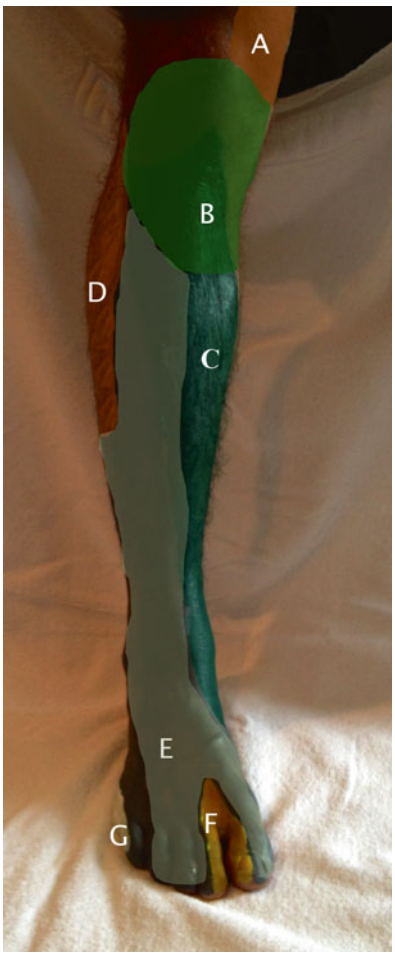

Fig. 72.2 Areas of lower extremity nerve sensation. *A* obturator nerve, *B* infrapatellar saphenous nerve, *C* superficial saphenous nerve, *D* lateral sural cutaneous nerve, *E* superficial peroneal nerve, *F* deep peroneal nerve, *G* sural nerve (Image courtesy of Terri Dallas-Prunskis, MD)

Table 72.2 Lateral sural cutaneous nerve anatomy

Origin	Common peroneal (fibular) nerve (CFN)
General route	The LSCN arises from the CFN proximal to the fibular head and travels to the lateral calf as the lateral cutaneous nerve of the calf (LCNC); LSCN usually joins the medial sural cutaneous to make up the sural nerve
Sensory distribution	Posterolateral aspect of the lower leg and ankle as well as the dorsal aspect of the foot
Motor innervation	No motor function
Anatomic variability	Sometimes the sural communicating nerve comes off the CFN vs LSCN; sural may provide all the lateral calf sensation with minimal (or no) LSCN contribution
Other relevant structures	Popliteal crease, gastrocnemius, fibular head

> Detailed descriptions of the lateral sural cutaneous nerve are lacking. Most sural nerve reports omit details on the lateral sural cutaneous nerve, as it usually does not contribute directly to the sural nerve. Referring to those few existing and our own experience, the lateral sural cutaneous nerve typically originates subfascially from the common peroneal nerve. Its exact height varies—we found it most frequently at the popliteal crease from the posteromedial quadrant of the common peroneal nerve. It courses down between the lateral head of the gastrocnemius and the crural fascia. In our series, it passed approximately 1 cm behind the fibular head and penetrated the fascia within the proximal calf—sometimes as a single nerve, but more frequently (n =19 [68 percent]) after ramification into its terminals to the lateral-dorsal calf. The lateral sural cutaneous nerve may—exceptionally— represent the sural nerve (patterns IV and V). Except for the peroneal communicating branch, the lateral sural cutaneous nerve branches are highly variable; no report or anatomical textbook specifically characterized or labeled them [3].

Huelke's staggering dissection series of the sural nerve complex (550 limbs) from 1957 to 1958 is frequently referenced as the benchmark dataset for these nerves [4, 5]. In his 1958 series, he reported that of 198 limbs, 159 of them (roughly 80 %) contained a sural nerve derived from the medial sural cutaneous nerve (MSCN) and what he calls the peroneal communicating nerve (PCN) (Fig. 72.4). In 93 of these 159 specimens, the LSCN was associated with the PCN, either with the PCN as the parent nerve (56 %), derivative branches of the PCN (22 %), or arising from a trunk common to both the LSCN and the PCN (21 %). In the remaining 66 specimens, the LSCN was not associated with the PCN but arose completely distinctly from the CFN. In 35 specimens (22 % overall), no LSCN was found, and its normal territory appeared to be served by the posterior femoral cutaneous nerve and the saphenous nerve.

More recent dissection series [6–10] are fraught with difficulties in both interpretation and comparison due to lack of uniform nomenclature; however, two common themes are seen:

- A communication from the CFN (sometimes labeled the LSCN) contributes to the sural nerve in 75 % of cases, on average (standard error of the mean = 5.4 %).

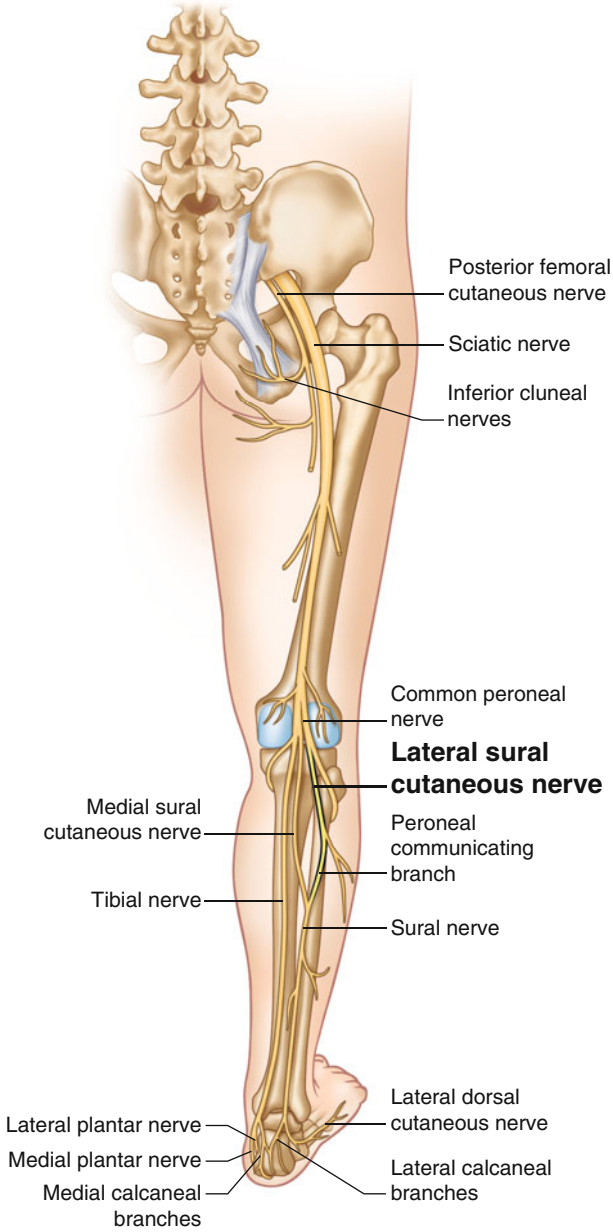

Fig. 72.3 Nerves of the lower extremity (Image courtesy of Springer)

- There is complete heterogeneity of the location of the anastomosis in terms of the level of the calf.

Entrapment

Only three reports of entrapment of the LSCN were found in the literature, and all three referred to the nerve as the lateral cutaneous nerve of the calf. The first report, in 1998, described entrapment of the nerve as it pierced the tendon of the biceps femoris muscle [11]. The second report, in 2006, described entrapment of the nerve by a peri-popliteal cystic bursitis [2]. In the most recent report, in 2013, Khalil et al. [1] reported a patient with an isolated LCNC entrapment from a fibrous band of tissue. The patient had temporary relief from local anesthetic and steroid injections; he opted for surgery (see below) with good relief 1 year later.

Physical Exam

Cutaneous sensitivity or hyperalgesia may be seen in the distribution of the LSCN. Careful palpation or Tinel's test over the course of the nerve from the popliteal fossa to the fibular head may occasionally reveal sensitivity of an entrapped or otherwise inflamed LSCN (Fig. 72.5). Motor function (specifically the tibialis anterior, tibialis posterior, and extensor hallucis longus muscles) and reflexes should be normal.

Differential Diagnosis (Table 72.3)

The most common cause of lateral calf pain is an L5 radiculopathy [1]; however, if the symptoms are purely sensory in nature, LSCN pathology may be the etiology. Given the significant heterogeneity in origin and course of all of the nerves of the sural complex, cutaneous pain in the posterolateral lower leg must be thought of in terms of all of the potential contributors to the region.

The superior border of the LSCN territory abuts that of the *lateral femoral cutaneous nerve* (see Chap. 61) anteriorly and the *posterior femoral cutaneous nerve* posteriorly (see Chap. 62). The anteromedial border of the LSCN territory abuts that of the *intermediate cutaneous nerve* of the thigh (and the *patellar plexus*) proximally and that of the *saphenous nerve* (see Chap. 59) distally. The posteromedial border of the LSCN territory overlaps significantly with that of the *medial sural cutaneous nerve* and the *sural nerve* itself (see Chap. 71). The inferior border of the LSCN abuts that of the *superficial fibular/peroneal nerve* (see Chap. 68). Pain emanating from a lesion of one of these neighbors should present with a distribution more typical of the nerve in question, but when confined to a "border zone," the diagnosis may become more elusive.

Diagnostic Tests (Table 72.4)

The diagnosis is usually confirmed by electrodiagnostics. Needle EMG should be normal, and there should be no delay in the sensory nerve action potential (SNAP) of the sural nerve or the common peroneal nerve across the fibular head. Campagnolo et al. [12] described a technique of NCV, placing the stimulating electrodes 2 cm posteriomedial and 4 cm proximal to the center of the fibular head, with the recording electrode placed 12 cm distally to the stimulation site.

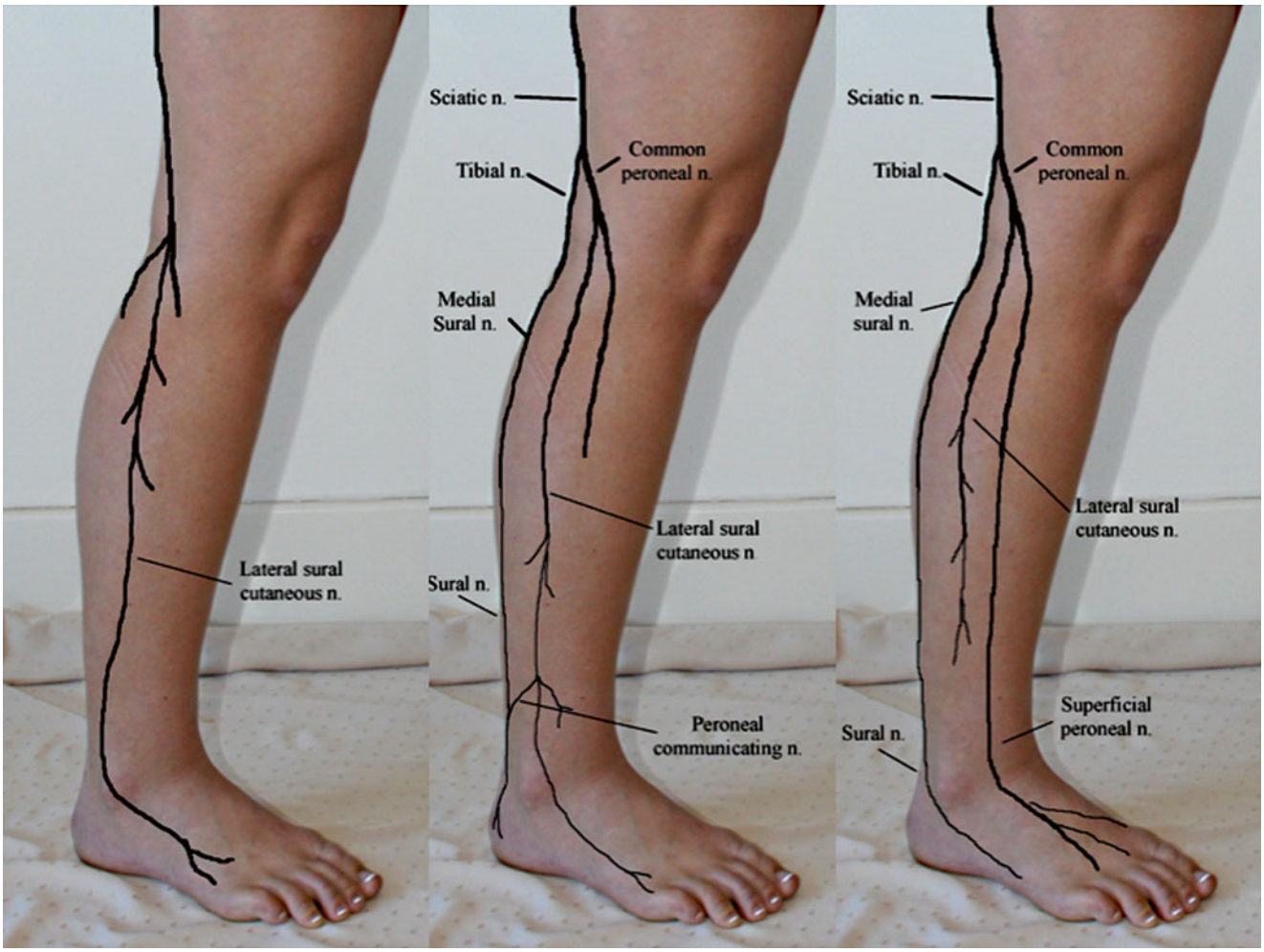

Fig. 72.4 Composite drawing of the variability of the lateral sural cutaneous nerve (Image inspired by Huelke [4], courtesy of Andrea Trescot, MD)

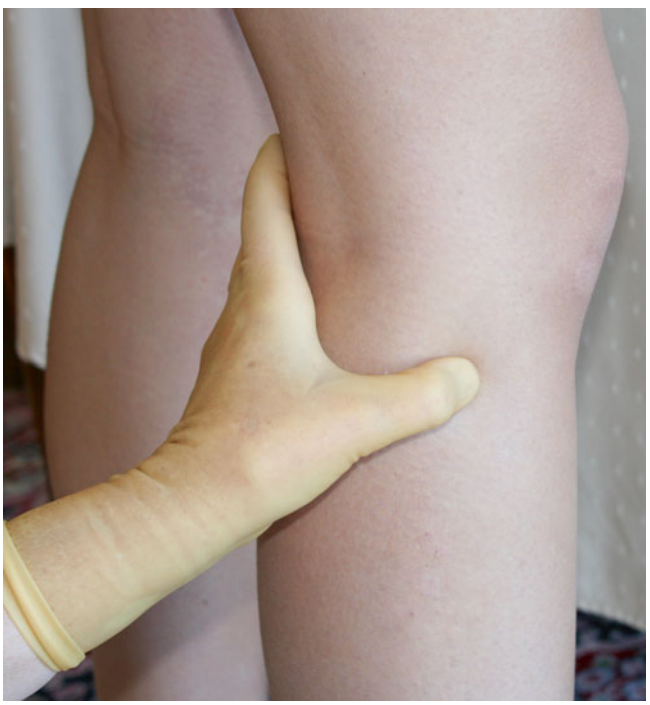

Fig. 72.5 Physical exam of the lateral sural cutaneous nerve (Image courtesy of Andrea Trescot, MD)

Table 72.3 Differential diagnosis of lateral calf pain

	Potential distinguishing features
Lateral femoral cutaneous nerve	Distribution to upper lateral thigh
Posterior femoral cutaneous nerve	Distribution to upper posterior thigh
Saphenous nerve	Distribution to medial thigh and calf to great toe
Superficial peroneal/fibular nerve	Distribution to dorsum of the foot
L5 radiculopathy	Weakness, reflex changes, MRI findings

Injection Techniques

Landmark-Guided Technique

Due to the highly variable course (and even existence) of this nerve, blind injection techniques cannot be recommended.

Table 72.4 Diagnostic tests for lateral sural cutaneous nerve

	Potential distinguishing features
Physical exam	Sensory deficit to upper 2/3rds posterolateral lower leg; Tinel's sign over fibular head; normal strength and reflexes [2]
Diagnostic injection	Necessary for diagnosis (usually with US and PNS)
Ultrasound	CFN is identified in the popliteal fossa and traced distally around the fibula; alternatively, trace the sural nerve cephalad
MRI	Lumbar MRI normal
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies	Antidromic and orthodromic SNAPs [1]

Fluoroscopic-Guided Technique

Because of the highly variable course (and even existence) of this nerve, and the lack of bony landmarks, fluoroscopy is of little utility in either locating or attempting to anesthetize this nerve.

Ultrasound-Guided Technique

Ultrasound is the imaging modality of choice for attempting to locate the LSCN. The *common fibular nerve* (CFN, historically known as the *common peroneal nerve*) (see Chap. 67), which is the "parent" nerve of the LSCN, may be easily visualized in the popliteal fossa after it divides from the tibial component of the sciatic nerve. The US linear probe is used to trace the CFN as it wraps around the head of the fibula (Fig. 72.6) in its anteroinferior course down the leg (Fig. 72.7). Continuous visualization of the CFN in the intervening area is difficult, but its course and depth may be extrapolated with careful examination. Between these two areas, it is sometimes possible to see a peroneal communicating nerve, or LSCN, or a common trunk take off in its course inferiorly, although, given the small diameter of these nerves (usually less than 1 mm), it is usually not visible. The fascial plane of the CFN in this intermediate zone, however, should be noted and scanned inferiorly to the region of the assumed course of the LSCN. The division of the gastrocnemius muscle should also be noted, as the *medial sural cutaneous nerve* (MSCN) travels down this line, and it is sometimes possible to observe the MSCN join the LSCN (or other contributor from the CFN) (Fig. 72.8).

Alternatively, one may trace the sural nerve cephalad from the ankle where it is usually found in close proximity to the posterior tibial nerve (see Chap. 73) and look for the two tributaries of the MSCN and the LSCN (or peroneal communicating nerve). This approach is more difficult, as the nerves become smaller as they move distally, and the multiple fascial planes of the calf render ultrasound more challenging.

Regardless of the approach taken, peripheral nerve stimulation is extremely useful, and often necessary, to find this nerve given its small size.

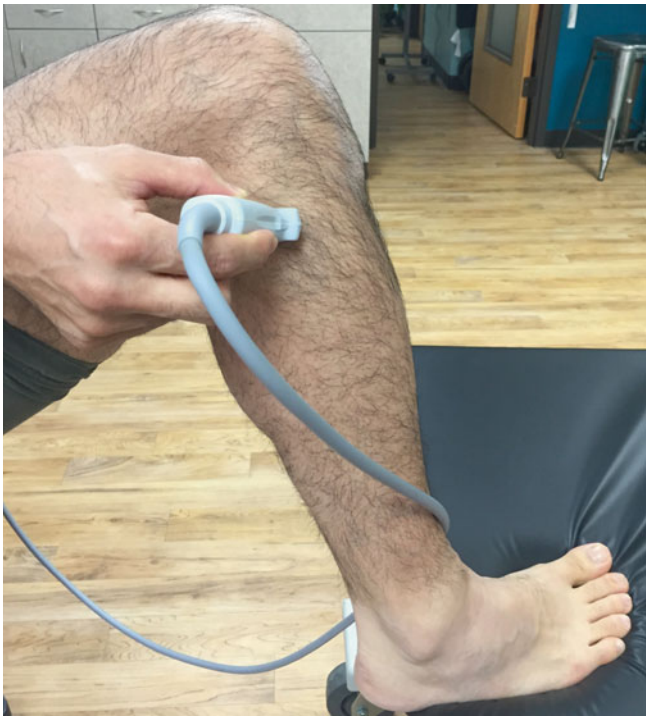

Fig. 72.6 Ultrasound probe at the lateral fibular head (Image courtesy of Heath McAnally, MD)

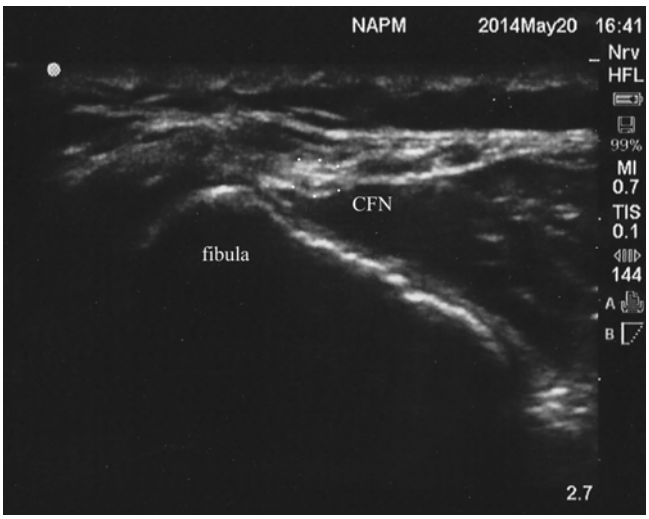

Fig. 72.7 Ultrasound image of the common fibular (peroneal) nerve at the fibular head. *CPN* common peroneal nerve (Image courtesy Heath McAnally, MD)

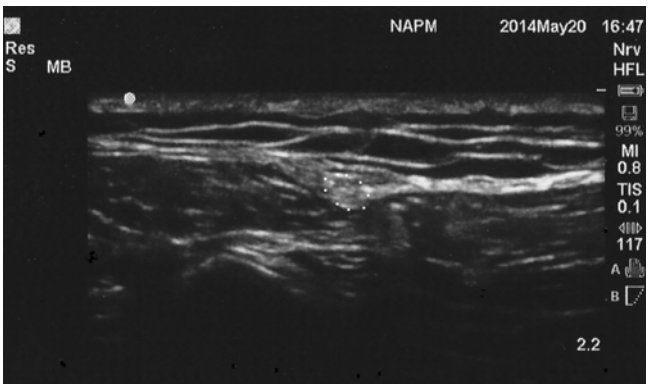

Fig. 72.8 Ultrasound image of the fascial plane containing the lateral sural cutaneous nerve (Image courtesy of Heath McAnally, MD)

Treatment of Perpetuating Factors

Avoid lateral compression of the leg in stirrups or during surgery.

Neurolytic/Surgical Technique

Cryoneuroablation

Cryoneuroablation has been an effective technique for many peripheral nerves [13, 14], though there have been no specific reports of cryoneuroablation of the LSCN. Since the LFCN is a purely sensory nerve, if low-volume diagnostic injections give temporary relief, cryoneuroablation could be an appropriate neurolytic technique.

Radio-frequency Lesioning

No reports of radio-frequency neurolysis are found in the literature currently; however, the author recently performed a radio-frequency ablation of the LSCN in a patient with intractable pain in its distribution [15]. The patient was a 57-year-old male who had persistent pain after total knee arthroplasty, and a neurologist localized his complaint to the LSCN by electromyography/nerve conduction velocity studies. He was placed prone on the procedure table, and his popliteal fossa was examined with ultrasound (linear high-frequency probe); the bifurcation of the sciatic nerve into the tibial nerve and CFN components was clearly seen above the knee. The CFN was then traced down below the knee, and what appeared to be the takeoff of the LSCN was noted. A 25-gauge needle was used to anesthetize the skin and subcutaneous tissue in the proposed probe pathway with 2 mL of 1 % lidocaine. A 20-guage, 10 cm (10 mm active tip) radio-frequency cannula was then advanced under ultrasonic guidance to the nerve, and sensory stimulation was carried out at 50 Hz and 0.5 V; after minute adjustments, he described exact reproduction/exacerbation of his pain in the lateral knee. Motor stimulation was commenced at 2 Hz, 2 V to confirm no motor component. Sensory stimulation was carried out once more as above to confirm proper placement, and then 1 mL of 1 % lidocaine was injected through the cannula. Radio-frequency ablation was then commenced at 60 °C for 70 s. Afterward, 5 mL of 0.5 % bupivacaine was injected into the site. He had near immediate relief of his typical pain 10 min after the local anesthetic was given, with sustained total relief for at least 18 months, at last follow-up.

Chemical Neurolysis

Chemical neurolytic techniques involve the use of alcohol, phenol, or botulinum toxin. There are no reports in the literature regarding chemical neurolysis of the LSCN.

Peripheral Nerve Stimulation

Neuromodulation in the form of peripheral nerve stimulation is another potentially effective treatment for LSCN. However, there are no reports of its use in LSCN neuralgia, and because of its location, it would be difficult to stabilize a peripheral stimulator lead.

Surgical Technique

Only isolated cases [1, 2, 11, 15] of entrapment of the LSCN exist in the literature, and only two of these cases progressed to surgery. Khalil et al. [1] described a 35-year-old man with LSCN entrapment diagnosed by nerve conduction velocity study, with temporary relief from local anesthetic and steroid, who underwent surgical release of a fibrous band around the nerve with "complete" relief postoperatively.

Complications

There is a risk of bleeding or infection with any percutaneous technique. Nerve damage and increases in pain may occur with any injections close to nerves.

Summary

The LSCN is a little-known cutaneous sensory nerve with marked anatomic variability. It is usually derived from both the tibial and fibular divisions of the sciatic nerve and supplies the lateral calf region. Isolated pain syndromes in this region should include consideration of the LSCN in the differential diagnosis.

References

1. Khalil NM, Nicotra A, Kaplan C, O'Neill KS. Entrapment of the lateral cutaneous nerve of the calf. BMJ Case Rep. 2013:online.
2. Ginanneschi F, Rossi A. Lateral cutaneous nerve of calf neuropathy due to peri-popliteal cystic bursitis. Muscle Nerve. 2006;34(4):503–4.
3. Riedl O, Frey M. Anatomy of the sural nerve: cadaver study and literature review. Plast Reconstr Surg. 2013;131(4):802–10.
4. Huelke DF. A study of the formation of the sural nerve in adult man. Am J Phys Anthropol. 1957;15(1):137–47.
5. Huelke DF. The origin of the peroneal communicating nerve in adult man. Anat Rec. 1958;132(1):81–92.
6. Mahakkanukrauh P, Chomsung R. Anatomical variations of the sural nerve. Clin Anat. 2002;15(4):263–6.
7. Kavyashree AN, Subhash LP, Asha KR, Bindu Rani MK. Anatomical variations in formation of sural nerve in adult Indian cadavers. J Clin Diagn Res. 2013;7(9):1838–41.
8. Aktan Ikiz ZA, Ucerler H, Bilge O. The anatomic features of the sural nerve with an emphasis on its clinical importance. Foot Ankle Int. 2005;26(7):560–7.
9. Pyun SB, Kwon HK. The effect of anatomical variation of the sural nerve on nerve conduction studies. Am J Phys Med Rehabil. 2008;87(6):438–42.
10. Nuri T, Ueda K, Maeda S, Otsuki Y. Anatomical study of medial and lateral sural cutaneous nerve: implications for innervated distally-based superficial sural artery flap. J Plast Surg Hand Surg. 2012;46(1):8–12.
11. Hackam DG, Zwimpfer TJ. Congenital entrapment of the lateral cutaneous nerve of the calf presenting as a personal sensory neuropathy. Can J Neurol Sci Le J Canadien des Sci Neurol. 1998;25(2):168–70.
12. Campagnolo DI, Romello MA, Park YI, Foye PM, Delisa JA. Technique for studying conduction in the lateral cutaneous nerve of calf. Muscle Nerve. 2000;23(8):1277–9.
13. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
14. Caporusso EF, Fallat LM, Savoy-Moore R. Cryogenic neuroablation for the treatment of lower extremity neuromas. J Foot Ankle Surg. 2002;41(5):286–90.
15. McAnally H. Radiofrequency ablation of the lateral sural cutaneous nerve: a case report. 2016 (submitted).

Tibial Nerve Entrapment

73

Michael N. Brown, Beth S. Pearce, Andrea M. Trescot, and Helen W. Karl

Introduction

Patients with foot pain are not uncommon in clinical practice. A recent systematic review revealed that 24 % of older adults (≥45 years) have frequent foot pain; forefoot pain is more common in women and causes at least moderate disability in most patients [1]. Heel pain is also common and has a variety of causes including nerve entrapment [2].

The *tibial nerve* (TN), often called the *posterior tibial nerve* (PTN) as it nears the ankle, is vulnerable to entrapment at two separate anatomic sites. The more well known of these locations is the *tarsal tunnel* (TT), also known as the *tibiotalocalcaneal tunnel*, *calcaneal tunnel*, or *Richet's tunnel* [3], where the PTN travels under the *flexor retinaculum* (previously known as the *lancinate ligament*) of the ankle [4]. Entrapment of the PTN or its branches at this site is called *tarsal tunnel syndrome* (TTS) [5, 6]. We address entrapment of the individual branches of the TN in subsequent chapters (Chaps. 74, 75, 76, and 77).

In 1981, Mastaglia et al. [1] described a proximal entrapment of the tibial nerve at the tendinous arch between the two heads of the soleus muscle behind the knee. This is becoming known as the *soleal sling syndrome* (SSS) [7–9] and can exacerbate TTS symptoms via a "double crush" effect (see Chap. 1). SSS presents with pain in the popliteal fossa and proximal calf (aggravated by active and passive dorsiflexion of the foot), inability to bear weight, weakness of toe flexion, and sensory deficits on the sole of the foot that are aggravated by walking.

The tarsal tunnel is a major corridor at the posteromedial ankle between the distal tibia and the plantar foot, allowing nerves, blood vessels, and tendons to cross into the foot. The taxonomy of the tarsal tunnel can be confusing. In the radiology literature, it has been divided into "upper" (tibiotalar) and "lower" (talo-caneal) tunnels, with the dividing line at the *sustentaculum talus* [2–4]. However, surgeons as a group divide the tarsal tunnel into "proximal," the portion deep to the *flexor retinaculum*, and "distal," the portion deep to the *abductor hallucis* (AbH) muscle [5–7]. In this and subsequent chapters, we will follow the surgical convention.

TTS was thought to be clinically and pathologically comparable to carpal tunnel syndrome [8, 9] (Chap. 37), although the tarsal tunnel is now understood as more analogous to the forearm compartment, while the medial plantar tunnel (Chap. 75) is more equivalent to the carpal tunnel [6, 10].

Clinical Presentation (Table 73.1)

TTS is a relatively common problem, especially in patients with biomechanical faults of intrinsic foot structure that lead to overpronation [13]. Foot pronation increases the pressure in the tarsal tunnel [26] and decreases its volume [27], thereby chronically compressing the nerve. Patients with increased foot pronation may have additional proximal musculoskeletal complaints such as *shin splints*, *patellofemoral syndrome*, or *peritrochanteric hip pain* because the same biomechanical faults can be the root cause of all these conditions.

It is also seen in athletes [18–22] and those with occupations which require extensive standing or walking [8]. There

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Table 73.1 Occupation/exercise/trauma history relevant to tibial nerve entrapment

Compression	Synovial cysts [11, 12]
	Neurilemmoma [12]
	Foot pronation [13]
	Varicose veins [11]
	Talocalcaneal coalition [11]
	Accessory flexor digitorum longus [14]
	Hemodialysis [15]
Inflammation	Thrombophlebitis [16]
	Tenosynovitis [16]
	Osteomyelitis [17]
Trauma	Fracture or dislocation of foot bones [16]
	Posttraumatic edema [16]
	Athletes [18–23], especially in those sports that require repetitive foot dorsiflexion that increases the pressure in the tarsal tunnel
Others	Obesity is associated with a greater incidence of flat foot [24] Diabetes and other peripheral neuropathies [25]

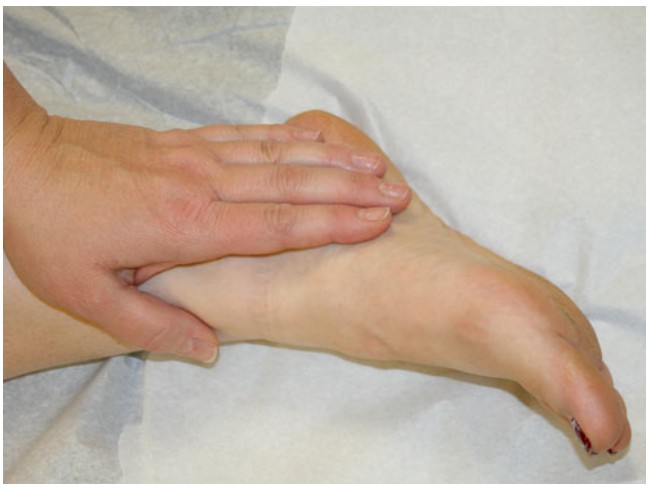

Fig. 73.1 Patient pain complaint from tarsal tunnel syndrome (Image courtesy of Andrea Trescot, MD)

is an even distribution of genders exhibiting TTS [9]. Acute trauma or repetitive microtrauma can generate entrapment and pain. Other contributing triggers may include diabetes or chemotherapy [28].

The symptoms of TTS often include burning pain and paresthesias at the medial ankle, though it may extend through the heel or the sole of the foot, with radiation proximally along the medial calf [10] (Fig. 73.1). Tenderness below the medial malleolus is much more common than on the plantar foot [13] (Fig. 73.2). Depending on the compression site and the specific nerve branch or branches involved, clinical symptoms may vary. Numbness can lead to loss of balance and falls [28], and PTN dysfunction can progress to paresis of the small muscles of the foot.

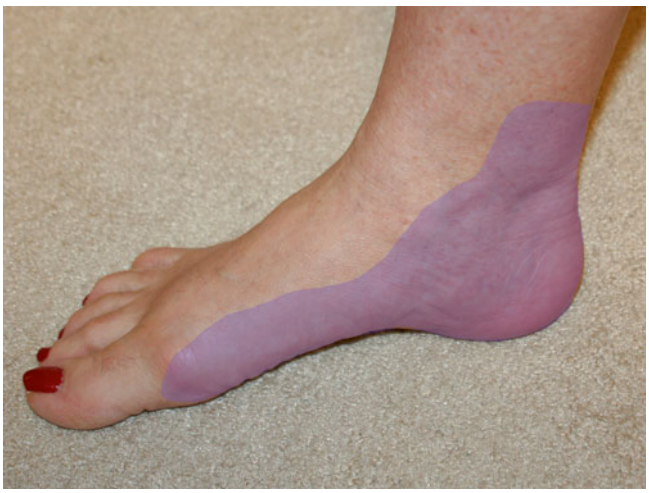

Fig. 73.2 Pattern of pain from tarsal tunnel syndrome (Image courtesy of Andrea Trescot, MD)

Although it is typically aggravated by activity [10] and relieved by rest, TTS pain often worsens at night and may actually awaken the patient from sleep [13]. Night pain may be the result of venous stasis when the patient is inactive, and patients may obtain relief by getting up or hanging the leg over the side of the bed [9].

In contrast to TTS, patients with SSS have pain in the back of the knee and proximal calf that is exacerbated by active plantar flexion.

Anatomy (Table 73.2)

The TN is the larger of the two terminal branches of the sciatic nerve, traveling down the back of the leg to the foot (Fig. 73.3). It gives off branches to the muscles of the thigh and then crosses the middle of the *popliteal fossa* and under the tendinous arch of the *soleus muscle* to supply the calf and foot flexor muscles in the deep posterior compartment. The TN accompanies the *posterior tibial branch of the popliteal artery* and one or more veins through the popliteal fossa.

The TN becomes the PTN in the lower leg; it divides into its plantar divisions in or near the fibro-osseous *tarsal tunnel* that stretches from the posteromedial ankle below the *medial malleolus* of the distal tibia down to the medial aspect of the plantar region of the foot (Figs. 73.4 and 73.5). The *malleolar-calcaneal axis* from the center of the medial malleolus to the center of the calcaneus is a useful constant reference point for the center of the tarsal tunnel [30, 31]. The ceiling of the tunnel is the *flexor retinaculum*, and its bony floor consists of the posteromedial *talus*, the medial *navicular*, and the medial *calcaneus* [34]. It contains three tendons: the tibialis posterior (TP), the flexor digitorum longus (FDL), and the flexor hallucis longus (FHL), in addition to the neurovascular bundle of the PTN and

Table 73.2 Tibial nerve anatomy

Origin	L4-S4 ventral rami form the *sciatic nerve*
General route	The sciatic nerve divides in the distal thigh into the *tibial nerve* (TN) and *common peroneal* (*fibular*) *nerve* (CPN) (see Chap. 67); the TN continues down through the deep posterior compartment of the leg, changing its name to the *posterior tibial nerve* (*PTN*)
	The TN divides within (usually) or near the tarsal tunnel behind and inferior to medial malleolus into *the lateral plantar nerve* (LPN), *medial plantar nerve* (MPN), and *medial calcaneal nerve* (MCN)
	Both the MPN and LPN run deep to the *abductor hallucis* (AbH) *muscle* in the direction of the toes within the middle layer of the soft tissues of the plantar side of the foot
	A fibrous septum between the calcaneus and the deep fascia of the AbH separates the MPN from the LPN [6, 10, 22]
Sensory distribution	*LPN*: the skin of the lateral sole, plantar surface, and lateral side of the fourth and all of the fifth toe as well as the anterior calcaneus
	MPN: the skin of the medial sole, the plantar surface, and sides of toes one to three, medial fourth toe, and the tarsal/metatarsal joints
	MCN: the skin and deep structures of the heel
Motor innervation	*Tibial nerve*: popliteus, soleus, gastrocnemius, plantaris, tibialis posterior, flexor digitorum longus, and flexor hallucis longus muscles
	LPN: quadratus plantae (QP), abductor digiti minimi (AbDM), adductor hallucis, flexor digiti minimi brevis, interosseous, and second to fourth lumbrical muscles
	MPN: flexor digitorum brevis (FDB), abductor hallucis (AbH), flexor hallucis brevis, and first and second lumbrical muscles
Anatomic variability	Site of the sciatic nerve branching into TN, the common peroneal nerve
	Relationships between TN and the hamstring tendons, skin, blood vessels, and its own branch points in and near the popliteal fossa [29]
	Site of the tibial N branching into MPN and LPN: in the tarsal tunnel in 93 % of 68 feet dissections, 7 % were more proximal [30, 31]
Other relevant structures	*Soleus muscle*: originates from the posterior fibular head and the soleal line of the tibia; these two origins are connected by a tendinous arch, a potential site of TN entrapment [32]
	Popliteal fossa: an area between the tendons of the hamstring muscles
	Flexor retinaculum: a specialized area of the deep fascia of the leg that runs between the anteromedial medial malleolus and the medial tuberosity of the calcaneus; it forms a tunnel described as oval [5] or conical [6] and continues distally as the fascia surrounding the AbH muscle
	Malleolar-calcaneal axis: the line between the center of the medial malleolus and the center of the calcaneus; a useful reference point since the specialized fascia like the flexor retinaculum blends into the nearby fascia, thereby making exact measurements difficult [30, 31]
	Contents of the tarsal tunnel: the posterior tibial artery and veins (superficial to the nerves), as well as three tendons in their synovial sheaths (tibialis posterior, flexor digitorum longus, and flexor hallucis longus)
	Abductor hallucis (*AbH*) *muscle*: the continuation of the "roof" of the tarsal tunnel; its hypertrophy may contribute to PTN entrapment [33]

the *posterior tibial artery* and veins (Fig. 73.6). The mnemonic "Tom, Dick & Harry" helps one remember the positions of the tendons at the level of the medial malleolus from anterior to posterior, with the "&" representing the neurovascular bundle.

In the classic description, the PTN at the ankle trifurcates into its three terminal branches: the *lateral plantar nerve* (LPN) (Chap. 74), the *medial plantar nerve* (MPN) (Chap. 75), and the *medial calcaneal nerve* (MCN) (Chap. 77), within the tarsal tunnel [34]. Further work has revealed that the MCN may branch from the PTN proximal to the tarsal tunnel [33, 34], from the LPN [35] (see Chap. 74) or the MPN [36] (see Chap. 77). In this chapter, the authors will assume that the PTN bifurcates into the MPN and LPN deep to the flexor retinaculum, within 2 cm of the malleolar-calcaneal axis, as it does in almost all (93–95 %) subjects [30, 31] (Fig. 73.6), and that the MCN origin is variable (thereby accounting for the variable presence of heel pain in TTS).

The MPN and LPN exit the tarsal tunnel to innervate the sole of the foot (Fig. 73.7). As the branches continue distally, deep to the AbH, they become separated by a fascial septum known as the *medial septum* [37] that runs between the medial calcaneus (the tunnel floor) and deep fascia of the AbH (the tunnel roof), and each divides into smaller soft tissue tunnels (Fig. 73.8) [5, 6, 10].

Ultrasound Anatomy of the Tibial Nerve Just Proximal to the Tarsal Tunnel

On ultrasound, the PTN is an approximately 4 mm hyperechoic oval with an internal honeycomb of hypoechoic structures that

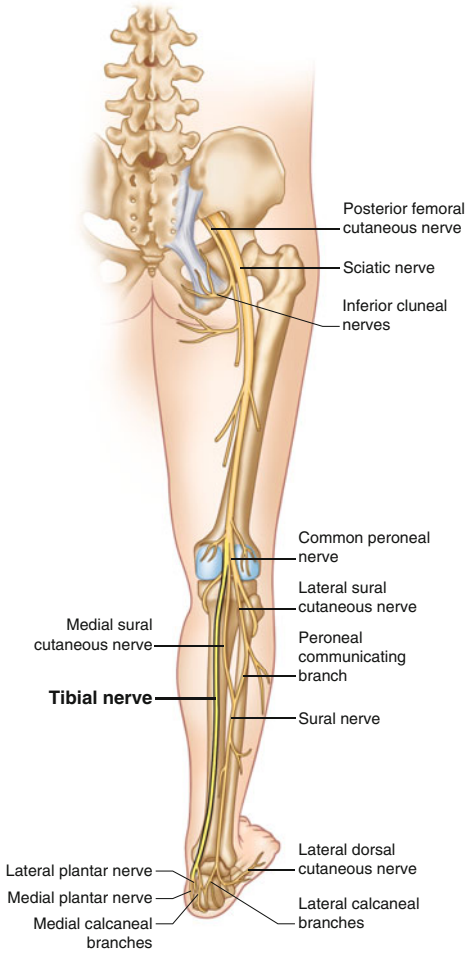

Fig. 73.3 Anatomy of the posterior leg (Image by Springer)

lies posterior to its accompanying blood vessels (Fig. 73.9) [38, 39]. The posterior tibial artery usually has a vein on either side of it, creating a vein-artery-vein configuration. The FHL tendon is also hyperechoic and posterior to the vessels, but it has finer fibrils and will move when the great toe is flexed.

Entrapment

The TN is most commonly compressed in the tarsal tunnel, deep to the flexor retinaculum at the medial ankle [40]. Its branches, the MPN and LPN, can be trapped separately in the two tunnels created by the fascial septum deep to the AbH [10], as well as at the medial calcaneal tunnel (Chaps. 74, 75, 76, and 77). Pressure around the PTN is increased significantly with dorsiflexion and hind-foot eversion, as well as with pronation [25, 26, 41]. Changes in ankle position increase the pressure in the medial and lateral plantar tunnels even more than the pressure in the tarsal tunnel itself; therefore, more distal compression is likely [25].

A more proximal entrapment of the TN occurs where it passes through a tunnel between the superficial and deep posterior compartments of the leg at the origin of the soleus muscle [1, 32, 42–44]. Patients report numbness of the sole of the foot and great toe, along with tenderness of the posterior proximal calf. There can be a positive Tinel's sign at the posterior calf approximately 5 cm below the medial tibial plateau. MRI images will show flattening of the TN and thickening of the soleal arch at the site of entrapment [45].

The TN can also be entrapped at the popliteal fossa by a mass such as a Baker's cyst or popliteal artery aneurysm [46]

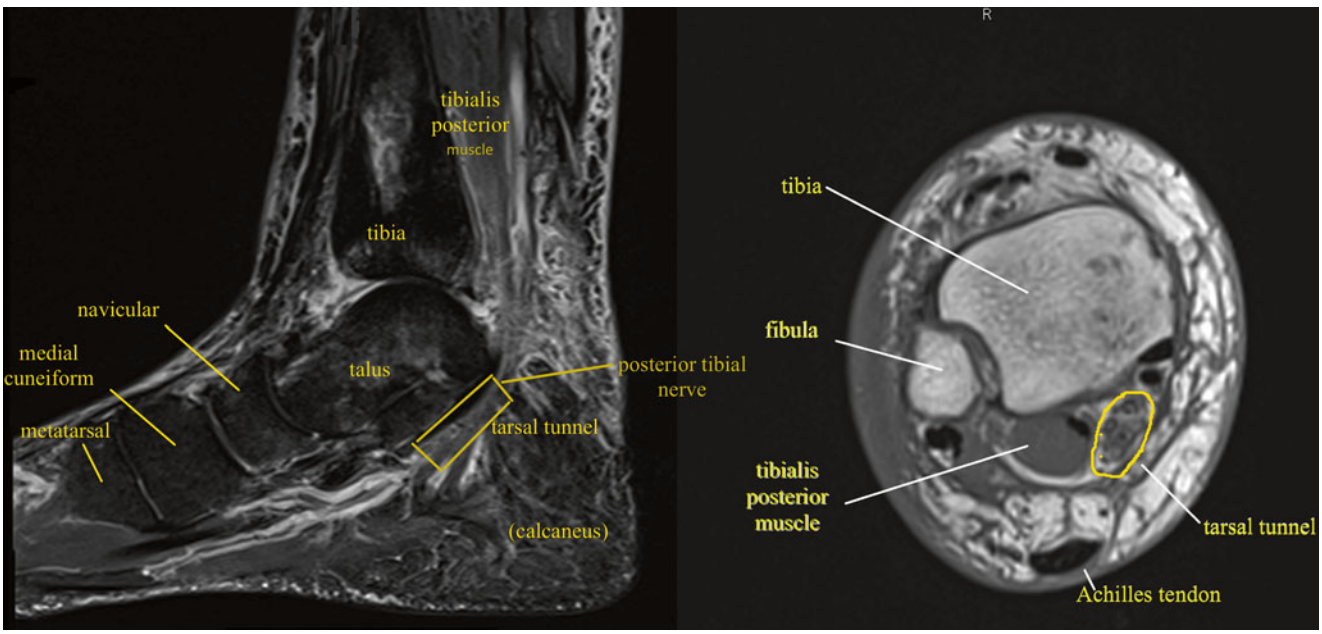

Fig. 73.4 MRI (sagittal and axial) of the tarsal tunnel (Image courtesy of Andrea Trescot, MD)

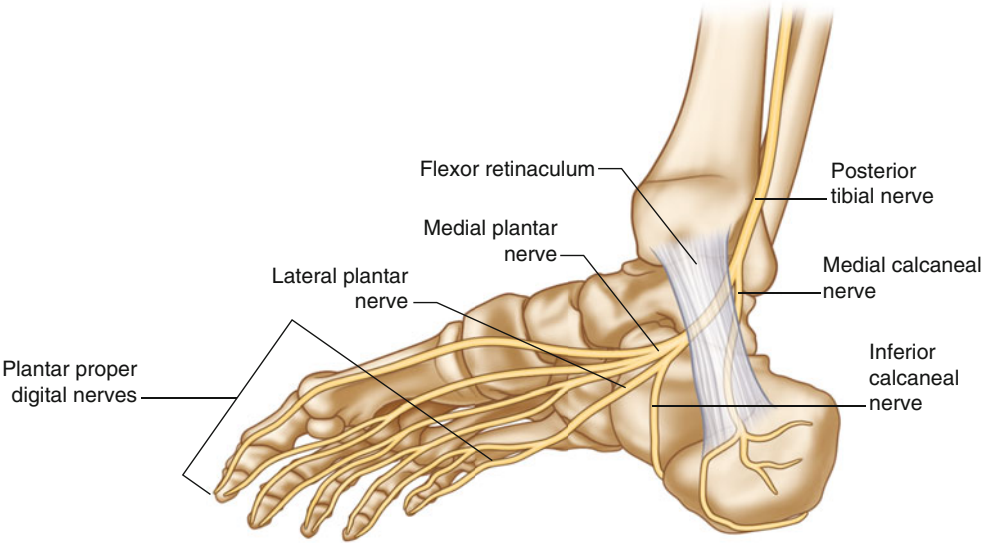

Fig. 73.5 Anatomy of the distal tibial nerve and its branches (Image by Springer)

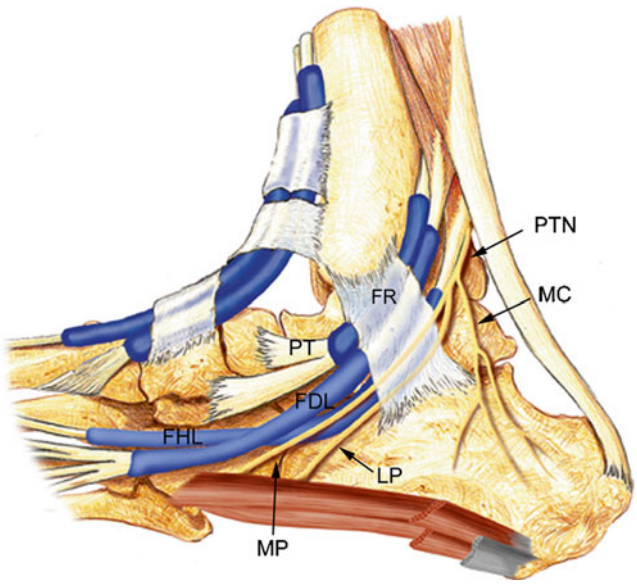

Fig. 73.6 Anatomy of the medial foot. *PTN* posterior tibial nerve, *MC* medial calcaneal nerve, *LP* lateral plantar nerve, *MP* medial plantar nerve, *PT* tibialis posterior tendon, *FDL* flexor digitorum longus tendon, *FHL* flexor hallucis longus tendon, *FR* flexor retinaculum. Note the parallel courses of the PTN and the FHL tendon (From Beltran et al. [65]. Reprinted with permission from Thieme)

and more distally by the lower edge of the gastrocnemius muscle [47] or by synovial cysts in the tarsal tunnel (Fig. 73.10).

Physical Examination

If TTS is suspected, begin with the surface anatomy of the foot (Fig. 73.11). There can be local tenderness at the medial ankle over the tarsal tunnel [9] (Video 73.1) (Fig. 73.12c),

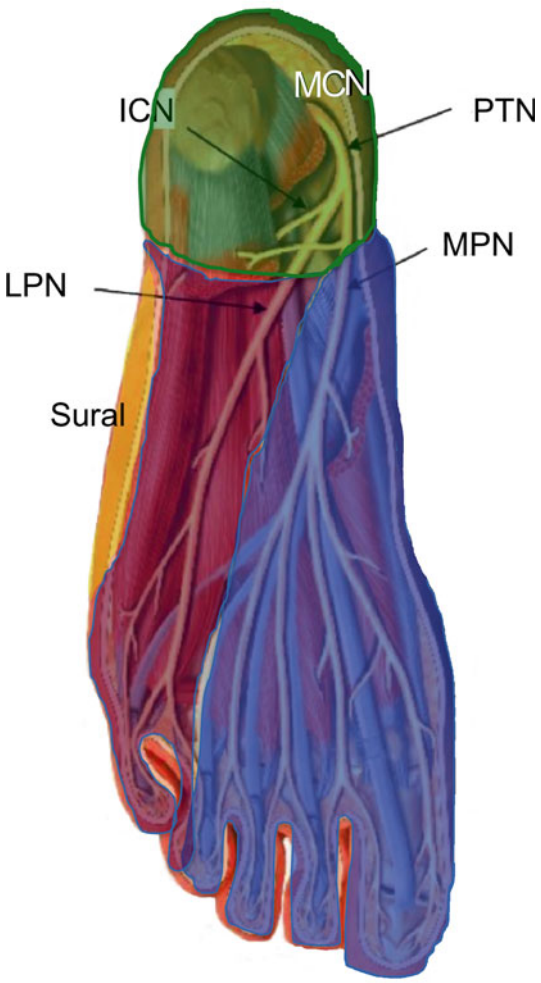

Fig. 73.7 Schematic representation of the plantar nerves and their cutaneous territories. *LCN* lateral calcaneal nerve, *MCN* medial calcaneal nerve, *PTN* posterior tibial nerve, *MPN* medial plantar nerve, *LPN* lateral plantar nerve, *ICN* inferior calcaneal nerve, *PF* plantar fascia (Image courtesy of Michael Brown, MD)

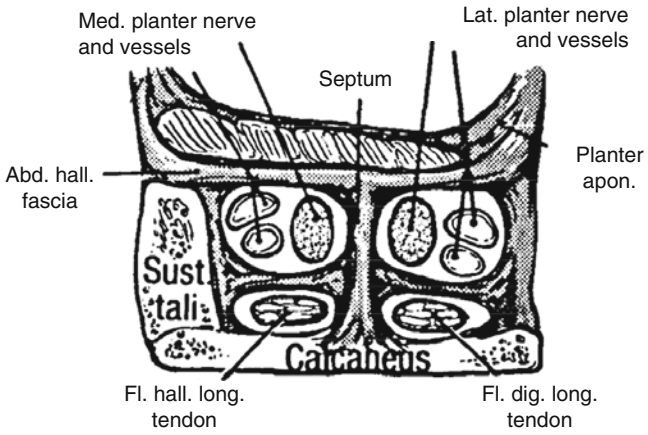

Fig 73.8 Cross section through the region immediately distal to the proximal tarsal tunnel. The medial and lateral plantar tunnels are separated by a septum between the tunnel roof (the deep abductor hallucis fascia) and the tunnel floor (the calcaneus). *Apon* aponeurosis, *dig* digital, *fl* flexor, *hall* hallucis, *lat* lateral, *long* longus, *med* medial, *sust* sustentaculum (From Dellon [10]. Reprinted with permission from Elsevier Limited)

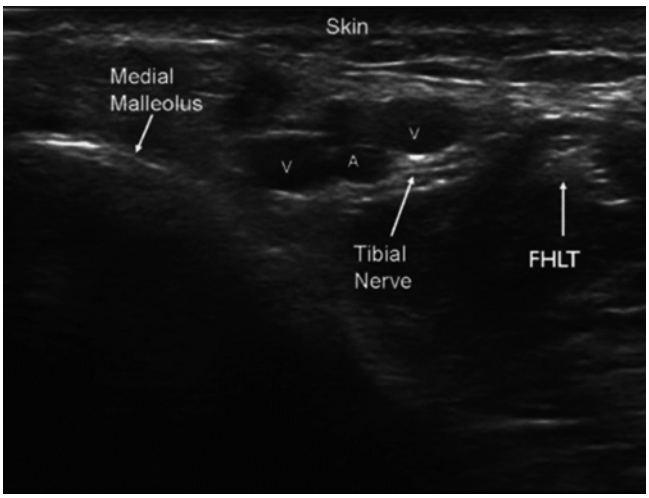

Fig. 73.9 Ultrasound anatomy of the tarsal tunnel at the level of the ankle (short axis view). *A* posterior tibial artery, *FHLT* flexor hallucis longus tendon, *V* vein. Screen right is posterior, and screen left is anterior. Note the vein-artery-vein configuration near the nerve and the proximity of the flexor hallucis longus tendon (FHLT); the latter can be distinguished from the nerve by watching it move when the great toe is flexed (From Redborg et al. [38]. Reprinted with permission from Wolters Kluwer Health, Inc)

with sensory loss over the bottom of the foot. A Tinel's sign may be elicited with the fingers (Fig. 73.12a) or a reflex hammer (Fig. 73.12b) over the PTN at this site [13]; if it is present, it provides an 88 % positive predictive value of a good to excellent response to surgery [10]. However, its absence may indicate an even greater degree of injury [28].

The *triple compression stress test* (TCST) (also known as *dorsiflexion/eversion test*) is a provocative test for TTS. Place

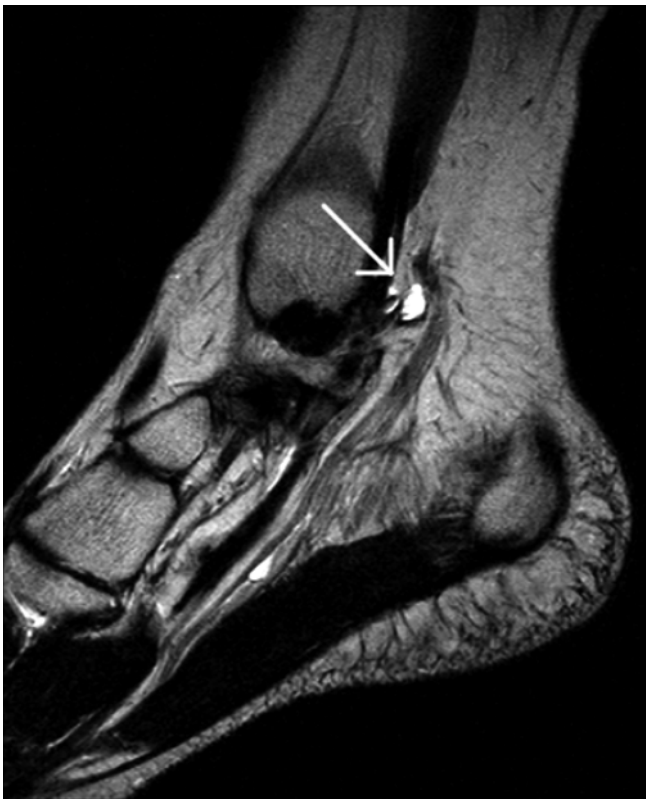

Fig. 73.10 T2 sagittal MRI image showing a synovial cyst impinging the tarsal tunnel (*white arrow*) (Image courtesy of Andrea Trescot, MD)

the ankle in full dorsiflexion, evert the foot, and apply digital pressure over the PTN to replicate paresthesias and pain (Fig. 73.12d) [26, 41]. The TCST has been shown to have a sensitivity of 85.9 % and specificity of 100 % for TTS diagnosis [48]. Symptoms may also be increased by a blood pressure cuff inflated to create venous occlusion [9].

Biomechanical assessment of the intrinsic structure of the foot, including the subtalar position and the *pes planus foot*, *rear foot,* and *forefoot position*, gait analysis, and the cause of foot pronation may be helpful in revealing possible causes of increased PTN tension in the tarsal tunnel. One should evaluate whether or not the patient has maintained the ability to abduct the fifth toe (a function of the *abductor digiti minimi* (AbDM)) or abduct the greater toe (a function of the AbH). There may be weak toe flexion and sensory changes over the sole of the foot. Frank motor weakness, however, is a late finding [9]. If the AbH is compressing a nerve or part of one, the patient's symptoms may be reproduced by tensing it [33].

Evaluation of the TN near the popliteal fossa is also an important part of the examination, as impingement at this level may exacerbate TTS. A very tender spot is found on deep palpation of an area about one fifth of the way down the back of the leg. A Tinel's test at this point may travel to the medial plantar foot; if it goes to the lateral foot, the sural nerve may also be entrapped (Chap. 71) [32].

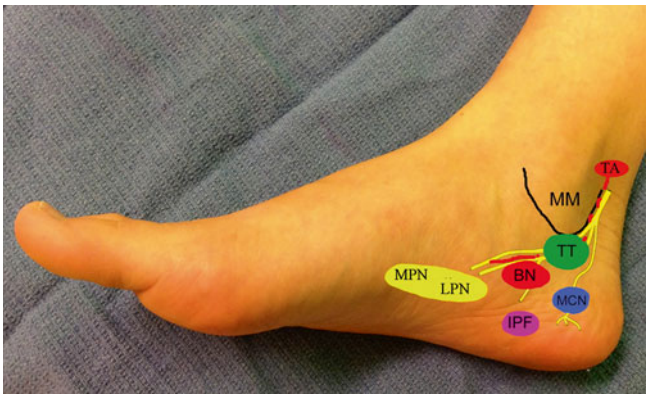

Fig. 73.11 Surface anatomy of the medial ankle. *MM* medial malleolus, *TT* proximal tarsal tunnel, *BN* Baxter's *nerve* inferior calcaneal nerve, *MCN* medical calcaneal nerve, *MPN* medial plantar nerve, *LPN* lateral plantar nerve, *TA* tibial artery, *IPF* insertion of the plantar fascia (Image courtesy of Michael Brown, MD)

Differential Diagnosis (Tables 73.3 and 73.4)

The differential diagnosis of TTS includes plantar fasciitis, Achilles tendonitis, retrocalcaneal bursitis (Fig. 73.12), and infection (e.g., calcaneal osteomyelitis) [17]. Passive dorsiflexion and eversion will provoke pain, tingling, numbness, and burning, which typifies tarsal tunnel syndrome. TTS may present as a complex regional pain syndrome (CRPS) as a complication of trauma. As discussed in Chap. 3, CRPS may be triggered by nerve entrapment; this is not actually a differential diagnosis, but rather an etiology.

Diagnostic Tests (Table 73.5)

Imaging

Plain radiography, supplemented as needed with CT, is useful for evaluating the patient's underlying foot structure and assessing other bony abnormalities that can cause compression [51]. MRI of the foot and ankle (Fig. 73.13) can also be useful in identifying suspected space-occupying soft tissue lesions within the tarsal tunnel [51, 52]. Ultrasound is another way to evaluate the PTN and potential sources of entrapment [11], and it is an excellent way to guide placement of local anesthetic around the PTN or its branches (see below).

EMG/NCV

Electrodiagnostic studies can help diagnose and quantify the severity of entrapment neuropathies. Goodgold et al. [16] published a normal range of TN conduction velocities

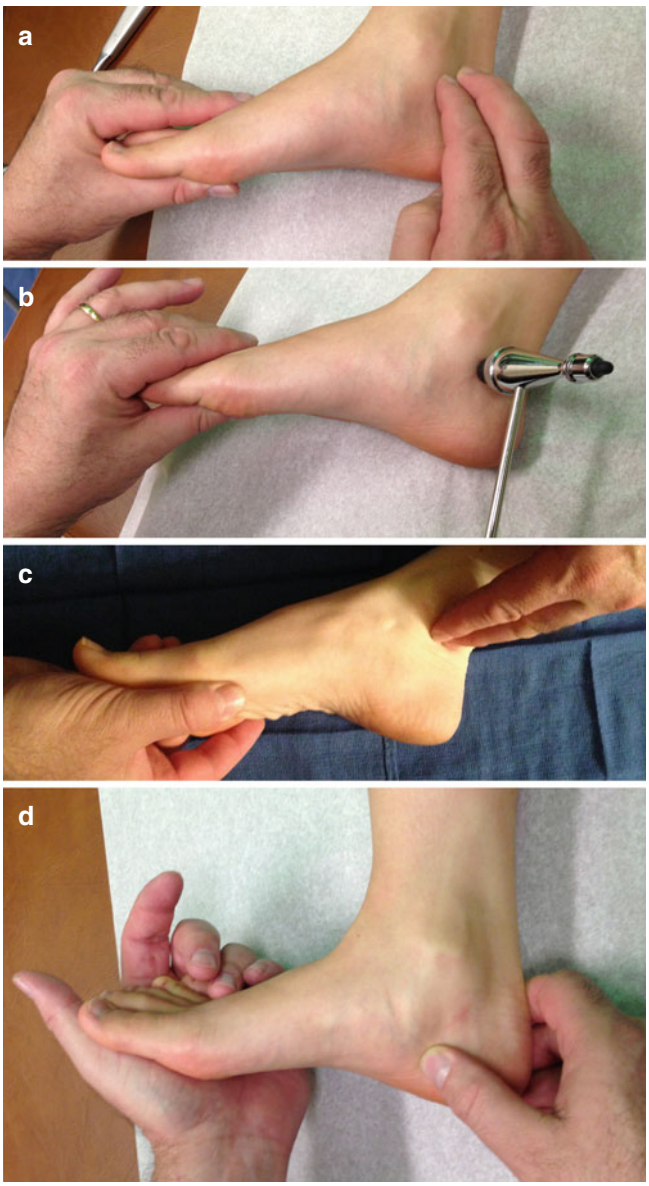

Fig. 73.12 Physical exam of tarsal tunnel syndrome. (**a**) Tinel's sign elicited with fingers; (**b**) Tinel's with a reflex hammer; (**c**) palpation of the tarsal tunnel; (**d**) triple compression stress test (Image courtesy of Michael Brown, MD)

in 1965. More recently, an "*electrophysiologic severity scale*" to grade TTS has been proposed [53]. These may be useful, but a systematic review has revealed that the level of evidence (as of 2005) is weak [54]; some authors think that they are not reliable, particularly because many patients without TTS have abnormal results [10]. In complex situations, however, they may be useful to rule out a radiculopathy [32].

The *pressure-specified sensory device* (PSSD) is an objective, non-painful method to measure nerve function; the pressure required for a person to distinguish one from two static points is the most sensitive gauge of nerve dysfunction

Table 73.3 Differential diagnosis of plantar pain and paresthesias

	Potential distinguishing features
Plantar fasciitis	Tenderness at the attachment of the flexor retinaculum on the calcaneus
Achilles tendonitis	Tenderness more posteriorly at the Achilles tendon attachment
Retrocalcaneal bursitis	Tenderness on palpation at medial and/or lateral Achilles attachment
Osteomyelitis	Fever, elevated WBC, increased ESR, + culture
Ganglion cyst [49]	MRI or ultrasound showing cystic structure
Interdigital neuroma [50]	See Chap. 70
Infection (osteomyelitis) [17]	Bone scan or MRI showing increased uptake and edema
Varicosities	US evaluation
Tenosynovitis	Tenderness along the tendon sheath

Table 73.4 Comparison of plantar pain from common causes

Etiology	Distinguishing characteristics	Location of pain and tenderness
Plantar fasciitis	Insidious onset	From the anterior calcaneus as far as the MTP joints
	Decreased range of motion of the great toe	
	"First step" pain	
Tarsal tunnel syndrome	Pain worse at night	The heel and entire plantar surface, although the heel may be spared if the MCN diverges proximal to the flexor retinaculum
Medial calcaneal nerve dysfunction	History of trauma to the medial border of the calcaneus	Anterior to the medial heel
		Tenderness over the medial calcaneus possibly referred to the anterior calcaneus
Medial plantar nerve dysfunction	Pain aggravated by arch supports	May spread to the AbH origin on the posteromedial calcaneus
Lateral plantar nerve dysfunction	Rare motor dysfunction	Pain and numbness to lateral foot
Soleal sling	Numbness at the sole of the foot	Pain at the back of the knee, approx. 5 cm below the medial tibial plateau
	Tinel's sign at the mid-calf	

Table 73.5 Diagnostic tests for tibial nerve entrapment

	Tarsal tunnel syndrome	Soleal sling
Physical exam	Clinical symptoms, sensory abnormalities, and provocative tests are the gold standard for diagnosis [10]	Foot and heel pain and numbness plus calf pain
		Tenderness and positive Tinel's sign of the tibial nerve under the soleal sling, ~5 cm distal to the popliteal fossa
Provocative tests	Passive dorsiflexion and eversion cause pain	Increased calf pain with active plantar flexion
	Triple compression stress test (TCST)	
Injection	Resolution of pain after diagnostic injection at the tarsal tunnel	Resolution of pain after diagnostic injection at the soleal sling
X-ray	Best for assessing bony abnormalities that can cause compression [51]	None
Ultrasound	Can show small mass lesions or bony abnormalities [11]	Not reported
	May show compression of the nerve	
	Use high-resolution techniques [51]	
MRI/MR neurography	May show mass lesions, bony abnormalities, or compression of the nerve [2, 64]	May show flattening of the TN and thickening of the soleal arch [45]
Arteriography	Not useful	Not useful
Electrodiagnostic studies	Needle EMG of foot muscles may show denervation	To rule out radiculopathy [32]
	False negative results may lead to underdiagnosis [40]	

from chronic compression [55]. Data for the MPN (big toe) and MCN (heel) has been recently reported in normal subjects and patients diagnosed with tarsal tunnel syndrome [56], and it may be a useful adjunct to diagnosis and treatment [57, 58].

Identification and Treatment of Contributing Factors

The cause of a particular patient's TTS should be sought in order to plan a management strategy. According to Ahmad et al. [40], causes can be divided into intrinsic factors involving localized compression or inflammation and extrinsic factors such as ill-chosen footwear [3], foot abnormalities,

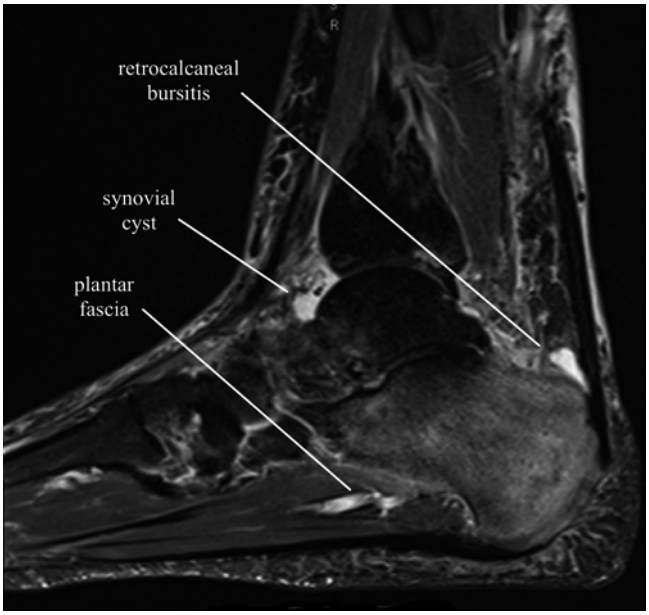

Fig. 73.13 STIR MRI sagittal image of the ankle showing retrocalcaneal bursitis, a synovial cyst, and plantar fascial irritation (Image courtesy of Andrea Trescot, MD)

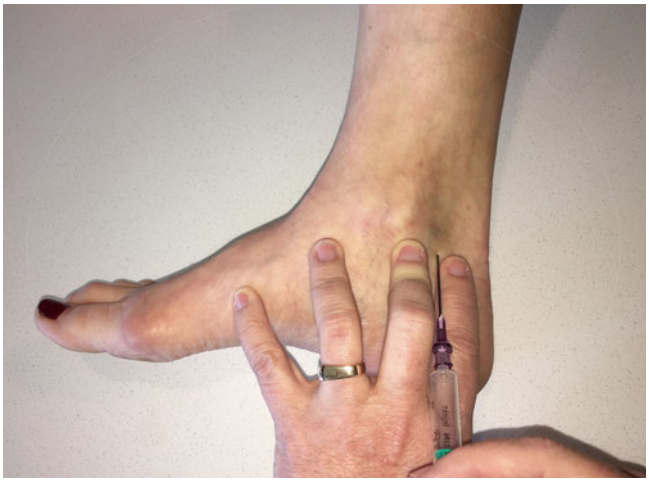

Fig. 73.14 Landmark-guided tarsal tunnel injection (Image courtesy of Andrea Trescot, MD)

and edema due to pregnancy or venous congestion, systemic diseases, and trauma, including postsurgical scarring (Table 73.1) [40].

The pain is idiopathic in 40 % of the cases [59]. Early diagnosis is necessary to minimize injury [51]. It is best to fix the entrapment before the onset of motor involvement [50]. Treatment of underlining perpetuating factors predominately involves the pathomechanics of gait, and correcting rear foot and forefoot position with orthotics can reduce the tension placed on the nerve through the tarsal tunnel and may do a better job of resolving the problem long term than surgery. Injection therapy can be a component of short-term symptomatic management, while treatment of the underlying biomechanical faults and contributing factors will provide better long-term resolution.

Injection Techniques

Landmark-Guided Injection

The patient is placed in a lateral position with the medial side of the foot facing upward. After a sterile skin-prep and drape, palpate the posterior tibial artery pulse between the medial malleolus and the Achilles tendon (Fig. 73.11). Using a 27- or 25-gauge, 1.5 inch needle, direct the needle from the region just behind the artery anteriorly toward the pulse and insert it 1–2 cm (Fig. 73.14) (Video 73.2). The nerve may be encountered posterior to the artery, and paresthesias may occur if the needle tip touches the nerve. Typically, if the purpose of the injection is a field block, use 3–5 cc of local anesthetic if a paresthesia is encountered and 7–10 cc if it is not. If the injection is a diagnostic or therapeutic one, a volume less than 2 cc should be used.

Ultrasound-Guided Injection

The patient is placed supine with the knee flexed and the hip externally rotated or lying on their side to expose the medial side of the foot. The *malleolar-calcaneal axis* (MCA) from the center of the medial malleolus to the center of the calcaneus is a useful constant reference point for the center of the tarsal tunnel [30, 31]. A 7.5–15 MHz linear probe is placed obliquely along the MCA, with the notch toward the medial malleolus (Fig. 73.15) and the other end directed toward the heel. You should see the pulsation of the posterior tibial artery with veins on either side of it (Fig. 73.9). Anterior to the three vessels are two tendons, the PT and FDL, along with the muscular portion of the FHL. The nerve is usually just posterior or medial to the vein, and the transducer can be slightly tilted back and forth to pick up its usual honeycomb appearance. If the nerve has already branched, one will see at least two nerves (the MPN and LPN); the LPN is the most posterior.

Direct a 27- or 25-gauge needle toward the nerve in the tarsal tunnel using an in-plane or out-of-plane approach. The in-plane approach provides a means of coming behind the vascular structures, if the nerve is found posterior to the vein

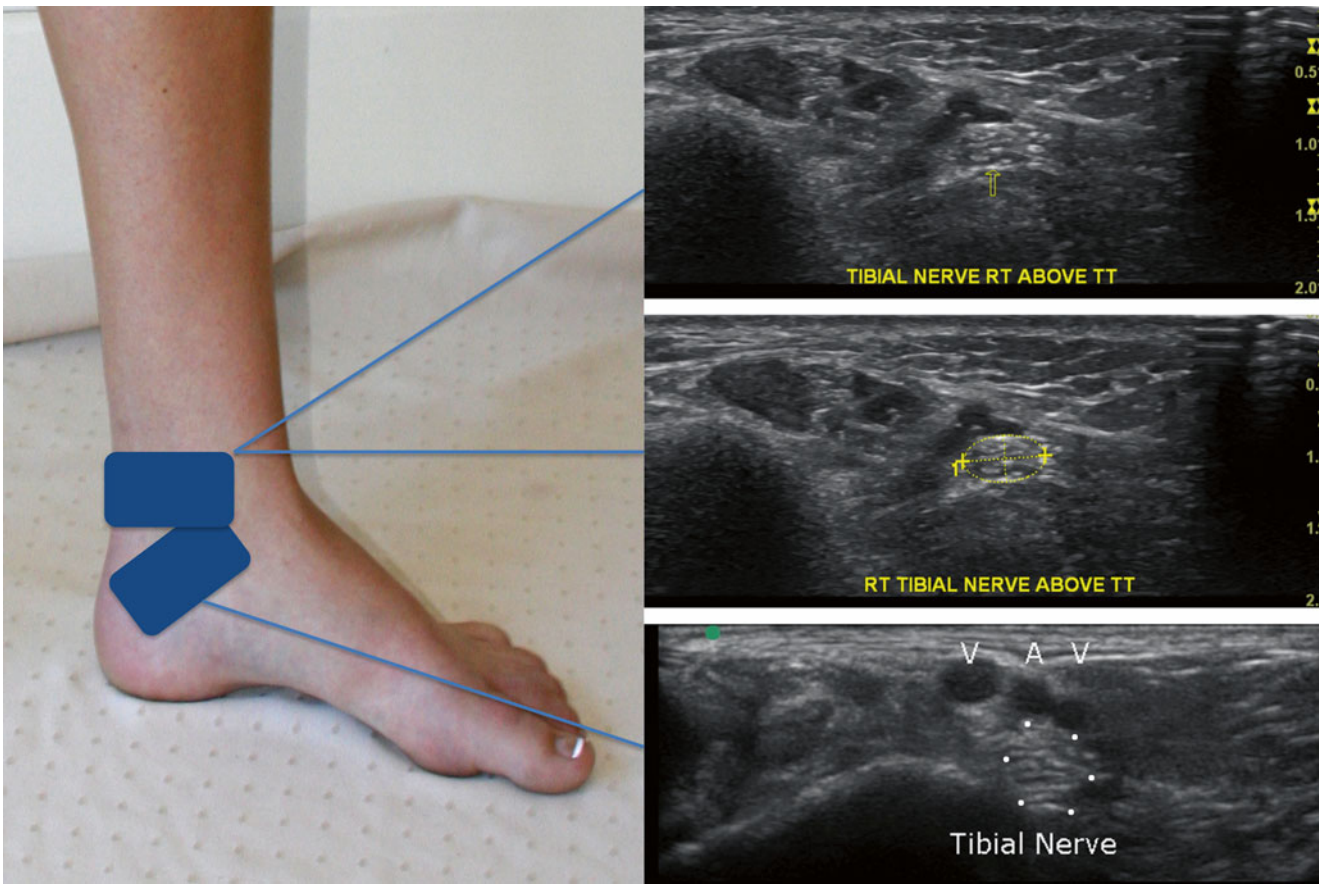

Fig. 73.15 Ultrasound evaluation of the tarsal tunnel (Image courtesy of Michael Brown, MD)

(Fig. 73.16). An out-of-plane approach is satisfactory if the nerve is found next to the vein.

Tibial Nerve Hydrodistention (See Chap. 7)

Before contemplating surgical decompression of the tarsal tunnel, perineural hydrodistention can be considered. The authors report this technique based on positive clinical experience; its efficacy for tarsal tunnel entrapment neuropathies will need to be verified in controlled trials.

Because of the vascular structures in this area, it is important to use US guidance. Identify the structures in the proximal tarsal tunnel, and inject a small aliquot of buffered lidocaine with a 30-gauge, 0.5 inch needle for skin anesthesia. Attach a 10 cc syringe filled with 7 cc D5W, 2 cc 1 % lidocaine, and 1 cc dexamethasone 30 mg/cc onto a 22- or 25-gauge needle. Slide the needle just posterior to the PTN (or the MPN and LPN, if the TN bifurcates proximally), and rapidly inject 3–4 cc, watching the fluid fill the space around the PTN. Reposition the needle anterior to the nerve, behind the vein-artery-vein bundle (Fig. 73.16), and repeat the procedure, taking care to avoid injuring the vascular structures.

Fluoroscopic-Guided Injections

In the authors' opinion, fluoroscopy provides little benefit for PTN injections, since there are no good bone landmarks.

Neurolytic Technique

Cryoneuroablation

Cryoneuroablation is not performed on the PTN, since it is a large mixed nerve, innervating multiple intrinsic foot muscles. However, cryoneuroablation can be used to treat smaller, more distal branches such as *inferior calcaneal nerve* (Chap. 76) or pure sensory nerves such as the *sural* (Chap. 71) or *distal saphenous* (Chap. 59) nerves of the foot.

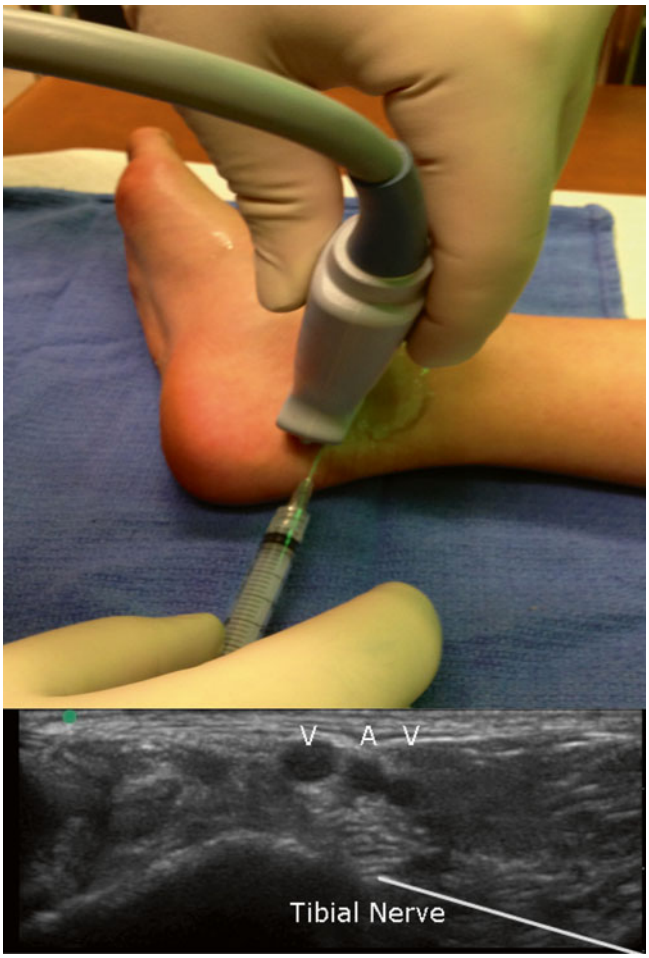

Fig. 73.16 Ultrasound-guided needle placement behind the tibial nerve (Image courtesy of Michael Brown, MD)

Pulsed Radio Frequency (RF)

No specific pulsed RF procedures to treat TTS have been described.

Neuromodulation

Although no specific neuromodulation techniques have been described for the tarsal tunnel, peripheral nerve stimulation for the lower extremities has been rapidly expanding [60, 61]. Dr. Porter McRoberts noted good relief with stimulation of the tarsal tunnel (personal communication) (Fig. 73.17).

Surgical Technique

Surgical treatment of TTS involves release of the PTN and its branches [10, 62]. Patients with coexisting radiculopathy or generalized neuropathy are more likely to have a poor outcome [63].

Barker et al. [25] measured proximal and distal TT pressures in cadavers placed in a variety of ankle positions. Dividing the flexor retinaculum decreased TT pressure in all positions; excising the septum between the medial and lateral plantar tunnels led to optimum pressure change. The same group [63] also described 44 patients (46 ankles) who had undergone prior, unsuccessful surgical release of the TT. These patients were treated with division of the roof of the medial and lateral plantar tunnels and of the septum between them. They underwent neurolysis of the MPN and

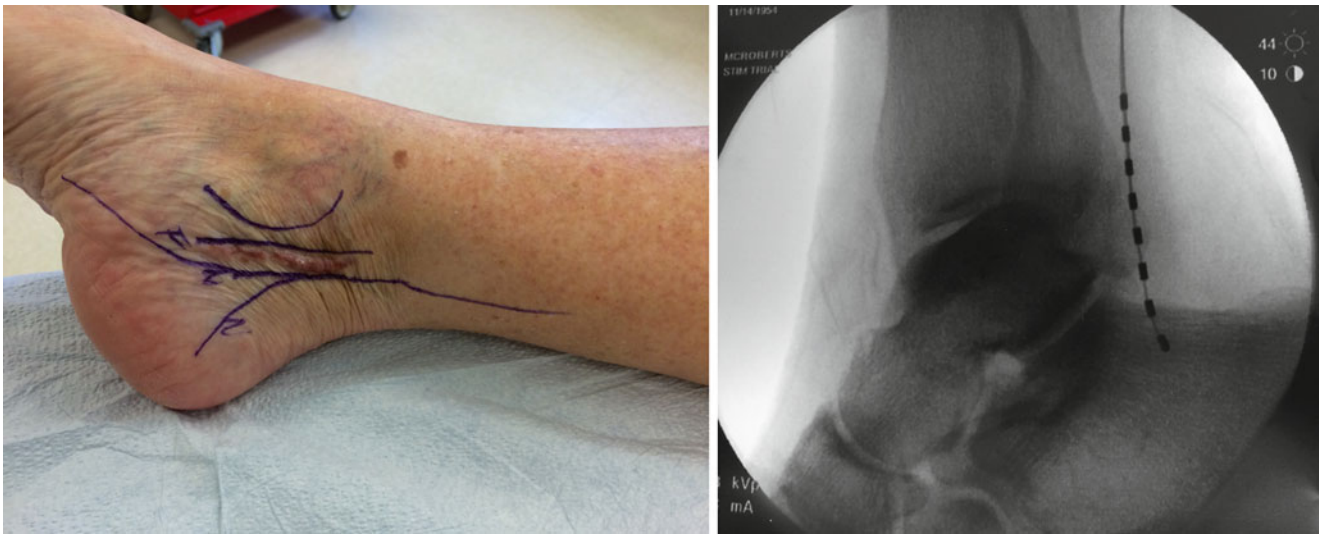

Fig. 73.17 Electrode placement for peripheral nerve stimulation (Image courtesy of W. Porter McRoberts, MD)

LPN as needed and were then followed for an average of 2.2 years. Fifty-four percent rated their pain relief as "excellent," 24 % noted "good" relief, and 9 % had "poor" outcomes.

Complications

As with any procedure, there are risks of infection, hematoma, vascular puncture, or nerve injury. The most common associated problem is incomplete nerve release or ablation leading to increased pain. In this circumstance, repeating the procedure may lead to resolution of symptoms. The skin is very thin at this site, so injections or surgery can result in skin injury or atrophy.

Summary

Tibial nerve entrapment can cause a variety of foot and heel pains, depending on the site of entrapment. Knowledge of the clinical presentation, the anatomy, and the treatment options can provide patients with needed pain relief.

References

1. Mastaglia FL, Venerys J, Stokes BA, Vaughan R. Compression of the tibial nerve by the tendinous arch of origin of the soleus muscle. Clin Exp Neurol. 1981;18:81–5.
2. Erickson SJ, Quinn SF, Kneeland JB, Smith JW, Johnson JE, Carrera GF, et al. MR imaging of the tarsal tunnel and related spaces: normal and abnormal findings with anatomic correlation. AJR Am J Roentgenol. 1990;155(2):323–8.
3. Delfaut EM, Demondion X, Bieganski A, Thiron MC, Mestdagh H, Cotten A. Imaging of foot and ankle nerve entrapment syndromes: from well-demonstrated to unfamiliar sites. Radiographics. 2003; 23(3):613–23.
4. Lopez-Ben R. Imaging of nerve entrapment in the foot and ankle. Foot Ankle Clin. 2011;16(2):213–24.
5. Heimkes B, Posel P, Stotz S, Wolf K. The proximal and distal tarsal tunnel syndromes. An anatomical study. Int Orthop. 1987;11(3): 193–6.
6. Mackinnon SE, Dellon AL. Homologies between the tarsal and carpal tunnels: implications for surgical treatment of the tarsal tunnel syndrome. Contemp Orthop. 1987;14(4):75–8.
7. Franson J, Baravarian B. Tarsal tunnel syndrome: a compression neuropathy involving four distinct tunnels. Clin Podiatr Med Surg. 2006;23(3):597–609.
8. Keck C. The tarsal-tunnel syndrome. J Bone Joint Surg Am. 1962;44A:180–2.
9. Lam SJS. A tarsal-tunnel syndrome. Lancet. 1962;2:1354–5.
10. Dellon AL. The four medial ankle tunnels: a critical review of perceptions of tarsal tunnel syndrome and neuropathy. Neurosurg Clin N Am. 2008;19(4):629–48, vii.
11. Nagaoka M, Matsuzaki H. Ultrasonography in tarsal tunnel syndrome. J Ultrasound Med. 2005;24(8):1035–40.
12. Taguchi Y, Nosaka K, Yasuda K, Teramoto K, Mano M, Yamamoto S. The tarsal tunnel syndrome. Report of two cases of unusual cause. Clin Orthop Relat Res. 1987;217:247–52.
13. Radin EL. Tarsal tunnel syndrome. Clin Orthop Relat Res. 1983;181:167–70.
14. Wittmayer BC, Freed L. Diagnosis and surgical management of flexor digitorum accessorius longus-induced tarsal tunnel syndrome. J Foot Ankle Surg. 2007;46(6):484–7.
15. Imai K, Ikoma K, Imai R, Fujiwara H, Kido M, Hara Y, et al. Tarsal tunnel syndrome in hemodialysis patients: a case series. Foot Ankle Int. 2013;34(3):439–44.
16. Goodgold J, Kopell HP, Spielholz NI. The tarsal-tunnel syndrome. Objective diagnostic criteria. N Engl J Med. 1965;273(14):742–5.
17. Baghla DP, Shariff S, Dega R. Calcaneal osteomyelitis presenting with acute tarsal tunnel syndrome: a case report. J Med Case Rep. 2010;4:66.
18. Meadows JR, Finnoff JT. Lower extremity nerve entrapments in athletes. Curr Sports Med Rep. 2014;13(5):299–306.
19. McCrory P, Bell S, Bradshaw C. Nerve entrapments of the lower leg, ankle and foot in sport. Sports Med. 2002;32(6):371–91.
20. Kinoshita M, Okuda R, Yasuda T, Abe M. Tarsal tunnel syndrome in athletes. Am J Sports Med. 2006;34(8):1307–12.
21. Kennedy JG, Baxter DE. Nerve disorders in dancers. Clin Sports Med. 2008;27(2):329–34.
22. Schon LC, Baxter DE. Neuropathies of the foot and ankle in athletes. Clin Sports Med. 1990;9(2):489–509.
23. Murphy PC, Baxter DE. Nerve entrapment of the foot and ankle in runners. Clin Sports Med. 1985;4(4):753–63.
24. Lee MS, Vanore JV, Thomas JL, Catanzariti AR, Kogler G, Kravitz SR, et al. Diagnosis and treatment of adult flatfoot. J Foot Ankle Surg. 2005;44(2):78–113.
25. Barker AR, Rosson GD, Dellon AL. Pressure changes in the medial and lateral plantar and tarsal tunnels related to ankle position: a cadaver study. Foot Ankle Int. 2007;28(2):250–4.
26. Trepman E, Kadel NJ, Chisholm K, Razzano L. Effect of foot and ankle position on tarsal tunnel compartment pressure. Foot Ankle Int. 1999;20(11):721–6.
27. Bracilovic A, Nihal A, Houston VL, Beattie AC, Rosenberg ZS, Trepman E. Effect of foot and ankle position on tarsal tunnel compartment volume. Foot Ankle Int. 2006;27(6):431–7.
28. Dellon AL. The Dellon approach to neurolysis in the neuropathy patient with chronic nerve compression. Handchir Mikrochir Plast Chir. 2008;40(6):351–60.
29. Driban JB, Swanik CB, Barbe MF. Anatomical evaluation of the tibial nerve within the popliteal fossa. Clin Anat. 2007;20(6):694–8.
30. Dellon AL, Mackinnon SE. Tibial nerve branching in the tarsal tunnel. Arch Neurol. 1984;41(6):645–6.
31. Havel PE, Ebraheim NA, Clark SE, Jackson WT, DiDio L. Tibial nerve branching in the tarsal tunnel. Foot Ankle. 1988;9(3):117–9.
32. Williams EH, Williams CG, Rosson GD, Dellon LA. Anatomic site for proximal tibial nerve compression: a cadaver study. Ann Plast Surg. 2009;62(3):322–5.
33. Ghosh SK, Raheja S, Tuli A. Potential sites of compression of tibial nerve branches in foot: a cadaveric and imaging study. Clin Anat. 2013;26(6):768–79.
34. Horwitz MT. Normal anatomy and variations of the peripheral nerves of the leg and foot. Application in operations for vascular diseases: study of one hundred specimens. Arch Surg. 1938;36(4):626–36.
35. Louisia S, Masquelet AC. The medial and inferior calcaneal nerves: an anatomic study. Surg Radiol Anat. 1999;21(3):169–73.
36. Dellon AL, Kim J, Spaulding CM. Variations in the origin of the medial calcaneal nerve. J Am Podiatr Med Assoc. 2002;92(2): 97–101.
37. Singh G, Kumar VP. Neuroanatomical basis for the tarsal tunnel syndrome. Foot Ankle Int. 2012;33(6):513–8.
38. Redborg KE, Antonakakis JG, Beach ML, Chinn CD, Sites BD. Ultrasound improves the success rate of a tibial nerve block at the ankle. Reg Anesth Pain Med. 2009;34(3):256–60.
39. Gray AT. Atlas of ultrasound-guided regional anesthesia. 2nd ed. Philadelphia: Elsevier Saunders; 2013.

40. Ahmad M, Tsang K, Mackenney PJ, Adedapo AO. Tarsal tunnel syndrome: a literature review. Foot Ankle Surg Off J Eur Soc Foot Ankle Surg. 2012;18(3):149–52.
41. Kinoshita M, Okuda R, Morikawa J, Jotoku T, Abe M. The dorsiflexion-eversion test for diagnosis of tarsal tunnel syndrome. J Bone Joint Surg Am. 2001;83-A(12):1835–9.
42. Mastaglia FL. Tibial nerve entrapment in the popliteal fossa. Muscle Nerve. 2000;23(12):1883–6.
43. Iida T, Kobayashi M. Tibial nerve entrapment at the tendinous arch of the soleus: a case report. Clin Orthop Relat Res. 1997;334:265–9.
44. Williams EH, Rosson GD, Hagan RR, Hashemi SS, Dellon AL. Soleal sling syndrome (proximal tibial nerve compression): results of surgical decompression. Plast Reconstr Surg. 2011;129(2):454–62.
45. Chhabra A, Williams EH, Subhawong TK, Hashemi S, Soldatos T, Wang KC, et al. MR neurography findings of soleal sling entrapment. AJR Am J Roentgenol. 2011;196(3):W290–7.
46. Kashani SR, Moon AH, Gaunt WD. Tibial nerve entrapment by a baker cyst: case report. Arch Phys Med Rehabil. 1985;66(1):49–51.
47. Baxter D. Compressive neuropathies of the foot and ankle. Oper Tech Sports Med. 1994;2(1):18–23.
48. Abouelela AA, Zohiery AK. The triple compression stress test for diagnosis of tarsal tunnel syndrome. Foot (Edinb Scotl). 2012; 22(3):146–9.
49. Fujita I, Matsumoto K, Minami T, Kizaki T, Akisue T, Yamamoto T. Tarsal tunnel syndrome caused by epineural ganglion of the posterior tibial nerve: report of 2 cases and review of the literature. J Foot Ankle Surg. 2004;43(3):185–90.
50. Lam SJ. Tarsal tunnel syndrome. J Bone Joint Surg (Br). 1967; 49(1):87–92.
51. Hochman MG, Zilberfarb JL. Nerves in a pinch: imaging of nerve compression syndromes. Radiol Clin N Am. 2004;42(1):221–45.
52. Duran-Stanton AM, Bui-Mansfield LT. Magnetic resonance diagnosis of tarsal tunnel syndrome due to flexor digitorum accessorius longus and peroneocalcaneus internus muscles. J Comput Assist Tomogr. 2010;34(2):270–2.
53. Mondelli M, Morana P, Padua L. An electrophysiological severity scale in tarsal tunnel syndrome. Acta Neurol Scand. 2004;109(4): 284–9.
54. Patel AT, Gaines K, Malamut R, Park TA, Toro DR, Holland N, et al. Usefulness of electrodiagnostic techniques in the evaluation of suspected tarsal tunnel syndrome: an evidence-based review. Muscle Nerve. 2005;32(2):236–40.
55. Dellon AL, Keller KM. Computer-assisted quantitative sensorimotor testing in patients with carpal and cubital tunnel syndromes. Ann Plast Surg. 1997;38(5):493–502.
56. Tassler PL, Dellon AL. Pressure perception in the normal lower extremity and in the tarsal tunnel syndrome. Muscle Nerve. 1996;19(3):285–9.
57. Dellon AL. Deciding when heel pain is of neural origin. J Foot Ankle Surg. 2001;40(5):341–5.
58. Rose JD, Malay DS, Sorrento DL. Neurosensory testing of the medial calcaneal and medial plantar nerves in patients with plantar heel pain. J Foot Ankle Surg. 2003;42(4):173–7.
59. Donovan A, Rosenberg ZS. MRI of ankle and lateral hindfoot impingement syndromes. AJR Am J Roentgenol. 2010;195(3): 595–604.
60. Huntoon MA, Huntoon EA, Obray JB, Lamer TJ. Feasibility of ultrasound-guided percutaneous placement of peripheral nerve stimulation electrodes in a cadaver model: part one, lower extremity. Reg Anesth Pain Med. 2008;33(6):551–7.
61. Huntoon MA, Burgher AH. Ultrasound-guided permanent implantation of peripheral nerve stimulation (PNS) system for neuropathic pain of the extremities: original cases and outcomes. Pain Med. 2009;10(8):1369–77.
62. Gondring WH, Shields B, Wenger S. An outcomes analysis of surgical treatment of tarsal tunnel syndrome. Foot Ankle Int. 2003;24(7):545–50.
63. Barker AR, Rosson GD, Dellon AL. Outcome of neurolysis for failed tarsal tunnel surgery. J Reconstr Microsurg. 2008;24(2): 111–8.
64. Martinoli C, Court-Payen M, Michaud J, Padua L, Altafini L, Marchetti A, et al. Imaging of neuropathies about the ankle and foot. Semin Musculoskelet Radiol. 2010;14(3):344–56.
65. Beltran LS, Bencardino J, Ghazikhanian V, Beltran J. Entrapment neuropathies III: lower limb. Semin Musculoskelet Radiol. 2010; 14(5):501–11.

Lateral Plantar Nerve Entrapment

74

Michael N. Brown, Beth S. Pearce, Thais Khouri Vanetti, Andrea M. Trescot, and Helen W. Karl

Introduction

Patients with foot pain are not uncommon in clinical practice. A recent systematic review revealed that 24 % of older adults (≥45 years) have frequent foot pain; foot pain is more common in women, especially in the forefoot, and causes at least moderate disability in most patients [1].

The *posterior tibial nerve* (PTN) travels down to the foot, through the tarsal tunnel, and then divides into the *lateral plantar nerve* (LPN) and the *medial plantar nerve* (MPN) (see Chap. 75). The LPN is the smaller of the two plantar nerves. It has a consistent cutaneous sensory distribution, so diagnosis of LPN dysfunction may be straightforward. Normal LPN activity is important for balance in walking and running [2–4].

Isolated injury of the main trunk of this nerve is very rare; only 8 % (2/25) of patients with foot pain due to tarsal tunnel syndrome were considered clinically to have LPN entrapment, though that number increased to 52 % (13/25, some with coexistent MPN dysfunction) after sensory nerve testing [5], which identified subclinical pathology.

This chapter will focus on the main trunk of the LPN. Entrapment of its first branch, the *inferior calcaneal nerve* (ICN), is more common [6] and is discussed in detail in Chap. 76.

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Clinical Presentation (Table 74.1)

Patients with LPN dysfunction usually have burning pain, paresthesias, and numbness in the lateral side of the sole (Fig. 74.1) that can extend to the lateral toes (Fig. 74.2) [9, 10, 12–14]. Sensory disturbances over the lateral toes are common, but motor deficit (the inability to abduct the little toe) is rare [12]. Patients often have a history of trauma [12] or previous surgery [7, 13], particularly failed tarsal tunnel release [15]. Complaints of burning, sharp, shooting heel and lateral foot pains are common, typically worse during or after weight-bearing activities and improved with rest (though the pain can occur during rest as well) [16].

LPN entrapment can coexist with compression of the tibial nerve at a variety of levels (*double crush syndrome*; see Chap. 1) or simultaneous entrapment of other PTN branches, most commonly the MPN [17]. Thus, the clinical picture may be multifaceted. Evaluation of LPN dysfunction can be

Table 74.1 Occupation/exercise/trauma history relevant to lateral plantar nerve entrapment

Trauma/surgery	Surgical harvest of the FHL tendon for Achilles or posterior tibial tendon repair (2 of 24 cases), although MPN injury is more likely (6 of 24 cases) [7, 8]
Mass lesion	Neurilemmoma [9, 10] Pseudoganglion [11]
Intrinsic foot anomaly	Increased foot pronation, midtarsal joint laxity, forefoot varus, rear foot eversion, pes planus, and cavovarus foot

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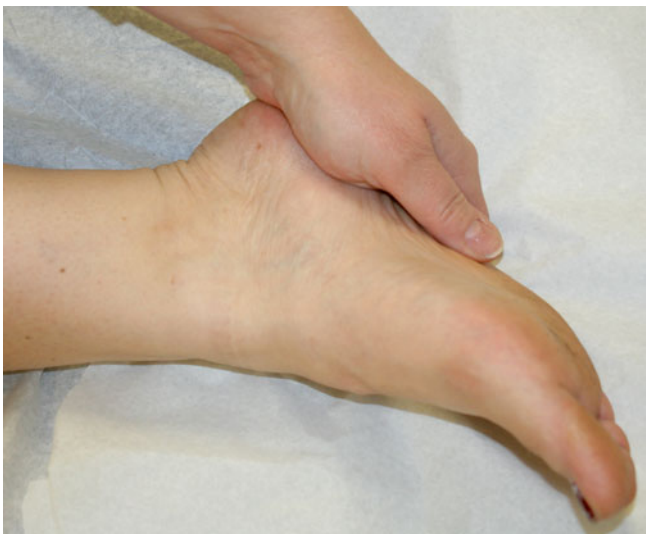

Fig. 74.1 Patient complaint of pain from lateral plantar nerve entrapment (Image courtesy of Andrea Trescot, MD)

particularly challenging in the setting of coexisting small fiber peripheral neuropathy, as seen in diabetes mellitus and other conditions.

Anatomy (Table 74.2)

The sciatic nerve travels down the leg and splits into the *common peroneal nerve* and the *tibial nerve* near the popliteal fossa. The tibial nerve at the ankle is often called the *posterior tibial nerve* (PTN, Chap. 73). It enters the proximal tarsal tunnel (deep to the *flexor retinaculum*, also known as the *laciniate ligament*) at the medial ankle (Fig. 74.3), where it divides into the LPN, the MPN, and the *medial calcaneal nerve* (MCN) (see Chap. 77) (Fig. 74.4). In the distal tarsal tunnel, deep to the *abductor hallucis muscle* (AbH), a connective tissue septum known as the *medial septum* [22] lies between the *medial calcaneus* (the floor of the tarsal tunnel) and the deep fascia of the AbH (the tunnel roof), and creates separate and independent medial and lateral plantar tunnels. These tunnels allow the tendons, vessels, and nerves to continue their transition further into the foot (Fig. 74.5) [24–27]. Recent anatomic investigations have expanded previous understanding of the myofascial compartments of the foot, as a basis for surgical decompression of compartment syndrome of the foot [23] and to provide additional understanding of the anatomy of the MPN and LPN [22]. A change in girth of any structure that crosses these confined areas can cause a compressive neuropathy.

The LPN exits the proximal tarsal tunnel posterior to the MPN (Fig. 74.6) and goes through the deep fascia of the AbH and the *medial plantar septum* [22] into the *lateral plantar tunnel* (Figs. 74.5 and 74.7) between the AbH and

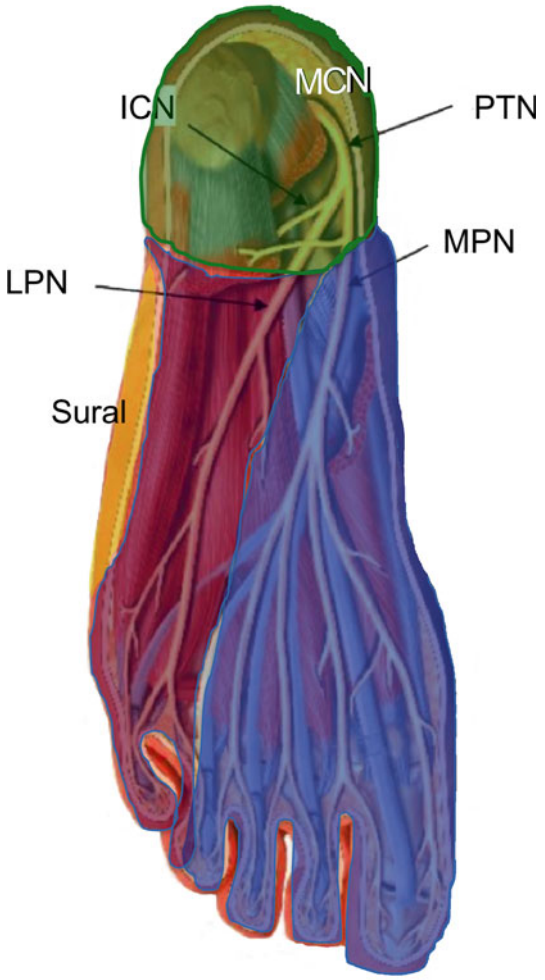

Fig. 74.2 Sensory map of the plantar nerves. *MCN* medical calcaneal nerve (*green*), *PTN* posterior tibial nerve, *MPN* medial plantar nerve (*blue*), *ICN* inferior calcaneal nerve, *LPN* lateral plantar nerve (*red*), *sural* sural nerve (*orange*) (Image courtesy of Michael Brown, MD)

the calcaneus (between the AbH and the *flexor digitorum brevis muscle* (FDB), as it moves diagonally across the foot toward the toes). The *lateral plantar tunnel* communicates with the central compartment of the sole of the foot and is sometimes termed the *porta pedis* [28]. Unfortunately, this term is confusing as it has been used loosely to refer to a variety of parts of the tarsal tunnel. For this reason, that term is avoided in this book.

The LPN emerges from the lateral border of the AbH (sometimes abbreviated AHM) about 2 cm distal to the caudal edge of the flexor retinaculum, approximately 1 cm proximal to the average site of MPN emergence [29]. The LPN then runs diagonally between the flexor digitorum brevis muscle (FDB) and the *quadratus plantae muscle* (QP) (also known as the *flexor accessorius muscle*) toward the medial base of the fifth metatarsal [22, 30] (Fig. 74.8).

Table 74.2 Lateral plantar nerve anatomy

Origin	L4–S4 ventral rami from the sciatic nerve
General route	The sciatic nerve divides in the distal thigh into the tibial (Chap. 73) and peroneal (fibular) (see Chap. 67) nerves, and the tibial nerve (TN) continues down through the deep posterior compartment of the leg
	The TN divides within or occasionally near the tarsal tunnel behind and inferior to medial malleolus into the lateral plantar (LPN), medial plantar (MPN), and medial calcaneal (MCN) nerves
	The first branch of the LPN is usually the inferior calcaneal nerve (ICN, Baxter's nerve)
	The LPN travels under the abductor hallucis (AbH) and diagonally across the sole between the flexor digitorum brevis (FDB) and quadratus plantae (QP) muscles. It divides into its terminal branches at the mid-lateral edge of the FDB [3]
	The deep motor branch innervates the lumbrical and adductor hallucis (AdH) muscles, and the superficial branch further divides into the digital nerves
Sensory distribution	*LPN*: fat pad over the fifth metatarsal head [3], skin of the lateral sole, plantar surface, and lateral side of the fourth and all of the fifth toe
	ICN: periosteum at the medial calcaneal tubercle (MCT), long plantar ligament
Motor innervation	*LPN*
	Flexor digiti minimi brevis (FDMB): from the base of the fifth metatarsal and long plantar ligament to the base of the proximal phalanx and little toe; flexes the MTP joint of the little toe
	Flexor accessorius (FA, also called *opponens digiti minimi*): from the long plantar ligament and sheath of the fibularis longus tendon to the fifth metatarsal; pulls the little toe slightly medial and plantar
	Adductor hallucis (AdH): from oblique and transverse heads to the base of the first proximal phalanx; adducts the great toe and supports both arches of the foot
	The third and fourth interosseus muscles
	ICN:
	Flexor digitorum brevis (FDB): from the anterior portion of the calcaneal tuberosity and the plantar aponeurosis to the sides of the second–fifth toes; flexes second–fifth toes and supports the longitudinal arch
	Quadratus plantae (QP): from the anterior portion of the calcaneal tuberosity to the lateral border of the flexor digitorum longus (FDL) tendon; augments FDL action
	Abductor digiti minimi (AbDM): from heads on the lateral process of the calcaneal tuberosity and the plantar aponeurosis to the base of the little toe; abducts and flexes the little toe
Anatomic variability	Presence of a connecting branch between the LPN and the MPN that penetrates the oblique head of the AdH in 86 % (19/22) [18]
	Other more lateral and superficial connections to the MPN were found in 28 % (14 of 50 cases) (27), 100 % (20 of 20 cases) [19], and 27 % (19 of 71 cases) [20]
Other relevant structures	*Tarsal tunnel*: deep to the flexor retinaculum (see Chap. 73)
	Abductor hallucis (*AbH*): from the distal flexor retinaculum, medial calcaneal tubercle, and the plantar aponeurosis to the base of the great toe; it flexes and abducts the great toe
	Plantar fascia (*plantar aponeurosis*): a strong, superficial layer of fascia (three cords) [21] that runs from the calcaneal tuberosity to the toes; posteriorly, it covers the FDB, which covers the QP, which in turn covers the long plantar ligament
	Medial calcaneal tubercle (*medial process of the calcaneal tuberosity*, *MCT*): the origin of the central band of plantar fascia, AH, FDB, and, more laterally, the AbDM
	Medial plantar septum: runs between the plantar fascial aponeurosis and the calcaneus, navicular, and medial cuneiform bones [22, 23]

The LPN ultimately divides into deep and superficial branches. The deep branch innervates the *flexor digiti minimi brevis muscle* (FDMB) (which has also been called the *flexor brevis minimi digiti muscle* or the *flexor digiti quinti brevis muscle*), the QP, and the *adductor hallucis muscle* (AdH), all of which help to maintain the longitudinal arch, flex the toes at the metatarsophalangeal (MTP) joints, and adduct the toes [31], counteracting the pull of the flexor digitorum longus (FDL). According to Arakawa et al. [11], there is often (19 of 22 specimens, 86 %) a connecting branch between the deep branches of the LPN and the MPN, in the space between the FHB muscle and FHL tendon, where it is subject to friction during walking or running.

The superficial branch gives off the two lateral *common digital nerves*, which further divide into the *plantar proper digital nerves* (Fig. 74.4, see Chap. 70) innervating the plantar aspect of the lateral fourth toe and the whole fifth toe (Fig. 74.2). The direction of the nerve's path toward the medial base of the fifth metatarsal helps direct the needle trajectory during a block of this nerve. Connections between the LPN and MPN at this level have also been identified (Fig. 74.8) [8–20].

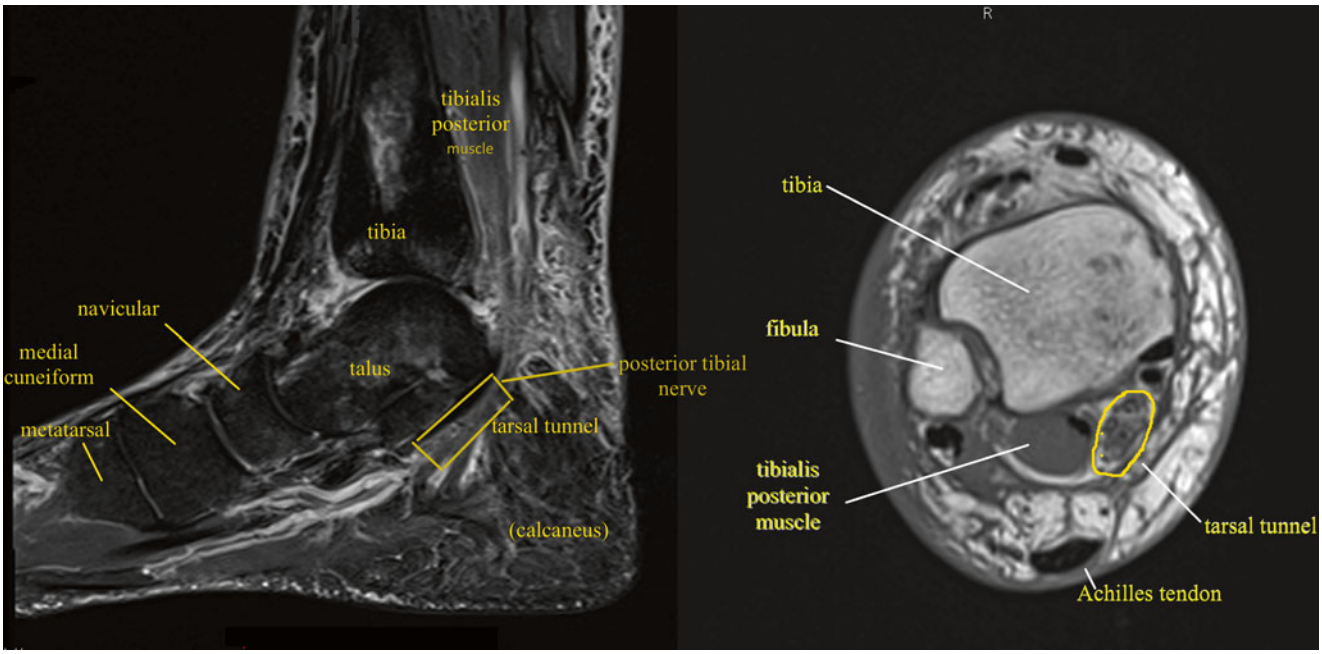

Fig. 74.3 MRI (sagittal and axial) of the tarsal tunnel (Image courtesy of Andrea Trescot, MD)

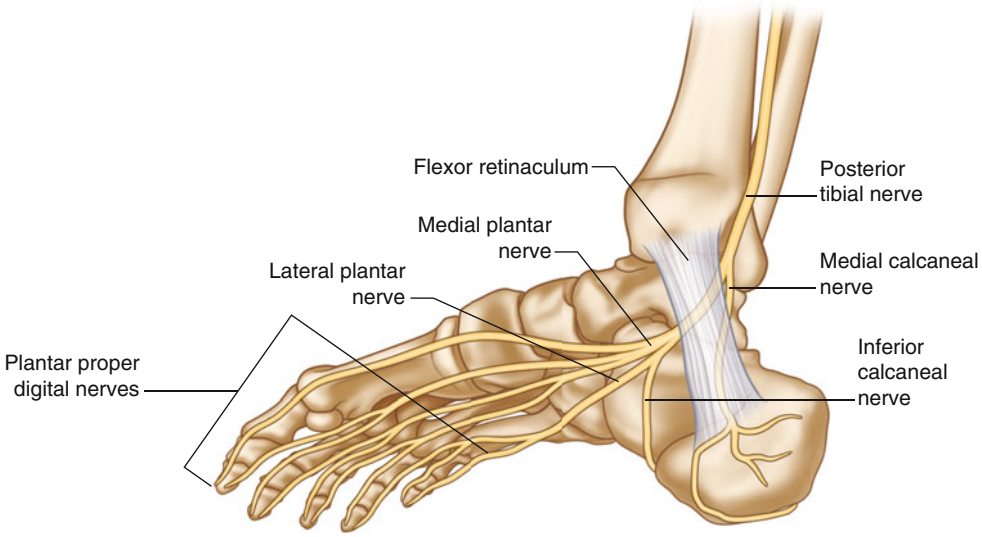

Fig. 74.4 Anatomy of the branches of the posterior tibial nerve. Note the relationship between the lateral plantar nerve and the medial base of the fifth metatarsal (Image courtesy of Springer)

Entrapment

Any space-occupying lesion along the LPN, including ganglia, bone and joint abnormalities, or tenosynovitis, may cause compression [9, 32]. It may be part of a tarsal tunnel syndrome that includes other branches of the PTN [33].

The LPN can be subject to stretch injury due to a hypermobile pronating foot [34], although the contribution of pronation to entrapment of the PTN and its branches is controversial [35–38].

Pronation of the ankle with or without plantar flexion (to simulate walking on a pronated foot) significantly elevates the pressure in the medial and lateral plantar tunnels as well as in the tarsal tunnel in cadavers [39] and surgical patients [40]. The lateral plantar tunnel is functionally analogous to Guyon's canal at the wrist (see Chap. 38) [39].

Physical Exam

On examination, the patient may have exquisite tenderness and paresthesias when the AbH is palpated (Fig. 74.9). Tinel's sign should be elicited at the proximal and distal tarsal tunnel. If the AbH is compressing a nerve or part of one,

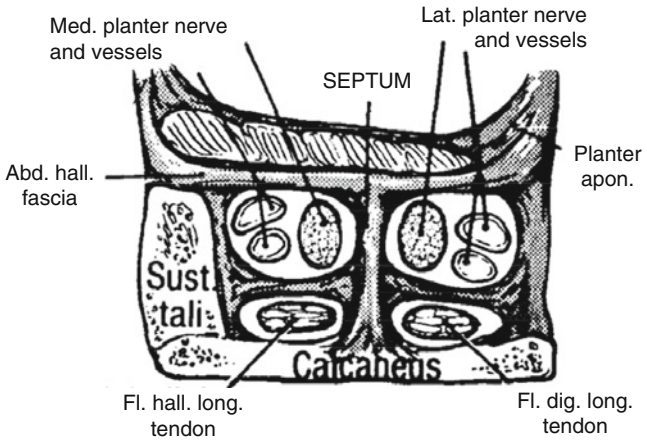

Fig 74.5 Cross section through the region immediately distal to the proximal tarsal tunnel. The medial and lateral plantar tunnels are separated by a septum (also known as the medial septum) between the tunnel roof (the deep abductor hallucis fascia) and the tunnel floor (the calcaneus). *Apon* aponeurosis, *dig* digital, *fl* flexor, *hall* hallucis, *lat* lateral, *long* longus, *med* medial, *sust* sustentaculum (From Dellon [26]. Reprinted with permission from Elsevier Limited)

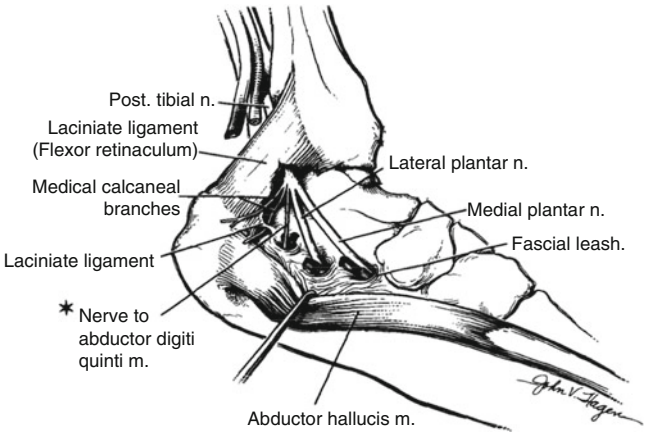

Fig. 74.6 Anatomy of the branches of the posterior tibial nerve. Note the lateral and medial plantar nerves as they cross the thick deep fascia of the abductor hallucis muscle. Note also the nerve to the *abductor digiti quinti muscle* (*inferior calcaneal nerve*) as it migrates under the deep fascia of the abductor hallucis and the medial calcaneal branches (nerves) as they cross the flexor retinaculum (*lancinate ligament*) to become more superficial (From Baxter and Thigpen [27]. Reprinted with permission from SAGE Publications)

the patient's symptoms may be reproduced by tensing it [29]. The *plantar flexion-inversion test* may reproduce symptoms (the foot is plantar flexed and inverted with pressure applied under the AbH) (Fig. 74.10a) [41], since this movement increases the pressure on the nerves within the tarsal tunnel [35]. Subsequent investigators have described the *dorsiflexion-eversion test*. The authors prefer the latter test, with pressure over the proximal tarsal tunnel (Fig. 74.10b). This maneuver has been shown to narrow the width of the tarsal

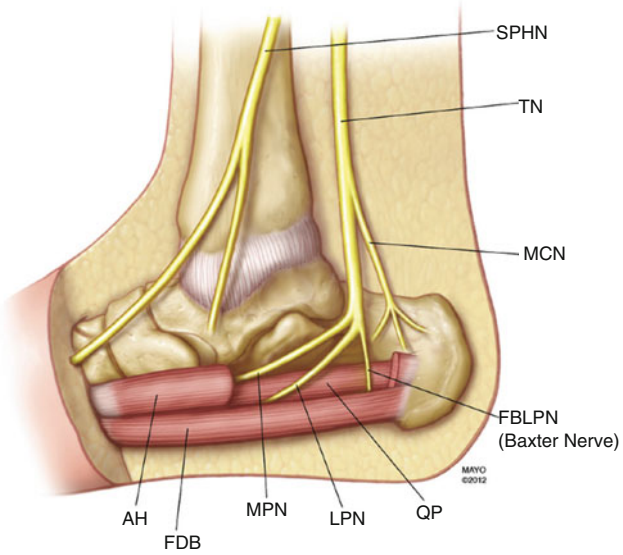

Fig. 74.7 Drawing of the medial ankle showing the branching pattern of the tibial nerve in the proximal and distal tarsal tunnels. The abductor hallucis has been partially removed to show the vertical course of the inferior calcaneal nerve, as well as the space between the abductor hallucis (removed) and quadratus plantae muscles. *SPHN* saphenous nerve, *TN* tibial nerve (posterior tibial nerve), *MCN* medial calcaneal nerve, *FBLPN* first branch of the lateral plantar nerve (inferior calcaneal nerve), *QP* quadratus plantae, *LPN* lateral plantar nerve, *MPN* medial plantar nerve. *FDB* flexor digitorum brevis, *AH* abductor hallucis (From Presley et al. [53]. Reprinted with permission from the American Institute of Ultrasound in Medicine)

tunnel and compress the LPN, first branch of the LPN, and the MPN [41]. Cadaver studies showed a significant increase in tension in the tibial nerve and the LPN within the tarsal tunnel during dorsiflexion, eversion, and combined dorsiflexion and eversion [42].

Differential Diagnosis (Table 74.3)

Plantar heel pain can sometimes be difficult to diagnose. The differential diagnosis includes plantar fasciitis, calcaneal fracture, plantar fascia rupture, and atrophy of the plantar fat pad [16], as well as S1 radiculopathy.

Diagnostic Tests (Table 74.4)

Weight-bearing plain radiographs can help assess foot alignment and exclude fractures or joint degeneration [45]. MRI may be helpful in identifying a potential cause for compressive neuropathy (Figs. 74.11 and 74.12), such as masses (especially between the QP, FDB, and AbH muscles), tendonitis, and muscle denervation [43].

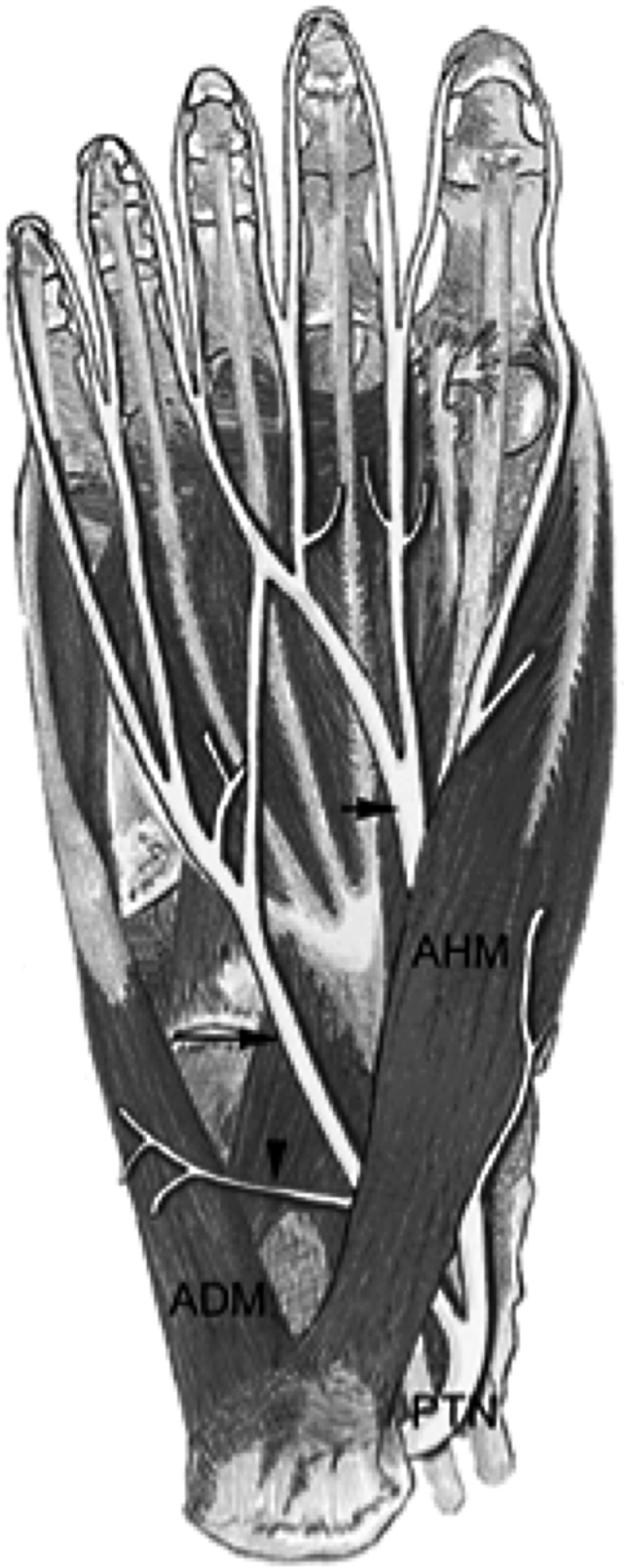

Fig. 74.8 Anatomy of the plantar nerves. *Short arrow* medial plantar nerve, *long arrow* lateral plantar nerve, *arrowhead* inferior calcaneal nerve, *PTN* posterior tibial nerve, *AHM* abductor hallucis muscle, *AbDM* abductor digiti minimi. Note the relationship of the plantar nerves to the abductor hallucis and the connecting branch between the lateral and medial plantar nerves (From Beltran et al. [30]. Reprinted with permission from Thieme)

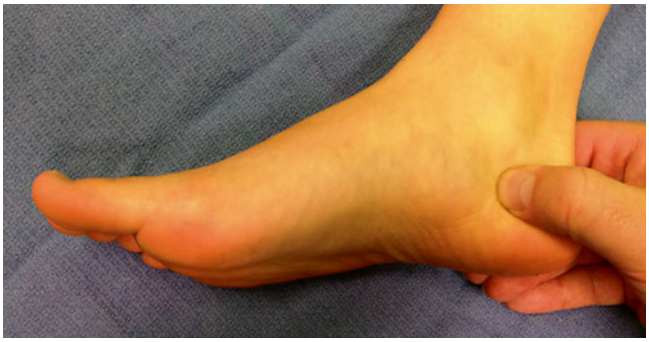

Fig. 74.9 Physical exam of the proximal plantar nerves in the tarsal tunnel (Image courtesy of Michael Brown, MD)

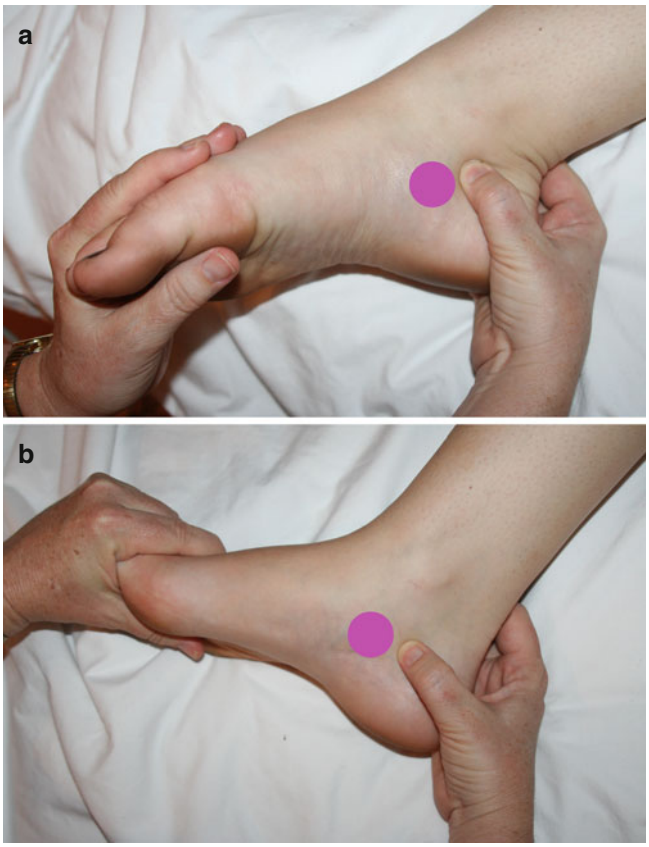

Fig. 74.10 (**a**) Plantar flexion-inversion with palpation; (**b**) dorsiflexion and foot eversion with pressure applied over the tarsal tunnel. The *pink dot* is centered on the malleolar-calcaneal axis (Image courtesy of Andrea Trescot, MD)

Nerve conduction velocity and electrodiagnostic testing may be helpful (Table 74.5) [12, 17]. Needle EMG may identify abnormalities of the QP, FDMB, and AbH muscles.

Ultrasonography (US) can show tendonitis, bursitis, masses, and nerve injuries. US is widely available, inexpensive, and quick; however, image quality is operator dependent and may be limited in the presence of dense scarring [15].

Table 74.3 Differential diagnosis of lateral foot pain

Condition	Potential distinguishing features
Tarsal tunnel syndrome	Presence of additional symptoms attributable to involvement of other tibial nerve branches
	Tenderness over the proximal tarsal tunnel
	Tinel's sign elicited over the medial ankle
S1 radiculopathy [24]	Proximal sensory loss
	Loss of Achilles reflex
	Weakness of dorsiflexion
Plantar fasciitis [17]	Tenderness at the fascial attachment
	Edema at attachment on MRI and US
Fat pad atrophy [17]	Palpable decreased heel pad height
Neuroma	Visible on US or MRI

Table 74.4 Diagnostic tests for lateral plantar nerve entrapment

	Potential distinguishing features
Physical exam	Tenderness over the medial calcaneal tubercle
Diagnostic injection	Landmark- or US-guided injections are diagnostic
Ultrasound	Used to identify the nerve and impingement structures
MRI	High-resolution sagittal MRI shows the LPN between the ICN and MPN; the veins between the LPN and ICN can become varicosities [43]; the LPN is found more posterior and lateral to the MPN and may show denervation changes of the AbDM if the ICN is involved [44]
Arteriography	Not useful
X-ray	May show bony abnormalities
Electrodiagnostic studies	Inconsistent results; may show denervation changes of the FDBM, QPM, and AbDM

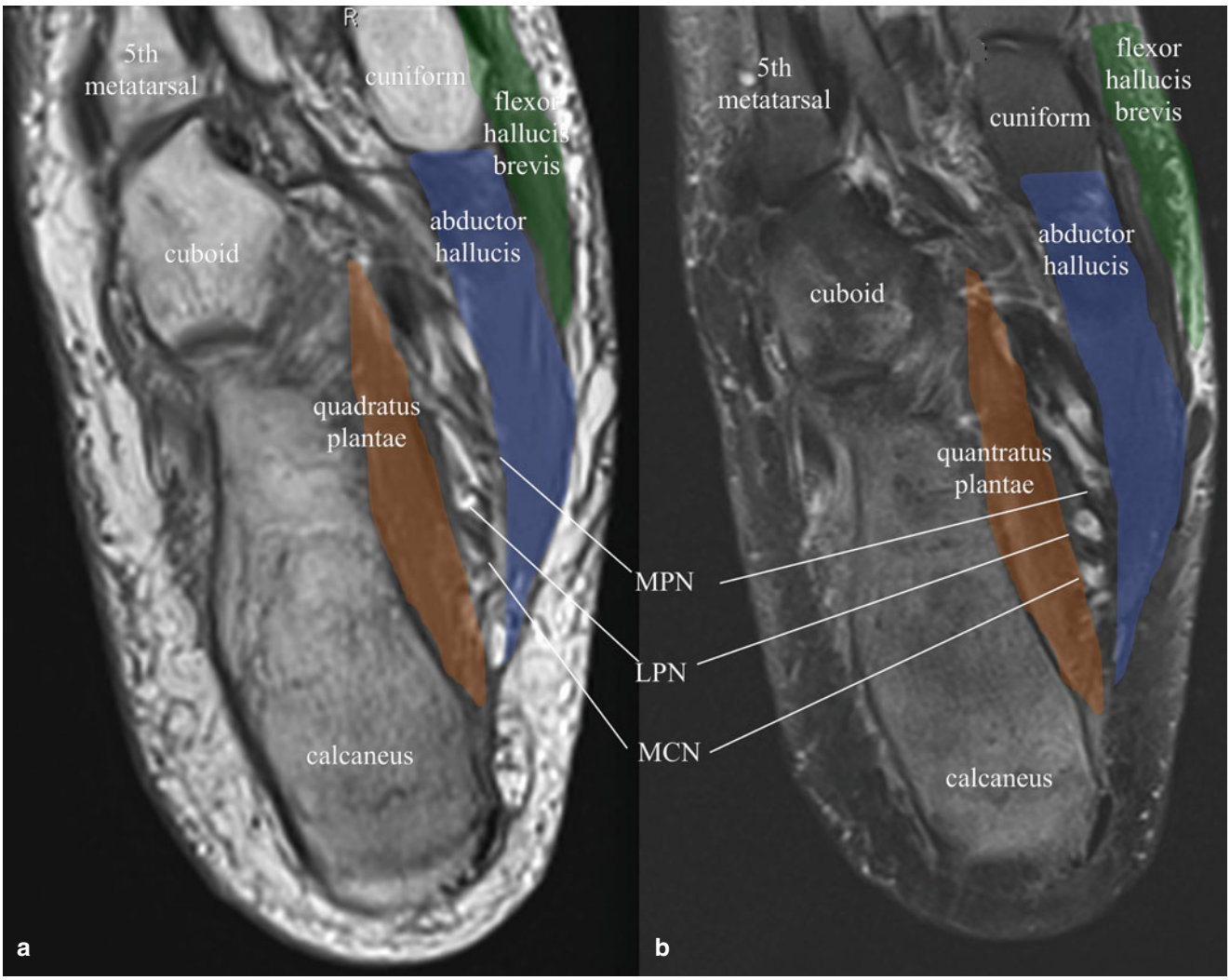

Fig. 74.11 Axial MRI images of the foot. (**a**) T1-weighted image; (**b**) T2-weighted image; *MPN* medial plantar nerve, *LPN* lateral planter nerve, *MCN* medial calcaneal nerve (Image courtesy of Andrea Trescot, MD)

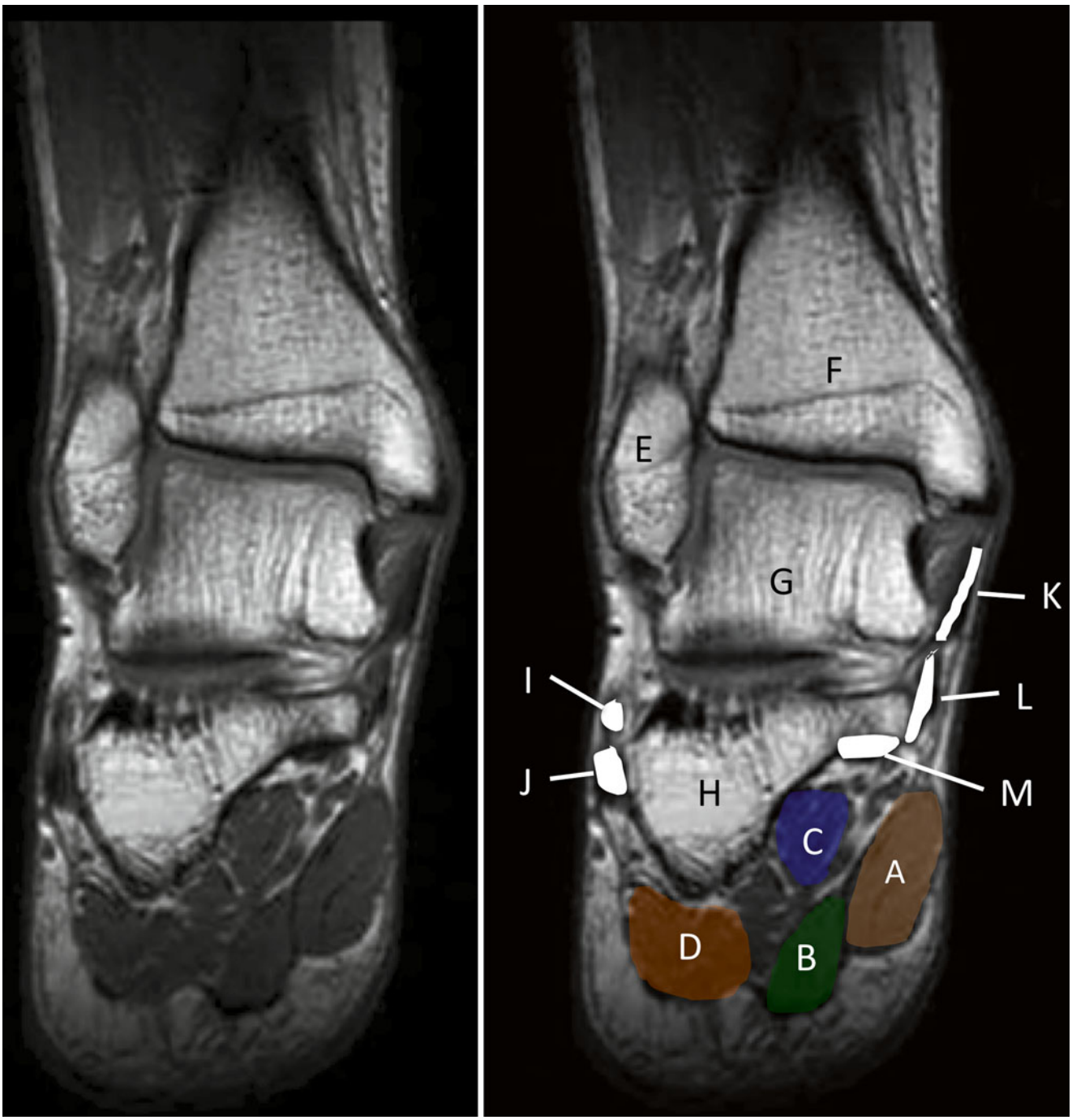

Fig. 74.12 Coronal MRI images of the foot. *A* abductor hallucis, *B* flexor digitorum brevis, *C* quadratus plantae, *D* abductor digiti minimi, *E* fibula, *F* tibia, *G* talus, *H* calcaneus, *I* peroneus brevis tendon, *J* peroneus longus tendon, *K* tibialis posterior tendon, *L* flexor digitorum longus tendon, *M* flexor hallucis longus (Image courtesy of Andrea Trescot, MD)

Identification and Treatment of Contributing Factors

Obesity, or any intrinsic foot anomaly that may contribute to increased foot pronation such as a midtarsal joint laxity, forefoot varus, rear foot eversion, pes planus, cavovarus foot, and more, may increase the risk of LPN entrapment. Treatment of perpetuating factors is similar to the treatment of those for plantar fasciitis. Correction of the passive mechanics of the foot with orthotics should be used when needed. Proper shoe wear and stretching of the plantar fascia and Achilles tendon will reduce stress on the ligaments and muscles. Weight reduction and physical therapy to strengthen the foot flexor and posterior tibialis muscles are additional important strategies to decrease hyperpronation [2]. Runners are at particular risk for LPN entrapment due to hypertrophy of the AbH [46].

Table 74.5 Diagnostic tests for lateral plantar neuralgia

	Potential distinguishing features
Physical exam	Tenderness over the medial calcaneal tubercle
Diagnostic injection	Landmark or US guidance injections are diagnostic
Ultrasound	Hypoechoic and thickened plantar fascia may not rule out ILPN entrapment
MRI	May show denervation changes of the ADM c/w lateral plantar entrapment [27, 44], but 6 % of asymptomatic patients have ADM atrophy [17]
	LPN is found more posterior and lateral to the MPN
Arteriography	Not useful
X-ray	May show bony abnormalities such as spurs
Electrodiagnostic studies	Inconsistent results; may show denervation changes of the FDBM, QPM, and ADM

Harvest of the FHL tendon for surgical repair of the Achilles tendon at the knot of Henry has been theoretically associated with LPN damage [47].

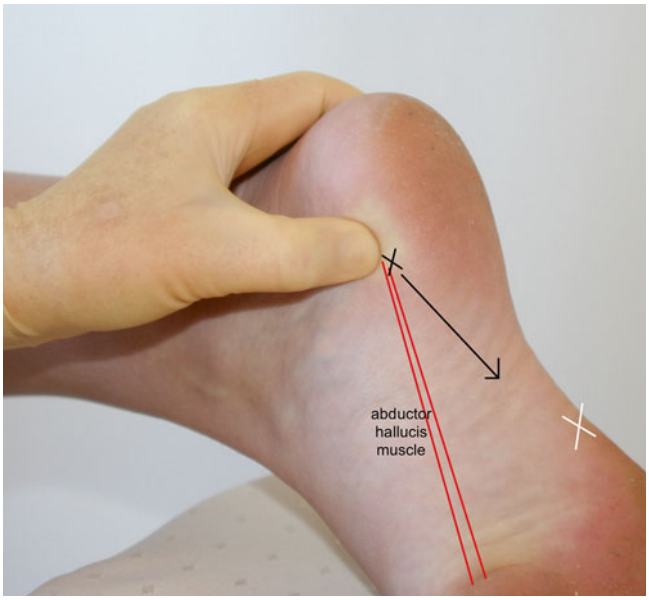

Fig. 74.13 Landmark-guided lateral plantar nerve injection. *Black X* needle insertion site, *white X* medial base of the fifth metatarsal, *arrow* direction of injection, *red line* abductor hallucis muscle (Image courtesy of Andrea Trescot, MD)

Injection Techniques

Successful low-volume local anesthetic injections provide diagnostic information and aid in planning for definitive therapeutic procedures (Chap. 7). Sensory testing will confirm that the local anesthetic injection reached the LPN, as a successful block will result in a sensory deficit along the lateral one third of the plantar aspect of the foot.

Landmark-Guided Injection

The patient is placed in a lateral position with the medial side of the foot facing up. A 27-gauge 1.5-in. needle, a 25-gauge 2-in. needle, or, when the patient has very small feet, a 30-gauge, 1-in. needle can be used. Mark the medial base of the fifth metatarsal to visualize the direction of the needle. Palpate the area just inferior to the medial malleolus, and slide the thumb downward, intermittently squeezing until contacting the first muscle bundle, the AbH (Fig. 74.9). When the patient spreads their toes, one can feel the AbH move. The LPN usually runs just cephalad to the AbH until it enters the lateral plantar tunnel; it then becomes deep and progressively lateral to it (Fig. 74.8).

Place a small skin wheal of local anesthetic just above the AbH, and insert the chosen needle. Direct it at an oblique angle across the foot toward the medial base of the fifth metatarsal (Fig. 74.13). The AbH is no wider than about 2–3 cm, so insert the needle to about that depth and inject 1 cc of therapeutic solution, usually local anesthetic and deposteroid, to address possible entrapment at the AbH fascia. The injection site needs to be posterior to and slightly deeper than that for MPN injection, to bypass the region where the MPN and LPN travel together (Fig. 74.6). After the injection, test for cutaneous numbness in the LPN distribution to confirm the block.

Fluoroscopic-Guided Technique

In the authors' opinion, fluoroscopy provides little benefit for LPN injections, since there are no appropriate bony landmarks.

Ultrasound-Guided Injections

US evaluation and injection may be more effective than landmark-guided injections for diagnosis and treatment of LPN entrapment. Mark the top of the AbH and the medial base of the fifth metatarsal as with the landmark-guided injection. Ultrasound provides the point of needle insertion.

Out-of-Plane Technique

The *malleolar-calcaneal axis* (MCA) from the center of the medial malleolus to the center of the calcaneus is a useful constant reference point for the center of the tarsal tunnel [48, 49]. Place a 7.5–15 MHz linear transducer obliquely over the MCA, with the notch toward the medial

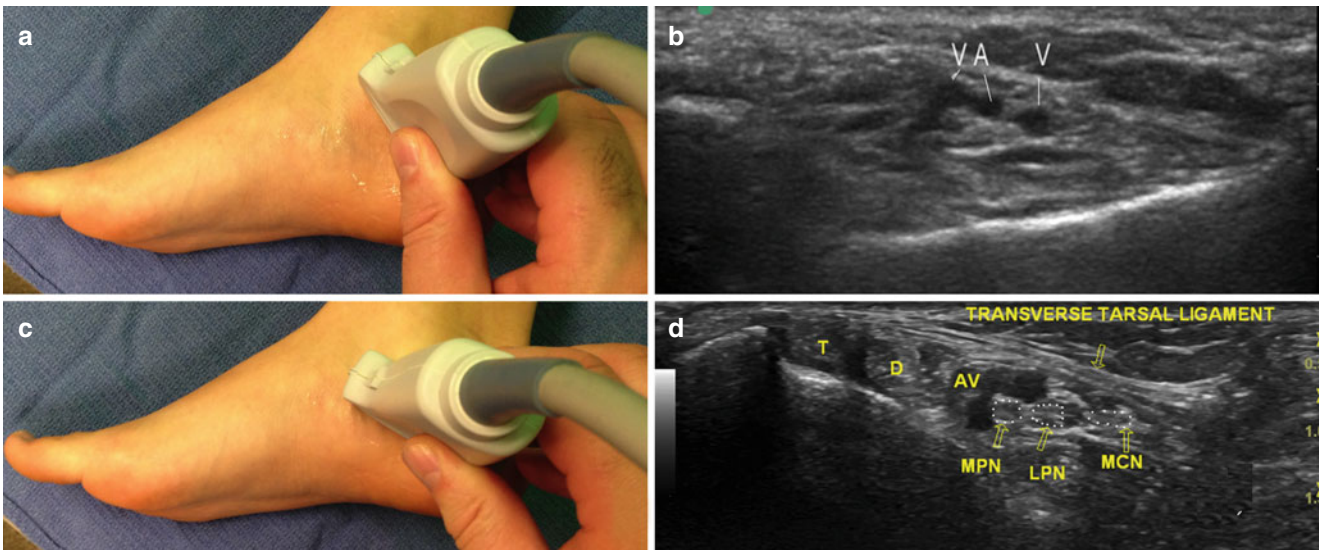

Fig. 74.14 Ultrasound localization of the plantar nerves. (**a**) Proximal tarsal tunnel ultrasound probe placement; (**b**) ultrasound image of the proximal tarsal tunnel (*A* artery, *V* vein); (**c**) ultrasound probe placement to show the plantar nerves as they enter the distal tarsal tunnel; (**d**) ultrasound identification of the plantar nerves; *T* posterior tibialis tendon, *D* flexor digitorum longus tendon, *AV* artery/vein complex, *MPN* medial plantar nerve, *LPN* lateral plantar nerve, *MCN* medial calcaneal nerve; transverse tarsal ligament = flexor retinaculum (Image courtesy of Michael Brown, MD)

malleolus (Fig. 74.14a), and find the vein-artery-vein (VAV) configuration of the vascular structures within the proximal tarsal tunnel (Fig. 74.14b) (Chap. 73). Move the transducer caudad and rotate it slightly (Fig. 74.14c) to identify the MPN (most anterior), LPN, and MCN (Fig. 74.14d). Rotate it further until a transverse view of the AbH and FHB centered on the LPN comes into view. Confirm the position of the AbH by having the patient spread their toes; the upper muscle will contract, confirming that it is the AbH. Just plantar to this muscle is the FHB; have the patient flex the great toe to see it contracting below the AbH. The advantage of this transducer position is that one can clearly see the transition zone above the AbH to direct the needle over the muscle. Once the transducer is in position, provide skin anesthesia just above the AbH with a 30-gauge needle. Insert a 25- or 27-gauge needle and direct it over the AbH across the foot toward the medial base of the fifth metatarsal. Inject 0.1–0.2 cc aliquots of saline to track the position of the needle tip as it is advanced, keeping it in view at all times. At this location, the tissue planes can be distended by hydrodissection as needed for better visualization and injection. Use normal saline, not local anesthetic, for this purpose so as not to confound a diagnostic block. Advance the needle in the fascial plane over the AbH, and direct it toward the medial base of the fifth metatarsal. Once the needle tip is beyond the AbH, inject 1–1.5 cc of local anesthetic (with or without deposteroid) for the block. After the injection, test for cutaneous numbness in the LPN distribution to confirm the block (Fig. 74.2).

In-Plane Technique

Place the transducer longitudinally over the AbH (Fig. 74.15). Move it over the AbH until its anterior edge is just in front of the medial calcaneal tubercle. Confirm its position by functionally testing the muscles: abduction of the great toe against resistance should contract the AbH, and passive flexion and extension of the lesser toes will move the QP (Fig. 74.16). Slide the transducer slightly toward the sole to allow room for the needle over the AbH. Use a 27- or 25-gauge needle and direct it across the foot toward the medial base of the fifth metatarsal. Once the needle is past the AbH fascia, inject 0.5–1 cc of therapeutic solution. After the injection, test for cutaneous numbness in the LPN distribution to confirm the block (Fig. 74.2).

Neurolytic/Surgical Technique

Cryoneuroablation

The LPN provides significant motor function to the foot and would not normally be a target for a neurolytic technique such as cryoneuroablation. However, because of the reversible nature of cryoneuroablation, unlike conventional radiofrequency or alcohol/phenol neurolytic techniques (see Chap. 8), there may be some clinical situations where a relatively temporary (3 month) muscle dysfunction with longer-term (6–12 month) analgesia might be appropriate. Cryoneuroablation of the ICN, the first branch of the LPN, is

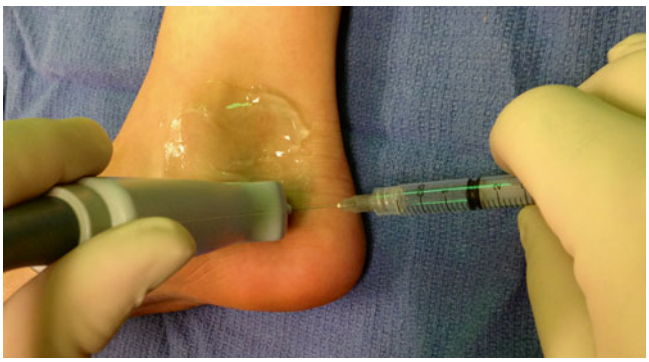

Fig. 74.15 Ultrasound probe and needle position for in-plane injection (Image courtesy of Michael N. Brown, MD)

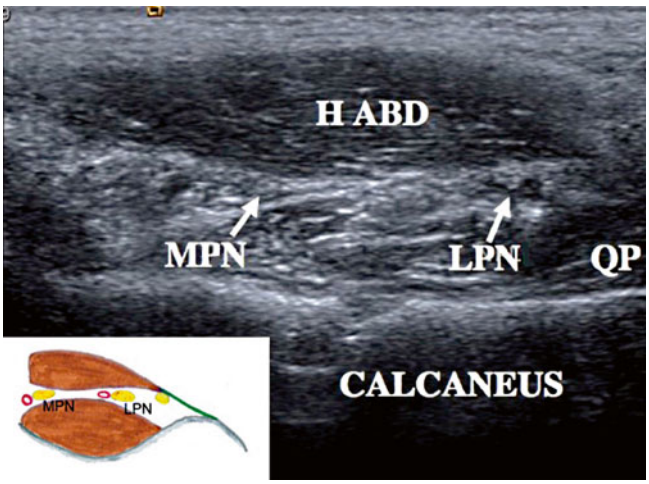

Fig. 74.16 Ultrasound anatomy of the distal tarsal tunnel, axial scan. *LPN* lateral plantar nerve, *MPN* medial plantar nerve, *QP* quadratus plantae muscle, *H ABD* abductor hallucis muscle (From Fantino [54]. Reprinted with permission from Springer)

discussed in Chap. 76. Placement of the probe for cryoneuroablation of the LPN should be as distal as possible to limit motor loss. An in-plane US approach gives the best view of the end of the probe; use of the built-in stimulator can help with positioning as close to the nerve as possible.

Surgery

Surgical release of LPN entrapment is performed in conjunction with release of the PTN and its other branches [26, 50]. Patients with coexisting radiculopathy or generalized neuropathy are more likely to have a poor outcome [51].

Complications

As with any procedure, there are risks of infection, hematoma, vascular puncture, or nerve injury. The most common associated problem is incomplete nerve release of the tarsal tunnel, leading to increased pain. In this circumstance, repeating the procedure may lead to resolution of symptoms. The skin is very thin at this site, so injections or surgery can result in skin injury or atrophy. There is a report of LPN injury after a steroid injection for plantar fasciitis [52].

Summary

Entrapment of the LPN is a possible cause of or contributor to lateral foot pain. A careful history (describing the triggers of pain), physical exam (showing tenderness over the tarsal tunnel or the LPN), and diagnostic injections can help the clinician to recognize and treat LPN entrapment.

References

1. Thomas MJ, Roddy E, Zhang W, Menz HB, Hannan MT, Peat GM. The population prevalence of foot and ankle pain in middle and old age: a systematic review. Pain. 2011;152(12):2870–80.
2. McKeon PO, Hertel J, Bramble D, Davis I. The foot core system: a new paradigm for understanding intrinsic foot muscle function. Br J Sports Med. 2015;49(5):290.
3. Unlü RE, Orbay H, Kerem M, Esmer AF, Tüccar E, Sensöz O. Innervation of three weight-bearing areas of the foot: an anatomic study and clinical implications. J Plast Reconstr Aesthet Surg. 2008;61(5):557–61.
4. Dellon AL, Steck JK. Reversal of toe clawing in the patient with neuropathy by neurolysis of the distal tibial nerve. Microsurgery. 2008;28(5):303–5.
5. Oh SJ, Kim HS, Ahmad BK. The near-nerve sensory nerve conduction in tarsal tunnel syndrome. J Neurol Neurosurg Psychiatry. 1985;48(10):999–1003.
6. Baxter DE, Pfeffer GB. Treatment of chronic heel pain by surgical release of the first branch of the lateral plantar nerve. Clin Orthop Relat Res. 1992;279:229–36.
7. Lui TH. Lateral plantar nerve neuropraxia after FHL tendoscopy: case report and anatomic evaluation. Foot Ankle Int. 2010;31(9):828–31.
8. Mulier T, Rummens E, Dereymaeker G. Risk of neurovascular injuries in flexor hallucis longus tendon transfers: an anatomic cadaver study. Foot Ankle Int. 2007;28(8):910–5.
9. Coulter KR, Gerbert J, Shea TP. Neurilemmoma of the lateral plantar nerve: a case report. J Am Podiatry Assoc. 1978;68(10):721–4.
10. Kwok KB, Lui TH, Lo WN. Neurilemmoma of the first branch of the lateral plantar nerve causing tarsal tunnel syndrome. Foot Ankle Spec. 2009;2(6):287–90.
11. Arakawa T, Sekiya S, Terashima T, Miki A. Pseudoganglion on the connecting branch between the deep branch of the lateral plantar nerve and medial plantar nerve. Clin Anat. 2011;24(5):646–51.
12. Oh SJ, Kwon KH, Hah JS, Kim DE, Demirci M. Lateral plantar neuropathy. Muscle Nerve. 1999;22(9):1234–8.
13. Rao SB, Crawford AH. Lateral plantar nerve entrapment following rush rod fixation for congenital pseudarthrosis of the tibia. Orthopedics. 1996;19(12):1043–5; discussion 6.
14. Belding RH. Neurilemmoma of the lateral plantar nerve producing tarsal tunnel syndrome: a case report. Foot Ankle. 1993;14(5):289–91.
15. Chalian M, Soldatos T, Faridian-Aragh N, Williams EH, Rosson GD, Eng J, et al. 3T magnetic resonance neurography of tibial nerve pathologies. J Neuroimaging. 2013;23(2):296–310.

16. Alshami AM, Souvlis T, Coppieters MW. A review of plantar heel pain of neural origin: differential diagnosis and management. Man Ther. 2008;13(2):103–11.
17. Schon LC, Glennon TP, Baxter DE. Heel pain syndrome: electrodiagnostic support for nerve entrapment. Foot Ankle. 1993;14(3): 129–35.
18. Govsa F, Bilge O, Ozer MA. Anatomical study of the communicating branches between the medial and lateral plantar nerves. Surg Radiol Anat. 2005;27(5):377–81.
19. Jones JR, Klenerman L. A study of the communicating branch between the medial and lateral plantar nerves. Foot Ankle. 1984;4(6):313–5.
20. Levitsky KA, Alman BA, Jevsevar DS, Morehead J. Digital nerves of the foot: anatomic variations and implications regarding the pathogenesis of interdigital neuroma. Foot Ankle. 1993;14(4):208–14.
21. Hoffman D, Bianchi S. Sonographic evaluation of plantar hindfoot and midfoot pain. J Ultrasound Med. 2013;32(7):1271–84.
22. Singh G, Kumar VP. Neuroanatomical basis for the tarsal tunnel syndrome. Foot Ankle Int. 2012;33(6):513–8.
23. Ling ZX, Kumar VP. The myofascial compartments of the foot: a cadaver study. J Bone Joint Surg (Br). 2008;90(8):1114–8.
24. Heimkes B, Posel P, Stotz S, Wolf K. The proximal and distal tarsal tunnel syndromes. An anatomical study. Int Orthop. 1987;11(3): 193–6.
25. Mackinnon SE, Dellon AL. Homologies between the tarsal and carpal tunnels: implications for surgical treatment of the tarsal tunnel syndrome. Contemp Orthop. 1987;14(4):75–8.
26. Dellon AL. The four medial ankle tunnels: a critical review of perceptions of tarsal tunnel syndrome and neuropathy. Neurosurg Clin N Am. 2008;19(4):629–48, vii.
27. Baxter DE, Thigpen CM. Heel pain–operative results. Foot Ankle. 1984;5(1):16–25.
28. Sarrafian SK, Kelikian AS. Retaining systems and compartments. In: Kelikian AS, editor. Sarrafian's anatomy of the foot and ankle: descriptive, topographic, functional. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2011.
29. Ghosh SK, Raheja S, Tuli A. Potential sites of compression of tibial nerve branches in foot: a cadaveric and imaging study. Clin Anat. 2013;26(6):768–79.
30. Beltran LS, Bencardino J, Ghazikhanian V, Beltran J. Entrapment neuropathies III: lower limb. Semin Musculoskelet Radiol. 2010;14(5):501–11.
31. Kopell HP, Thompson WA. Peripheral Entrapment Neuropathies. Baltimore: Williams and Wilkins; 9163.
32. Peri G. The "critical zones" of entrapment of the nerves of the lower limb. Surg Radiol Anat. 1991;13(2):139–43.
33. Oh SJ, Lee KW. Medial plantar neuropathy. Neurology. 1987;37(8):1408–10.
34. Schon LC, Baxter DE. Neuropathies of the foot and ankle in athletes. Clin Sports Med. 1990;9(2):489–509.
35. Trepman E, Kadel NJ, Chisholm K, Razzano L. Effect of foot and ankle position on tarsal tunnel compartment pressure. Foot Ankle Int. 1999;20(11):721–6.
36. Mann RA. Increased pressures in the tarsal tunnel with various foot positions. Foot Ankle Int. 2000;21(7):616–7.
37. Franson J, Baravarian B. Tarsal tunnel syndrome: a compression neuropathy involving four distinct tunnels. Clin Podiatr Med Surg. 2006;23(3):597–609.
38. Francis H, March L, Terenty T, Webb J. Benign joint hypermobility with neuropathy: documentation and mechanism of tarsal tunnel syndrome. J Rheumatol. 1987;14(3):577–81.
39. Barker AR, Rosson GD, Dellon AL. Pressure changes in the medial and lateral plantar and tarsal tunnels related to ankle position: a cadaver study. Foot Ankle Int. 2007;28(2):250–4.
40. Rosson GD, Larson AR, Williams EH, Dellon AL. Tibial nerve decompression in patients with tarsal tunnel syndrome: pressures in the tarsal, medial plantar, and lateral plantar tunnels. Plast Reconstr Surg. 2009;124(4):1202–10.
41. Hendrix CL, Jolly GP, Garbalosa JC, Blume P, DosRemedios E. Entrapment neuropathy: the etiology of intractable chronic heel pain syndrome. J Foot Ankle Surg. 1998;37(4):273–9.
42. Daniels TR, Lau JT, Hearn TC. The effects of foot position and load on tibial nerve tension. Foot Ankle Int. 1998;19(2):73–8.
43. Farooki S, Theodorou DJ, Sokoloff RM, Theodorou SJ, Trudell DJ, Resnick D. MRI of the medial and lateral plantar nerves. J Comput Assist Tomogr. 2001;25(3):412–6.
44. Chundru U, Liebeskind A, Seidelmann F, Fogel J, Franklin P, Beltran J. Plantar fasciitis and calcaneal spur formation are associated with abductor digiti minimi atrophy on MRI of the foot. Skeletal Radiol. 2008;37(6):505–10.
45. Lareau CR, Sawyer GA, Wang JH, DiGiovanni CW. Plantar and medial heel pain: diagnosis and management. J Am Acad Orthop Surg. 2014;22(6):372–80.
46. Donovan A, Rosenberg ZS, Cavalcanti CF. MR imaging of entrapment neuropathies of the lower extremity. Part 2. The knee, leg, ankle, and foot. Radiographics. 2010;30(4):1001–19.
47. Amlang M, Rosenow MC, Friedrich A, Zwipp H, Rammelt S. Direct plantar approach to Henry's knot for flexor hallucis longus transfer. Foot Ankle Int. 2012;33(1):7–13.
48. Dellon AL, Mackinnon SE. Tibial nerve branching in the tarsal tunnel. Arch Neurol. 1984;41(6):645–6.
49. Havel PE, Ebraheim NA, Clark SE, Jackson WT, DiDio L. Tibial nerve branching in the tarsal tunnel. Foot Ankle. 1988;9(3):117–9.
50. Gondring WH, Shields B, Wenger S. An outcomes analysis of surgical treatment of tarsal tunnel syndrome. Foot Ankle Int. 2003;24(7):545–50.
51. Barker AR, Rosson GD, Dellon AL. Outcome of neurolysis for failed tarsal tunnel surgery. J Reconstr Microsurg. 2008;24(2): 111–8.
52. Snow DM, Reading J, Dalal R. Lateral plantar nerve injury following steroid injection for plantar fasciitis. Br J Sports Med. 2005;39(12):e41.
53. Presley JC, et al. Sonographic visualization of the first branch of the lateral plantar nerve (Baxter nerve): technique and validation using perineural injections in a cadaveric model. J Ultrasound Med. 2013;32(9):1643–52.
54. Fantino O. Role of ultrasound in posteromedial tarsal tunnel syndrome: 81 cases. J Ultrasound. 2014;17(2):99–112.

Medial Plantar Nerve Entrapment

75

Michael N. Brown, Beth S. Pearce, Andrea M. Trescot, and Helen W. Karl

Introduction

Patients with medial foot and arch pain are not uncommon in clinical practice. A recent systematic review revealed that 24 % of older adults (≥45 years) have frequent foot pain; it is more common in women, in the forefoot, and causes at least moderate disability in most patients [1].

The *medial plantar nerve* (MPN) (also known as the *internal plantar nerve*) is the larger of the two plantar nerves. It crosses the midfoot deep to the junction of the talus and navicular bones, making patients who hyperpronate or those with a high medial arch vulnerable to compression and irritation of the MPN [2]. Normal MPN activity is important for balance, walking, and many sports [3–6].

Isolated entrapment of the MPN is relatively rare. Only 5 % (1 out of 21) of patients with ankle and foot pain diagnosed with nerve entrapment were considered to have MPN entrapment [7]. However, forty percent (10 out of 25) of patients with foot pain due to tarsal tunnel syndrome were considered clinically to have MPN entrapment, and that number increased to 96 % (24 out of 25, some with coexistent LPN dysfunction) after sensory nerve testing [8].

The MPN has a consistent cutaneous sensory distribution, so the clinical diagnosis of MPN entrapment or dysfunction may be straightforward.

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Clinical Presentation (Table 75.1)

Patients with MPN entrapment complain of burning pain, dysesthesias, and aching in the medial arch of the foot that increases with activity and may radiate to the plantar aspect of the first and second toes or to the heel (Fig. 75.1) [3, 21]. They will be tender over the medial arch, just posterior to the navicular tuberosity, and may have decreased sensation on the sole of the foot behind the great toe (Fig. 75.2) [3, 4, 9, 21].

Their discomfort may be aggravated by arch supports [3, 9], by running on a slanted track or road that increases pronation [4], and by inappropriate footwear such as narrow shoes with tight laces. They may have a history of intrinsic foot problems or may have had surgical harvesting of the

Table 75.1 Occupation/exercise/trauma history relevant to medial plantar nerve entrapment

Heel valgus/ excessive pronation	Running or walking in unsupportive shoes ("jogger's foot") [3] New orthosis [9]
Intrinsic foot pathology	Hallux rigidus (stiffness of the great toe) [2, 9]
Compression	Ganglion cysts, schwannoma [10], neurofibromas [11], neurilemoma [12], peripheral nerve sheath tumor [13], pseudoganglion [14]
	Hypertrophic muscles
	Varicosities [2]
	Inappropriate shoes
Inflammation	Tenosynovitis of the FDL, FHL [4]
Surgery	Surgical harvesting of FHL for Achilles tendon repair [15–17] Repair of a FHL laceration [18] Repair of a distal tibial fracture [19]
Occupation	Joggers [3]
	Chronic vibration (pneumatic drill) [20]
	Military recruits [4]
	Used stair climbing machine [4]
	Standing on a ladder in thin shoes [2]

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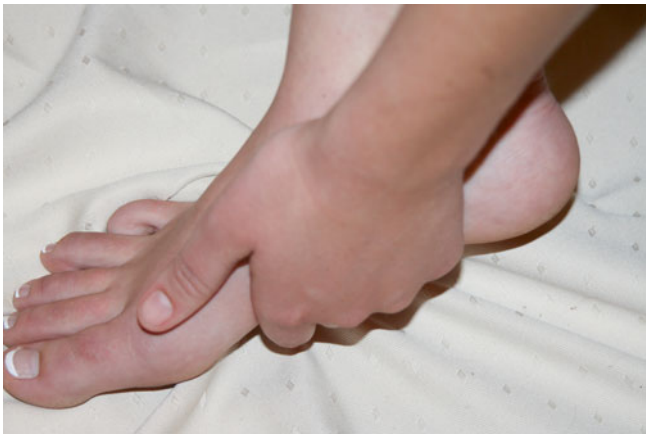

Fig. 75.1 Patient pain complaint from medial plantar nerve entrapment (Image courtesy of Andrea Trescot, MD)

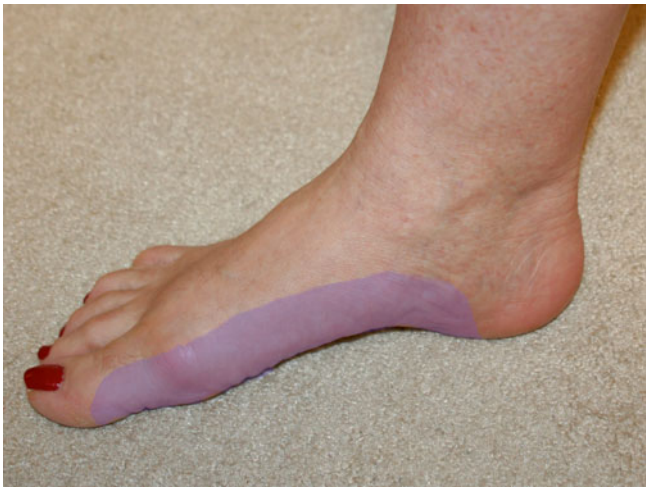

Fig. 75.2 Pattern of pain from medial plantar nerve entrapment (Image courtesy of Andrea Trescot, MD)

flexor hallucis longus (FHL) tendon for Achilles tendon repair [15].

MPN entrapment can coexist with compression of the tibial nerve at a variety of levels (*double crush syndrome*; see Chap. 1) [22] or with simultaneous entrapment of other PTN branches, most commonly the *medial calcaneal nerve* (MCN) [21, 23]. Thus, the clinical picture may be multifaceted. Evaluation of MPN dysfunction can be particularly challenging in the setting of coexisting small fiber neuropathy as seen in diabetes mellitus and other conditions.

Anatomy (Table 75.2)

The *sciatic nerve* travels down the leg and splits into the peroneal and tibial nerves near the popliteal fossa. The tibial nerve at the ankle is often called the *posterior tibial nerve* (PTN, see Chap. 73). It enters the proximal *tarsal tunnel* (deep to the *flexor retinaculum*) at the medial ankle where it divides into the *lateral plantar nerve* (LPN, see Chap. 74), the MPN, and the *medial calcaneal nerve* (MCN, see Chap. 77) (Fig. 75.3). Interestingly, a study of 50 cadaveric legs by Brzezinski and Meyn [31] found the bifurcation of the PTN to be very variable, ranging from 9.6 cm proximal to 1.6 cm distal to the ankle. In the distal tarsal tunnel, deep to the *abductor hallucis* (AbH) muscle, a connective tissue septum (known as the *medial septum*) [29] lies between the medial calcaneus (the tunnel floor) and the deep fascia of the AbH (the tunnel roof), to create separate and independent medial and lateral plantar tunnels. These tunnels allow the tendons, vessels, and nerves to continue their transition further into the foot (Fig. 75.4) [32–34]. Recent anatomic investigations have expanded previous understanding of the myofascial compartments of the foot, as a basis for surgical decompression of compartment syndrome of the foot [30] and to provide additional understanding of the anatomy of the MPN and LPN [29]. A change in girth of any structure that crosses these confined areas can cause a compressive neuropathy.

After it leaves the PTN, the MPN runs anterior to the LPN through an opening in the deep fascia of the AbH (Fig. 75.5) into the *medial plantar tunnel*, also known as the *abductor tunnel* [35]. The MPN remains deep or close to the AbH until it divides into its terminal branches in about half of the population. In the remainder, it moves more lateral to a small space under the *flexor digitorum brevis* (FDB), between the AbH and the crossing of the FHL and *flexor digitorum longus* (FDL) tendons, an area under the junction of the *talus* and *navicular* bones known as the *knot of Henry* or the *chiasma tendineum plantare* [15] (Figs. 75.6, 75.7, and 75.8) [26]. The variable position of the MPN in relation to the AbH is important when considering placement of needles for injection.

The terminal branches of the MPN are the *medial plantar cutaneous nerve of the hallux* and the three medial *common digital nerves*, which further divide into the *proper digital nerves* (Fig. 75.9, see Chap. 70) [36]. The MPN supplies the FDB, AbH, *flexor hallucis brevis* (FHB), and the first *lumbrical* muscles; these plantar muscles flex the toes, especially at the MTP joint of the great toe [2]. It also carries sensation from the medial two thirds of the plantar surface of the foot (including the plantar sides of the first to third toes) and the medial half of the fourth toe as the *proper digital nerves*. Sensation from the lateral half of the fourth toe as well as the fifth toe comes from the proper digital nerves of the LPN (Fig. 75.10) [2].

There is often (86 %, 19 of 22 specimens) a connecting branch between the deep branch of the LPN and the MPN, in the space between the FHB muscle and FHL tendon that is subject to friction during walking or running [14]. Other superficial connections have also been identified (Fig. 75.9) [27, 28, 37].

Table 75.2 Medial plantar nerve anatomy

Origin	L4–S4 ventral rami form the sciatic nerve
General route	The sciatic nerve divides in the distal thigh into the tibial (Chap. 73) and peroneal (fibular, see Chap. 67) nerves, and the tibial nerve (TN) continues down through the deep posterior compartment of the leg
	The TN divides within or occasionally near the tarsal tunnel behind and inferior to medial malleolus into the lateral plantar nerve (LPN), medial plantar nerve (MPN), and medial calcaneal nerve (MCN)
	The MPN travels anterior to the LPN under the abductor hallucis (AbH) muscle and the junction of the talus and the navicular bones
	It becomes plantar between the AbH and the *flexor digitorum brevis* (FDB), gives off the *medial plantar cutaneous nerve of the hallux* (to the medial great toe), and ends in three *common digital nerves*, each of which divides into two *proper digital nerves*
Sensory distribution	The fat pad over the first metatarsal head [5], the tarsal/metatarsal joints, the skin of the medial sole, and the plantar surface and sides of the first to third toes and the medial fourth toe
Motor innervation	*Abductor hallucis* (*AbH*): from the distal flexor retinaculum, medial calcaneal tubercle, and the plantar aponeurosis to the base of the great toe; it flexes and abducts the great toe and may also have contributions from the inferior calcaneal nerve (ICN) [24]
	Flexor hallucis brevis (*FHB*): from the two heads of the muscle to the sesamoid bones at the base of the proximal phalanx of the great toe
	Flexor digitorum brevis (*FDB*, *also innervated by the ICN*): from the anterior portion of the calcaneal tuberosity and the plantar aponeurosis to the sides of the second to fifth toes; flexes second to fifth toes and supports the longitudinal arch
	First lumbrical muscle: from the medial border of the FDL tendon to the dorsal aponeurosis of the second toe; moves the toes closer together
Anatomic variability	*Branches of the MPN*: MCN can arise from the MPN [24, 25]
	Relationship to the AbH: MPN ran under the AbH in the sole for its entire course in 27 % (32/120) or emerged from its lateral edge and stayed close to the AbH in 24 % (29/120) (61/120 = 51 %); in the other 49 % (59/120), it went further lateral to the space under the FDM to the crossover of the FHL and FDL tendons [26]
	The *presence of a connecting branch* between the deep branch of the LPN and the MPN that penetrates the oblique head of the ADH in 86 % (19 of 22 specimens) [14]
	The presence of more lateral and superficial connections between the LPN and MPN in 28 % (14 of 50 specimens) [27] to 100 % (20 of 20) [28]
	Relationship to the medial plantar septum: remains medial in 84 % (16/19) but crosses it in the remainder [29]
Other relevant structures	*Knot of Henry* (*also called the chiasma tendineum plantare*): where the FHL tendon crosses deep to the FDL tendon [15, 16]; this area is plantar to the talonavicular joint
	Navicular tuberosity (*tubercle*): the medial portion of the tuberosity is easily palpated about 2.5 cm anteroinferior to the tip of the medial malleolus, at the superior edge of the AbH, just above the border between the skin of the foot and that of the sole; the plantar aspect of the tuberosity, the main attachment site of the tibialis posterior tendon, is deeper
	FHL tendon: MPN runs along its plantar surface
	Medial plantar septum: runs between the plantar fascial aponeurosis and the calcaneus, navicular and medial cuneiform [29, 30]

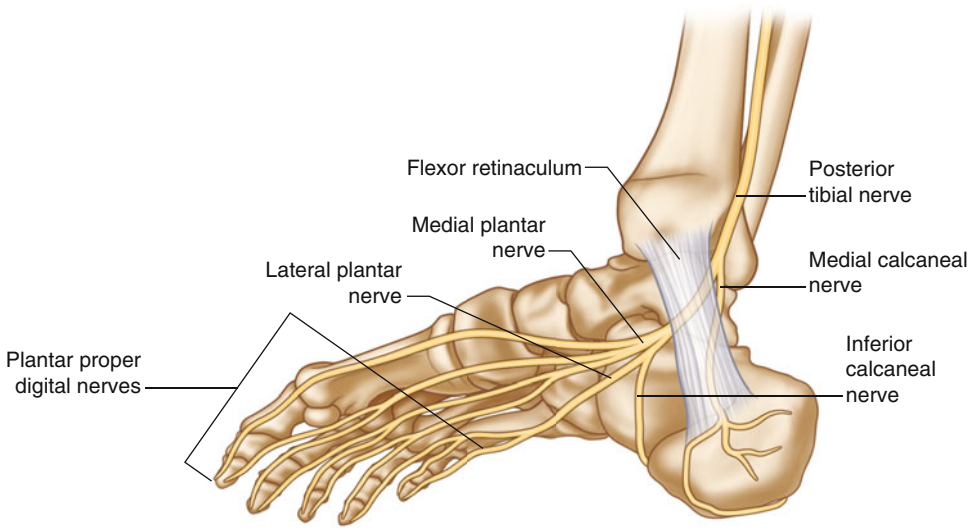

Fig. 75.3 Anatomy of the branches of the posterior tibial nerve. Note the proximity of the medial plantar nerve to the navicular tuberosity (Image courtesy of Springer)

Entrapment

Proximally, the MPN can be entrapped close to its origin at the proximal tarsal tunnel, deep to the flexor retinaculum (Chap. 73). It may also be part of a tarsal tunnel syndrome that includes other branches of the PTN [21].

Further along its course, the MPN can be entrapped at the distal tarsal tunnel as it passes through the deep fascia of the AbH to enter the medial plantar tunnel (Fig. 75.5). MPN entrapment at this level is analogous to entrapment of the median nerve in the *carpal tunnel* (Chap. 37) [33, 34]. Abnormal foot structure such as a high arch (*pes cavus*) or excessive pronation can constrict the openings in the AbH fascia and stretch the MPN against the tunnel [2].

There is also a fascial sling at the *knot of Henry* that can trap the nerve beneath the talus and the plantar prominence of the *navicular tuberosity* (Figs. 75.6 and 75.7), a condition

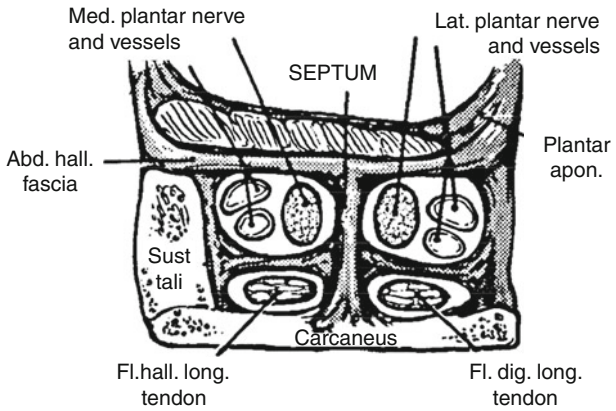

Fig 75.4 Cross section through the region immediately distal to the proximal tarsal tunnel. The medial and lateral plantar tunnels are separated by a septum (also known as the medial septum) between the tunnel roof, the deep abductor hallucis fascia, and the tunnel floor, the calcaneus. *Apon* aponeurosis, *dig* digital, *fl* flexor, *hall* hallucis, *lat* lateral, *long* longus, *med* medial, *sust* sustentaculum (From Dellon [34]. Reprinted with permission from Elsevier Limited)

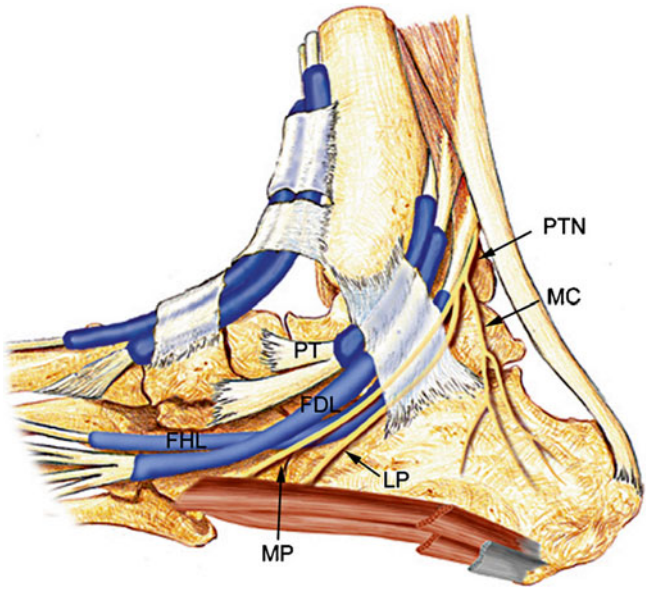

Fig. 75.6 Anatomy of the medial foot. *PTN* posterior tibial nerve, *MC* medial calcaneal nerve, *LP* lateral plantar nerve, *MP* medial plantar nerve, *PT* tibialis posterior tendon, *FDL* flexor digitorum longus tendon, *FHL* flexor hallucis longus tendon. Note the parallel courses of the MPN and the FHL tendon, as well as the knot of Henry where the FDL and FHL tendons cross (From Beltran et al. [36]. Reprinted with permission from Thieme

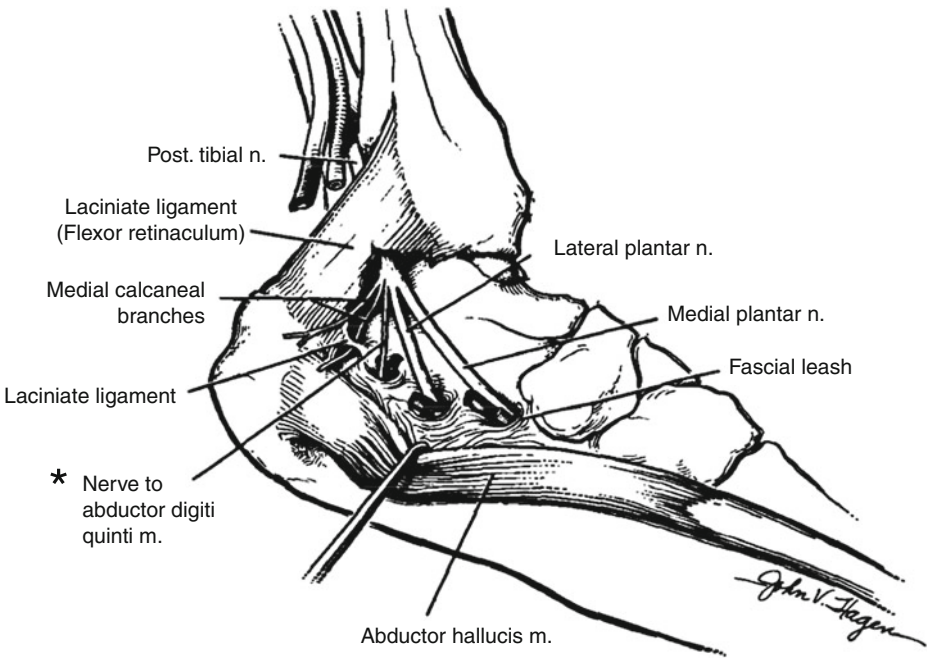

Fig. 75.5 Anatomy of the branches of the posterior tibial nerve. Note the lateral and medial plantar nerves as they cross the thick deep fascia of the abductor hallucis muscle. Note also the nerve to the *abductor digiti quinti muscle* (*inferior calcaneal nerve*) as it migrates under the deep fascia of the abductor hallucis and the medial calcaneal branches (nerves), while they cross the flexor retinaculum (*lancinate ligament*) to become more superficial (From Baxter [62]. Reprinted with permission from SAGE Publications)

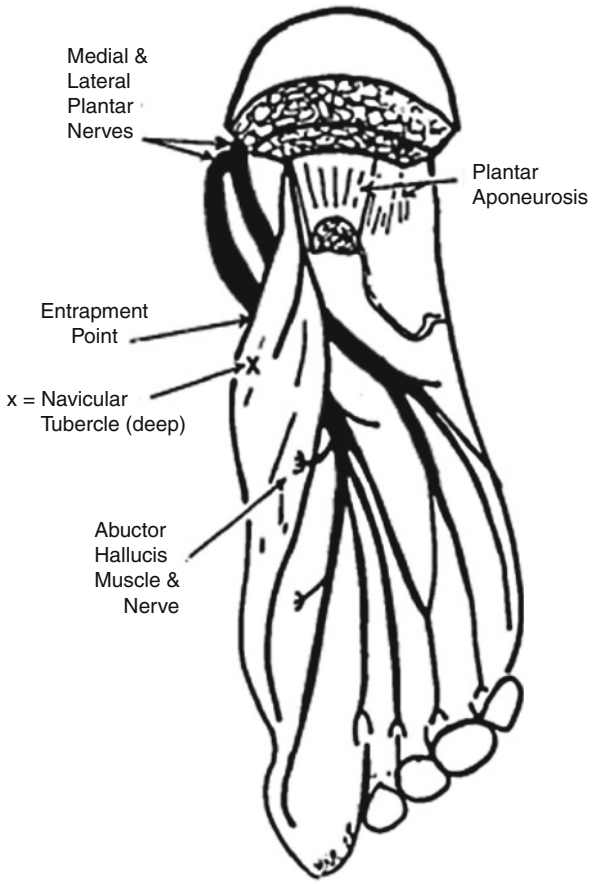

Fig. 75.7 The structures of the plantar aspect of the right foot. The plantar entrapment point of the medial plantar nerve is just posterior to the plantar navicular tubercle (tuberosity) (From Rask [3]. Reprinted with permission from Wolters Kluwer Health)

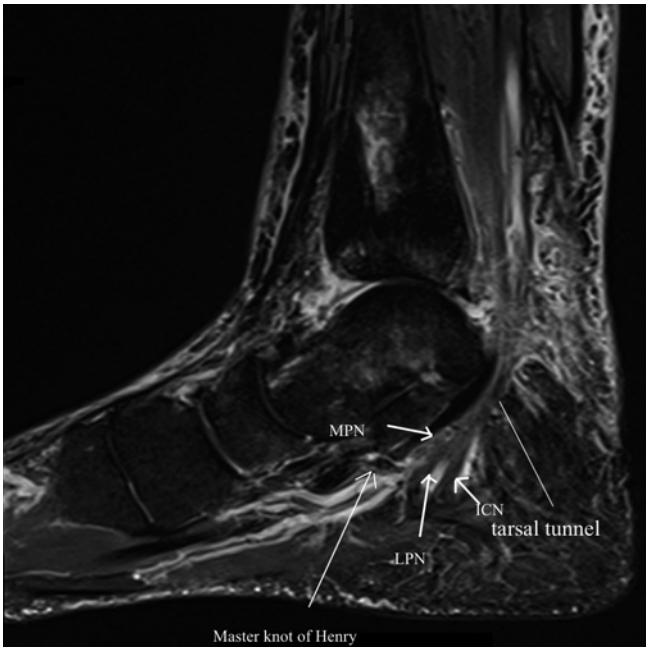

Fig. 75.8 STIR sagittal MRI of the medial malleolus showing the proximal and distal tarsal tunnels and the knot of Henry. *MPN* medial plantar nerve, *LPN* lateral plantar nerve, *ICN* inferior plantar nerve (Image courtesy of Andrea Trescot, MD)

known as "jogger's foot" [3, 4, 7]. Eversion of the foot, collapse of the arch, and repetitive motion of the *talonavicular joint* above the nerve can injure the MPN at this location [3].

In the occasional situation where the MPN crosses the medial plantar septum (16 %, 3 of 19 specimens), it could theoretically be entrapped there [29], although this has not been reported.

Pronation of the ankle with or without plantar flexion (which simulates walking on a pronated foot) significantly elevates the pressure in the medial and lateral plantar tunnels as well as in the tarsal tunnel in cadavers [38] and surgical patients [39].

Physical Examination

Physical examination begins with the patient standing. Specific attention should be given to observation of the patient's intrinsic foot structure and degree of foot pronation. Ask them to demonstrate the particular activity that increases the pain [40] and to stand on the ball of the foot to see if this replicates the pain [9]. The patient is then placed prone with the foot in the *subtalar* neutral position, so that information regarding forefoot position in relation to rear-foot position can be evaluated for clues as to why the patient pronates. Since some patients with MPN entrapment may not have obvious foot pronation, evaluation for a *cavovarus* foot may also be helpful. Gait analysis is likely to show foot supination to avoid pressure on the arch [16].

Palpate the entire tibial nerve from the popliteal fossa to the medial heel and along each of the major branches, looking for masses and areas of tenderness. With MPN entrapment, tenderness is often maximal over the medial plantar arch from near the superior edge of the AbH to the medial and plantar portions of the navicular tuberosity (Figs. 75.7 and 75.11) [3, 4, 9, 21].

Tinel's test performed just behind the plantar aspect of the navicular tuberosity is often positive with MPN entrapment [9, 21]. Compressing the nerve with one finger and tapping on that finger with the other hand may be particularly effective [4]. This test is usually negative in entrapment of the *inferior calcaneal nerve* (ICN, see Chap. 76) [41, 42].

Because the MPN has a relatively consistent cutaneous innervation (Fig. 75.10), sensory testing with a Semmes-Weinstein monofilament (Chap. 2) for light touch and pain is an important portion of the clinical examination, although decreased sensation may only be present after exercise [9].

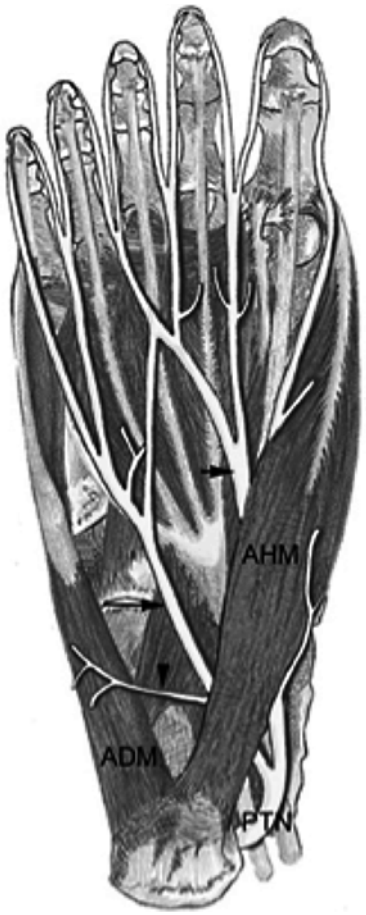

Fig. 75.9 Anatomy of the plantar nerves. *Short arrow* medial plantar nerve, *long arrow* lateral plantar nerve, *arrowhead* inferior calcaneal nerve, *PTN* posterior tibial nerve, *AHM* abductor hallucis muscle, *ADM* abductor digiti minimi. Note the relationship of the plantar nerves to the abductor hallucis and the connecting branch between the lateral and medial plantar nerves (From Beltran et al. [36]. Reprinted with permission from Thieme)

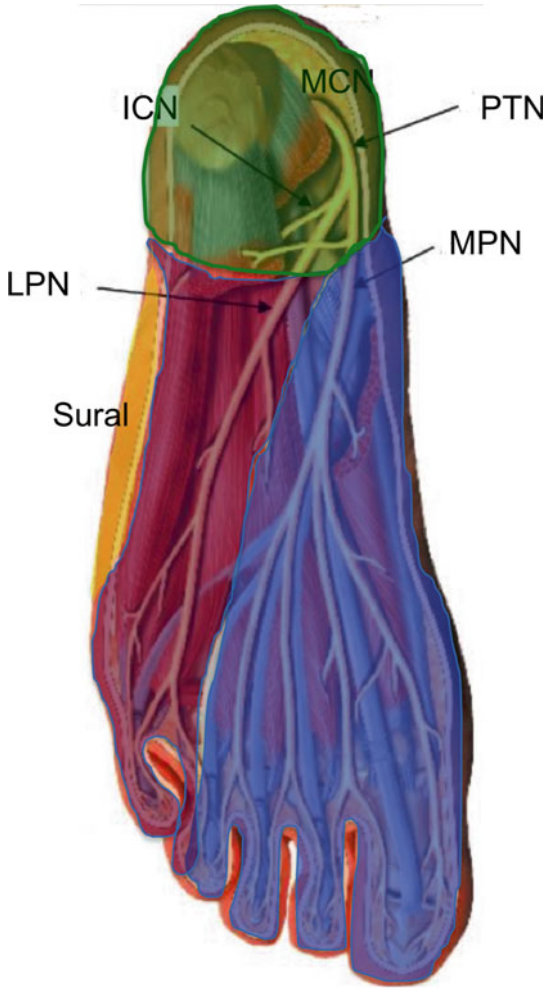

Fig. 75.10 Sensory map of the plantar nerves. *MCN* medical calcaneal nerve (*green*), *PTN* posterior tibial nerve, *MPN* medial plantar nerve (*blue*), *ICN* inferior calcaneal nerve (note the lack of cutaneous territory), *LPN* lateral plantar nerve (*red*), *sural* sural nerve (*orange*) (Image courtesy of Michael Brown, MD)

Passive hyperextension of the toes and toe flexion against resistance should not increase the pain of MPN dysfunction, although pain would be increased by these movements if the patient has tenosynovitis [4]. If the AbH is compressing a nerve or part of one, the patient's symptoms may be reproduced by tensing it [26].

Placing the foot in flexion and then inversion (the plantar flexion-inversion test) (Fig. 75.12a) is a provocative maneuver that increases pressure within the tarsal tunnel [43]. Subsequent investigators have described the *dorsiflexion-eversion test* [39], a maneuver that simulates pronation, a position that has been shown to increase pressure in the medial and lateral plantar tunnels as well as in the tarsal tunnel [38]. The authors prefer the latter test, with pressure over the proximal tarsal tunnel (Fig. 75.12b).

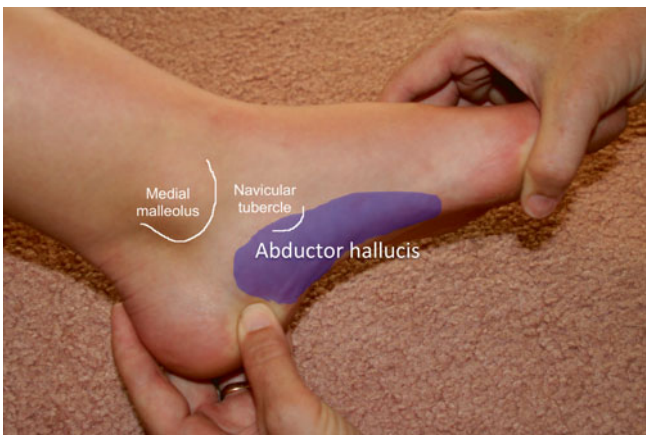

Fig. 75.11 Physical examination of the foot, showing the location of the abductor hallucis muscle (Image courtesy of Andrea Trescot, MD)

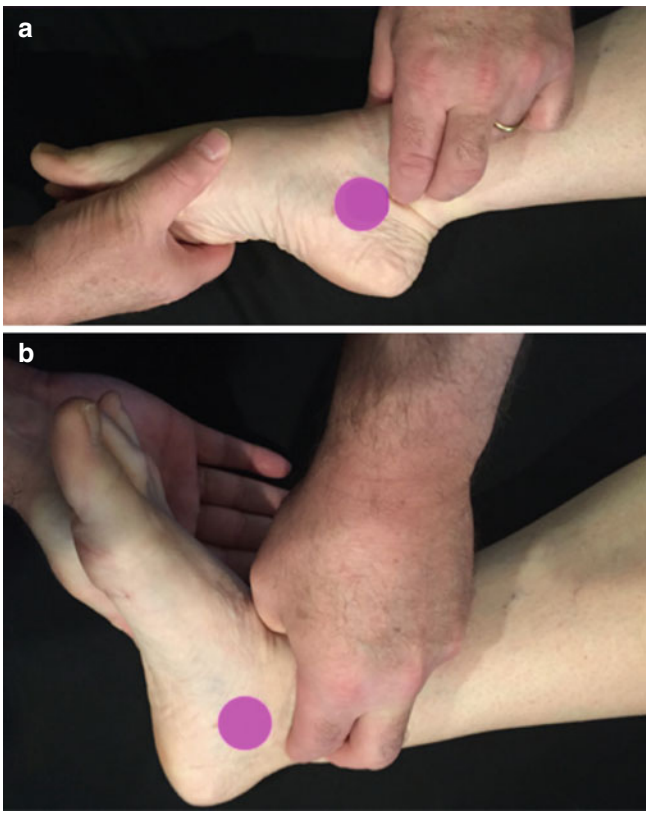

Fig. 75.12 (**a**) Plantar flexion-inversion with palpation; (**b**) dorsiflexion and foot eversion with pressure applied over the proximal tarsal tunnel. The *pink dot* is centered on the malleolar-calcaneal axis (Image courtesy of Michael Brown, MD)

Differential Diagnosis (Table 75.3)

There are many causes of medial plantar arch and foot pain. If the pain is near the anterior calcaneal tubercle, it may be confused with ICN entrapment (see Chap. 76). However, a patient with MPN dysfunction can have a deficit of cutaneous sensation in the first, second, and third toes [21], while cutaneous sensation for those with ICN entrapment will be like that of the contralateral foot. Patients with MPN dysfunction sometimes lose the ability to abduct the great toe but retain the ability to abduct the little toe, which may be absent or reduced in ICN entrapment.

MPN entrapment can mimic *plantar fasciitis*, although patients with MPN dysfunction usually will not experience the "first-step" foot pain characteristic of that disorder and will be tender more distally. It can also be confused with intermetatarsal (Morton's) "neuroma" (Chap. 70) [11].

Diagnostic Tests (Table 75.4)

Although most of the diagnosis is made by history and physical examination, variable anatomy and the possible simultaneous involvement of several branches of the PTN often make imaging and other tests useful [22, 44–46].

Table 75.3 Differential diagnosis of medial arch and heel pain

Condition	Potential distinguishing features
Inferior calcaneal nerve entrapment	Heel pain, no cutaneous distribution
Tarsal tunnel syndrome	Presence of additional symptoms attributable to involvement of other tibial nerve branches
	Tenderness over the superior tarsal tunnel
	Tinel's sign elicited over the medial ankle
Plantar fasciitis	"First-step" pain (*post-static dyskinesia*)
Flexor digitorum longus or flexor hallucis longus tenosynovitis [4]	Passive hyperextension of the toes or toe flexion against resistance is painful
Neuroma	Visible on US or MRI
Medical calcaneal nerve entrapment	Pain is more in the heel than the arch, with tenderness and Tinel's sign present more posteriorly
S1 radiculopathy [32]	Proximal pain, loss of Achilles reflex

Table 75.4 Diagnostic tests for medial plantar nerve entrapment

	Potential distinguishing features
Physical exam	Tenderness over the navicular tuberosity
	Sensory changes over the medial sole and toes
	Tinel's test
Provocative tests	Foot dorsiflexion-eversion with pressure over the tarsal tunnel
Diagnostic injection	Landmark or US guided
Ultrasound	Locate a space-occupying mass
MRI	Magnetic resonance neurography (MRN) can show anatomy and the degree and location of nerve injury, as well as demonstrate a mass [44, 45]
Arteriography	Not useful
X-ray	To evaluate bony abnormalities and alignment
Electrodiagnostic studies	The pressure-sensitive sensory device (PSSD) is painless and quick; nerve conduction velocity and needle EMG may be useful in complex situations

Weight-bearing plain radiographs can help assess foot alignment and exclude fractures or joint degeneration [47].

Ultrasonography (US) and MRI can show tenosynovitis, bursitis, masses, and nerve injuries. US is widely available, inexpensive, and quick; however, image quality is operator dependent and may be limited in the presence of dense scarring [45].

With MPN injury, MRI may also reveal edema-like changes in signal intensity or fatty infiltration atrophy in the AbH or FDB muscles indicative of denervation [44], while more distal damage may affect the FHB and first lumbrical muscles [46]. High-resolution magnetic resonance neurography (MRN) can show the anatomy of branches as small as 3 mm as well as the degree and location of nerve injury [45].

This technique has been shown to correlate well with intraoperative findings [44].

Axial plane images are thought to be most useful for evaluating nerve pathologies of the tarsal tunnel and plantar foot, while T1-weighted views were best for nerve anatomy and evaluation of branching abnormalities, and T2-weighted images were best for nerve inflammation, neuromas, or muscle edema (Fig. 75.13) [44].

The pressure-specified sensory device (PSSD) is an objective, non-painful method to measure nerve function; the pressure required for a person to distinguish one from two static points is the most sensitive gauge of nerve dysfunction from chronic compression [48]. Data for the MPN (big toe) and MCN (heel) has been recently reported in normal subjects and patients diagnosed with tarsal tunnel syndrome [49], which can be a useful adjunct to diagnosis and treatment [23, 50].

Electrodiagnostic studies of the tibial nerve and its branches may help to differentiate tarsal tunnel syndrome from MPN entrapment [22, 35], but a systematic review has revealed that the level of evidence (as of 2005) is weak [51]. Some authors think that those tests are not reliable, particularly because many patients without TTS also have abnormal results [34] or because symptoms that occur during strenuous activity may not be seen on measurements taken at rest [7]. In complex situations, however, nerve conduction studies and needle EMG testing of the AbH, FHB, FDB, and first lumbrical muscles may be helpful [22], particularly to rule out a radiculopathy [52].

Sensory nerve action potentials (SNAPs) of the MPN are more sensitive than standard nerve testing in diagnosing peripheral neuropathy [53–55].

Identification and Treatment of Contributing Factors

Many patients with MPN dysfunction have intrinsic foot abnormalities that increase foot pronation. This condition may improve with supportive shoe wear, appropriately

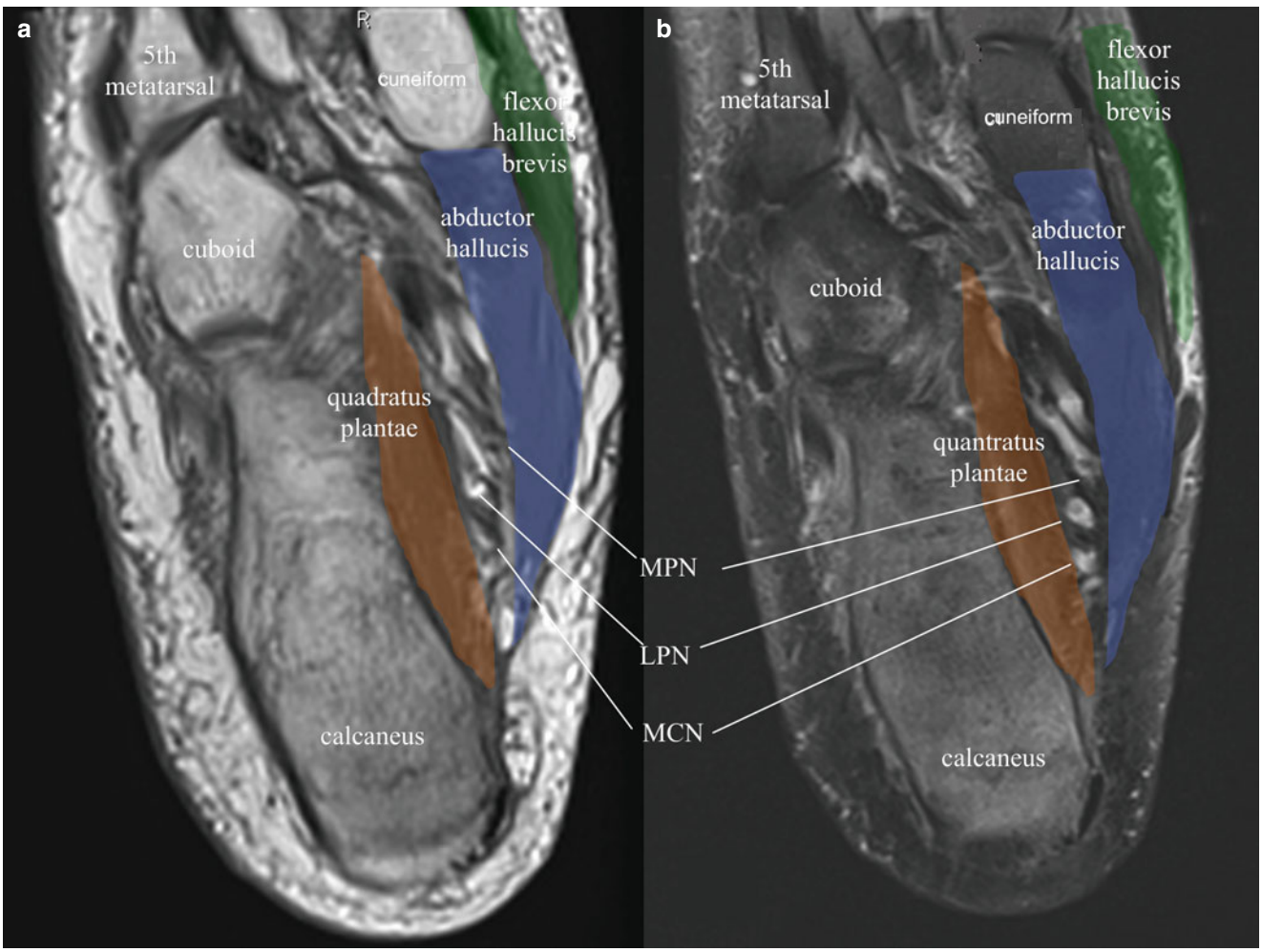

Fig. 75.13 Axial MRI images; (**a**) T1-weighted image; (**b**) T2-weighted image; *MPN* medial plantar nerve, *LPN* lateral planter nerve, *MCN* medial calcaneal nerve (Image courtesy of Andrea Trescot, MD)

designed orthotics, changed running posture of the foot, and NSAIDs [3]. However, one has to be careful because an overcorrected arch or aggressive arch support can make the problem worse. Many of these patients have a mobile midfoot with ligamentous laxity and can collapse over the arch, worsening the pressure over the MPN, so the focus should be on a more appropriate posting of the varus position of the foot. Proper shoe wear, as well as stretching of plantar fascia and Achilles tendon, will reduce stress on the ligaments and muscles. Weight reduction and physical therapy to strengthen the foot flexor and tibialis posterior muscles are additional important strategies [6].

Harvest of the FHL tendon for surgical repair of the Achilles tendon at the knot of Henry has been associated with MPN damage; a surgical technique specifically designed to protect the MPN from this injury has been described [15].

Injection Techniques

The techniques described below are for MPN injections at the entrance of the medial plantar tunnel. If the entrapment is thought to be at the knot of Henry ("jogger's foot"), the injection should be more distal, just proximal to the area under the navicular bone. If a landmark- or US-guided procedure is planned, consider using a peripheral nerve stimulator to improve the accuracy of low-volume injections. Successful low-volume local anesthetic injections provide diagnostic information and aid in planning for definitive therapeutic procedures (Chap. 7). Sensory testing will confirm that the local anesthetic injection reached the MPN (Fig. 75.10), since a successful block will result in a sensory deficit along the medial two thirds of the plantar aspect of the foot.

Landmark-Guided Injections

The patient is placed in a lateral position with the medial side of the foot facing up. The authors prefer a 27-gauge, 1.5 in. needle for MPN injection. Palpate the area just inferior to the tarsal tunnel, and then slide the thumb downward, intermittently squeezing until you contact the first muscle bundle, the AbH (Fig. 75.11). When the patient spreads their toes, one can feel the AbH move. Place a small skin wheal of local anesthetic just above the muscle, and direct the needle at an oblique angle toward the fourth and fifth toes (Fig. 75.14). The MPN usually runs superior to the AbH until it enters the medial plantar tunnel (Fig. 75.5) and then deep and progressively lateral to it. Slide the needle just over the muscle, and inject 1 cc of therapeutic solution, usually local anesthetic and deposteroid, to address possible entrapment at the AbH fascia. Next, aim the needle slightly deeper, still directing it obliquely toward the fourth and fifth toes, and inject another 1 cc to cover possible entrapment at the knot of Henry. After the injection, test for cutaneous numbness in the MPN distribution to confirm the block.

Fluoroscopy-Guided Injection

In our opinion, fluoroscopy provides little benefit for MPN injections.

Ultrasound-Guided Injections

Out-of-Plane Technique

The *malleolar-calcaneal axis* (MCA) from the center of the medial malleolus to the center of the calcaneus is a useful constant reference point for the center of the tarsal tunnel [56, 57]. Place a linear US transducer obliquely over the MCA, with the notch toward the medial malleolus (Fig. 75.15a), and identify the vein-artery-vein (VAV) relationship in the proximal tarsal tunnel (Fig. 75.15b). Move the transducer caudad so that one end is approximately at the mid-heel, and rotate it (clockwise if you are imaging the left heel and counterclockwise if you are imaging the right heel) (Fig. 75.15c) to identify the MPN, LPN, and MCN (Fig. 75.15d). As the transducer is moved inferiorly, the ICN (Baxter's nerve) will come into view (Fig. 75.16).

Rotate the transducer even more, until you can see two muscles with a septum between them (Fig. 75.17). Confirm the anatomy by having the patient spread their great toe against resistance and watch the superior muscle, the AbH, move. If the AbH is causing the entrapment and the MPN is visible, abduction of the great toe may darken (increase the hypogenicity) its signal [26]. Then, have the patient flex their toes to show contraction of the inferior muscle group, the FDB, as well as a tendon that flexes independently, the FHL. This transducer position shows these muscles in the longitudinal plane (Fig. 75.18).

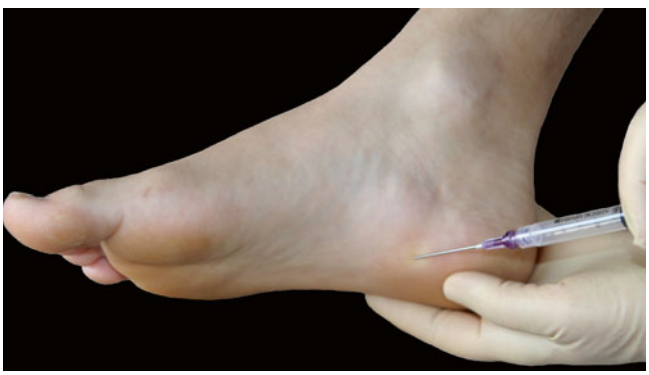

Fig. 75.14 Landmark-guided injection of the medial plantar nerve (Image courtesy of Andrea Trescot, MD)

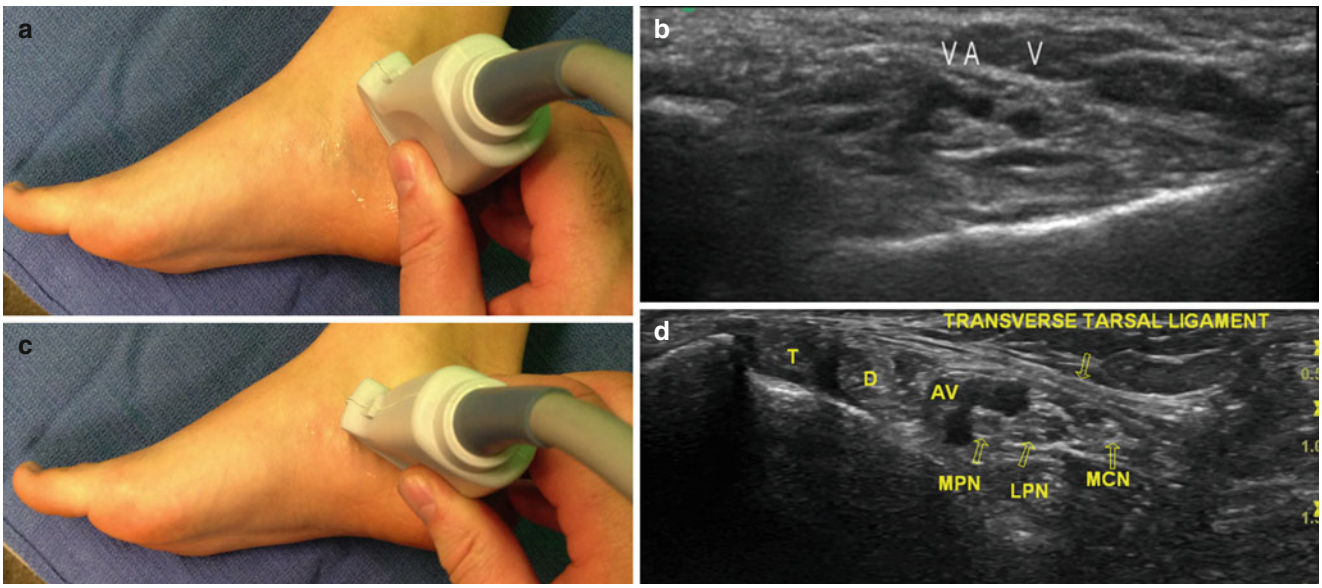

Fig. 75.15 Ultrasound localization of the medial plantar nerve. (**a**) Initial US probe placement; (**b**) US image showing the vein-artery-vein configuration of the posterior tibial artery and veins; (**c**) rotated probe position; (**d**) ultrasound image of the probe at position (**c**); *MPN* medial plantar nerve, *LPN* lateral plantar nerve, *MCN* medial calcaneal nerve, *AV* artery and vein, *D* flexor digitorum longus tendon, *T* tibialis posterior tendon; transverse tarsal ligament = flexor retinaculum (Image courtesy of Michael Brown, MD)

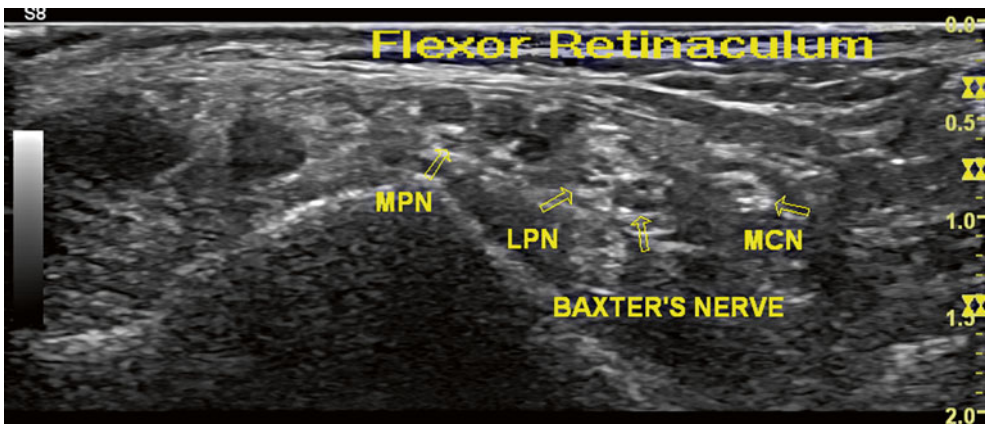

Fig. 75.16 Ultrasound image of the medial plantar nerve. *MPN* medial plantar nerve, *LPN* lateral plantar nerve, *MCN* medial calcaneal nerve; Baxter's nerve = inferior calcaneal nerve (Image courtesy of Michael Brown, MD)

When using an out-of-plane approach, inject 0.1–0.2 cc aliquots of saline to track the position of the needle tip, keeping it in view at all times. Insert the needle directly over the AbH, where the tissue planes can be distended by hydrodissection if needed for better visualization. Use normal saline, not local anesthetic, for this purpose so as not to confound a diagnostic block. Advance the needle in the fascial plane over the AbH, and direct it toward the fourth and fifth toes; at a depth of approximately 1 cm, inject 1 cc of local anesthetic and steroid, watching the spread of the medication. Then, advance another 1–2 cm in the same direction, and inject another 1 cc of therapeutic solution. This will provide local anesthetic to both places at which the MPN may be entrapped in the foot. After the injection, test for cutaneous numbness in the MPN distribution to confirm the block.

In-Plane Technique

Place the transducer over the AbH and FHB, and move it until the anterior edge is just in front of the *navicular tubercle* (Fig. 75.19a). Confirm its position by functionally testing the muscle: abduction of the great toe against resistance should contract the AbH. Slide the transducer slightly toward the sole to allow room for the needle over the AbH. Use a 27- or 25-gauge needle, and direct it across the foot toward the fourth and fifth toes (Fig. 75.19b). Once the needle is past the AbH fascia, inject 0.5–1 cc of therapeutic solution, watching the spread of medication. This will provide a local anesthetic block of the more superficial portion of the MPN. Next, insert the needle 2–3 cm deeper in the same direction, and inject another 1–1.5 cc of solution to block the

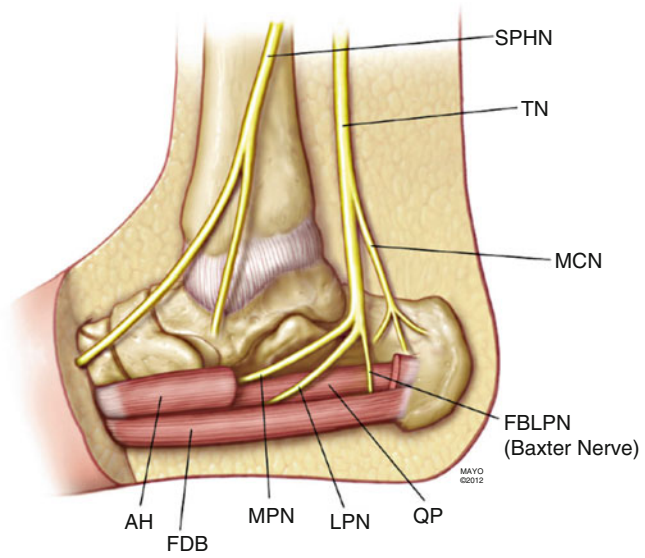

Fig. 75.17 Drawing of the medial ankle showing the branching pattern of the tibial nerve in the proximal and distal tarsal tunnels. The AH has been partially removed to show the vertical course of the ICN and the interval between the AH (removed) and QP muscles. *SPHN* saphenous nerve, *TN* tibial nerve, *MCN* medial calcaneal nerve, *FBLPN* first branch of the lateral calcaneal nerve = inferior calcaneal nerve, *QP* quadratus plantae, *LPN* lateral plantar nerve, *MPN* medial plantar nerve, *FDB* flexor digitorum brevis, *AH* abductor hallucis. Note the vertical course of the ICN perpendicular to the parallel paths of the AH and the FDB (From Presley et al. [61]. Reprinted with permission from the American Institute of Ultrasound in Medicine)

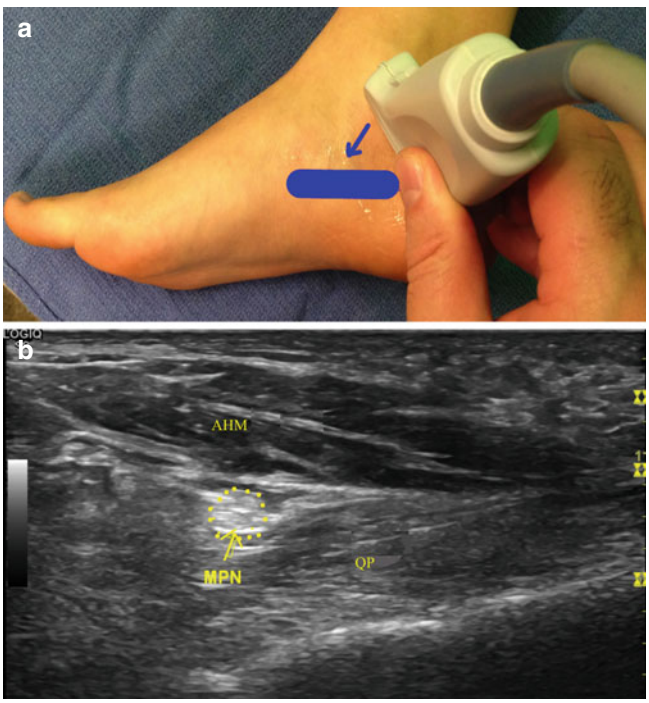

Fig. 75.18 In-plane ultrasound evaluation of the medial plantar nerve. (**a**) Surface landmarks; (**b**) ultrasound evaluation of the medial arch of the foot; *MPN* medial plantar nerve, *AHM* abductor hallucis muscle, *QP* quadratus plantae (Image courtesy of Michael Brown, MD)

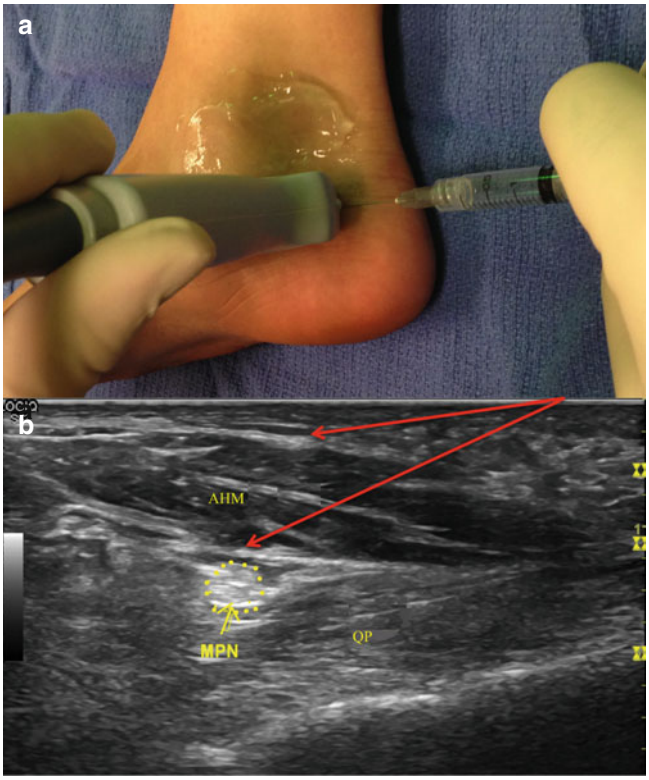

Fig. 75.19 In-plane injection of the medial plantar nerve. (**a**) Surface landmarks; (**b**) ultrasound evaluation of the medial arch of the foot with simulated needle (*red arrows*) showing injection above and below the abductor hallucis muscle; *AHM* abductor hallucis muscle, *QP* quadratus plantae (Image courtesy of Michael Brown, MD)

nerve at the knot of Henry. After the injection, test for cutaneous numbness in the MPN distribution to confirm the block.

Neurolytic Procedures

Because of the significant motor function of the MPN, neurolytic procedures are not usually employed.

Neurostimulation

Peripheral neurostimulation is not usually considered a treatment option for foot pain, and spinal cord stimulation may be a challenge because of the difficulties of stimulating the conus (the traditional stimulation site for the foot). However, Mobbs and Lazarro [58] described the successful use of a peripheral nerve stimulator to treat a medial plantar neuralgia, which had triggered complex regional pain syndrome (CRPS) following a metatarsal fracture (see Chap. 1).

Surgery

Injuries of the PTN and its branches may coexist in the proximal and distal tarsal tunnels, and specific locations may be difficult to pinpoint. Surgical decompression of the MPN may be indicated after a thorough trial of nonoperative therapies. The locations of points of maximum tenderness and Tinel's sign should be documented preoperatively [4, 34]. The incision should begin proximal to the flexor retinaculum and medial plantar septum and should be long enough to allow release of all constricting structures, including the deep fascia of the AbH that surrounds each distal branch. Minimal manipulation of the nerve and its accompanying blood vessels to avoid nerve damage is important for a successful outcome [4].

Surgical release of MPN entrapment is performed in conjunction with release of the PTN and its other branches [34, 59]. Patients with coexisting radiculopathy or generalized neuropathy are more likely to have a poor outcome [60].

Complications

As with any procedure, there are risks of infection, hematoma, vascular puncture, or nerve injury. The most common associated problem is incomplete nerve release or ablation leading to increased pain. In this circumstance, repeating the procedure may lead to resolution of symptoms. The skin is very thin at this site, so injections or surgery can result in skin injury or atrophy.

Summary

Patients with pain at the medial arch of the foot are not uncommon in clinical practice. The MPN entrapment is a possible cause of this kind of foot pain and may be confused or coexist with entrapments of other branches of the tibial nerve. A careful history and physical examination, as well as a diagnostic injection, can help the clinician recognize and treat MPN entrapments.

References

1. Thomas MJ, Roddy E, Zhang W, Menz HB, Hannan MT, Peat GM. The population prevalence of foot and ankle pain in middle and old age: a systematic review. Pain. 2011;152(12):2870–80.
2. Kopell HP, Thompson WA. Peripheral Entrapment Neuropathies. Baltimore: Williams and Wilkins; 1976.
3. Rask MR. Medial plantar neurapraxia (jogger's foot): report of 3 cases. Clin Orthop Relat Res. 1978;134:193–5.
4. Schon LC. Nerve entrapment, neuropathy, and nerve dysfunction in athletes. Orthop Clin North Am. 1994;25(1):47–59.
5. Unlü RE, Orbay H, Kerem M, Esmer AF, Tüccar E, Sensöz O. Innervation of three weight-bearing areas of the foot: an anatomic study and clinical implications. J Plast Reconstr Aesthet Surg. 2008;61(5):557–61.
6. McKeon PO, Hertel J, Bramble D, Davis I. The foot core system: a new paradigm for understanding intrinsic foot muscle function. Br J Sports Med. 2015;49(5):290.
7. Murphy PC, Baxter DE. Nerve entrapment of the foot and ankle in runners. Clin Sports Med. 1985;4(4):753–63.
8. Oh SJ, Kim HS, Ahmad BK. The near-nerve sensory nerve conduction in tarsal tunnel syndrome. J Neurol Neurosurg Psychiatry. 1985;48(10):999–1003.
9. Schon LC, Baxter DE. Neuropathies of the foot and ankle in athletes. Clin Sports Med. 1990;9(2):489–509.
10. Spinner RJ, Scheithauer BW, Amrami KK. Medial plantar nerve compression by a tibial artery schwannoma. Case report. J Neurosurg. 2007;106(5):921–3.
11. D'Orazi V, Venditto T, Panunzi A, Anichini S, Manzini G, Tallarico A, et al. Misdiagnosis of plexiform neurofibroma of the medial plantar nerve: case report. Foot (Edinb). 2014;24(3):143–5.
12. Odom RD, Overbeek TD, Murdoch DP, Hosch JC. Neurilemoma of the medial plantar nerve: a case report and literature review. J Foot Ankle Surg. 2001;40(2):105–9.
13. Kwon JH, Yoon JR, Kim TS, Kim HJ. Peripheral nerve sheath tumor of the medial plantar nerve without tarsal tunnel syndrome: a case report. J Foot Ankle Surg. 2009;48(4):477–82.
14. Arakawa T, Sekiya S, Terashima T, Miki A. Pseudoganglion on the connecting branch between the deep branch of the lateral plantar nerve and medial plantar nerve. Clin Anat. 2011;24(5):646–51.
15. Amlang M, Rosenow MC, Friedrich A, Zwipp H, Rammelt S. Direct plantar approach to Henry's knot for flexor hallucis longus transfer. Foot Ankle Int. 2012;33(1):7–13.
16. Herbst SA, Miller SD. Transection of the medial plantar nerve and hallux cock-up deformity after flexor hallucis longus tendon transfer for Achilles tendinitis: case report. Foot Ankle Int. 2006;27(8):639–41.
17. Mulier T, Rummens E, Dereymaeker G. Risk of neurovascular injuries in flexor hallucis longus tendon transfers: an anatomic cadaver study. Foot Ankle Int. 2007;28(8):910–5.
18. Frenette JP, Jackson DW. Lacerations of the flexor hallucis longus in the young athlete. J Bone Joint Surg Am. 1977;59(5):673–6.
19. Johal S, Sawalha S, Pasapula C. Post-traumatic acute hallux valgus: a case report. Foot (Edinb). 2010;20(2–3):87–9.
20. Hiratai M, Sakakibara H, Toibana N. Medial plantar nerve conduction velocities among patients with vibration syndrome due to rock-drill work. Ind Health. 2004;42(1):24–8.
21. Oh SJ, Lee KW. Medial plantar neuropathy. Neurology. 1987;37(8):1408–10.
22. Schon LC, Glennon TP, Baxter DE. Heel pain syndrome: electrodiagnostic support for nerve entrapment. Foot Ankle. 1993;14(3):129–35.
23. Rose JD, Malay DS, Sorrento DL. Neurosensory testing of the medial calcaneal and medial plantar nerves in patients with plantar heel pain. J Foot Ankle Surg. 2003;42(4):173–7.
24. Davis TJ, Schon LC. Branches of the tibial nerve: anatomic variations. Foot Ankle Int. 1995;16(1):21–9.
25. Dellon AL, Kim J, Spaulding CM. Variations in the origin of the medial calcaneal nerve. J Am Podiatr Med Assoc. 2002;92(2):97–101.
26. Ghosh SK, Raheja S, Tuli A. Potential sites of compression of tibial nerve branches in foot: a cadaveric and imaging study. Clin Anat. 2013;26(6):768–79.
27. Govsa F, Bilge O, Ozer MA. Anatomical study of the communicating branches between the medial and lateral plantar nerves. Surg Radiol Anat. 2005;27(5):377–81.

28. Jones JR, Klenerman L. A study of the communicating branch between the medial and lateral plantar nerves. Foot Ankle. 1984; 4(6):313–5.
29. Singh G, Kumar VP. Neuroanatomical basis for the tarsal tunnel syndrome. Foot Ankle Int. 2012;33(6):513–8.
30. Ling ZX, Kumar VP. The myofascial compartments of the foot: a cadaver study. J Bone Joint Surg (Br). 2008;90(8):1114–8.
31. Brzezinski PF, Meyn NP. The level of bifurcation of the tibial nerve into the medial and lateral plantar nerves. J Am Podiatry Assoc. 1977;67(8):553–5.
32. Heimkes B, Posel P, Stotz S, Wolf K. The proximal and distal tarsal tunnel syndromes. An anatomical study. Int Orthop. 1987;11(3): 193–6.
33. Mackinnon SE, Dellon AL. Homologies between the tarsal and carpal tunnels: implications for surgical treatment of the tarsal tunnel syndrome. Contemp Orthop. 1987;14(4):75–8.
34. Dellon AL. The four medial ankle tunnels: a critical review of perceptions of tarsal tunnel syndrome and neuropathy. Neurosurg Clin N Am. 2008;19(4):629–48, vii.
35. Oh SJ. Neuropathies of the foot. Clin Neurophysiol. 2007; 118(5):954–80.
36. Beltran LS, Bencardino J, Ghazikhanian V, Beltran J. Entrapment neuropathies III: lower limb. Semin Musculoskelet Radiol. 2010;14(5):501–11.
37. Levitsky KA, Alman BA, Jevsevar DS, Morehead J. Digital nerves of the foot: anatomic variations and implications regarding the pathogenesis of interdigital neuroma. Foot Ankle. 1993;14(4):208–14.
38. Barker AR, Rosson GD, Dellon AL. Pressure changes in the medial and lateral plantar and tarsal tunnels related to ankle position: a cadaver study. Foot Ankle Int. 2007;28(2):250–4.
39. Rosson GD, Larson AR, Williams EH, Dellon AL. Tibial nerve decompression in patients with tarsal tunnel syndrome: pressures in the tarsal, medial plantar, and lateral plantar tunnels. Plast Reconstr Surg. 2009;124(4):1202–10.
40. Kinoshita M, Okuda R, Morikawa J, Jotoku T, Abe M. The dorsiflexion-eversion test for diagnosis of tarsal tunnel syndrome. J Bone Joint Surg Am. 2001;83-A(12):1835–9.
41. Baxter DE, Pfeffer GB. Treatment of chronic heel pain by surgical release of the first branch of the lateral plantar nerve. Clin Orthop Relat Res. 1992;279:229–36.
42. Fredericson M, Standage S, Chou L, Matheson G. Lateral plantar nerve entrapment in a competitive gymnast. Clin J Sport Med. 2001;11(2):111–4.
43. Trepman E, Kadel NJ, Chisholm K, Razzano L. Effect of foot and ankle position on tarsal tunnel compartment pressure. Foot Ankle Int. 1999;20(11):721–6.
44. Chhabra A, Subhawong TK, Williams EH, Wang KC, Hashemi S, Thawait SK, et al. High-resolution MR neurography: evaluation before repeat tarsal tunnel surgery. AJR Am J Roentgenol. 2011;197(1):175–83.
45. Chalian M, Soldatos T, Faridian-Aragh N, Williams EH, Rosson GD, Eng J, et al. 3T magnetic resonance neurography of tibial nerve pathologies. J Neuroimaging. 2013;23(2):296–310.
46. Donovan A, Rosenberg ZS, Cavalcanti CF. MR imaging of entrapment neuropathies of the lower extremity. Part 2. The knee, leg, ankle, and foot. Radiographics. 2010;30(4):1001–19.
47. Lareau CR, Sawyer GA, Wang JH, DiGiovanni CW. Plantar and medial heel pain: diagnosis and management. J Am Acad Orthop Surg. 2014;22(6):372–80.
48. Dellon AL, Keller KM. Computer-assisted quantitative sensorimotor testing in patients with carpal and cubital tunnel syndromes. Ann Plast Surg. 1997;38(5):493–502.
49. Tassler PL, Dellon AL. Pressure perception in the normal lower extremity and in the tarsal tunnel syndrome. Muscle Nerve. 1996;19(3):285–9.
50. Dellon AL. Deciding when heel pain is of neural origin. J Foot Ankle Surg. 2001;40(5):341–5.
51. Patel AT, Gaines K, Malamut R, Park TA, Toro DR, Holland N, et al. Usefulness of electrodiagnostic techniques in the evaluation of suspected tarsal tunnel syndrome: an evidence-based review. Muscle Nerve. 2005;32(2):236–40.
52. Williams EH, Williams CG, Rosson GD, Dellon LA. Anatomic site for proximal tibial nerve compression: a cadaver study. Ann Plast Surg. 2009;62(3):322–5.
53. Ponsford SN. Sensory conduction in medial and lateral plantar nerves. J Neurol Neurosurg Psychiatry. 1988;51(2):188–91.
54. Squintani G, Zoppini G, Donato F, Pineschi E, Donini D, Stoico V, et al. Antidromic sensory nerve conduction study of the digital branches of the medial plantar nerve: a novel method to detect early diabetic sensory axonal polyneuropathy. Muscle Nerve. 2014; 50(2):193–9.
55. Altun Y, Demirkol A, Tumay Y, Ekmekci K, Unsal I, Koyluoglu AC, et al. The medial plantar and medial peroneal cutaneous nerve conduction studies for diabetic polyneuropathy. Neurol Sci. 2011;32(5):849–54.
56. Dellon AL, Mackinnon SE. Tibial nerve branching in the tarsal tunnel. Arch Neurol. 1984;41(6):645–6.
57. Havel PE, Ebraheim NA, Clark SE, Jackson WT, DiDio L. Tibial nerve branching in the tarsal tunnel. Foot Ankle. 1988;9(3):117–9.
58. Mobbs RJ, Lazarro A. Stimulation of the medial plantar nerve for complex regional pain syndrome. J Clin Neurosci Off J Neurosurg Soc Australas. 2010;17(11):1421–2.
59. Gondring WH, Shields B, Wenger S. An outcomes analysis of surgical treatment of tarsal tunnel syndrome. Foot Ankle Int. 2003;24(7):545–50.
60. Barker AR, Rosson GD, Dellon AL. Outcome of neurolysis for failed tarsal tunnel surgery. J Reconstr Microsurg. 2008;24(2): 111–8.
61. Presley JC, Maida E, Pawlina W, Murthy N, Ryssman DB, Smith J. Sonographic visualization of the first branch of the lateral plantar nerve (Baxter nerve): technique and validation using perineural injections in a cadaveric model. J Ultrasound Med. 2013;32(9): 1643–52.
62. Baxter DE, Thigpen CM. Heel pain – operative results. Foot Ankle. 1984;5(1):16–25.

Inferior Calcaneal Nerve Entrapment

76

Michael N. Brown, Beth S. Pearce, and Helen W. Karl

Introduction

Patients with heel pain are common in clinical practice [1]. One potential source of medial heel pain is entrapment of the *inferior calcaneal nerve* (ICN), often referred to as *Baxter's nerve* [2]. Some of its other names include the *first branch of the lateral plantar nerve* (FBLPN), *deep calcaneal nerve*, and *nerve to the abductor digiti minimi (quinti)* muscle. It has been estimated that up to 20 % of chronic heel pain may be due to ICN entrapment [3]. Heel pain can have a variety of other causes including plantar fasciitis (the most common cause of heel pain [4]), plantar fascia rupture, heel fat pad atrophy, calcaneal stress fractures, and some rheumatologic diseases [5]. ICN entrapment can mimic or coexist with plantar fasciitis, which is now thought to be a degenerative process due to repetitive microtrauma of the plantar fascia together with acute and chronic inflammation [4, 6].

Clinical Presentation (Table 76.1)

Patients with ICN entrapment have burning medial heel pain that sometimes radiates to the arch of the foot or to the ankle (Fig. 76.1). Pain worsens the longer they are standing or walking and may awaken them from sleep [2]. The pain usually has a gradual onset without a specific traumatic event. These patients segregate into two discrete groups: avid runners and older individuals, often obese, with occupations that require a lot of standing [2]. They often walk with exaggerated foot supination to avoid weight on the medial heel and are tender over a region near the *medial calcaneal tubercle* (MCT) that is similar to the tenderness of plantar fasciitis (Fig. 76.2). Some causes of ICN entrapment are listed in Table 76.1.

Patients may have symptoms and signs of neurogenic heel pain with objective findings of plantar fasciitis such as hypoechoic and thickened plantar fascia on ultrasound (US) examination [13]. Evidence of plantar fasciitis does not rule out an entrapment neuropathy; the edema and thickening of the plantar fascia may itself compress the ICN [6]. The clinical picture may be further confounded by medical comorbidities such as diabetic peripheral neuropathy.

The presence of coexisting back pain should alert providers to the possibility that the primary lesion is an L5-S1 radiculopathy [14] or other proximal tibial nerve injuries, perhaps contributing to a "double crush" (Chap. 1), or that a compensatory antalgic gait has led to painful spasms of the back muscles [15]. Bilateral heel pain, especially when combined with pain in multiple joints or sites of ligament insertions, implies a probable rheumatologic origin [14].

Table 76.1 Occupation/exercise/trauma history relevant to inferior calcaneal nerve entrapment

Repetitive microtrauma	Occupations involving prolonged standing or walking: "Jogger's heel, tennis heel, policeman's heel, checkout girl's heel, etc." [2]
	Plantar fasciitis
	Fat pad atrophy [7]
Acute trauma	Direct trauma from a fall or stepping on a hard object
	Scar tissue after plantar fasciotomy
Impingement	Bony exostosis, calcaneal spur
	Neuroma, neurilemmoma [8–10]
Stretch injury	Hypermobile pronating foot
	Abductor hallucis hypertrophy, especially in runners [11, 12]
Conditions	Diabetic peripheral neuropathy

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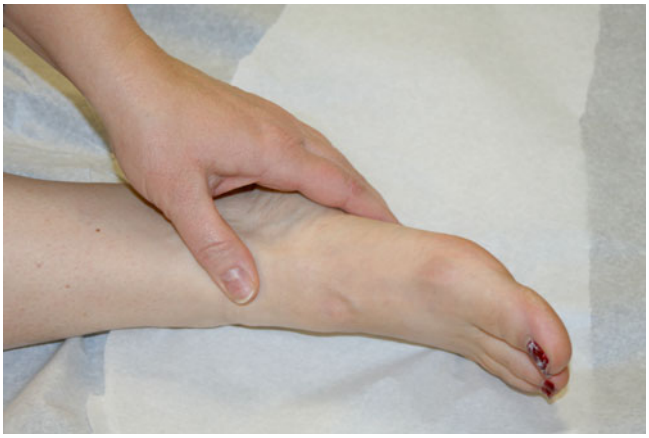

Fig. 76.1 Patient pain complaint from inferior calcaneal nerve entrapment (Image courtesy of Andrea Trescot, MD)

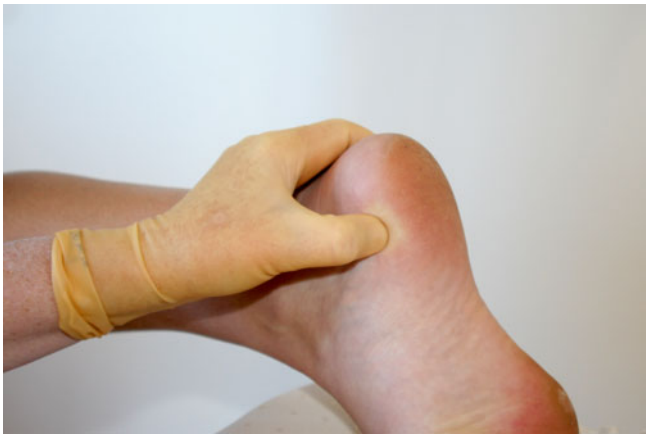

Fig. 76.2 Tenderness of the medial calcaneal tubercle (Image courtesy of Andrea Trescot, MD)

Table 76.2 Inferior calcaneal nerve anatomy

Origin	L4-S4 ventral rami form the sciatic nerve
General route	The sciatic nerve divides in the distal thigh into the tibial (Chap. 73) and peroneal (fibular, Chap. 67) nerves, and the tibial nerve (TN) continues down through the deep posterior compartment of the leg
	The TN divides within or occasionally near the tarsal tunnel behind and inferior to the medial malleolus into the lateral plantar (LPN), medial plantar (MPN), and medial calcaneal (MCN) nerves
	The LPN gives off the inferior calcaneal nerve (ICN) near the medial malleolus
	The ICN travels vertically between the abductor hallucis (AbH) and quadratus plantae (QP) muscles, then turns laterally and gives off its terminal branches
Sensory distribution	Periosteum at the medial calcaneal tubercle (MCT)
	Long plantar ligament
Motor innervation	*Abductor hallucis* (*AbH*, *primarily innervated by the MPN* [16]): from the distal flexor retinaculum, medial calcaneal tubercle, and the plantar aponeurosis to the base of the great toe; it flexes and abducts the great toe
	Flexor digitorum brevis (*FDB*, *also innervated by the MPN*): from the anterior portion of the calcaneal tuberosity and the plantar aponeurosis to the sides of the 2nd–5th toes; flexes 2nd–5th toes
	Quadratus plantae (*QP*): from the anterior portion of the calcaneal tuberosity to the lateral border of the flexor digitorum longus (FDL) tendon; augments FDL action
	Abductor digiti minimi (*AbDM*): from heads on the lateral process of the calcaneal tuberosity and the plantar aponeurosis to the base of the little toe; abducts and flexes the little toe
Anatomic variability	Origin of the ICN: LPN 82 % (119/145), TN 12 % (17/145), common trunk of LPN and MCN 4 % (6/145), common trunk of the branch to QP 2 % (3/145) [17]; other authors found the origin to be 100 % from the LPN: 30/30 [15], 34/34 [18]
	Branches of the ICN: one 11 %, two 87 %, three 2 % of 145 ft [17]
	Distance between the ICN and the medial calcaneal tubercle (MCT): averages 5.5 mm (−8 to 17 mm) [15]
	The MCN occasionally (3/15 = 20 %) branches from the LPN, so the ICN will be the 2nd branch of the LPN, not the first [19]
Other relevant structures	Plantar fascia (plantar aponeurosis): a strong superficial layer of fascia (3 cords [20]) that runs from the calcaneal tuberosity to the toes; posteriorly, it covers the FDB, which covers the QP, which covers the long plantar ligament
	Medial calcaneal tubercle (medial process of the calcaneal tuberosity, MCT): the origin of the central band of plantar fascia, AbH, FDB, and, more laterally, the AbDM
	Plantar heel spur: an exostosis at the junction of the plantar aponeurosis and the calcaneus; often present in asymptomatic patients

Anatomy (Table 76.2)

The sciatic nerve travels down the leg and splits into the peroneal and tibial nerves near the popliteal fossa. The tibial nerve at the ankle is often called the *posterior tibial nerve* (PTN, see Chap. 73). It enters the proximal *tarsal tunnel* (deep to the flexor retinaculum) at the medial ankle where it divides into the *lateral plantar nerve* (LPN, see Chap. 74), the *medial plantar nerve* (MPN, see Chap. 75), and the *medial calcaneal nerve* (MCN, see Chap. 77) (Fig. 76.3).

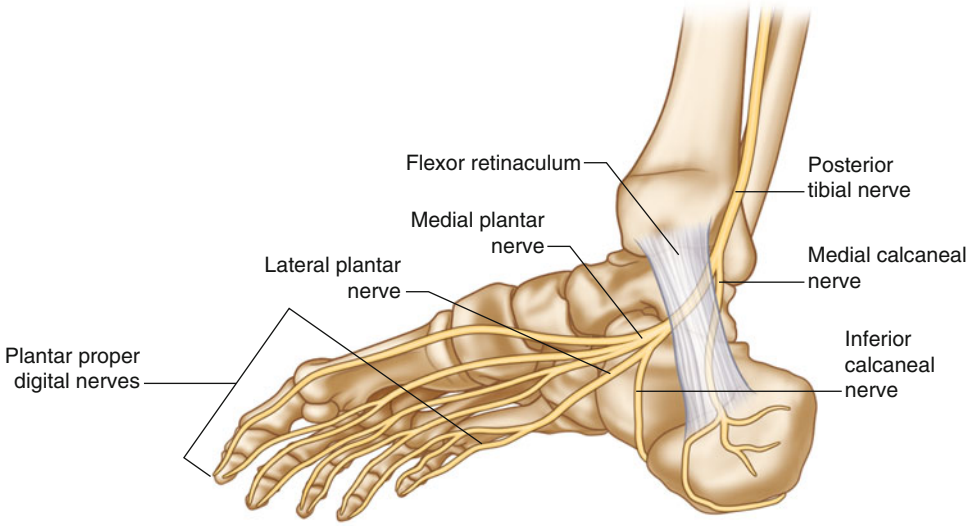

Fig. 76.3 Anatomy of the branches of the posterior tibial nerve. Note the inferior calcaneal nerve crossing deep and anterior to the medial calcaneal tubercle and the medial calcaneal nerve on the lateral face of the calcaneus (Image courtesy of Springer)

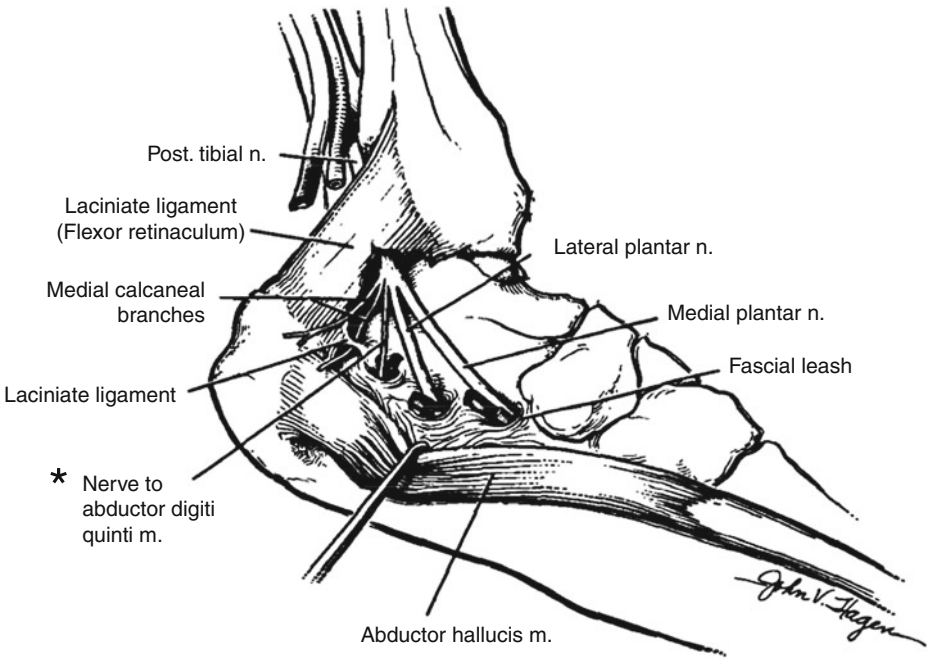

Fig. 76.4 Anatomy of the branches of the posterior tibial nerve. Note the nerve to the abductor digiti quinti muscle (inferior calcaneal nerve) as it migrates under the deep fascia of the abductor hallucis muscle. Note also the medial calcaneal branches (nerves) as they cross the lanciniate ligament (flexor retinaculum) to become more superficial and the lateral and medial plantar nerves as they cross the thick deep fascia of the abductor hallucis muscle (From Baxter and Thigpen [2]. Reprinted with permission from SAGE Publications)

The ICN is usually (in >80 % of patients) [15, 19] the first branch of the LPN (FBLPN); it diverges near the *malleolar-calcaneal axis* [21] and continues a vertical course toward the deep structures of the foot (Fig. 76.4). It lies between the abductor hallucis (AbH) and the quadratus plantae (QP) muscles (the *AbH-QP interval*) (Fig. 76.5), a relationship that is important when doing US-guided ICN blocks [5].

The ICN then runs between the QP and *flexor digitorum brevis* (FDB) (Fig. 76.5), innervating them as it becomes horizontal and heads for the abductor digiti minimi muscle (AbDM). Beyond the lateral margin of the QP, the ICN passes very close (mean of 5.5 mm) to the calcaneus, the MCT, and the long plantar ligament (Fig. 76.6) [15]. The terminal branches of the ICN continue laterally to the *abductor digiti minimi* (AbDM, also called the *abductor digiti quinti*) [22], accompanied by small arteries and veins. The ICN gives additional sensory branches to the periosteum of the MCT and the long plantar ligament [15, 18].

Ultrasound Anatomy of the Inferior Calcaneal Nerve

When the ICN is the first branch of the LPN, it is seen as a 1–2 mm structure splitting from the posterior border of the larger LPN (Fig. 76.7). Its vertical course can be followed from the origin to the AbH-QP interval; thereafter, the signal is attenuated as it becomes deeper and horizontal to cross

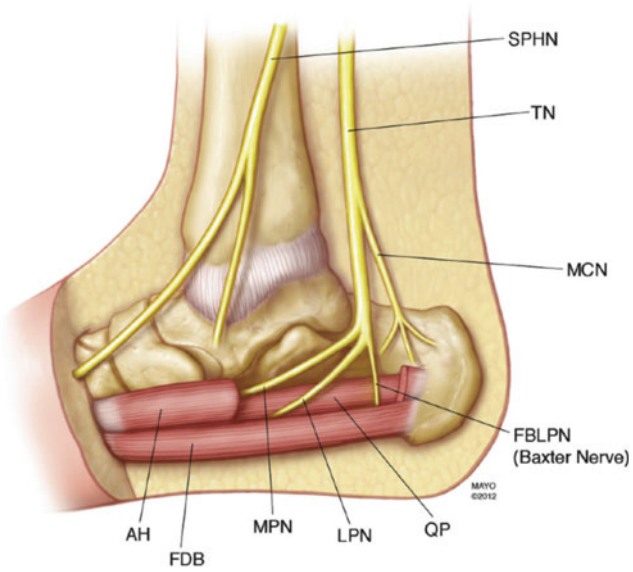

Fig. 76.5 Drawing of the medial ankle showing the branching pattern of the tibial nerve in the proximal and distal tarsal tunnels. The abductor hallucis (AbH) has been partially removed to show the vertical course of the inferior calcaneal nerve (ICN) and the interval between the AbH (removed) and quadratus plantae muscles (QP). *SPHN* saphenous nerve, *TN* tibial nerve, *MCN* medial calcaneal nerve, *FBLPN* first branch of the lateral calcaneal nerve (inferior calcaneal nerve), *QP* quadratus plantae, *LPN* lateral plantar nerve, *MPN* medial plantar nerve, *FDB* flexor digitorum brevis, *AH* abductor hallucis. Note the vertical course of the ICN perpendicular to the parallel paths of the AH, FDB, and QP (From Presley et al. [5]. Reprinted with permission from American Institute of Ultrasound in Medicine)

toward the AbDM [5, 23]. It is important not to confuse the ICN with the MCN, which has multiple branches that travel obliquely, posteriorly, and superficially [5].

Entrapment

There are two common sites of ICN entrapment [2]. The first is between the dense deep fascia of the AbH and the medial aspect of the QP where the nerve changes direction from vertical to horizontal as it travels toward the lateral foot (Fig. 76.6, site 1) [5]. At this site, the ICN can be compressed by a hypertrophied AbH, which is particularly common in runners [11, 12]. Increased foot pronation can stretch the nerve where it takes the sharp turn at the QP [24].

The second, more distal location is where the ICN crosses anterior to the MCT (or heel spur if one is present), between the FDB plantarly and the QP dorsally (Fig. 76.6, site 2) [2, 5, 13, 18].

ICN neuropathy may also be iatrogenic. After a plantar fasciotomy, postoperative scar tissue or distal migration of the fascia may bind the nerve. Alternatively, it may be damaged during heel spur resection.

Physical Examination

Begin the physical examination of the heel by palpating the central band of the plantar fascia to check for distal fasciitis and the origin of the plantar fascia at the deep MCT for proximal fasciitis (Fig. 76.7, circle 2). Palpate the distal Achilles (calcaneal) tendon and retrocalcaneal bursa and then squeeze the heel to evaluate a potential stress fracture (Fig. 76.7, circle 4). Ask the patient to widely abduct all their toes and compare the strength of the great and little toes with the other side. Since the ICN innervates the AbDM, the patient's ability to abduct the little toe may be diminished; if the MCN is also involved, the AbH may be weaker on the affected side (Chap. 77).

Tenderness and paresthesias may be identified deep to the AbH (Fig. 76.7, circle 3), and the pain may radiate proximally and distally. Percuss the upper and lower tarsal tunnels over the AbH to elicit *Tinel's sign*, although this is often not present in patients with ICN dysfunction [22, 25]. If the foot is plantar flexed and inverted, pressure applied under the AbH is more likely to provoke the patient's symptoms [26]. Dorsiflexion of the foot and toes puts tension on the plantar fascia. The fascia is more likely to be the source of the patient's pain if tenderness is greater when it is under tension. If tenderness is greater when the fascia is relaxed, a subfascial structure such as the ICN is likely responsible [26]. If the AbH is compressing a nerve or part of one, the patient's symptoms may be reproduced by tensing it [27].

Since the ICN has no cutaneous territory, the presence of discrete areas of sensory change makes MCN dysfunction more likely [28, 29].

Differential Diagnosis (Tables 76.3 and 76.4)

The most common causes of plantar heel pain are *plantar fasciitis*, *heel pad atrophy*, *calcaneal stress fracture*, and nerve entrapments [1, 7]. The presence of bilateral pain should trigger investigation into an inflammatory source, and L5-S1 radiculopathy may be the cause in a patient with coexisting back pain [3].

Patients with ICN compression and plantar fasciitis present similarly, but subtle differences can provide some clues to the diagnosis (Table 76.3). The pain of ICN dysfunction is "neuralgic," with burning and sometimes tingling that often worsens the longer they are standing or walking, an indication of a biomechanical contribution to the pathophysiology. It also may be worse at night, which is not characteristic of plantar fasciitis. A biomechanical assessment of the foot and ankle will often demonstrate intrinsic foot abnormalities that cause increased foot pronation. Patients with ICN entrapment will not experience cutaneous sensory disturbance;

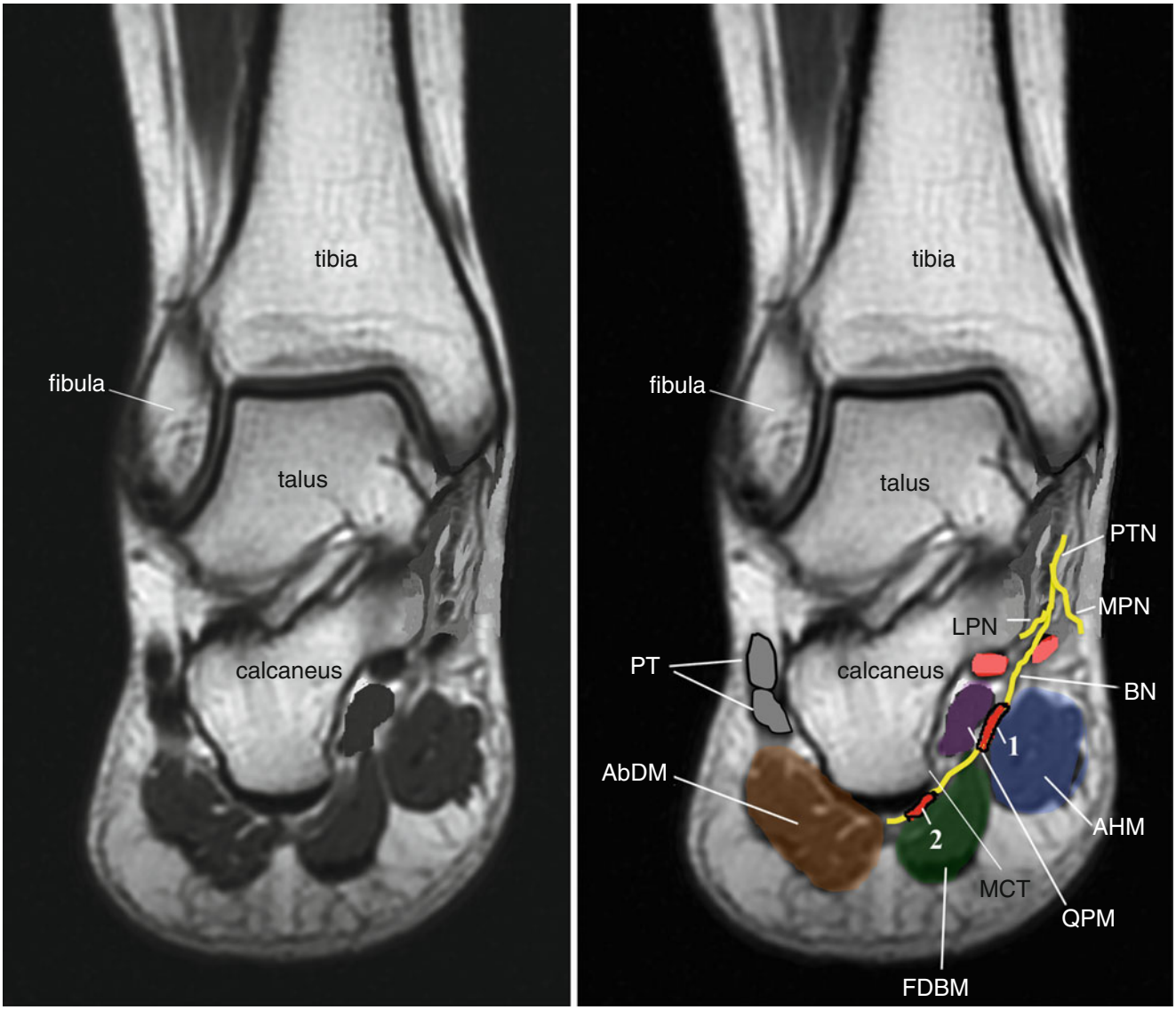

Fig. 76.6 Locations of inferior calcaneal nerve entrapment. *PTN* posterior tibial nerve, *MPN* medial plantar nerve, *LPN* lateral plantar nerve, *BN* Baxter's nerve (inferior calcaneal nerve), *AHM* abductor hallucis muscle, *QPM* quadratus plantae muscle, *FDBM* flexor digitorum brevis muscle, *AbDM* abductor digiti minimi muscle, *MCT* medial calcaneal tubercle, *PT* peroneus (fibularis) brevis and longus tendons. *1* Entrapment site between the AHM and QPM. *2* Entrapment site between the MCT and the FDBM (Image courtesy of Andrea Trescot, MD)

however, some may have anatomical variants where it will pick up some fibers from the lateral plantar nerve and demonstrate numbness of the lateral two toes. They may also have denervation changes in the AbDM on MRI [6, 11], although this finding is not specific to ICN dysfunction [33]. Since an ICN block abolishes sensation at the periosteum of the MCT, it could block the pain of plantar fasciitis. Therefore, one may not be able to differentiate ICN compression neuropathy from plantar fasciitis by simply blocking the ICN.

Patients with plantar fasciitis experience the greatest pain with the first several steps they take in the morning or after rest and then gradually improve as they bear weight.

The list of other possible diagnoses is quite long, and it can be divided into those that lead to plantar heel pain, medial heel pain, and more general heel pain (Table 76.4).

Diagnostic Tests (Table 76.5)

Patients may have symptoms of neurogenic heel pain mixed with objective findings of plantar fasciitis, such as US findings of hypoechoic and thickened (>4 mm) plantar fascia [13]. However, the presence of signs of plantar fasciitis does not rule out an entrapment neuropathy. Electrodiagnostic testing is inconsistent in the diagnosis of ICN entrapment [34–36].

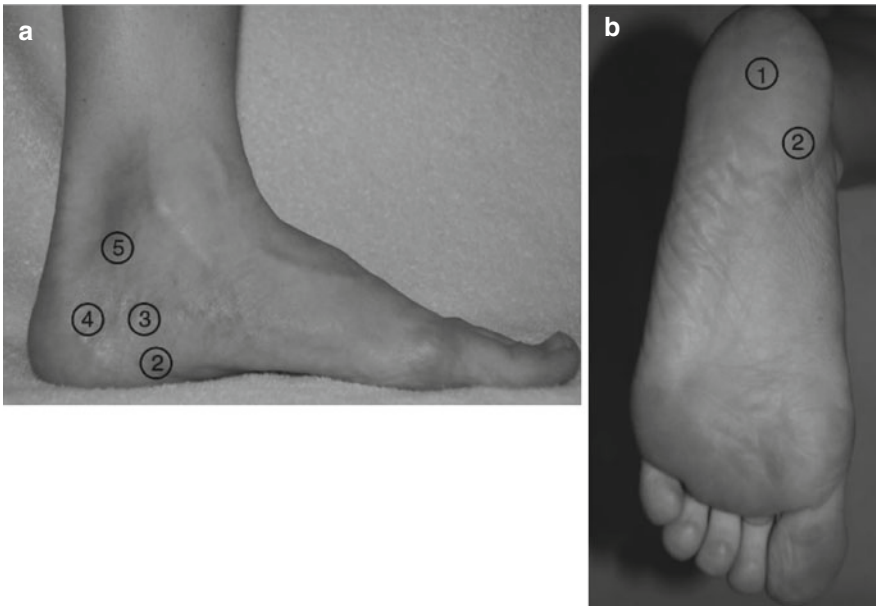

Fig. 76.7 Locations of tenderness for five diagnoses of heel pain: *1* heel pad atrophy, *2* plantar fasciitis, *3* ICN entrapment, *4* calcaneal stress fracture, *5* tarsal tunnel syndrome (From Lareau et al. [7]. Reprinted with permission from Wolters Kluwer Health)

Table 76.3 Clinical comparison of inferior calcaneal nerve entrapment and plantar fasciitis

Inferior calcaneal nerve entrapment	Plantar fasciitis
Pain is worse after activity and at night	"First step" pain: worse first thing in the morning or after a period of inactivity
Pain gets worse with walking	Pain gets better with walking
"Neuralgic" pain: sharp and burning	"Mechanical" pain: a deep ache
Maximum tenderness at the medial calcaneal tuberosity is likely to radiate to the medial ankle and/or to the lateral foot	Maximum tenderness at the insertion of the plantar fascia at the medial calcaneal tuberosity and along its medial edge where thickened fascia may be felt
Heel pad sensation should be like that on the contralateral side	Heel pad sensation should be like that on the contralateral side

Table 76.4 Differential diagnosis of heel pain

Plantar heel pain	Medial heel pain	Others
Plantar fasciitis [30, 31]	Flexor digitorum longus tenosynovitis [32]	Rheumatoid arthritis [30, 31], especially if bilateral [3]
Plantar fascia tear	Flexor hallucis longus tenosynovitis	Reiter's syndrome
Heel pad atrophy [30]	Tibialis posterior tenosynovitis	Ankylosing spondylitis [31]
Tarsal tunnel syndrome, although the heel may be spared if the MCN origin is high	Tarsal tunnel syndrome	Psoriatic arthritis
Inferior calcaneal nerve entrapment, dysfunction, or neuroma [3, 8]	Medial calcaneal nerve entrapment, dysfunction, or neuroma [31]	Gout [30, 31]
Calcaneal stress fracture [7, 31]	Inferior calcaneal nerve entrapment	Posterior enthesopathies
Calcaneal cyst, tumor, osteomyelitis, or edema	Retrocalcaneal bursitis [8]	Behçet's syndrome
Calcaneal apophysitis (Sever's disease)		Systemic lupus erythematosus
Heel spur [31]		Fibromyalgia
Vascular insufficiency, congestion, or varicosities [8]		L5-S1 radiculopathy [3]

Chundru et al. [6] reviewed the MRIs of 100 patients with heel pain and AbDM atrophy and then matched them with an equal number of patients with heel pain and no AbDM atrophy. There were significantly more patients with advanced age, calcaneal spurs, and plantar fasciitis in the group whose MRIs showed AbDM atrophy. However, selective atrophy of the AbDM is not rare; it was seen in 6 % of 602 foot and ankle MRIs, and only one of those patients was thought to

Table 76.5 Diagnostic tests for inferior calcaneal nerve entrapment

	Potential distinguishing features
Physical exam	Tenderness over the medial calcaneal tubercle
Diagnostic injection	Landmark- or US-guided
Ultrasound	Hypoechoic and thickened plantar fascia may coexist with inferior calcaneal nerve (ICN) entrapment
MRI	Abductor digiti minimi muscle atrophy indicates possible ICN dysfunction; MRI is recommended before surgical intervention [6]
Arteriography	Not useful
X-ray	To rule out bony causes
	Heel spurs are nonspecific: they are often present in asymptomatic patients [2]
Electrodiagnostic studies	Inconsistent results

have ICN dysfunction [33]. Thus, AbDM atrophy is suggestive of but not specific for ICN dysfunction. Precise injection of a small amount of local anesthetic at the ICN may assist the clinician in determining whether the patient would benefit from its ablation (see section below "Neurolytic techniques").

Treatment of Contributing/Perpetuating Factors

Obesity and/or an intrinsic foot anomaly that contributes to increased foot pronation (midtarsal joint laxity, forefoot varus, rear foot eversion, *pes planus*, *cavovarus foot*) can contribute to plantar fasciitis and ICN dysfunction [4, 6]. Initial treatment is similar for both conditions, including correction of passive mechanics of the foot with orthotics when needed, proper shoes, weight reduction, and stretching the plantar fascia and Achilles tendon.

Injection Techniques

Landmark-Guided Injection

ICN entrapment can occur at two separate sites: where the nerve crosses the sharp edge of the AbH fascia and just medial to the MCT (Fig. 76.6) [2]. Careful palpation may allow one to distinguish whether the point of maximum tenderness is more superficial, at the AbH, or deeper, near the plantar fascia. The injection for entrapment above the AbH is shallower than the one performed at the transition zone at the MCT.

The authors' preference is to have the patient lying on their side with the medial foot facing up. Place your thumb on the posterior aspect of the medial malleolus, and slide it toward the heel. The first muscle that you feel will be the AbH, a key anatomic landmark that will move when the great toe is abducted. Find the depression just cephalad to the AbH: this is where the ICN travels under the muscle as it crosses the foot towards the MCT (Fig. 76.8a). Palpate and mark the region of the MCT and then get a sense of its depth by resting the needle on the skin (Fig. 76.8b).

Direct the needle over the AbH, toward the MCT (Fig. 76.8c). Check the needle depth and compare it to your previous estimate. If you contact the bone, then you have reached the plantar fascia or QP attachments. Pull the needle

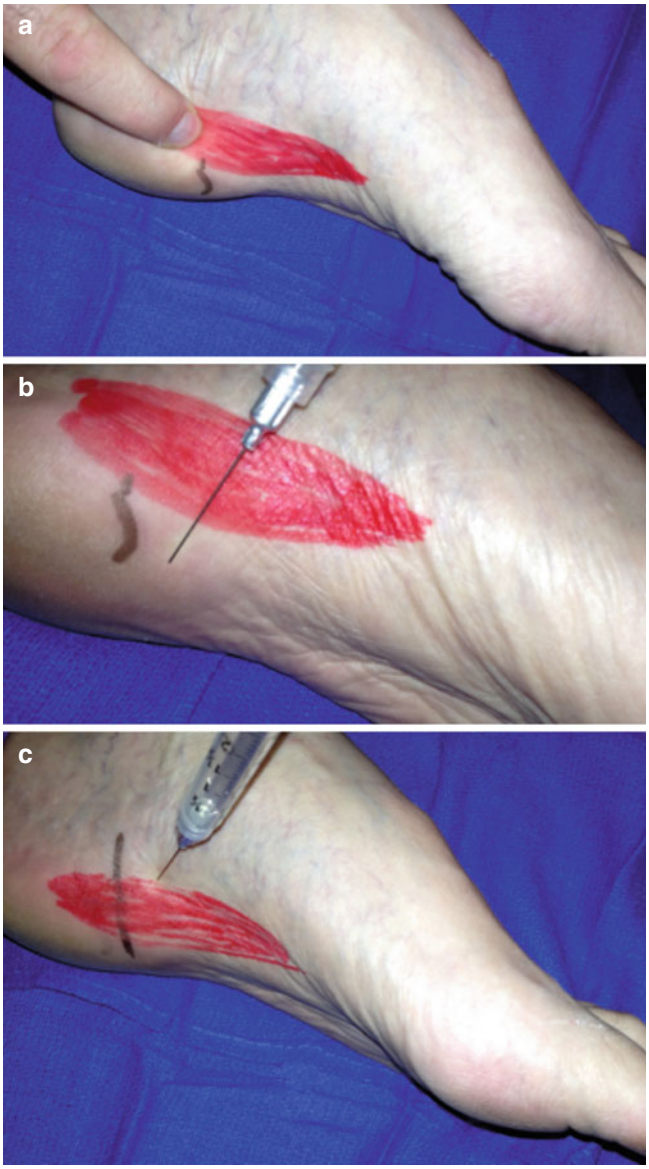

Fig. 76.8 Landmark-guided inferior calcaneal nerve injection. (**a**) palpation of injection site; (**b**) depth of injection; (**c**) inferior calcaneal nerve injection; *Red* structure, outline of the abductor hallucis muscle (Image courtesy of Michael N. Brown, MD)

back half a centimeter to place its tip near the ICN, and inject 1–2 cc of local anesthetic.

The authors prefer a 27-gauge, 1.5-in. needle, though a 25-gauge needle or on occasion a 30-gauge, 1-in. needle can also be used. If the injection is too shallow, one may not obtain an adequate block.

Fluoroscopy-Guided Injection

In the authors' opinion, fluoroscopy provides little benefit for ICN injections, since there are no bony landmarks.

Ultrasound-Guided Injections

The plantar aponeurosis and surrounding muscles are thought likely to keep local anesthetic placed around the ICN localized to the deep tissues [15]. For purposes of diagnosis, a low-volume local anesthetic injection at the ICN, even proximal to the entrapment site, can be useful, so the *in-plane approach* described below may be the simplest. If therapeutic or hydrodistention maneuvers are planned, an *out-of-plane approach* may be preferred. Initial US views of the ICN involve placing the US probe horizontally across the MCT (Fig. 76.9) and then rotating the probe vertically (Fig. 76.10). In this position, the ABH and QP are visible with the neurovascular bundle (Fig. 76.11).

In-Plane Technique

The *in-plane approach* to the ICN from posterior to anterior is easiest, allows the clinician to avoid the blood vessels anterior to the ICN, and has been documented to provide complete coverage of the ICN in 91 % of injections (11 of 12 injections in one study) [5]. This approach is the authors' preference.

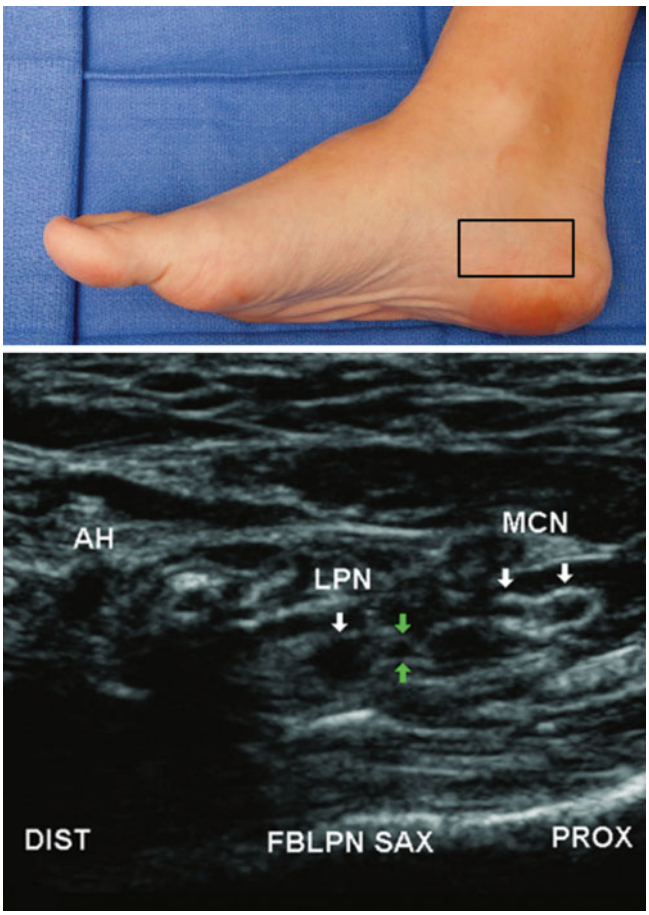

Fig. 76.9 Short-axis sonographic view of the inferior calcaneal nerve. *LPN* lateral plantar nerve; *MCN* medial calcaneal nerve, *green arrows* ICN, *AH* abductor hallucis muscle; bony acoustic margin at the bottom, calcaneus. The ICN lies just posterior to the much larger LPN and is vertical at this level. The two visible branches of the MCN should not be confused with the ICN. The MCN travels obliquely posteriorly and superficially. The QP is just becoming visible between the ICN and the calcaneus (From Presley et al. [5]. Reprinted with permission from the American Institute of Ultrasound in Medicine)

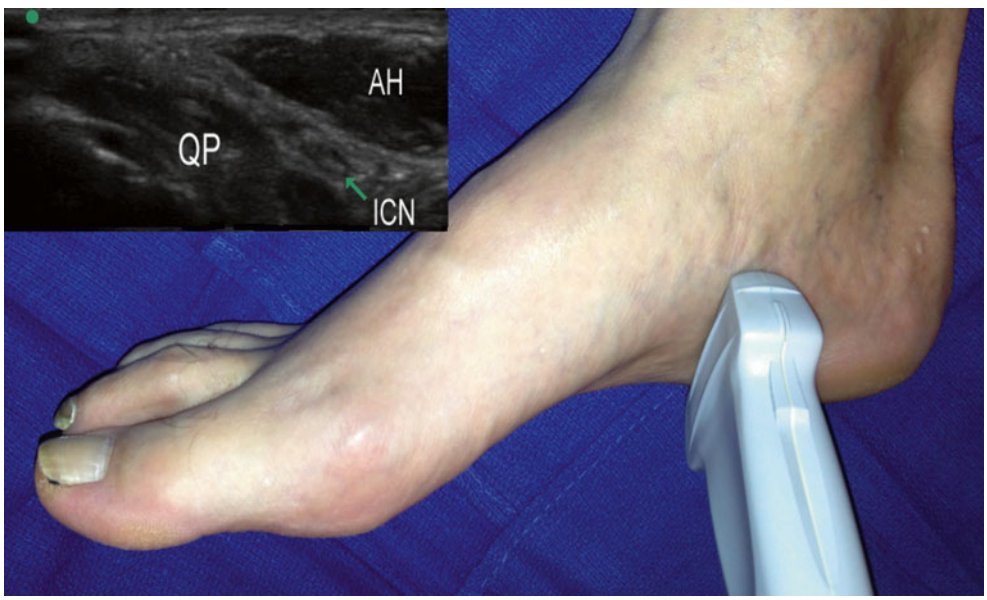

Fig. 76.10 In-plane ultrasound image showing the probe location and the neurovascular bundle that includes the inferior calcaneal nerve. *AH* adductor hallucis muscle, *QP* quadratus plantae muscle, *ICN* inferior calcaneal nerve (Image courtesy of Michael Brown, MD)

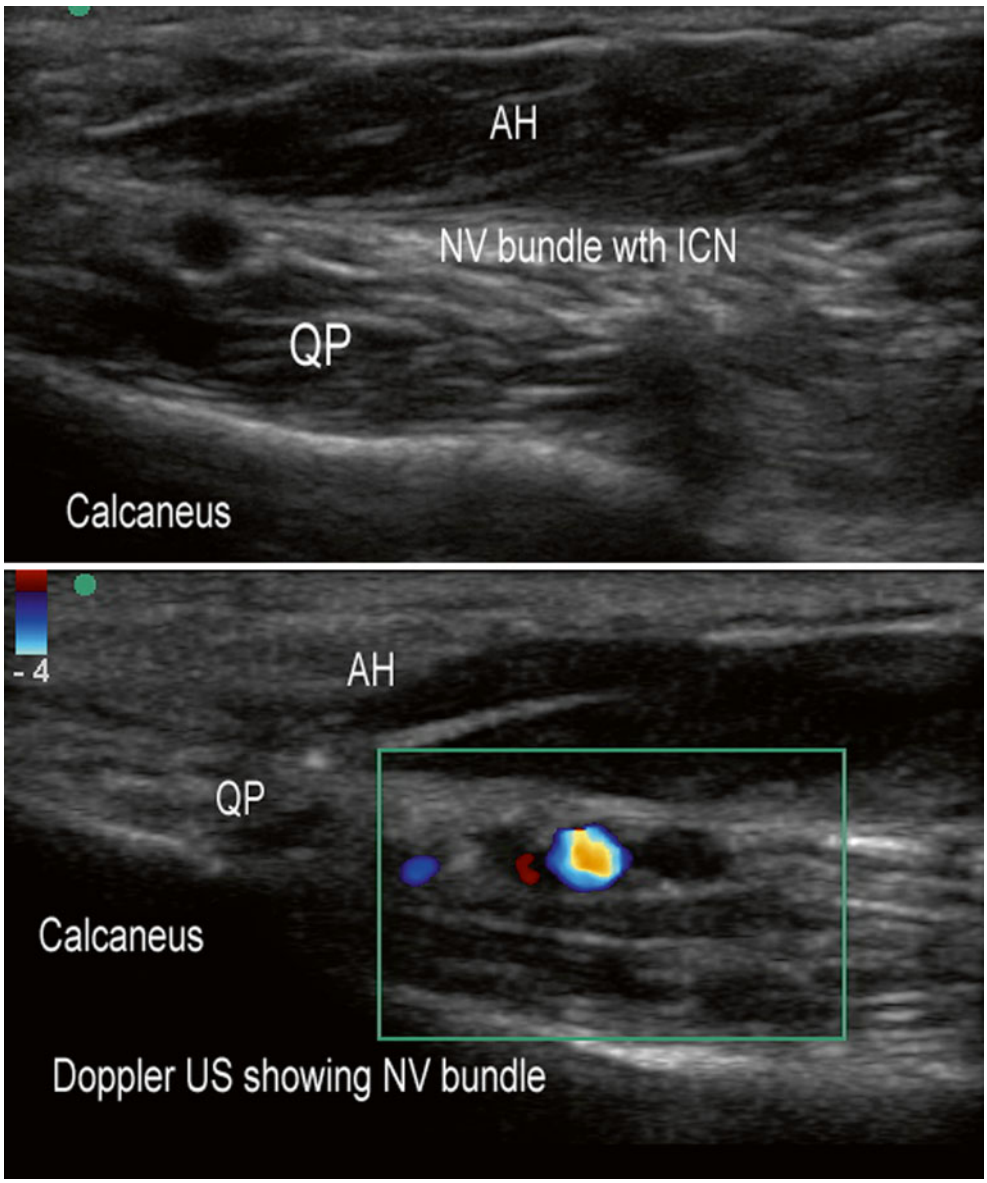

Fig. 76.11 In-plane ultrasound image of the neurovascular bundle containing the inferior calcaneal nerve. *AH* adductor hallucis muscle, *QP* quadratus plantae muscle, *ICN* inferior calcaneal nerve (Image courtesy of Michael Brown, MD)

Anatomical dissections have revealed the presence of a fascial plane through which the first branch of the lateral plantar nerve courses [5]. One of the problems in performing an injection of this nerve is the cranial flow that occurs with local anesthetics, even with low injection volumes (1 cc) [5]. This may be important when considering the specificity of diagnosis. Placing the injection in a more caudal location within the AbH-QP interval or using smaller injectate volumes (0.5 cc) may improve diagnostic specificity. It has also been shown that the injectate does not flow into the plantar fascia region, providing possible differential diagnostic value with respect to the ICN versus plantar fasciitis [5].

Place a linear transducer in the position used to see the proximal PTN, confirm the vein-artery-vein (VAV) relationship to the PTN (see Chap. 73), and follow the LPN until the ICN diverges from its posterior surface (Fig. 76.9). Follow the ICN, moving the transducer until you can identify the AbH-QP interval (Fig. 76.10). Accentuate the slight separation between the AbH and QP by passively dorsiflexing and plantar flexing the lesser toes to move the QP [5]. Clear identification of the AbH-QP interval allows an accurate injection, even if the ICN itself is poorly seen. This is also the location for the neural fascial hydrodistention and cryoneuroablation techniques described below.

After appropriate skin anesthesia (1 % buffered lidocaine injected next to the transducer with a 30-gauge, 0.5-in. needle), direct a 27-gauge, 1.5-in. needle or 25-gauge, 2-in. needle from posterior to anterior for the most accurate placement of the injectate (Fig. 76.12) [5]. Watch the needle approach the AbH-QP interval and the ICN; once it is in position, inject local anesthetic with deposteroid or hydrodistention solution.

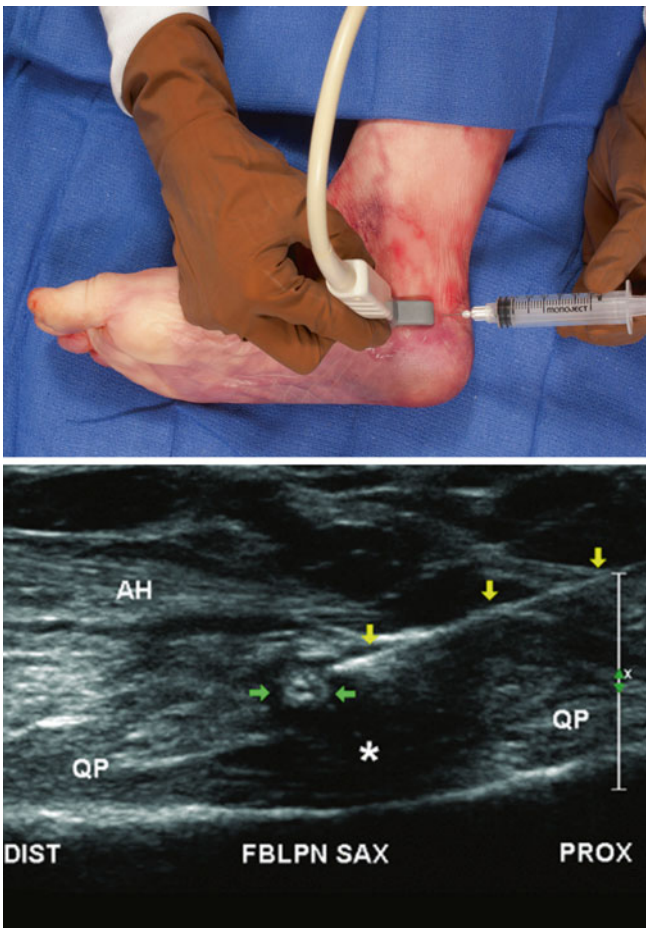

Fig. 76.12 Short-axis sonographic view of the inferior calcaneal nerve. *AH* abductor hallucis muscle, *QP* quadratus plantae muscle, *green arrows* ICN, * = plantar fascia. The vertical ICN lies between the AH and the QP. Note the in-plane needle entering posteriorly (From Presley et al. [5]. Reprinted with permission from American Institute of Ultrasound in Medicine)

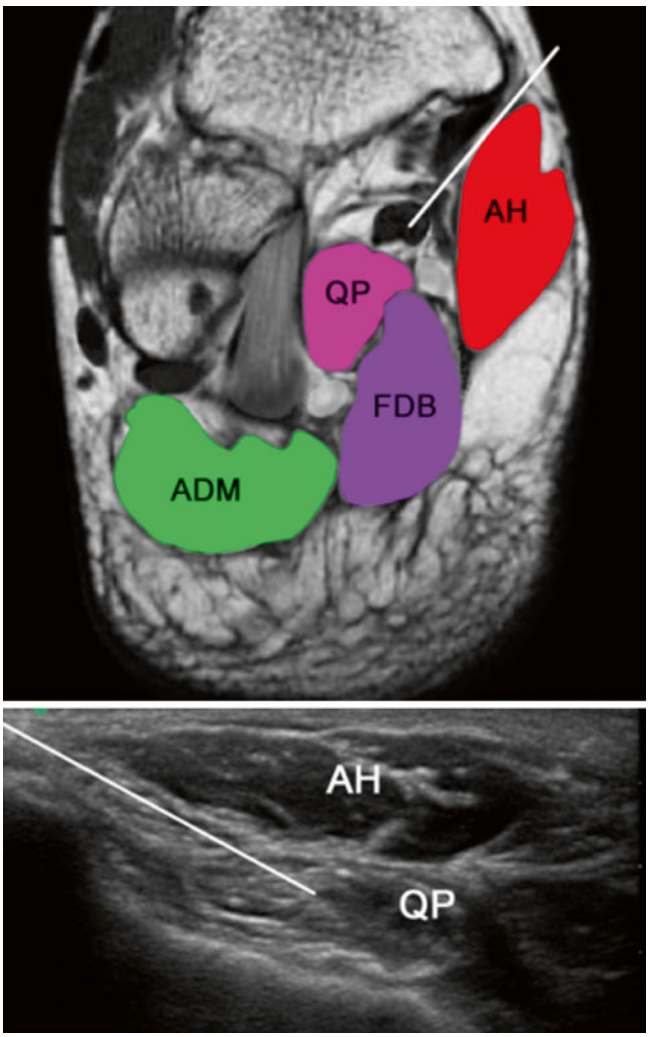

Fig. 76.13 Composite images of the heel structures, coronal cross-sectional technique. (**a**) MRI cross section with simulated needle; (**b**) ultrasound transverse image; *ADM* abductor digiti minimi muscle, *AH* abductor hallucis, *FHB* flexor hallucis brevis, *QP* quadratus plantae (Image courtesy of Brian Shilpe, DO)

Out-of-Plane Technique

The variable thickness of the heel fat pad and AbH aponeurosis makes the exact location of the MCT difficult to detect with palpation alone. A simple way to find it with US is to use a bent paper clip under a 7.5–15 MHz linear US transducer (see Chap. 7) [37]. The paper clip will create an echo artifact that reflects down to the MCT. By moving the paper clip, the artifact can be lined up on the MCT and the skin marked. The paperclip can be autoclaved if one prefers to use it after sterilizing the skin.

After positioning the patient similar to that for the landmark-guided injection (see above), locate the AbH by palpation (Fig. 76.8a). Place a linear transducer in the position used to see the proximal PTN, and confirm the vein-artery-vein (VAV) relationship to the PTN (see Chap. 73). Next, follow the LPN as it travels toward the distal tarsal tunnel, and rotate it as shown in Fig. 76.10. You should now be able to see the AbH, FHB, and QP in cross section and confirm their identities by moving the appropriate digits (Fig. 76.13).

Inject local anesthetic for skin anesthesia with a 30-gauge, 0.5-in. needle, in line with the previous skin mark anterior to the MCT and over the AbH. Insert a 27-gauge, 1.5-in. needle or 25-gauge, 2-in. needle, and place the transducer on top of it to see the needle and guide its trajectory (Fig. 76.14). This approach can be used to pass a needle over the AbH or through AbH-QP interval. Another technique is to direct the needle toward the region 0.5–1 cm anterior to the MCT. One cc of local anesthetic and deposteroid can be injected in either location.

Hydrodissection

Before opting for surgical decompression, perineural hydrodissection can be considered (see Chap. 7). The

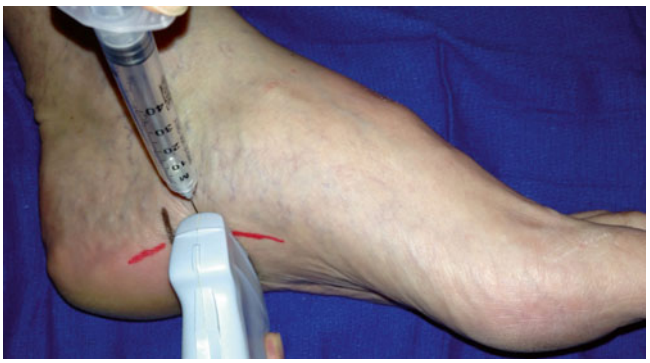

Fig. 76.14 In-plane ultrasound technique of ICN injection (Image courtesy of Michael N. Brown, MD)

efficacy of this technique for tarsal tunnel and ICN entrapment neuropathies will need to be verified in controlled clinical trials. However, the authors will report the procedure based on their experience and hope it may spark ongoing discussion.

Use the in-plane US approach as described above. Inject 1 % buffered lidocaine with a 30-gauge, 0.5-in. needle posterior to the transducer for skin anesthesia, and advance a 25-gauge, 2-in. needle at the AbH-QP interval above the neurovascular bundle near the ICN. Then, inject 3–4 cc of hydrodissection solution (Chap. 7), and pull the needle back slightly to repeat the procedure with the needle under the nerve.

Neurolytic Technique

Cryoneuroablation

In circumstances where incapacitating pain is clearly due to ICN compression, a decision may be made to sacrifice AbDM function and proprioception for the sake of pain relief. If cryoneuroablation of the ICN is elected, the cryoprobe can be placed using either of the approaches described above.

Surgical Technique

A description of the many surgical approaches to the tarsal tunnel, plantar fasciitis, and the plantar and calcaneal nerves is outside the scope of this chapter. The release of any entrapped nerve requires specific and precise identification of the entrapment location. In this area, "double-crush" phenomena are common (see Chap. 1), and the possibility of more than one entrapment needs to be considered. Incomplete decompression may be the reason for the relatively high failure rate of surgery in this area [38].

Complications

As with any procedure, there are risks of infection, hematoma, vascular puncture, or nerve injury. The most common associated problem is incomplete nerve release or ablation leading to increased pain. In this circumstance, repeating the procedure may lead to resolution of symptoms. The skin is very thin at this site, so injections or surgery can result in skin atrophy or injury.

Summary

Finding an exact cause of heel pain can be difficult. Meticulous history and physical examination reveal likely pathology, while precise injections often confirm the diagnosis.

References

1. Thomas JL, Christensen JC, Kravitz SR, Mendicino RW, Schuberth JM, Vanore JV, et al. The diagnosis and treatment of heel pain: a clinical practice guideline-revision 2010. J Foot Ankle Surg. 2010;49(3 Suppl):S1–19.
2. Baxter DE, Thigpen CM. Heel pain–operative results. Foot Ankle. 1984;5(1):16–25.
3. Baxter DE, Pfeffer GB, Thigpen M. Chronic heel pain. Treatment rationale. Orthop Clin North Am. 1989;20(4):563–9.
4. Buchbinder R. Clinical practice. Plantar fasciitis. N Engl J Med. 2004;350(21):2159–66.
5. Presley JC, Maida E, Pawlina W, Murthy N, Ryssman DB, Smith J. Sonographic visualization of the first branch of the lateral plantar nerve (Baxter nerve): technique and validation using perineural injections in a cadaveric model. J Ultrasound Med. 2013;32(9):1643–52.
6. Chundru U, Liebeskind A, Seidelmann F, Fogel J, Franklin P, Beltran J. Plantar fasciitis and calcaneal spur formation are associated with abductor digiti minimi atrophy on MRI of the foot. Skeletal Radiol. 2008;37(6):505–10.
7. Lareau CR, Sawyer GA, Wang JH, DiGiovanni CW. Plantar and medial heel pain: diagnosis and management. J Am Acad Orthop Surg. 2014;22(6):372–80.
8. Shandles ID, Pruchniewski J, Reynolds KL. Heel neuroma: the enigma of recalcitrant heel pain and an innovative approach highlighting sixty surgical cases and a review of two hundred and fifty-seven symptomatic but non-surgical cases. Foot. 2002;12(1):10–20.
9. Marui T, Yamamoto T, Akisue T, Hitora T, Kawamoto T, Nagira K, et al. Neurilemmoma in the foot as a cause of heel pain: a report of two cases. Foot Ankle Int. 2004;25(2):107–11.
10. Kwok KB, Lui TH, Lo WN. Neurilemmoma of the first branch of the lateral plantar nerve causing tarsal tunnel syndrome. Foot Ankle Spec. 2009;2(6):287–90.
11. Beltran LS, Bencardino J, Ghazikhanian V, Beltran J. Entrapment neuropathies III: lower limb. Semin Musculoskelet Radiol. 2010;14(5):501–11.
12. Erickson SJ, Quinn SF, Kneeland JB, Smith JW, Johnson JE, Carrera GF, et al. MR imaging of the tarsal tunnel and related spaces: normal and abnormal findings with anatomic correlation. AJR Am J Roentgenol. 1990;155(2):323–8.

13. Tsai WC, Chiu MF, Wang CL, Tang FT, Wong MK. Ultrasound evaluation of plantar fasciitis. Scand J Rheumatol. 2000; 29(4):255–9.
14. Rosenbaum AJ, DiPreta JA, Misener D. Plantar heel pain. Med Clin North Am. 2014;98(2):339–52.
15. Arenson DJ, Cosentino GL, Suran SM. The inferior calcaneal nerve: an anatomical study. J Am Podiatry Assoc. 1980; 70(11):552–60.
16. Davis TJ, Schon LC. Branches of the tibial nerve: anatomic variations. Foot Ankle Int. 1995;16(1):21–9.
17. del Sol M, Olave E, Gabrielli C, Mandiola E, Prates JC. Innervation of the abductor digiti minimi muscle of the human foot: anatomical basis of the entrapment of the abductor digiti minimi nerve. Surg Radiol Anat. 2002;24(1):18–22.
18. Rondhuis JJ, Huson A. The first branch of the lateral plantar nerve and heel pain. Acta Morphol Neerl Scand. 1986;24(4):269–79.
19. Louisia S, Masquelet AC. The medial and inferior calcaneal nerves: an anatomic study. Surg Radiol Anat. 1999;21(3):169–73.
20. Hoffman D, Bianchi S. Sonographic evaluation of plantar hindfoot and midfoot pain. J Ultrasound Med. 2013;32(7):1271–84.
21. Dellon AL, Mackinnon SE. Tibial nerve branching in the tarsal tunnel. Arch Neurol. 1984;41(6):645–6.
22. Baxter DE, Pfeffer GB. Treatment of chronic heel pain by surgical release of the first branch of the lateral plantar nerve. Clin Orthop Relat Res. 1992;279:229–36.
23. Fantino O. Role of ultrasound in posteromedial tarsal tunnel syndrome: 81 cases. J Ultrasound. 2014;17(2):99–112.
24. Schon LC, Baxter DE. Neuropathies of the foot and ankle in athletes. Clin Sports Med. 1990;9(2):489–509.
25. Fredericson M, Standage S, Chou L, Matheson G. Lateral plantar nerve entrapment in a competitive gymnast. Clin J Sport Med. 2001;11(2):111–4.
26. Hendrix CL, Jolly GP, Garbalosa JC, Blume P, DosRemedios E. Entrapment neuropathy: the etiology of intractable chronic heel pain syndrome. J Foot Ankle Surg. 1998;37(4):273–9.
27. Ghosh SK, Raheja S, Tuli A. Potential sites of compression of tibial nerve branches in foot: a cadaveric and imaging study. Clin Anat. 2013;26(6):768–79.
28. Dellon AL, Kim J, Spaulding CM. Variations in the origin of the medial calcaneal nerve. J Am Podiatr Med Assoc. 2002; 92(2):97–101.
29. Rose JD, Malay DS, Sorrento DL. Neurosensory testing of the medial calcaneal and medial plantar nerves in patients with plantar heel pain. J Foot Ankle Surg. 2003;42(4):173–7.
30. Diers DJ. Medial calcaneal nerve entrapment as a cause for chronic heel pain. Physiother Theory Pract. 2008;24(4):291–8.
31. Plotkin D, Patel D, Roberto T. Heel neuroma: a case study. J Foot Ankle Surg. 2009;48(3):376–9.
32. Schon LC. Nerve entrapment, neuropathy, and nerve dysfunction in athletes. Orthop Clin North Am. 1994;25(1):47–59.
33. Recht MP, Grooff P, Ilaslan H, Recht HS, Sferra J, Donley BG. Selective atrophy of the abductor digiti quinti: an MRI study. AJR Am J Roentgenol. 2007;189(3):W123–7.
34. Patel AT, Gaines K, Malamut R, Park TA, Toro DR, Holland N, et al. Usefulness of electrodiagnostic techniques in the evaluation of suspected tarsal tunnel syndrome: an evidence-based review. Muscle Nerve. 2005;32(2):236–40.
35. Oztuna V, Ozge A, Eskandari MM, Colak M, Gölpinar A, Kuyurtar F. Nerve entrapment in painful heel syndrome. Foot Ankle Int. 2002;23(3):208–11.
36. Colak T, Bamaç B, Gönener A, Ozbek A, Budak F. Comparison of nerve conduction velocities of lower extremities between runners and controls. J Sci Med Sport. 2005;8(4):403–10.
37. Gray AT. Atlas of ultrasound-guided regional anesthesia. 2nd ed. Philadelphia: Elsevier Saunders; 2013.
38. Dellon AL. The four medial ankle tunnels: a critical review of perceptions of tarsal tunnel syndrome and neuropathy. Neurosurg Clin N Am. 2008;19(4):629–48, vii.

Medial Calcaneal Nerve Entrapment

77

Michael N. Brown, Beth S. Pearce, Thais Khouri Vanetti, Andrea M. Trescot, and Helen W. Karl

Introduction

Patients with heel pain are common in clinical practice [1]. The *medial calcaneal nerve* -(MCN) also called the *calcaneal nerve* [2] or the *anterior calcaneal nerve* [3]) is one of several nerves that can be trapped at the ankle and heel; others include the *lateral plantar nerve* (LPN, Chap. 74), *inferior calcaneal nerve* (ICN, Chap. 76), and the *medial plantar nerve* (MPN, Chap. 75). The MCN is the nerve second most commonly reported to be involved in patients with plantar heel pain of neural origin [4], although dysfunction severe enough to require surgical relief is relatively rare (0.03 % in one series) [2]. Heel pain may be from a variety of other more common sources including *plantar fasciitis*, problems with the heel fat pad, and some rheumatologic diseases [1]. Entrapment neuropathies often present as heel pain that mimics that of plantar fasciitis, so misdiagnosis is not uncommon.

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Clinical Presentation (Table 77.1)

Patients with MCN entrapment have sharp "neuralgic" pain (Fig. 77.1), with burning and occasional tingling or sensory disturbances in the distribution of the MCN (Fig. 77.2). They often describe pain that worsens during the day or the longer they stand or walk. Pain may be worse at night. Night pain is thought to be due to slowing of venous outflow, resulting in edema that increases pressure on the nerve [4], although in one series, night pain was rare [2].

It is useful to make the distinction between neurogenic heel pain and plantar fasciitis, when possible. A patient with plantar fasciitis usually experiences the worst pain on the first several steps in the morning or after rest ("*first-step pain,*" also called *post-static dyskinesia* [17]). The pain of plantar fasciitis then gradually improves with activity and usually does not occur at night. Post-static dyskinesia is not limited to plantar fasciitis [18].

Table 77.1 Occupation/exercise/trauma history relevant to medial calcaneal nerve entrapment

Trauma	Previous foot or ankle surgery [5–7]
	Excessive calcaneal eversion [8]
	Playing soccer [3]
	Stepping on an object [8]
Intrinsic foot anomaly	Varus heel deformity with pronated forefoot; valgus heel with abducted forefoot [9]
	Fat pad atrophy [10]
Abductor hallucis hypertrophy [9]	Pes planus (flat foot) [11]
	Hallux valgus [12]
	Running or jogging [13]
	High protein intake [14]
Compression	Ganglion cyst
	Neuroma [15]
	Obesity [14]
	Increased foot pronation
	Midtarsal joint laxity
	External compression, such as OB stirrups [16]

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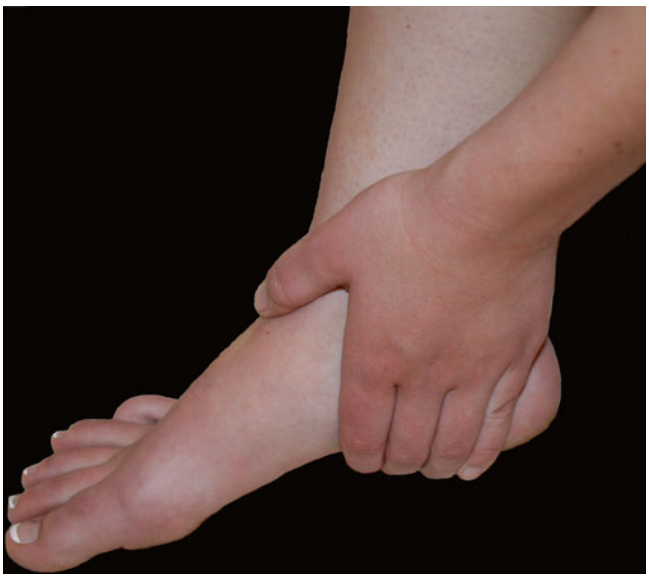

Fig. 77.1 Patient pain complaint of heel pain from medial calcaneal nerve entrapment (Image courtesy of Andrea Trescot, MD)

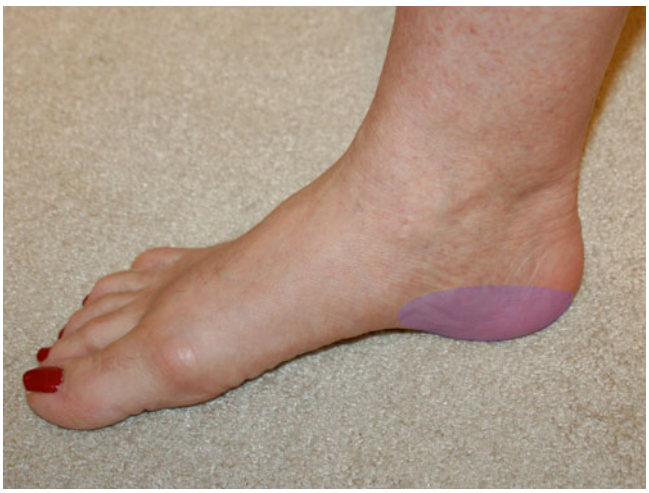

Fig. 77.2 Pattern of pain from medial calcaneal nerve entrapment (Image courtesy of Andrea Trescot, MD)

Repetitive microtrauma can cause MCN injury in athletes. Heel pain in runners is often due to plantar fasciitis with or without ICN dysfunction, whereas soccer players, with the sudden changes in direction required by that sport, are more likely to have MCN dysfunction due to the shoe pressing against the anteromedial part of the heel pad [3]. Biomechanical assessment of the foot and ankle will often demonstrate intrinsic foot abnormalities that cause increased foot pronation.

MCN entrapment can coexist with compression of the tibial nerve at a variety of levels (double-crush syndrome, Chap. 1) or simultaneous entrapment of other PTN branches, most commonly the MPN [19, 20]. This likely explains the heel pain that was prominent in the original description of MPN entrapment, a symptom that would not be expected with MPN entrapment alone [21].

The clinical picture of MCN entrapment is often multifaceted. A patient with a previous diagnosis of "plantar fasciitis," who has failed conservative management and has pain that worsens at night may instead have MCN entrapment neuropathy.

Anatomy (Table 77.2)

The sciatic nerve travels down the leg and splits into the peroneal and tibial nerves near the popliteal fossa. The tibial nerve at the ankle is often called the posterior tibial nerve (PTN, Chap. 73). It enters the proximal *tarsal tunnel* (deep to the flexor retinaculum) at the medial ankle where it divides into the *lateral plantar nerve* (LPN, Chap. 74), the *medial plantar nerve* (MPN, Chap. 75), and the MCN (Fig. 77.3). A change in girth of any structure that crosses this confined area can cause a compressive neuropathy.

While most of the nerves at the medial ankle and heel are variable in their courses, the MCN is particularly diverse [28, 30]. More than 70 % of the time (see Table 77.2), the MCN arises from the PTN within or proximal to the tarsal tunnel [5, 9, 22, 24, 28]. When the MCN branches from the main trunk of the PTN, it is superficial to the *abductor hallucis muscle* (AbH) and may go through or over the flexor retinaculum (as seen in Figs. 77.4 and 77.5). When it arises from the LPN, it goes through the tarsal tunnel, travels deep to the AbH, and becomes superficial at the inferior AbH border [9]. The MCN and its branches do not enter the plantar vault or the deep structures of the foot [22, 23]. Compared to the ICN, the MCN is posterior, more superficial, and thicker [22].

The MCN usually divides into two major branches: an anterior one to the proximal AbH and a posterior one to the skin overlying the medial aspect of the Achilles tendon, heel, and plantar fat pad (see below) [3, 5]. The relatively constant anterior terminal branch follows the contour of the medial aspect of the *medial calcaneal tubercle* (MCT) and innervates the superficial inferior heel (Fig. 77.5). It is often called the *cutaneous plantar nerve* [5] or the *anterior calcaneal nerve* [3].

The MCN and its branches do not innervate the skin of the medial ankle; the tibial nerve branches that innervate that region leave the main nerve about mid-calf [31].

Table 77.2 Medial calcaneal nerve anatomy

Origin	L4-S4 ventral rami form the sciatic nerve
General route	The sciatic nerve divides in the distal thigh into the tibial (Chap. 73) and peroneal (fibular, see Chap. 67) nerves, and the tibial nerve (TN) continues down through the deep posterior compartment of the leg
	The TN divides within or occasionally near the tarsal tunnel (TT) into the lateral plantar (LPN), medial plantar (MPN), and medial calcaneal (MCN) nerves
	The MCN runs postero-inferiorly, often through the flexor retinaculum, to end in the skin of the heel and the fat pad over the calcaneus [22], with an overall direction from deep to superficial [5, 9]
	The MCN and its branches are usually superficial to the abductor hallucis muscle (AbH) [5], and it does not enter the plantar vault or the deep structures of the foot [22, 23]; compared to the ICN, the MCN is posterior, more superficial, and thicker [22]
	The MCN usually divides into 2 major branches, an anterior one to the proximal abductor hallucis and a posterior one to the superficial soft tissues [3, 5]
	A relatively constant (15/15) terminal branch innervates the superficial inferior heel and is often called the cutaneous plantar nerve [5]
Sensory distribution	Medial aspect of the Achilles tendon, flexor retinaculum, medial heel, medial sole, and plantar fat pad [22]
Motor innervation	*Abductor hallucis muscle* (*AbH*) [5, 22]: from the distal flexor retinaculum, medial calcaneal tubercle, and the plantar aponeurosis to the base of the great toe; it flexes and abducts the great toe; the AbH is also innervated by the MPN and sometimes by a branch of the ICN [2, 24]
Anatomic variability	*MCN nerve of origin*: from the PTN in 100 % [25], 81 % (13/16) [22], 73 % (88/120) [9], 73 % (8/11) [26]
	From the LPN in 27 % (32/120) [9], 27 % (3/11) [26], 20 % (3/15) [5], 19 % (3/16) [22], 10 % (2/20) [24]; when the MCN is a branch of the LPN, it crosses deep to the AbH to appear from its inferior border distal to the flexor retinaculum [9]
	From the MPN in 46 % (39/85 operative notes) [27], 15 % (3/20) [24]
	MCN level of origin: proximal to the TT in 40 % (8/20), one of two branches proximal to the TT in 25 % (5/20), within the TT in 35 % (7/20) [28]; up to 5 cm [22] or 10 cm [5, 24] proximal to the PTN bifurcation
	Number of MCN terminal branches: one in 13 % (2/15) [5] – 36 % (31/85) [27], two in 41 % (35–85) [27] – 60 % (9/15) [5], three in 13 % (2/15) [5] – 19 % (16/85) [27], and four in 4 % (3/85) [27] – 13 % (2/15) [5]
	Relationship to the AbH: superficial to the AbH when MCN is a branch of the PTN; deep to the AbH when MCN is a branch of the LPN [9]
Other relevant structures	Plantar fascia (plantar aponeurosis): a strong, superficial layer of fascia (3 cords [29]) that runs from the calcaneal tuberosity to the toes; posteriorly, it is deep to but near the MCN and its branches
	Medial calcaneal tubercle (medial process of the calcaneal tuberosity, MCT): the origin of the central band of plantar fascia, AbH, FDB, and, more laterally, the AbDM

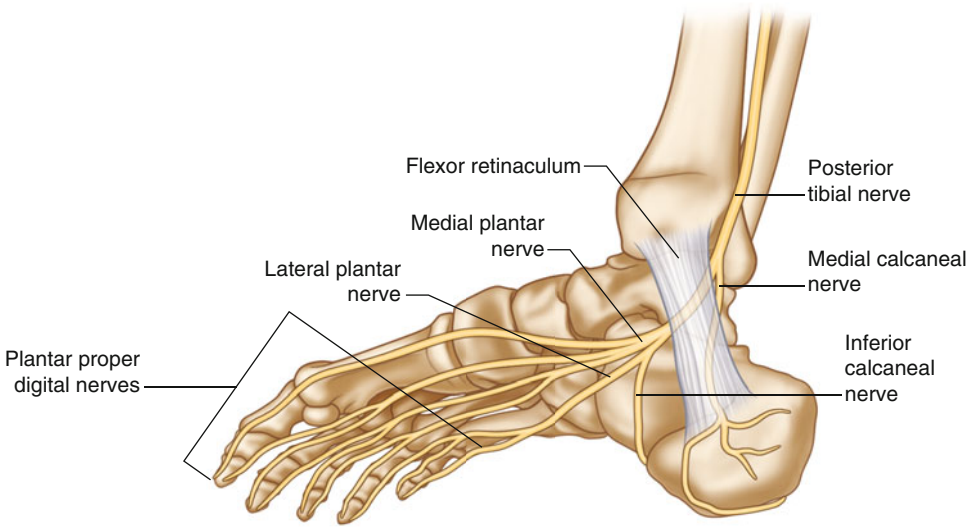

Fig. 77.3 Anatomy of the branches of the posterior tibial nerve. Note the relatively constant anterior terminal branch of the medial calcaneal nerve (often called the cutaneous plantar nerve [5]) on the medial face of the medial calcaneal tubercle (MCT) and the inferior calcaneal nerve crossing anterior and deep to the MCT (Image courtesy of Springer)

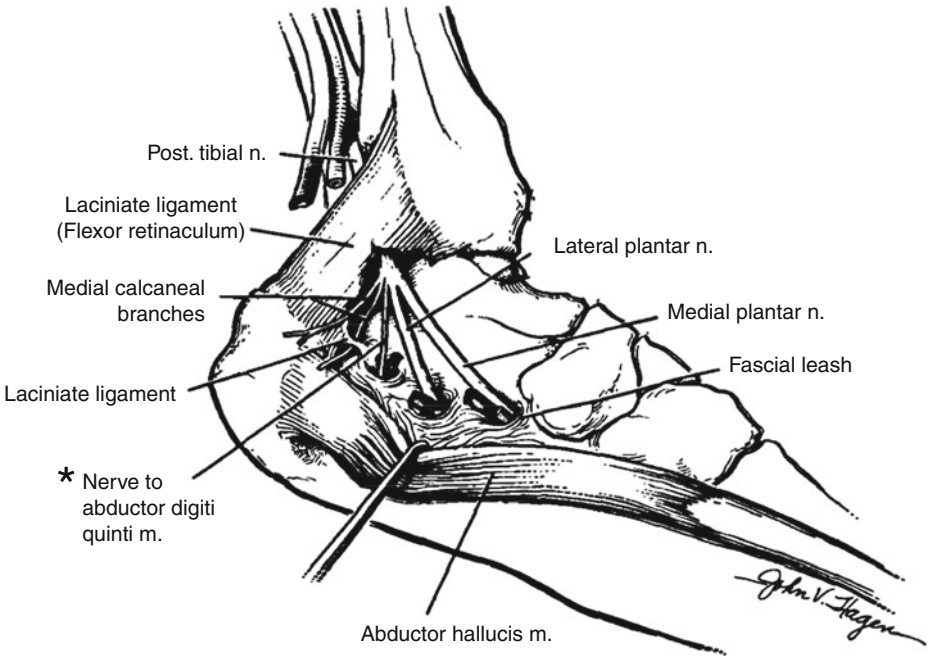

Fig. 77.4 Anatomy of the branches of the posterior tibial nerve. Note the medial calcaneal branches (nerves) as they cross the lancinate ligament (flexor retinaculum) to become more superficial. Note also the nerve to the abductor digiti quinti muscle (inferior calcaneal nerve) as it migrates under the deep fascia of the abductor hallucis and the lateral and medial plantar nerves as they cross the thick deep fascia of the abductor hallucis muscle (From Baxter [43]. Reprinted with permission from SAGE Publications)

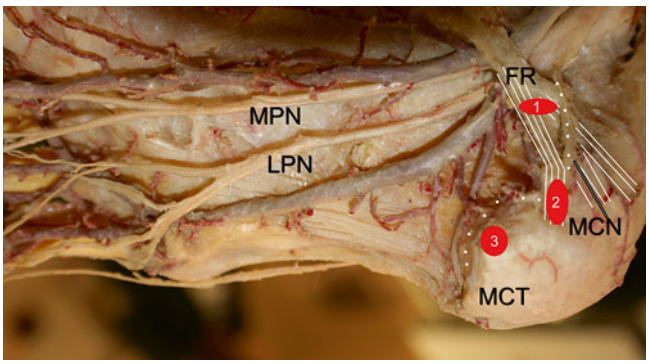

Fig. 77.5 Dissection of the medial ankle, from *Bodies*, *The Exhibition*, with permission. Circles represent possible sites of entrapment. Note the medial calcaneal nerve coming off *proximal to* the flexor retinaculum and the cutaneous plantar nerve (anterior terminal branch) following the contour of the calcaneus to the medial aspect of the medial calcaneal tubercle. Note also the position of the posterior tibial nerve and its branches deep to the blood vessels. The abductor hallucis muscle has been removed. *FR* flexor retinaculum, *MCN* medial calcaneal nerve (*white dots*), *MCT* medial aspect of the medial calcaneal tubercle, *LPN* lateral plantar nerve, *MPN* medial plantar nerve (Image courtesy of Andrea Trescot, MD)

Ultrasound Anatomy of the Medial Calcaneal Nerve

On ultrasound, the MCN is an approximately 1 mm structure that can be seen to separate from the TN as the probe is moved caudally (Fig. 77.6). Its variable branches travel obliquely, posteriorly, and superficially. It is important not to confuse the MCN with the ICN, which is vertical at this level, heading deep into the foot [32].

Entrapment

When the MCN travels in the proximal tarsal tunnel (under the flexor retinaculum) [28], it may be entrapped in the tunnel or at a site where it exits the tunnel through the flexor retinaculum (Fig. 77.5, circle 1).

When the MCN arises from the LPN, it travels deep to the AbH. Thus, AbH hypertrophy could entrap the MCN when it is a branch of the LPN (Fig. 77.5, circle 2) [9].

The anterior branch of the MCN can be entrapped where it passes through the deep fascia of the AbH origin at the medial corner of the MCT (Fig. 77.5, circle 3). Surgical release at this site was highly (90 %) successful in one series [3].

Physical Examination

Many structures at the medial heel and tarsal tunnel can be identified using surface anatomy (Fig. 77.7). Begin the physical examination of the heel by palpating the *heel pad* (Fig. 77.8, circle 1), then the central band of the *plantar fascia* to check for distal fasciitis, and then the origin of the plantar fascia at the deep MCT for proximal fasciitis (Fig. 77.8, circle 2). Palpate the distal *Achilles tendon* (*calcaneal tendon*) and *retrocalcaneal bursa*, then squeeze the heel to evaluate a potential stress fracture (Fig. 77.8, circle 4). Ask the patent to widely abduct all their toes, and compare the strength of the great and little toes with the other side. If the ICN is also involved (Fig. 77.8, circle 3), the abductor digiti minimi (AbDM) may be weaker on the affected side (Chap. 76).

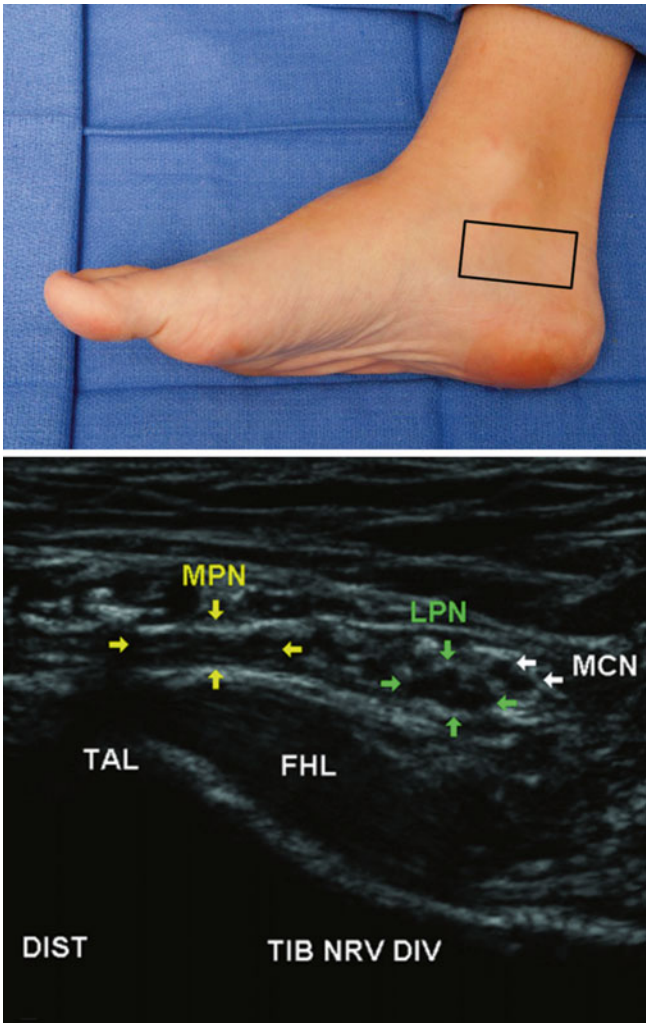

Fig. 77.6 Short-axis sonographic view of the medial calcaneal nerve. *MPN* medial plantar nerve (*yellow arrows*), *LPN* lateral plantar nerve (*green arrows*), *MCN* medial calcaneal nerve (*white arrows*), *FHL* flexor hallucis longus tendon, *TAL* medial talus. Two branches of the MCN are distinguishable and should not be confused with the ICN. MCN branches travel obliquely, superficially, and posteriorly; the ICN (not seen in this image) is vertical at this level and heads toward the *AbH-QP interval* (*abductor hallucis-quadratus plantus interval*) (From Presley et al. [32]. Reprinted with permission from American Institute of Ultrasound in Medicine)

Patients with MCN dysfunction may be tender along the anteromedial calcaneus, especially near the origin of the AbH (Fig. 77.9). The MCN can be tender at its entrapment sites (Fig. 77.5, circles 1, 2, and 3) and along its entire course from the medial malleolus to the medial MCT [2]. Paresthesias may be experienced with palpation over the AbH. Attempt to elicit Tinel's sign at the proximal and distal tarsal tunnel, as well as at the proximal AbH.

Sensory examination of the foot should be compared to the contralateral side (Fig. 77.10). In one study, Rose et al. documented sensory impairment in 72 % of heels in the distribution of the MCN or the MCN in combination with the MPN [20]. Local anesthetic block of the MCN, with or without ultrasound (US) guidance, can help make the diagnosis (see below). ICN dysfunction is not associated with changes in skin sensibility, so the absence of any sensory changes makes ICN dysfunction more likely [27].

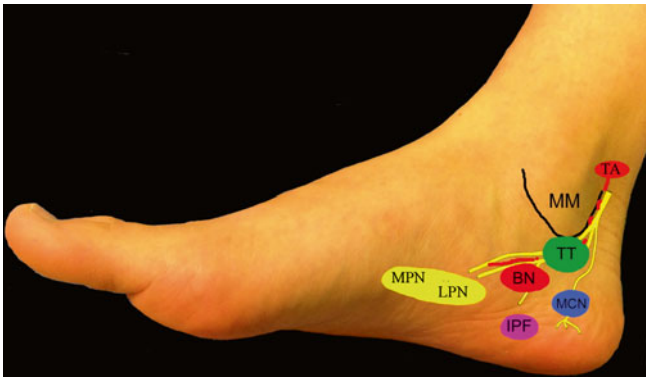

Fig. 77.7 Surface anatomy of the structures at the medial heel. *MM* medial malleolus, *TT* proximal tarsal tunnel, *TA* tibial artery, *MCN* medial calcaneal nerve, *BN* Baxter's nerve = inferior calcaneal nerve, *IPF* insertion of plantar fascia, *LPN* lateral plantar nerve, *MPN* medial plantar nerve (Image courtesy of Michael Brown, MD)

Differential Diagnosis (Tables 77.3 and 77.4)

The most common causes of plantar heel pain are *plantar fasciitis*, *heel fat pad atrophy*, *calcaneal stress fracture*, and nerve entrapments [1, 36]. The presence of bilateral pain should trigger investigation into an inflammatory source, and L5-S1 radiculopathy may be the cause in a patient with coexisting back pain [34].

A patient with heel pain from MCN entrapment may be tender near the MCT, similar to someone with plantar fasciitis, although the pain and tenderness may be more medial than plantar. They may have symptoms of neurogenic heel pain mixed with objective findings of plantar fasciitis, such as hypoechoic and thickened (>4 mm) fascia on US [37]. However, the presence of plantar fasciitis does not rule out an entrapment neuropathy, since the two conditions may coexist [38].

Fat pad atrophy is the second most common cause of plantar heel pain [39]. It is more common in older patients, particularly those with sudden weight loss or diabetes [40].

Evaluation of MCN dysfunction can be particularly difficult in the setting of coexisting small fiber neuropathy, as seen with diabetes mellitus.

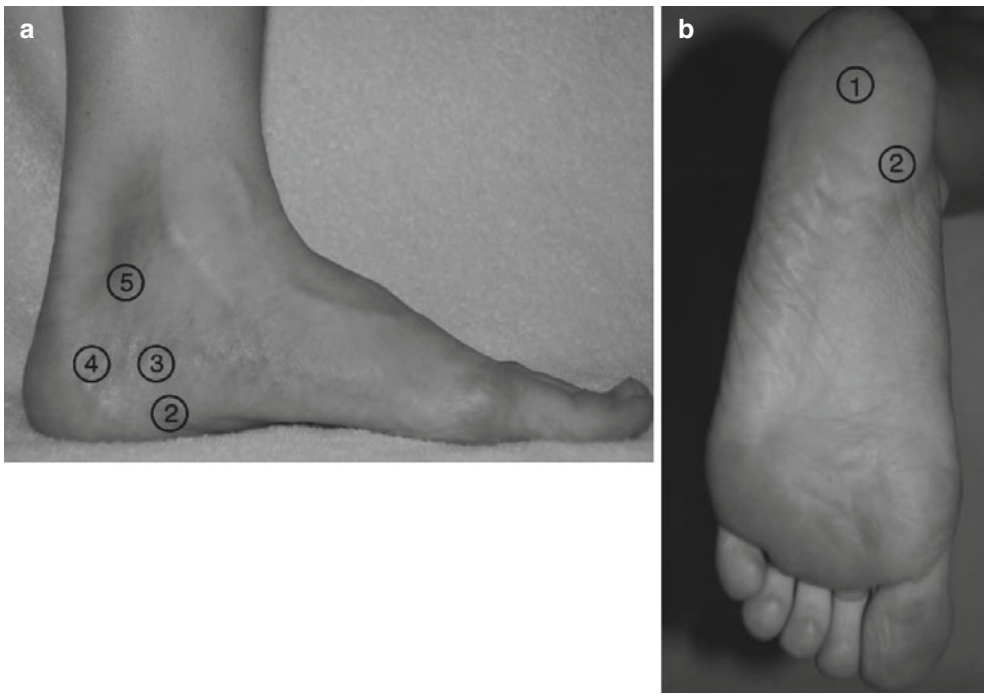

Fig. 77.8 Locations of tenderness for 5 diagnoses of heel pain: *1* heel pad atrophy, *2* plantar fasciitis, *3* ICN entrapment, *4* calcaneal stress fracture, *5* tarsal tunnel syndrome. The tenderness from MCN entrapment is between *circles 3* and *4* (From Lareau et al. [36]. Reprinted with permission from Wolters Kluwer Health)

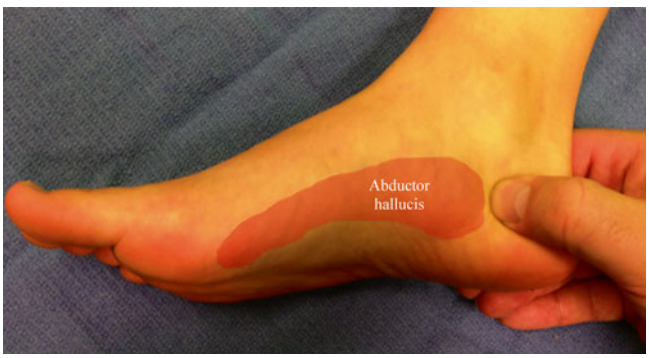

Fig. 77.9 Palpation of the medial calcaneal nerve at the proximal abductor hallucis muscle (Image courtesy of Michael Brown, MD)

Diagnostic Tests (Table 77.5)

Weight-bearing plain radiographs can help assess foot alignment and exclude fractures or joint degeneration [36].

US and MRI can show tenosynovitis, bursitis, masses, and nerve injuries. US is widely available, inexpensive, and quick; however, image quality is operator dependent and may be limited in the presence of dense scarring [44]. High-resolution MRI can show the structure of the nerves themselves (Fig. 77.11) and can be useful when planning interventions [45].

Electrodiagnostic testing can be helpful in identifying MCN entrapment [2]. Nerve conduction velocity (NCV) tests are part of a standard neurologic workup. Seo and Oh [46] described a "near-nerve" needle technique to evaluate orthodromic NCV and negative-peak NCV of the MCN; three of the four cases of MCN they studied were confirmed by this technique. Chang et al. [38] evaluated 26 patients with a clinical and US diagnosis of plantar fasciitis, as well as 30 control patients; 6 patients were identified by NCV to have MCN neuropathy.

The *pressure-specified sensory device* (PSSD) is an objective, nonpainful method to measure nerve function; the pressure required for a person to distinguish one from two static points is the most sensitive gauge of nerve dysfunction from chronic compression [47]. Data for the MPN (big toe) and MCN (heel) has been recently reported in normal subjects and patients diagnosed with *tarsal tunnel syndrome* [48], and it can be a useful adjunct to diagnosis and treatment [20, 49]. In one series, more than 70 % (70/97 ft) of patients with plantar heel pain evaluated with the PSSD showed abnormalities of the MCN [20].

Identification and Treatment of Contributing Factors

An intrinsic foot anomaly such as midtarsal joint laxity, forefoot varus, rear foot eversion, pes planus, and cavovarus foot may contribute to increased foot pronation. Differences in foot anatomy may be aggravated by obesity, although no significant correlation between BMI and MCN dysfunction has been identified [20]. As with plantar fasciitis, correcting the passive mechanics of the foot with orthotics may be helpful. Proper shoe wear and stretching of the plantar fascia and

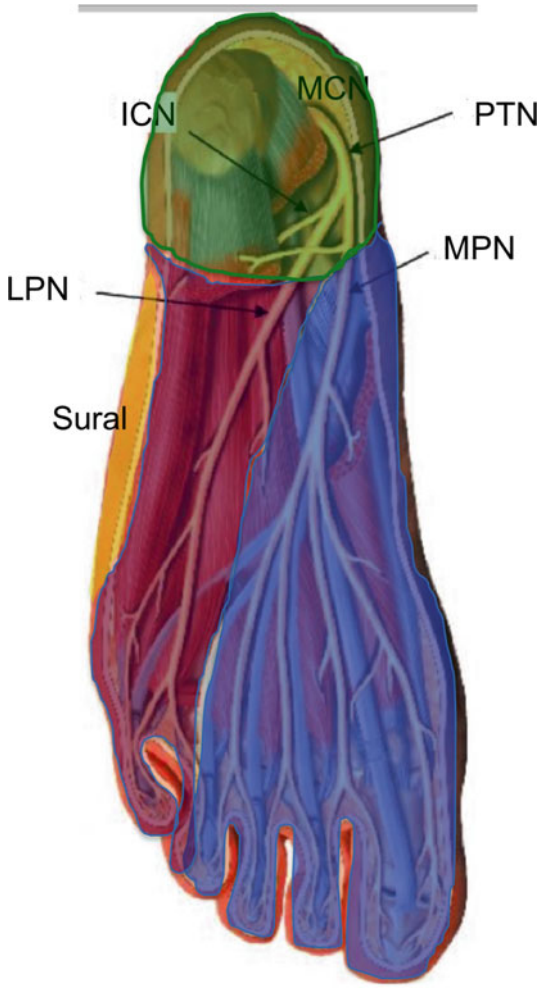

Fig. 77.10 Sensory map of the calcaneal and plantar nerves. *MCN* medical calcaneal nerve (*green*), *PTN* posterior tibial nerve, *MPN* medial plantar nerve (*blue*), *ICN* inferior calcaneal nerve (note the lack of cutaneous territory), *LPN* lateral plantar nerve (*red*), *sural* sural nerve (*orange*) (Image courtesy of Michael Brown, MD)

Achilles tendon will reduce stress on the ligaments and muscles. Weight reduction and physical therapy to strengthen the foot flexor and posterior tibialis muscles are additional important strategies [50].

Injection Technique

Landmark-Guided Injections

Anesthesiologists have traditionally done landmark-guided, large-volume blocks of the nerves around the foot and ankle for surgery. However, for pain evaluation and treatment, it is important to perform a diagnostic injection with a low volume of local anesthetic to prevent spillover to multiple nerves and soft tissues, which would interfere with interpretation of the injection. This is especially important when planning for

Table 77.3 Clinical comparison of medial calcaneal nerve entrapment and plantar fasciitis

Medial calcaneal nerve entrapment	Plantar fasciitis
Pain is worse after activity and at night	"First-step" pain: worse first thing in the morning or after a period of inactivity
Pain gets worse with walking	Pain gets better with walking
"Neuralgic" pain: sharp and burning	"Mechanical" pain: a deep ache
Maximum tenderness: over the anteromedial heel pad and "behind" the AbH; pressure there may cause pain to radiate toward the plantar heel	Maximum tenderness at the insertion of the plantar fascia and along its medial edge where thickened fascia may be felt
May have decreased sensation over the heel pad	Heel pad sensation should be like that on the contralateral side

Table 77.4 Differential diagnosis of heel pain

Plantar heel pain	Medial heel pain	Other
Plantar fasciitis [8, 15]	Flexor digitorum longus; tenosynovitis [33]	Rheumatoid arthritis [8, 15], especially if bilateral [34]
Plantar fascia tear	Flexor hallucis longus; tenosynovitis	Reiter's syndrome
Heel pad atrophy [8]	Tibialis posterior tenosynovitis	Ankylosing spondylitis [15]
Tarsal tunnel syndrome, though the heel may be spared if the MCN branches proximally	Tarsal tunnel syndrome	Psoriatic arthritis
Inferior calcaneal nerve entrapment, dysfunction, or neuroma [34, 35]	Medial calcaneal nerve entrapment, dysfunction, or neuroma [15]	Gout [8, 15]
Calcaneal stress fracture [15, 36]	Inferior calcaneal nerve entrapment	Posterior enthesopathies
Calcaneal cyst, tumor, osteomyelitis, or edema	Retrocalcaneal bursitis [35]	Behçet's syndrome
Calcaneal apophysitis (Sever's disease)		Systemic lupus erythematosus
Heel spur [15]		Fibromyalgia
Vascular insufficiency, congestion, or varicosities [35]		L5-S1 radiculopathy [34]

a neurolytic or surgical procedure. Consider using a nerve stimulator with successive approximation to provide better accuracy in targeting the injection.

The patient can be positioned prone, supine, or on their side. The authors prefer to have the patient lying on the symptomatic side with the medial foot facing upward. A 27-gauge, 1.5-in. needle is best, although a 25-gauge, 1.5-in. or a 30-gauge, 1-in. needle can also be used. Place a finger just behind the medial malleolus, then slide it down the posterior aspect of the malleolus until it lies over the middle of the calcaneus, still in line with the posterior malleolus (Fig. 77.9). Feel for the slight flair of the calcaneus, the MCT. After skin anesthesia with a 30-gauge, 0.5-in. needle, direct the chosen procedure needle straight down, perpendicular to the skin, toward the calcaneus, until it contacts bone (Fig. 77.12). Withdraw the needle 1–2 mm, and inject 0.5–1.0 cc of local anesthetic. Next, pull back a few more millimeters and inject another 0.5 cc of local anesthetic. This will place the anesthetic both deep and superficial to the

Table 77.5 Diagnostic tests for medial calcaneal nerve entrapment

	Potential distinguishing features
Physical exam	Primary diagnostic modality
	Tinel's sign [8]
	Pain and tenderness over the medial calcaneus
	Sensory deficit over plantar heel
Diagnostic injection	Diagnostic as well as potentially therapeutic
Ultrasound	Can diagnose mass lesions and guide injections
	MCN is difficult to see on US [41]
MRI	High-resolution sagittal MRI shows MCN [41, 42]
	Can demonstrate plantar fasciitis
Arteriography	Not useful
X-ray	To rule out bony causes of heel pain such as calcaneal fracture or tumors
	Heel spurs are nonspecific: they are often present in asymptomatic patients [43]
Electrodiagnostic studies	May help to diagnose and quantify the degree or severity of the entrapment neuropathy
	The predictable and superficial course of the middle branch of the MCN make it more accessible than the ICN [5]

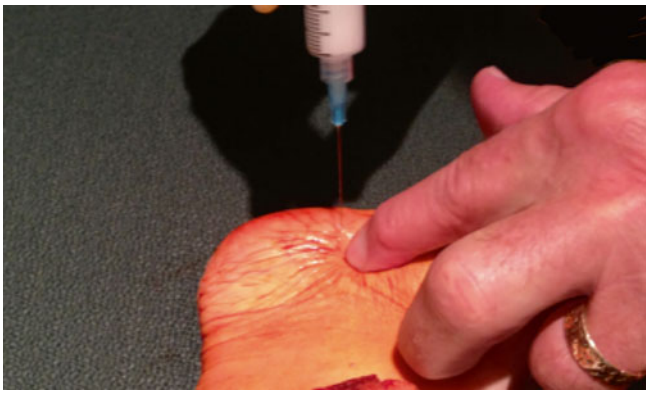

Fig. 77.12 Landmark-guided medial calcaneal nerve injection (Image courtesy of Gabor Racz, MD)

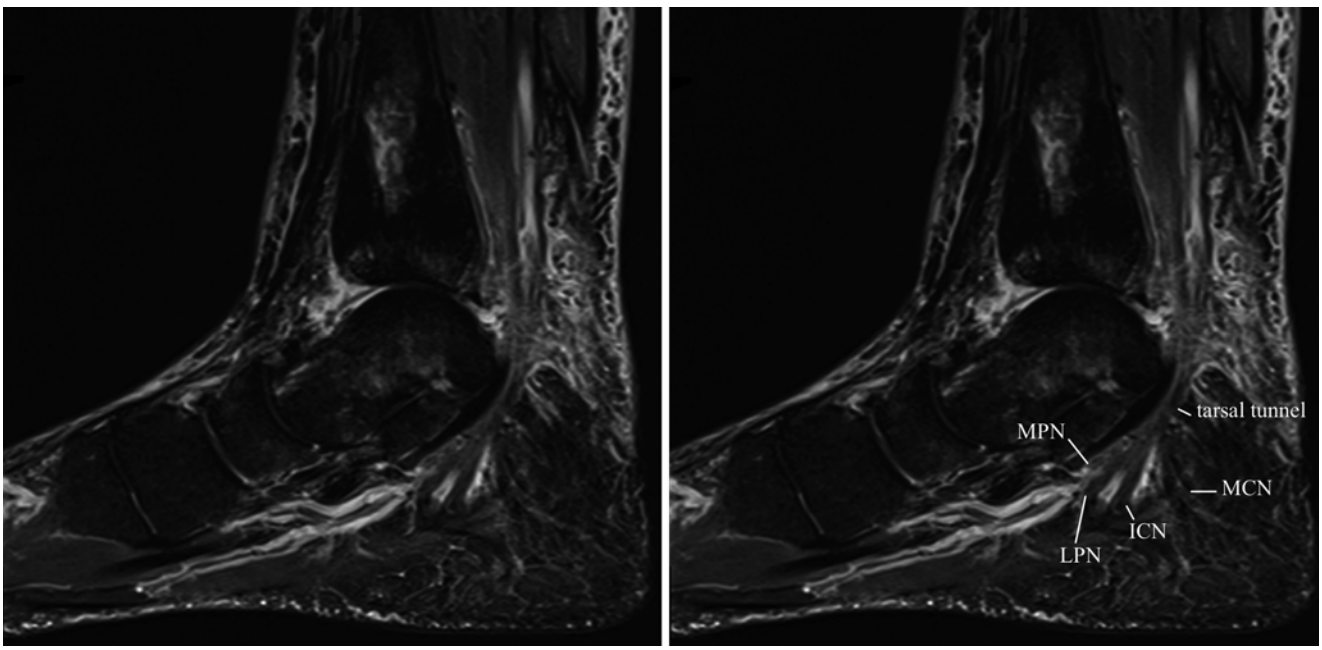

Fig. 77.11 STIR sagittal MRI of the medial malleolus showing the tarsal tunnel. *MCN* medial calcaneal nerve, *ICN* inferior calcaneal nerve, *LPN* lateral plantar nerve, *MPN* medial plantar nerve (Image courtesy of Andrea Trescot, MD)

flexor retinaculum and near the MCN. Allow sufficient time for anesthetic to take effect, and check for a sensory block in the distribution of the MCN (Fig. 77.10).

Fluoroscopy-Guided Injection

In the authors' opinion, fluoroscopy provides no benefit for MCN injections.

Ultrasound-Guided Medial Calcaneal Nerve Injection

Due to the small size of its multiple branches, it is difficult to see a normal MCN on US [41], and it may be confused with the ICN (Fig. 77.6) [32]. MCN branches travel obliquely, superficially, and posteriorly; the ICN (not seen in Fig. 77.6) is vertical at this level and heads toward the *AbH-QP interval* (*abductor hallucis-quadratus plantus interval*). Since there are no major vascular structures near the MCN, a landmark-guided injection may be sufficient.

After positioning similar to that for the landmark-guided injection (see above), place a linear transducer in the position used to see the proximal PTN, and confirm the vein-artery-vein (VAV) relationship to the PTN (Chap. 73). Move the transducer caudad so that the end is approximately at the mid calcaneus, and rotate it clockwise (if imaging the left heel) or counterclockwise (if imaging the right heel). The nerve in the most anterior position is the MPN; the middle nerve, lying behind the most posterior vein, is the LPN; and the third nerve, the MCN, lies over the calcaneus (Fig. 77.13).

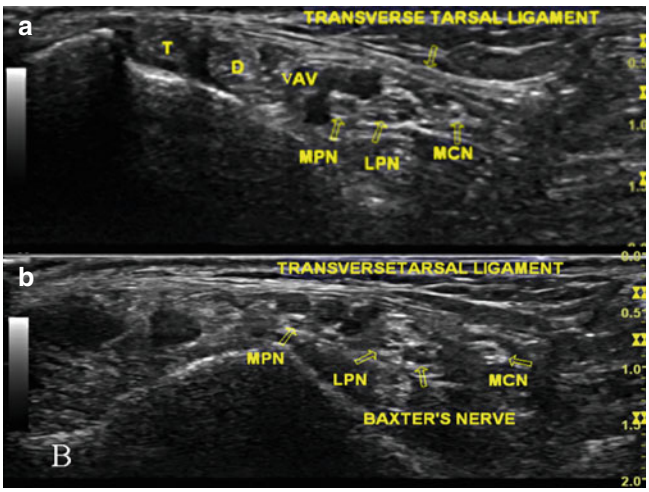

Fig. 77.13 Ultrasound location of the medial calcaneal nerve. *T* posterior tibialis tendon, *D* flexor digitorum longus tendon, *AV* posterior artery and vein, *MPN* medial plantar nerve, *LPN* lateral plantar nerve, *MCN* medial calcaneal nerve, *transverse tarsal ligament* flexor retinaculum (Image courtesy of Michael Brown, MD)

An out-of-plane injection (Fig. 77.14) approach may be preferable due to the superficial location of the nerve and its proximity to the calcaneus that will block the US signal. Attach a small syringe filled with normal saline to a 27-gauge, 1.5-in. needle, and inject 0.1–0.2 cc aliquots of saline to hydrodissect the tissue planes for better visualization of the anatomy and the needle tip. A small volume of saline is better than local anesthetic, which can confound a diagnostic block. Advance the needle in the fascial plane between the periosteum and subcutaneous tissues thus created, and inject 1–1.5 cc of therapeutic mixture. Perform a sensory examination before and after the injection to confirm the block of the MCN (Fig. 77.10).

For an in-plane approach to the MCN (Fig. 77.15), palpate the MCT, and place the transducer transversely over the AbH and FHB so that the lateral edge is just in front of the MCT, transversely overlying both the AbH and the FHB, and then move posteriorly.

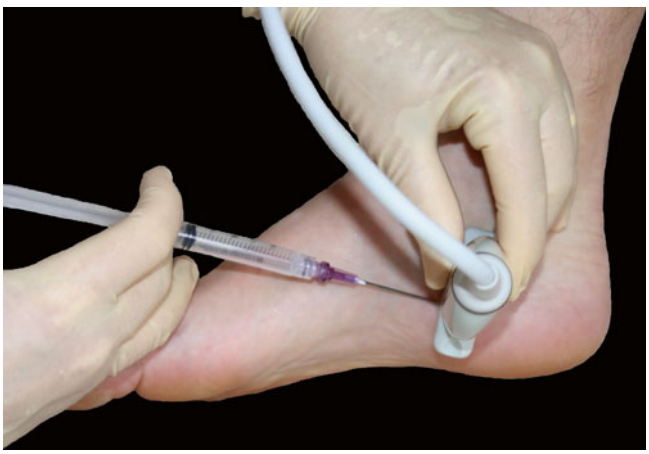

Fig. 77.14 Ultrasound for out-of-plane injection of the medial calcaneal nerve (Image courtesy of Andrea Trescot, MD)

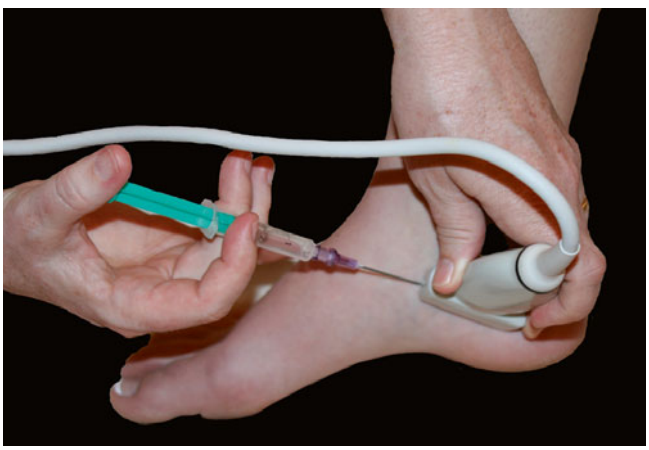

Fig. 77.15 Ultrasound for in-plane injection of the medial calcaneal nerve (Image courtesy of Andrea Trescot, MD)

Neurolytic Technique

Cryoneuroablation

Cryoneuroablation of the MCN is most easily performed just posterior to the MCT where the nerve can be palpated, assuming that a small-volume diagnostic injection has given good, but only temporary, relief [51]. After a sterile prep and drape, inject subcutaneous local anesthetic just cephalad to the MCT. A 27-gauge needle is advanced from cephalad to caudad, injecting about 1 cc of saline with epinephrine 1:200,000 along the periosteum and probe path. The introducer and then the probe are advanced to the edge of the medial calcaneus, and the nerve stimulator or US is used to locate the nerve (Fig. 77.16).

Radiofrequency Lesioning (RF)

Pulsed radiofrequency of the MCN has been reported to give prolonged relief from pain due to plantar fasciitis [52–54]. Review of these papers highlights some of the challenges of diagnosis and treatment of heel pain.

Surgery

Henricson and Westlin [3] reported entrapment of the *anterior branch of the MCN* in 10 athletes with 11 painful heels. Ten of the 11 heels were asymptomatic 58 months after surgical release, and patients returned to athletic participation an average of 5 weeks postoperatively.

Failures of tarsal tunnel surgery may be due to an untreated MCN entrapment. Yalcinkaya et al. [55] described eight patients with pain more than 12 months after tarsal tunnel release. All underwent open release of the MCN and ICN, as well as the MPN and LPN, with "good" to "excellent" results in 87.5 %.

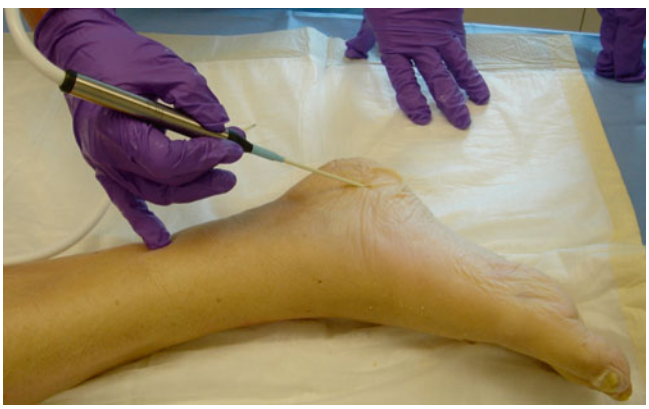

Fig. 77.16 Cryoneuroablation of the medial calcaneal nerve (Image courtesy of Andrea Trescot, MD)

Complications

As with any procedure, there are risks of infection, hematoma, vascular puncture, or nerve injury. The most common associated problem is incomplete nerve release or ablation leading to increased pain. In this circumstance, repeating the procedure may lead to resolution of symptoms. The skin is very thin at this site, so injections or surgery can result in skin injury or atrophy.

Summary

Heel pain is a common condition with multiple causes. MCN entrapment neuropathy should be differentiated from plantar fasciitis and other causes when possible. A careful history and physical exam, along with diagnostic tests and blocks, can help clinicians recognize and treat MCN entrapment.

References

1. Thomas JL, Christensen JC, Kravitz SR, Mendicino RW, Schuberth JM, Vanore JV, et al. The diagnosis and treatment of heel pain: a clinical practice guideline-revision 2010. J Foot Ankle Surg. 2010;49(3 Suppl):S1–19.
2. Schon LC, Glennon TP, Baxter DE. Heel pain syndrome: electrodiagnostic support for nerve entrapment. Foot Ankle. 1993;14(3):129–35.
3. Henricson AS, Westlin NE. Chronic calcaneal pain in athletes: entrapment of the calcaneal nerve? Am J Sports Med. 1984;12(2):152–4.
4. Alshami AM, Souvlis T, Coppieters MW. A review of plantar heel pain of neural origin: differential diagnosis and management. Man Ther. 2008;13(2):103–11.
5. Louisia S, Masquelet AC. The medial and inferior calcaneal nerves: an anatomic study. Surg Radiol Anat. 1999;21(3):169–73.
6. Kim J, Dellon AL. Neuromas of the calcaneal nerves. Foot Ankle Int. 2001;22(11):890–4.
7. Gamie Z, Donnelly L, Tsiridis E. The "safe zone" in medial percutaneous calcaneal pin placement. Clin Anat. 2009;22(4):523–9.
8. Diers DJ. Medial calcaneal nerve entrapment as a cause for chronic heel pain. Physiother Theory Pract. 2008;24(4):291–8.
9. Ghosh SK, Raheja S, Tuli A. Potential sites of compression of tibial nerve branches in foot: a cadaveric and imaging study. Clin Anat. 2013;26(6):768–79.
10. Kopell HP, Thompson WA. Peripheral entrapment neuropathies of the lower extremity. N Engl J Med. 1960;262:56–60.
11. Jung DY, Koh EK, Kwon OY. Effect of foot orthoses and short-foot exercise on the cross-sectional area of the abductor hallucis muscle in subjects with pes planus: a randomized controlled trial. J Back Musculoskelet Rehabil. 2011;24(4):225–31.
12. Wong YS. Influence of the abductor hallucis muscle on the medial arch of the foot: a kinematic and anatomical cadaver study. Foot Ankle Int. 2007;28(5):617–20.

13. Prior BM, Yang HT, Terjung RL. What makes vessels grow with exercise training? J Appl Physiol (Bethesda, Md : 1985). 2004;97(3):1119–28.
14. Rankin JW. Weight loss and gain in athletes. Curr Sports Med Rep. 2002;1(4):208–13.
15. Plotkin D, Patel D, Roberto T. Heel neuroma: a case study. J Foot Ankle Surg. 2009;48(3):376–9.
16. Kamphuis VP, Zegers MP, Koppen H. Bilateral heel numbness due to external compression during obstetric epidural analgesia. Case Rep Neurol. 2015;7(1):15–7.
17. Jolly GP, Zgonis T, Hendrix CL. Neurogenic heel pain. Clin Podiatr Med Surg. 2005;22(1):101–13, vii.
18. Watson TS, Anderson RB, Davis WH, Kiebzak GM. Distal tarsal tunnel release with partial plantar fasciotomy for chronic heel pain: an outcome analysis. Foot Ankle Int. 2002;23(6):530–7.
19. Oh SJ, Lee KW. Medial plantar neuropathy. Neurology. 1987;37(8):1408–10.
20. Rose JD, Malay DS, Sorrento DL. Neurosensory testing of the medial calcaneal and medial plantar nerves in patients with plantar heel pain. J Foot Ankle Surg. 2003;42(4):173–7.
21. Rask MR. Medial plantar neurapraxia (jogger's foot): report of 3 cases. Clin Orthop Relat Res. 1978;134:193–5.
22. Didia BC, Horsefall AU. Medial calcaneal nerve. An anatomical study. J Am Podiatr Med Assoc. 1990;80(3):115–9.
23. Arenson DJ, Cosentino GL, Suran SM. The inferior calcaneal nerve: an anatomical study. J Am Podiatry Assoc. 1980;70(11):552–60.
24. Davis TJ, Schon LC. Branches of the tibial nerve: anatomic variations. Foot Ankle Int. 1995;16(1):21–9.
25. Rondhuis JJ, Huson A. The first branch of the lateral plantar nerve and heel pain. Acta Morphol Neerl Scand. 1986;24(4):269–79.
26. Kim BS, Choung PW, Kwon SW, Rhyu IJ, Kim DH. Branching patterns of medial and inferior calcaneal nerves around the tarsal tunnel. Ann Rehabil Med. 2015;39(1):52–5.
27. Dellon AL, Kim J, Spaulding CM. Variations in the origin of the medial calcaneal nerve. J Am Podiatr Med Assoc. 2002;92(2):97–101.
28. Dellon AL, Mackinnon SE. Tibial nerve branching in the tarsal tunnel. Arch Neurol. 1984;41(6):645–6.
29. Hoffman D, Bianchi S. Sonographic evaluation of plantar hindfoot and midfoot pain. J Ultrasound Med. 2013;32(7):1271–84.
30. Havel PE, Ebraheim NA, Clark SE, Jackson WT, DiDio L. Tibial nerve branching in the tarsal tunnel. Foot Ankle. 1988;9(3):117–9.
31. Aszmann OC, Ebmer JM, Dellon AL. Cutaneous innervation of the medial ankle: an anatomic study of the saphenous, sural, and tibial nerves and their clinical significance. Foot Ankle Int. 1998;19(11):753–6.
32. Presley JC, Maida E, Pawlina W, Murthy N, Ryssman DB, Smith J. Sonographic visualization of the first branch of the lateral plantar nerve (Baxter nerve): technique and validation using perineural injections in a cadaveric model. J Ultrasound Med. 2013;32(9):1643–52.
33. Schon LC. Nerve entrapment, neuropathy, and nerve dysfunction in athletes. Orthop Clin North Am. 1994;25(1):47–59.
34. Baxter DE, Pfeffer GB, Thigpen M. Chronic heel pain. Treatment rationale. Orthop Clin North Am. 1989;20(4):563–9.
35. Shandles ID, Pruchniewski J, Reynolds KL. Heel neuroma: the enigma of recalcitrant heel pain and an innovative approach highlighting sixty surgical cases and a review of two hundred and fifty-seven symptomatic but non-surgical cases. Foot. 2002;12(1):10–20.
36. Lareau CR, Sawyer GA, Wang JH, DiGiovanni CW. Plantar and medial heel pain: diagnosis and management. J Am Acad Orthop Surg. 2014;22(6):372–80.
37. Tsai WC, Chiu MF, Wang CL, Tang FT, Wong MK. Ultrasound evaluation of plantar fasciitis. Scand J Rheumatol. 2000;29(4):255–9.
38. Chang CW, Wang YC, Hou WH, Lee XX, Chang KF. Medial calcaneal neuropathy is associated with plantar fasciitis. Clin Neurophysiol. 2007;118(1):119–23.
39. Yi TI, Lee GE, Seo IS, Huh WS, Yoon TH, Kim BR. Clinical characteristics of the causes of plantar heel pain. Ann Rehabil Med. 2011;35(4):507–13.
40. Wrobel JS, Najafi B. Diabetic foot biomechanics and gait dysfunction. J Diabetes Sci Technol. 2010;4(4):833–45.
41. Fantino O. Role of ultrasound in posteromedial tarsal tunnel syndrome: 81 cases. J Ultrasound. 2014;17(2):99–112.
42. Farooki S, Theodorou DJ, Sokoloff RM, Theodorou SJ, Trudell DJ, Resnick D. MRI of the medial and lateral plantar nerves. J Comput Assist Tomogr. 2001;25(3):412–6.
43. Baxter DE, Thigpen CM. Heel pain–operative results. Foot Ankle. 1984;5(1):16–25.
44. Chalian M, Soldatos T, Faridian-Aragh N, Williams EH, Rosson GD, Eng J, et al. 3T magnetic resonance neurography of tibial nerve pathologies. J Neuroimaging. 2013;23(2):296–310.
45. Chhabra A, Subhawong TK, Williams EH, Wang KC, Hashemi S, Thawait SK, et al. High-resolution MR neurography: evaluation before repeat tarsal tunnel surgery. AJR Am J Roentgenol. 2011;197(1):175–83.
46. Seo JH, Oh SJ. Near-nerve needle sensory conduction study of the medial calcaneal nerve: new method and report of four cases of medial calcaneal neuropathy. Muscle Nerve. 2002;26(5):654–8.
47. Dellon AL, Keller KM. Computer-assisted quantitative sensorimotor testing in patients with carpal and cubital tunnel syndromes. Ann Plast Surg. 1997;38(5):493–502.
48. Tassler PL, Dellon AL. Pressure perception in the normal lower extremity and in the tarsal tunnel syndrome. Muscle Nerve. 1996;19(3):285–9.
49. Dellon AL. Deciding when heel pain is of neural origin. J Foot Ankle Surg. 2001;40(5):341–5.
50. McKeon PO, Hertel J, Bramble D, Davis I. The foot core system: a new paradigm for understanding intrinsic foot muscle function. Br J Sports Med. 2015;49(5):290.
51. Trescot AM. Cryoanalgesia in interventional pain management. Pain Physician. 2003;6(3):345–60.
52. Liden B, Simmons M, Landsman AS. A retrospective analysis of 22 patients treated with percutaneous radiofrequency nerve ablation for prolonged moderate to severe heel pain associated with plantar fasciitis. J Foot Ankle Surg. 2009;48(6):642–7.
53. Cione JA, Cozzarelli J, Mullin CJ. A retrospective study of radiofrequency thermal lesioning for the treatment of neuritis of the medial calcaneal nerve and its terminal branches in chronic heel pain. J Foot Ankle Surg. 2009;48(2):142–7.
54. Thapa D, Ahuja V. Combination of diagnostic medial calcaneal nerve block followed by pulsed radiofrequency for plantar fascitis pain: a new modality. Indian J Anaesth. 2014;58(2):183–5.
55. Yalcinkaya M, Ozer UE, Yalcin MB, Bagatur AE. Neurolysis for failed tarsal tunnel surgery. J Foot Ankle Surg. 2014;53(6):794–8.

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