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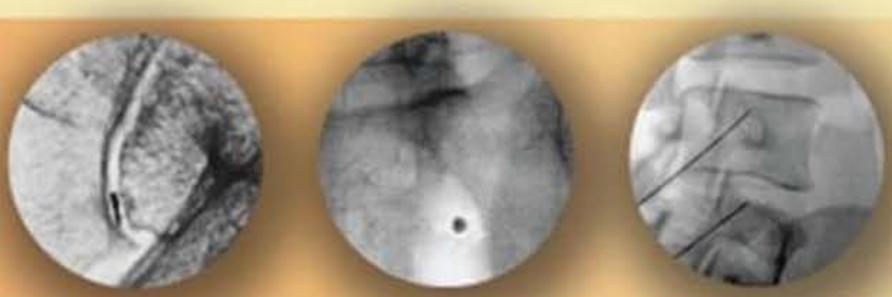

INTERVENTIONAL PAIN MANAGEMENT

Image-Guided Procedures

Second Edition

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Interventional Pain Management: Image-Guided Procedures

SECOND EDITION

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FOREWORD

It is a pleasure to write a Foreword for the second edition of Professor Raj's great book on interventional pain management. This world-renowned leader in pain treatment has again assembled a superb group of editors and contributors to create the definitive, authoritative text on interventional techniques for the management of acute and chronic pain. The book is profusely illustrated and is aimed at teaching physicians how to safely and effectively carry out the procedures that can be useful for pain management. Each chapter is written by an internationally recognized expert. This book is not a source of evidence-based medical studies; rather, it is a compendium of interventional strategies that have been found to be useful in properly selected patients.

Pain management began with the efforts of John J. Bonica, M.D. in the post-World War II era. He was able to launch pain research and management from his position as Professor and Chairman at the University of Washington and was the primary force behind the formation of IASP in 1975. As the field grew, many others earned leadership positions, but few achieved the successes of Prithvi Raj. His many publications reveal the importance of his work, and this book is the climax of those efforts. His friends and colleagues have all been willing contributors to his great efforts.

There are many approaches to the management of the patient with chronic or acute pain: pharmacologic, psychological, interventional, alternative medical, to name the most common. Each of these ways of treating patients has its advantages and disadvantages; patient selection is a critical part of deciding which strategies to employ for the patient's benefit. Knowing how to carry out a treatment strategy is also critically important, and that is the facet of pain management that this book addresses. There are numerous developments in our ability to image the human body during interventional procedures. These include ultrasound, fluoroscopy, CT scanning and the injection of agents that can be visualized on imaging studies. These advances are the cornerstone of this treatise on interventions for pain relief.

Professor Raj has devoted his career to the education and training of interventional pain specialists and this book is a glorious monument to his life-long educational efforts.

JOHN D. LOESER, M.D.
SEATTLE, WASHINGTON

Interventional Pain Management: Image-Guided Procedures is a remarkable publication completed by one of the marvelous minds of medicine in the modern world, Phulchand Prithvi Raj, M.D., fondly known to the world as Dr. Raj. Needless to say, it was both an honor and a privilege to be asked to write the Foreword to this seminal work, a textbook that I believe will be an essential, prized guide for all interventional pain physicians, irrespective of their specialties and countries of origin. Interventional pain management is a new specialty with its own identity and definition dating back to 2003. However, interventional pain management is not new to Dr. Raj. Since the 1970s, along with Drs. John Bonica, Gabor Racz, and Nikolai Bogduk, he has nurtured and raised interventional pain management to an energetic emerging specialty.

The first edition of Dr. Raj's book was published in 2002 to fulfill a clearly felt need. The need derived from a desire to raise standards, and to provide easy-to-follow materials. Through the years, Dr. Raj has shown us his remarkable ability to blend cutting edge clinical care with incredible common sense and creativity. In this atlas, Raj and others purposely supply the readers with a text that is designed to provide direct, simple instructions for those who desire to perform modern interventional pain management. The book is clearly arranged with up-to-date details, basic science, clinical relevance and radiographic figures, with appropriate conclusions.

The book is arranged in multiple sections for discriminate and interested readers detailing imaging, radiation safety, drugs, and a multitude of spinal and non-spinal interventional techniques. The book is the single-best source for interventional pain management physicians, and in my personal opinion, should be owned by each and every interventional pain physician, who must read and re-read this book in order to understand and practice interventional pain management. Each topic is arranged in a manner that is easy to understand and in an easy-to-follow format beginning with history, followed by anatomy, indications, contraindications, and descriptions of required equipment, drugs, preparation of the patient and procedural details. Further, each topic also describes complications and efficacy with appropriate and up-to-date references. One should have a copy of this book in the office, at home, and in the operating room for ready reference.

It would be impossible for one to practice modern interventional pain management without thinking of Dr. Raj. In his long career, he has tirelessly and diligently provided tools for physicians across the world to use in practicing interventional pain management. I met Dr. Raj as a first-year resident in 1977, while he was demonstrating the technical aspects of caudal epidural injections in San Francisco, California at an annual meeting of the International Anesthesia Research Society. It was quite a memorable event, meeting such a distinctive personality in the specialty who was also so kind, considerate and informative. No one can claim that Dr. Raj does not have original ideas. He is the author of the first comprehensive understandable textbook in pain management that has been followed by many other publications. He has guided literally hundreds of thousands of physicians, both directly and indirectly, to practice kind, comprehensive, modern interventional pain management over the years. Many of the developments we enjoy in interventional pain management today, including Comprehensive Competency Certification in Interventional Pain Management worldwide, are due to the dedication, unbelievable hard work, and the compassion of Dr. Raj. Needless to say, with delight and exuberance I credit most of my career developments to Dr. Raj, who has helped me with publications, research, and inspired me to start the American Society of Interventional Pain Physicians. I am very proud to let you know, those of you who do not know, that Dr. Raj received the first Lifetime Achievement Award presented by the American Society of Interventional Pain Physicians, as a small token of gratitude for his dedication to the specialty and what he has given to us which we can pass on to future generations.

Thanks to this book and the hard work and foundation laid out by Dr. Raj, the subspecialty of interventional pain management has developed from three epidurals in a recovery room, to a subspecialty and hopefully to a full-fledged specialty in the near future. This book brings many specialties together, discounting the dissenting voices of turf protection, dispelling the myths that one specialty can do it better than the other, and proving the necessity for the integrated practice of interventional pain management. Finally, this is a book that guides the reader in detail through the use of imaging to accomplish interventional pain management techniques. Dr. Raj, his coeditors and authors have provided us with hundreds of years of combined experience in this comprehensive encyclopedia. It was an honor for me to review a number of chapters, and I found them to be so well done, detailed, and clear, that a novice practitioner may feel like they can do any procedure. However, this book is not only for novices but also for well-experienced clinicians to be refreshed on techniques and to be exposed to varying perspectives.

Dr. Raj and his co-editors have produced a significant and comprehensive treatise on interventional pain management. After reading and reviewing advanced manuscripts submitted for this book, I know that Dr. Raj and his co-editors have succeeded in all their aims. Of course, it takes much more than a single book to provide complete knowledge and technical expertise required for the practice of interventional pain management, but this book lays out essential and valid foundation of knowledge. I congratulate Dr. Raj and all of those involved in the new edition of this seminal textbook, one that I believe will be a required, prized possession of all interventional pain physicians, irrespective of their specialty.

LAXMAIAH MANCHIKANTI, M.D., FIPP, ABIPP
PADUCAH, KENTUCKY

The book that you are about to read will certainly become the "bible" of interventional pain management techniques. The editors have succeeded in gathering a first class faculty around them, and they have done an excellent job. The result is a book in clear language, explaining the state-of-the-art of the various techniques for invasive pain treatment. A word of congratulation for the editors and for the faculty is in order.

The book has filled me with amazement about the rapid growth of invasive pain treatment. I started treating pain in 1972, and it was a very different world at that time. I vividly remember attending a meeting of the English Pain Society. There must have been an attendance of around 15 doctors, constituting almost the complete membership of the society. It was necessary to travel over the world to pick up techniques and wisdom, and often one came back poorer but not wiser.

I think that the Tsars of that period should be mentioned before they pass into oblivion, because we all have so much to thank them for, and they would all have loved to read this book. I shall do that in random order and without pretending completeness. There was Samson Lipton in Liverpool. Sam brought the percutaneous cordotomy from USA to England, and within a few years he had more experience with it than anyone in the world. He must have taught the technique to hundreds of doctors. They must all remember his wit and his charming personality.

My close friend Mark Mehta, from Norwich, was more interested in spinal pain and he left a great impression on his numerous visitors. John Lloyd, from Oxford, invented cryotherapy. A visit to Guido Moricca in Rome was an unforgettable experience. He injected alcohol into the hypophysis in cancer patients with widespread metastases. He was a great and courageous man. I am proud to have been awarded the prize that carries his name.

All these legendary men had one thing in common. They were always willing to receive visitors, to teach and to share their knowledge. They offered their time freely, without ever requesting an honorarium, because they realized how important the spread of knowledge is. This resulted in a tightly knit small community, where many personal friendships were born.

In the USA there was less activity. Whatever there was, was focal and mostly in the hands of neurosurgeons. There is nothing against that, but the fertile interdisciplinary exchange was more a European affair. Needless to say Prof. William Sweet played a prominent role, and we have to thank Norman Shealy for his inventive mind, introducing both radiofrequency treatment for spinal pain and epidural stimulation.

This is all about the seventies, and in retrospect the harvest of that period was modest, despite many individual efforts. At the end radiofrequency was only practiced by a handful of doctors, who were regarded as eccentrics by their colleagues. Epidural stimulation was still in its infancy. Possibly the lack of proper instrumentation was a decisive factor. Until 1980 the only electrode that was available for spinal work was the formidable Shealy electrode. As an example, the technique that was proposed by Uematsu to treat radicular pain in 1974 would have met a better fate if more advanced equipment would have been used.

The big boom only came after 1980. In 25 years invasive pain treatment evolved from the Middle Ages to where we are now. That may rightly be called an explosive growth. The question is now if these 25 years have led us on the right path. If you read this book, everything is fine and dandy. A diagnosis is made, if necessary we confirm it with a diagnostic block, we then do a logical treatment that we are fully justified to do because evidence based medicine tells us so. But is it really that simple?

I think that there are two viewpoints from which to look at invasive pain treatment. The first one is the simple one. We follow the steps as indicated above. The patient gets better, the insurance company pays the bill and everybody is happy. Is that good or is it not? Yes, of course it is. Many patients tell us that we have literally given them their life back and we can only be content that invasive pain treatment has grown so fast that a majority of patients can find a competent doctor not too far away.

But practicing invasive pain treatment as we do is like playing music on an old violin. The music is beautiful and we love finding new tunes to play, but we have no idea what makes the instrument produce such a wonderful tone. By the time that we feel that we need something new, we have no idea how to produce a new instrument, and progress is halted. From the second viewpoint therefore we are not only interested in the result, we also want to know what our motivation is and by what mechanism our procedure works.

Now life is no longer a rose garden. Let's start with our motivation. The thing that we don't realize in everyday medical praxis life is that our appreciation of a procedure is not governed by strict logic, it is part of a subtle system. The procedure must of course be generally accepted, not only in the medical world but also by the general public, who finds it a logical and understandable thing to do. Insurance companies pay for it, it is well within your technical capabilities. There is the factor of habitation. You've been used to doing this for years, and you have been using the vocabulary for years. We may even make up new words, because a good term gives substance and standing to a procedure or an idea. For example, some doctors search for instability following a spinal fusion, and when they don't find it and yet the patient hurts when he moves, they name it micro instability. The condition would be meaningless without the term. Then there is the industry, they don't sit still. Every now and then they send somebody to make you feel good and to brush up the image of the procedure.

Once this whole machinery has been set in motion it is almost impossible to stop, even if there comes a slight spot on the image. As an example, look at the root sleeve injections that are done by radiologists for patients with a herniated disc. Let me make it clear that I have the highest regards for radiologists and that I enjoy doing procedures under CT regularly. It is my contention however that this particular procedure must not be done under CT monitoring because with that type of monitoring there is no way to detect a partially intravascular injection. More than a few people have become hemiplegic because of this practice, but since the incidence is low and because the machinery is alive and kicking it is business as usual, until predictably the next complication will occur.

Another example is what happens around IDET. A number of patients have now acquired a cauda equina syndrome. Here is another low incidence but serious complication that is inherent to the method. If you burn seriously so close to the spinal canal, you can just wait for the next disaster. The chance that it will happen to you in a lifetime is minimal, so nobody thinks about stopping the machine. Ironically I just read in a journal a meta-analysis, stating that the procedure was very safe with a complication rate of just 0.8%. Just to remind you to stay alert while you are reading. It may be a good exercise to imagine yourself as being one of the victims. This will bring you to the conclusion that you must not be caught in automatisms, and that the *primum nil nocere* must prevail always.
Now let's turn to mechanisms. Hopefully it is superfluous to remind you that evidence based medicine ignores mechanisms. If tomorrow somebody publishes a randomized controlled trial showing that eating a cucumber a day prevents Alzheimer's disease, then insurance companies will instantly provide free cucumbers to anyone over 60. No questions asked.

The largest part of this book is taken up by three types of procedures: ablation, neuromodulation and steroid injections. For brevity, let me deal with the last item first. Steroid injections usually have a short lasting effect and they are dangerous. If injected intravascularly close to the spine, they may cause paraplegia, brain stem infarction and death. Long-term use may cause severe osteoporosis. The injection of steroids should therefore only be used as a last resort. Unfortunately steroid injections are an overused procedure, because the machinery around it is very much alive. It is my private opinion that we should collectively be ashamed.

Ablation is a procedure that appeals to the public opinion. If you tell a patient that you are going to burn the nerve that caused him his misery he relishes. But you did not tell the whole story, because in fact the situation is more complicated. The classic example of ablation is of course the destruction of the medial branch for facet pain, which is a form of nociceptive pain. Certainly burning the medial branch has a measure of success, be it for a limited period, and one may wonder why.

Success of an ablative procedure is a rare bird. I don't need to remind you that all the neurosurgical ablative procedures that were initiated during the seventies have without exception ended in failure, and in many instances in an exacerbation of pain. Cutting a nerve has never been a solution and finding the reason for that is not so difficult. If the archetype of chronic nociceptive pain, the myofascial trigger point, is injected with a local anesthetic solution, the pain goes away. If you now inject nalorphin, while the local anesthetic is still active, the pain returns. That means that in case of chronic pain there is a center in the dorsal horn or further up, that is able to fire spontaneously and to maintain the pain. It is only kept silent during the duration of action of the local anesthetic (or possibly shorter than that) by complex supraspinal mechanisms. This however is an acute situation, mediated by the sudden loss of peripheral input. In the chronic situation there is nothing to prevent firing of the dorsal horn focus.

Why does this not happen after burning the medial branch? We don't really know. The denervated area may be too small to cause trouble, or - more likely - the denervation may be incomplete despite the latest fashion of making large burns. If one looks at the innervation of the joint, complete denervation seems a tall order, not counting the capability of nature to restore what has been damaged. So, in summary, it works, but boasting that we have bravely denervated a joint is probably a bridge too far.

As for neuromodulation, despite many efforts the mechanism is still unclear. There is a chapter in this book about brain stimulation, and the author, Ricardo Ruiz-Lopez, is true to his character as I know him. He frankly states that we do implant electrodes in the brain but we don't know how it works.

The mode of action of pulsed radiofrequency is not clear either. Eric Cosman's new theory, that pulsed radiofrequency may cause Long Term Depression of the first synapse is a work of art, but it does not explain the delay in clinical improvement of a few weeks that we so often see. There must be another, possibly additional mechanism that is presently unknown.

So in summary, our music sounds reasonably well, and there are many tunes as you can read, but we make a very poor show of understanding our violin. We're playing in the concert hall now, but we cannot go on interminably this way. If we do, we'll end up being street fiddlers.

After reading all this, do you feel just a little uncomfortable? Excellent! You should, to be a responsible interventionist. Enjoy the book!

MENNO E. SLUIJTER, M.D., PH.D., FIPP
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PREFACE

It has been six years since the publication of the first edition of *Radiographic Imaging for Regional Anesthesia and Pain Management*. It was conceived to fill a void for a "how-to" manual for medical practitioners who wanted to develop their careers as interventional pain physicians. By all accounts the book was a success in that regard; however, several experts in interventional techniques noted that some procedures were not covered and that the book was geared more toward anesthesiologists than other medical specialists who practice interventional pain management.

We took all of our reader feedback into account when drafting the outline of the second edition. The table of contents was completely revised to include all the procedures relevant to the modern practice of interventional pain management. The coverage of regional anesthesia procedures for surgery was minimized and coverage of procedures for chronic pain management, such as catheter placement for various nerve blocks, was expanded. This book should be useful not only to anesthesiologists but to all those who practice neurosurgery, neurology, physical medicine and rehabilitation, orthopedics and interventional radiology who wish to include interventional pain techniques in their armamentarium of services.

The book contains 37 chapters, divided into a general section, a topographical section of common interventional procedures by body region, an advanced interventional procedure section, and an emerging techniques section. Although the general format of chapters remains unchanged from the first edition, the content of each chapter is either brand new or completely rewritten. Many chapters have benefited from expert illustrations by medical artist Meadow Green.

Presently, there is still a disparity between education and training of pain physicians around the world. To help standardize teaching and practice, experts in interventional pain procedures affiliated with the World Institute of Pain regularly conduct cadaver workshops in the United States and Europe. We also hope that this second edition will help set appropriate universal guidelines, based on best evidence and clinical experience, that can be followed and periodically reviewed.

The World Institute of Pain's Section of Pain Practice conducts an examination in Interventional Pain Procedures and certifies the successful candidate as a Fellow of Interventional Pain Practice (FIPP). The WIP Board of Examinations has endorsed this book as appropriate reading material for the theoretical and practical exam. We also hope that pain fellowship programs will make this book a part of their curriculum for interventional pain procedures.

During the writing and editing of this book one of our esteemed editors, Professor David Niv, passed away unexpectedly. His passing is an enormous loss to everyone involved in this project as Dr. Niv was a tremendous clinician, scientist, teacher and innovator. He will be missed by colleagues who worked with him, but more importantly by the patients he treated. With a heavy heart we mourn his loss with his family.

THE EDITORS

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We acknowledge with gratitude the hard work of Meadow Green, Susan Raj, and Marilyn Schwiers in organizing the table of contents, preparing manuscripts and illustrations, and editing the final chapters. Without their help this book would not have been possible.

We also want to thank the assistants to all our contributors for their hard work and input in getting the manuscripts completed. We are indebted to Elsevier and our publisher Natasha Andjelkovic for her advice and directions that helped us complete this book.

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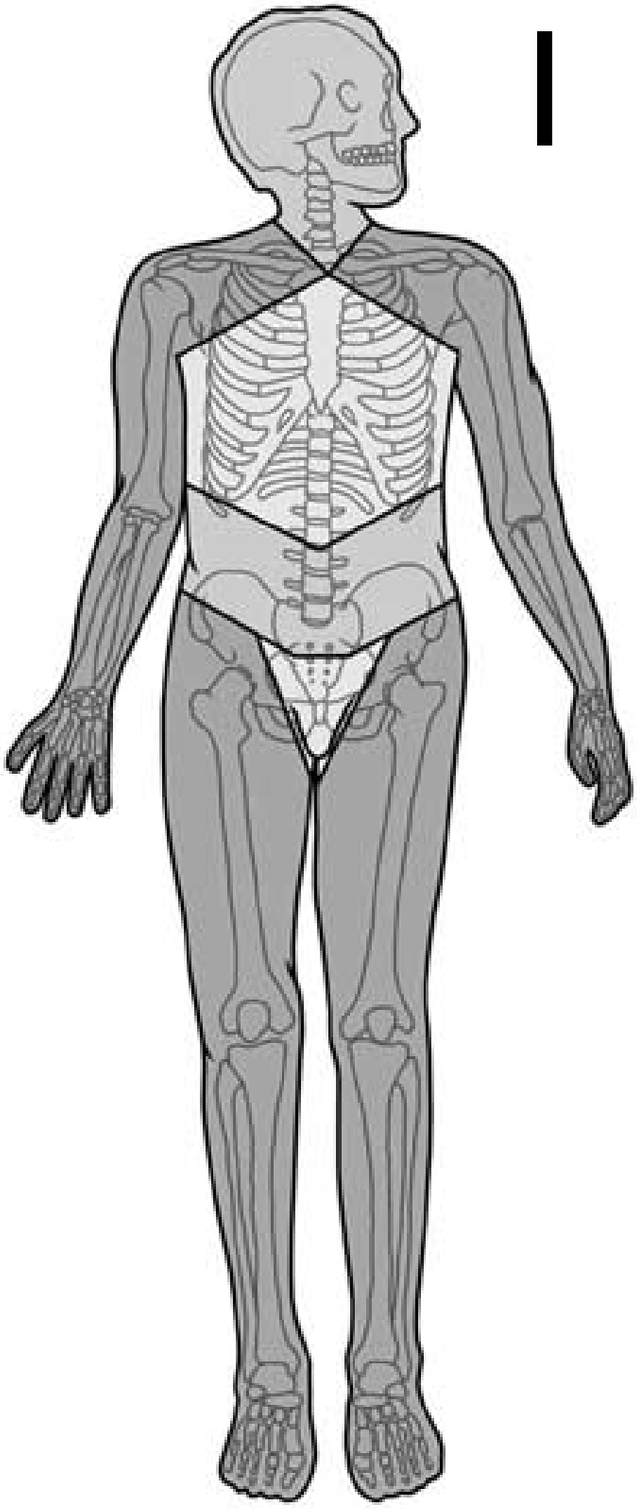

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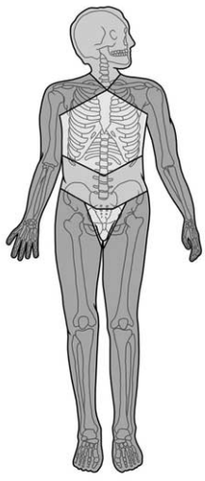

1 Imaging Techniques

LELAND LOU AND P. PRITHVI RAJ

HISTORY

Radiology began with the discovery of x-rays by William Conrad Roentgen in 1895, but it was not long after that the discovery of harmful effects was published. The first report of harmful physical effects was made in the *British Medical Journal*.[1]

The early workers who were developing the technique in the United Kingdom all had radiation injuries by 1903, and one died in 1911 after taking photographs of his hands showing progressive bony damage.

Public concern after the death of one of the first radiologists (William Ironside Bruce) from radiation-induced injuries in 1921 led to the establishment of the British X-Ray and Radium Protection Committee.[2] After the Second International Congress of Radiology in Stockholm in 1928, their recommendations were adopted and the International (X-ray and Radium) Protection Committee began. This was later to be renamed the International Commission on Radiation Protection and still exists today.

DEVELOPMENT OF INTERVENTIONAL RADIOLOGY

The possibility of intervention started with the development of angiography in the late 1920s by a Portuguese group. Many people have been involved in its progress since then, such as Werner Forssman, who performed the first cardiac catheter on himself in 1929, using a ureteric catheter, and Charles T. Dotter who introduced the concept of remodeling the artery by transluminal angioplasty in 1964.[3]

Coronary angioplasty began in humans with the first case in 1977 by Andreas Gruentzig and has since developed rapidly, at an annual increase of 8%; almost 2 million procedures were performed in 2001.[4] The successful use of angioplasty in an evolving myocardial infarct by Geoffrey Hartzler in 1980 and the development of stents in the late 1980s widened the indications and reduced the complications of the procedure, making it widely acceptable. It is now the most common interventional procedure in the world.

Interventional radiology in other anatomical systems has also developed dramatically and can be classified into types of procedure—for example, drainage, coil embolization, filter placement, stenting, and foreign body retrieval—or into anatomical systems, such as vascular, gastrointestinal, and urological. The most frequently performed angioplasties are in peripheral, as well as coronary arteries.

INTERVENTIONAL PROCEDURES FOR PAIN MANAGEMENT

Interventional procedures for pain management have been developing new techniques and precision techniques since 1960. What was previously the role of anesthesiologists to do such procedures is now open to many other specialists (physical medicine and rehabilitation, neurologists, neurosurgeons, and so on). Imaging techniques have become a part of this new advance and are considered a good practice for interventional procedures in pain management.

USE OF RADIOLOGY IN PAIN MANAGEMENT

The role of radiology in pain management is primarily diagnostic. In patients with pain symptoms, the goal is to establish the specific etiology of the pain to be able to direct proper therapeutic measures.

A proper radiological workup should provide a thorough, diagnostically accurate evaluation of the specific disorder. Consideration should be given to cost, availability, risk or side effects, and acceptability to the patient. A radiographic test should be obtained only when the results may alter the patient's subsequent management.

With the advent of multiple new radiographic modalities, the thoughtful selection and planning of radiological evaluation becomes crucial to efficiently derive the diagnostic benefits and control the substantial costs (and sometimes risk or discomfort) involved.

More than ever, it is important to consult with a radiologist when any doubt exists regarding the efficacy or

appropriateness of a planned radiological workup for a patient to avoid unnecessary studies. Furthermore, radiological examinations are often tailored to fit the individual patient to best answer the diagnostic question presented. Communication between clinician and radiologist thus directly results in a more accurate diagnosis and ultimately benefits the pa tient.

HISTORY OF RADIOLOGICAL IMAGING

Before the mid-1970s, plain film radiography, conventional tomography, and myelography with either gas or oily material as contrast agents were the only methods available for imaging abnormalities involving the vertebrae, intervertebral disks, spinal cord, or cauda equina. By 1975, a nonionic intrathecal contrast agent, metrizamide, was approved for clinical use. Unlike oily agents, nonionic contrast carried negligible risk for arachnoiditis and was absorbable, and thus eliminated the need for its removal from the thecal sac. Second, its neurotoxicity was minimal, compared with ionic water-soluble media, which never achieved widespread acceptance in the United States.

In 1977, the introduction of whole-body computed tomography (CT) permitted direct cross-sectional imaging of both spinal and paraspinal structures. However, the margins of the spinal cord could only be reliably demonstrated after the intrathecal administration of water-soluble contrast. This procedure is known as CT myelography (CTM). Because of the greater contrast sensitivity of CT, as compared with plain film myelographic technique, a smaller, less potentially neurotoxic dose of contrast agent could be administered for CTM. Nevertheless, this procedure still requires a lumbar puncture, with its attendant hazards to the patient.

By 1982, magnetic resonance imaging (MRI) became clinically feasible. MRI has proven to be superior to CT because the spinal cord and nerve roots could be visualized directly without the requirement for intrathecal contrast material. Most significantly, the parenchyma of the spinal cord could now be imaged and assessed for intrinsic pathology, such as multiple sclerosis plaques. These lesions may not alter the shape of the spinal cord, and, therefore, would be undetectable by CTM. Second, MRI provides multiplanar imaging, including sagittal and coronal orientations, with spatial and contrast resolution equivalent to the axial plane. Lastly, MRI poses no known health risk, as it uses only radiofrequency energy, not ionizing radiation as is the case with CT.

FUNDAMENTALS OF RADIOGRAPHY

ATOMS

Matter is composed of atoms that occupy space. Atoms can be further broken down into electrons, protons, and neutrons. All known substances, living and nonliving, are comprised of these elemental components. Combinations of these elemental particles determine atomic structure. Each element has an atomic number based on the number of protons.

Protons, which have a positive charge, and *neutrons*, which have a neutral charge, together form the *nucleus* of the atom. The *electrons* are often compared to "planets" that orbit the nucleus or "sun" of the atom (Figure 1-1). The negative charge from the electrons keeps them orbiting the nucleus in five electron shells labeled K, L, M, N, and O (Figure 1-2). The K shell is the strongest and requires the most energy to displace an electron from its orbit. If an electron is moved from a higher energy shell to a lower one, energy is released.

An atom in a nonionized state has an equal number of protons and electrons. A displaced orbital electron and the atom from which it originated is called an *ion pair*. This situation can occur with electron bombardment of matter, x-ray bombardment of matter, thermionic emission with electron release, and chemically, among other means. If the ionized electron is moved to a higher orbit, this is called *excitation*. In an excited state, the displaced electron will return to its original orbit or be replaced by another electron. Often the additional energy to ionize the atom is released as photons of electromagnetic energy, heat, or chemical energy.

ELECTROMAGNETIC RADIATION

Electromagnetic energy is arranged in an orderly fashion according to the wavelength. For medical x-rays, this range is from approximately 0.1 angstrom to 0.5 angstrom

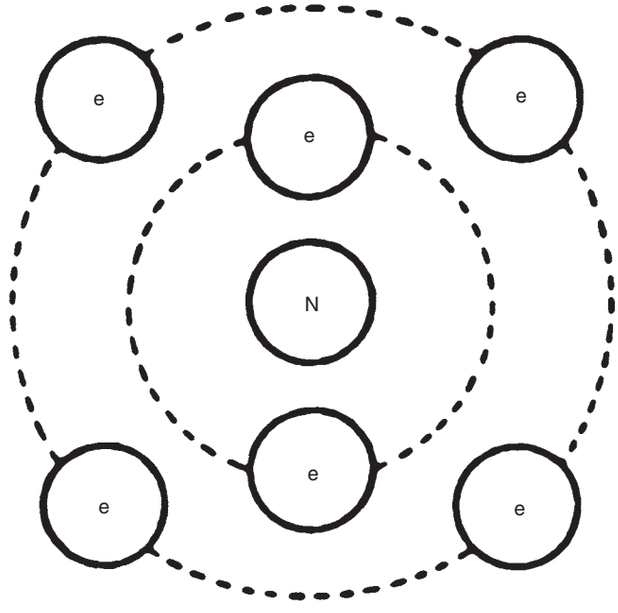

FIGURE 1-1

Atomic structure. A typical atom consists of a nucleus (N), which contains positively charged photons and neutrons (no charge) and negatively charged electrons (e). The electrons orbit around the nucleus. This representation of the carbon atom is not in scale. In actuality, neutrons and protons each constitute 1838 times more mass than electrons.

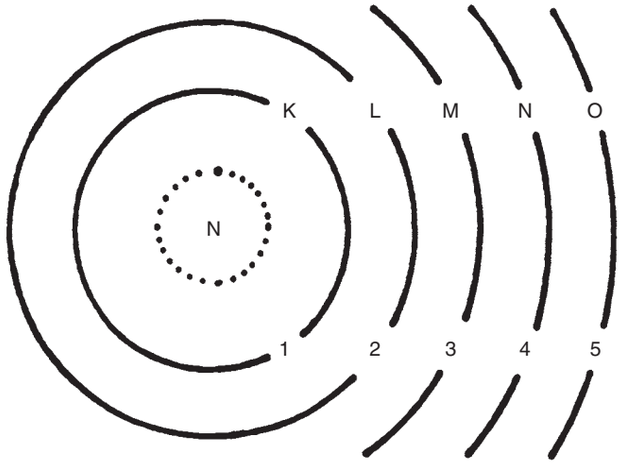

FIGURE 1-2

Electron shell arrangement and binding power. The binding force on the electron shells holding the electrons in orbit around the nucleus weakens as the number of shells increase. The five electron shells shown are labeled K, L, M, N, and O. The K shell possesses the strongest binding power. The electrons in the K shell require the most energy to dislodge from orbit, whereas the electrons in the peripheral shells are easier to displace.

(0.01–0.05 nm) (Figure 1-3). This energy travels in the form of sine wave–like oscillations at the speed of light. The oscillations are measured as amplitude, wavelength, and frequency. Amplitude is the height of the wave from the crest to midpoint or trough to midpoint. Wavelength (angstrom) is the distance from one wave to the next. Frequency (hertz) is the measurement of the number of waves passing by a specific point in a given unit of time (Figure 1-4). X-ray photons are commonly between 0.1 and 0.5 angstrom and 10^{18} to 10^{21} hertz.

ELECTRICAL ENERGY CONVERSION TO RADIANT ENERGY RADIATION

Alternating current (AC) is converted into direct current (DC) by an electrical transformer. DC is then put into motion (kinetic energy) from cathode to anode in the x-ray tube to produce heat (thermal energy) and x-radiation (radiant energy).

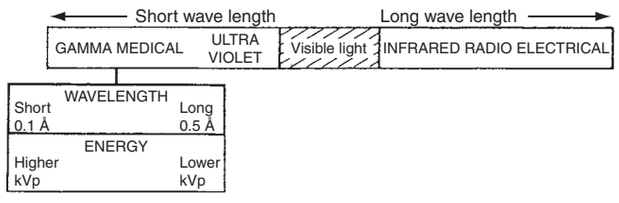

FIGURE 1-3

The relationship of medical x-ray to the electromagnetic spectrum. In this abbreviated illustration the electromagnetic spectrum runs from gamma radiation (short wavelength) to electrical waves (long wavelength). Within the medical x-ray portion of the spectrum, wavelengths may be short (0.1 A) or long (0.5A). In the medical x-ray range, a short wavelength will be produced with high kilovoltage values, whereas a long wavelength will be generated by low kilovoltage values.

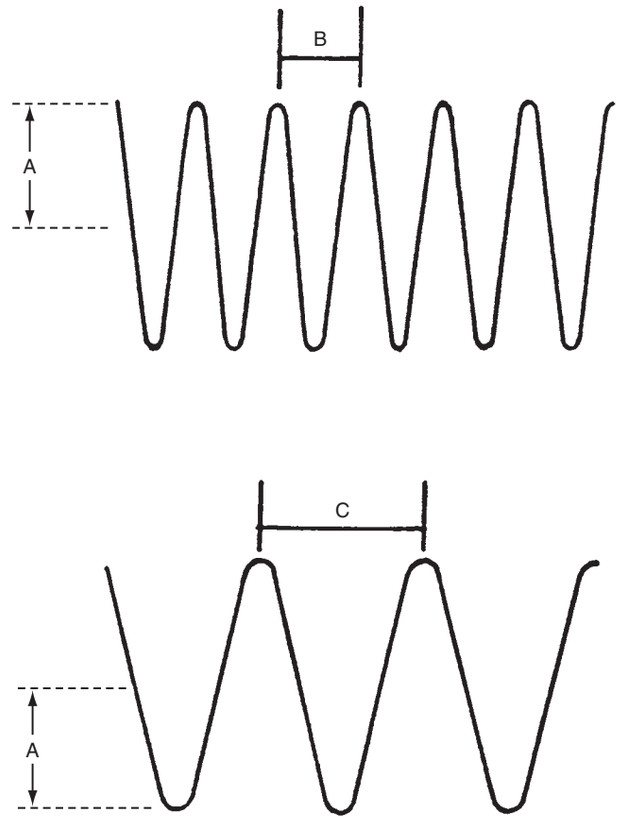

FIGURE 1-4

Sine wave. Electromagnetic energy is transported through space in the form of sine wave-like oscillations. This energy travels at the speed of light, about 186,300 miles per second, and can be schematically illustrated. Components of the sine wave include amplitude, wavelength and frequency. Amplitude refers to the height of the wave from crest to mean value (A). Wavelength describes the distance from one crest of the wave to another and represents the distance between two corresponding points on the wave (B and C). Frequency is determined by the number of crests or valleys passing through a specific point in a given time. There are more wavelengths in B compared with C. Shorter wavelengths (B) will result in increased frequency of the wave.

The filament (cathode) of the x-ray tube is heated to incandescence, causing electrons to "boil off" in a process known as *thermionic emission*. The electrons' energy is converted into heat and x-ray energy.

The milliampere (mA) setting selects the tube current and determines the heat of the filament. This setting determines the number of released electrons available for interaction. The range of the applied voltage (kVp) determines the wavelength and thus the energy of the x-ray photons. The relation of voltage and amperage to resistance can be expressed by Ohm's law, which states that

$$I = V/R$$

where I = amperage, V = voltage, and R = resistance.

Electron Interaction with Anode of X-ray Tube

More than 99% of the energy is converted to thermal energy (heat). The remaining energy is divided among bremsstrahlung and characteristic radiation. Heat is produced by the energy derived from the movement of the atoms and their quick return to a normal state. The greater the kinetic energy (energy of motion or vibration) produced, the greater the temperature.

Bremsstrahlung radiation is also known as *general radiation*, the *continuous spectrum*, or *white radiation*. Production of bremsstrahlung radiation is from the "braking" action that occurs as the electrons interact with the anode. This process involves electrons that pass by the heavy nuclei of the metallic atoms in the target material. The attraction between the negatively charged electrons and the positively charged nuclei causes the electrons to be deflected and decelerated from their original path and to lose energy. Since energy cannot be destroyed, the energy lost by the electrons is transformed and emitted as x-ray photons.

The considerable rate of deceleration causes the emission of short-wavelength radiation in the form of x-rays. As this braking action varies, so does the intensity of the resultant x-ray energy. In the 80- to 100-kVp range, using tungsten anode, these bremsstrahlung rays constitute about 90% of the radiation emitted as x-rays. For example, to produce characteristic radiation with a tungsten target, at least 70 kVp are required for K-shell interaction because the K-shell electron of tungsten is held with 69.53 effective kilo voltage (Figure 1-2). Characteristic radiation produced in the interaction of x-rays with matter is usually referred to as *secondary radiation* and is a form of scatter.

X-ray Interaction with Matter

In diagnostic radiology, there are three types of x-ray energies of importance: primary x-rays or photons emitted by the x-ray tube; scattered x-rays or photons produced when primary photons collide with electrons in matter; and remnant radiation, or x-rays that pass through the patient and strike the image detector.

When discussing x-ray interactions with matter, photoelectric and Compton effects are important. The photoelectric effect is the absorption of energy. When the x-ray photon collides with the inner shell electron of an atom, the photon may give off all its energy and the collision causes the photoelectric effect along with ionization.

The *Compton effect* refers to the scatter of the ions or radiation as it interfaces with different radiographic densities. If the incoming x-ray photon has increased energy resulting from increased kilo voltage applied to the x-ray tube, some of that energy is transferred to other atoms with the x-ray photon passing with a decreased energy and slower wavelength. This principle is relevant to the five basic medical radiographic densities: air, fat, water (soft tissue), bone, and metal.

ELECTRICAL CURRENT

Alternating current of sinusoidal wave shape results from the application of an alternating voltage with its polarity and values reversing direction at regularly occurring intervals, typically 60 times per second (60 Hz) in the United States. Electrical energy in the form of voltage and amperage is usually supplied by commercial power companies and delivered as alternating current because it is easier to produce and transfer from place to place in this form. AC can be greatly increased or decreased by employing a simple device called a *transformer* (see "Transformers" section).

Direct current may be steady or may be intermittent. The direction of flow does not change with direct current. To operate many devices, DC is created from AC. This current is easier to put to use but difficult to transmit over great distances.

TYPES OF ELECTRICAL CIRCUITS

In a series circuit, the current will pass consecutively through each individual component and can be expressed as $I = i^1 = i^2 = i^3$. In a parallel circuit, current flow is divided among the branches of the circuit and is expressed as $I = i^1 = i^2 = i^3$.

An electrical circuit is used to gather, carry, or direct flowing electron energy. Electrical energy is carried through the circuit by electrical current (electrons in motion). Volts (V) measure the potential difference from the start to the end of a path. Current (I) or electron flow is measured in amperes (A). There is resistance (opposition) to the electron flow in all circuits, with some absorption and thus loss of energy.

Electrical resistance (R) is measured in ohms (Ω). The term *resistance* is used in reference to simple DC. Impedance denotes resistance in AC. The resistance of a conductor is directly proportional to the resistivity of the material of which the conductor is formed. Resistance is also directly proportional to the length of the conductor, but inversely proportional to the width (cross-sectional area) of the conductor.

Conductors are materials that transport electrons at all levels of energy output. Some materials are able to conduct only when they receive a specific increment of energy; these are known as semiconductors. Materials that do not conduct are referred to as *insulators*.

TRANSFORMERS

A transformer does not produce energy; it transforms voltage and current by way of the ratios of respective windings.

Air-Core Transformer. By placing a coil of wire with current flowing through it, called a *solenoid*, adjacent to a second coil of wire, an air core transformer is formed. This is the simplest type of transformer.

Open-Core Transformer. To form an open core transformer, soft iron bars are placed in both the primary and secondary coils. The cores are not electrically connected; they only conduct field line.

Closed-Core Transformer. A continuous laminated iron bar forming a rectangular annulus is used to support the primary and secondary windings in a closed-core transformer. Again, there are no electrical connections between the coils.

Shell-Core Transformer. A continuous laminated iron bar forming a rectangular figure eight, with the primary and secondary wires wound around the center support, is called a shell-core transformer.

Step-Up Transformer. The ratio between the primary and secondary currents is related to the number of turns in the wires in the individual coils. If the number of turns in the wire of the secondary coil exceeds the number of turns in the wire of the primary coil, the transformer becomes a step-up transformer and voltage will be increased.

Step-Down Transformer. If more turns exist in the wire of the primary coil than in that of the secondary coil, the transformer is a step-down transformer and voltage will be reduced. The filament circuit uses a step-down transformer.

Three-Phase Transformers. Three-phase transformers are used for three-phase equipment to generate a more homogenous x-ray beam. Three separate circuits are required in an x-ray machine using three-phase power, one for each phase. Each circuit requires its own transformer, rectifiers, line voltage compensator, and so on.

The basic components of an x-ray generating system are illustrated in Figure 1-5.

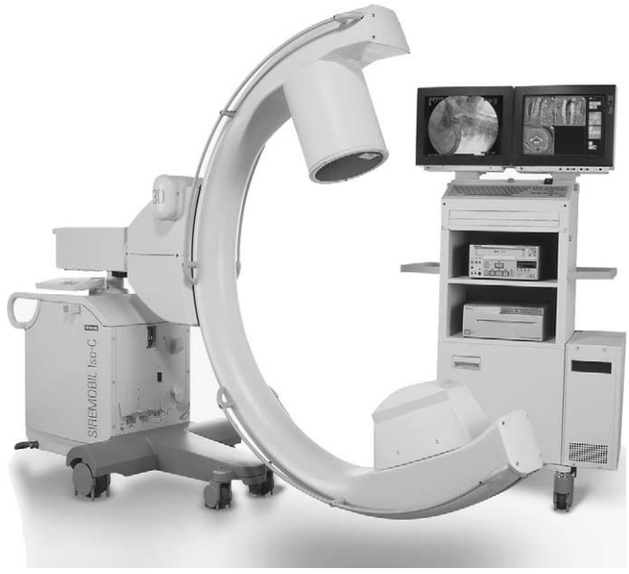

FIGURE 1-5
A typical fluoroscopy C-arm.

ELECTROMAGNETICS

Electrical charges may be static (at rest) or dynamic (in motion). Electrical current is always surrounded by a magnetic field, which exists only while the current is flowing. The process of electromagnetic induction can induce current induced in a second wire if the wires cut through the magnetic field lines produced by an electrical current. Since both coils are not electrically connected, placing a second coil of wire adjacent to the first coil induces an electrical current in the second coil by mutual induction. The force in the second wire loop is directly proportional to the number of turns in the first wire loop.

When the wire of a conductor is coiled, a helix is formed. A helix with current flowing through it is called a *solenoid*. The solenoid, an electromagnet, has a strong magnetic field in its center when current is flowing through the coiled wire.

Rheostats are controls used to add resistance to the circuit in order to adjust incoming voltage and amperage values. A break in the circuit can be achieved with switches that are used to control the length of time that the current may flow. Fuses or circuit breakers are protective devices that open at present levels of current and thus prevent circuit overloading and damage.

X-RAY CIRCUIT

The x-ray circuit is divided into subsections called primary (low voltage) and secondary (high voltage) circuits (Figure 1-6A and B).

The primary circuit consists of the following components:

1. Main switch: Power from an electrical source is turned off and on at this point.
2. Line voltage compensator: This is used to compensate for variations in power supply. It is important to monitor the incoming line voltage. Line voltage compensation is automatic in some units.
3. Fuses or circuit breakers: These are used to prevent equipment overload or tube damage.
4. An autotransformer: This is used to control voltage supplied to the primary of the step-up transformer, to allow for minimal variations in kilo voltage selection.
5. A prereading voltmeter: This indicates the amount of voltage being sent to the primary

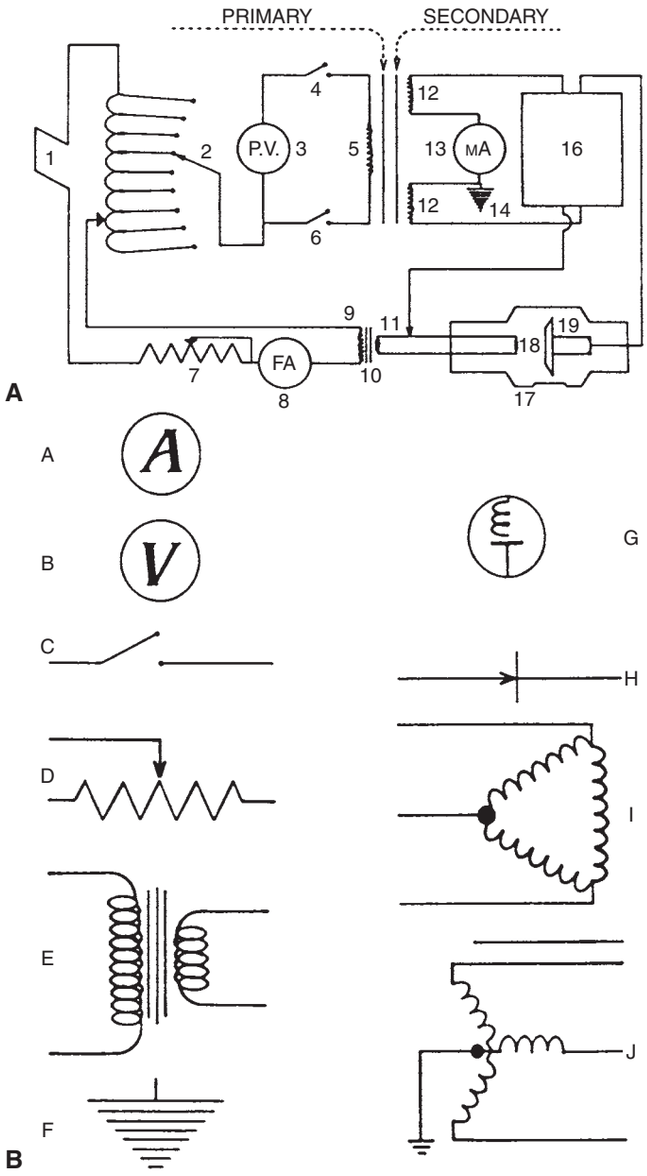

FIGURE 1-6

(A) The x-ray-generating circuit is divided into a primary (low-voltage) and secondary (high-voltage) circuit. The primary circuit consists of (1) a main switch (2) an autotransformer (3) a prereading voltmeter (4) fuses or circuit breakers (5) the primary coil of the step-up transformer (6) a timer that includes exposure switches (7) a filament circuit and rheostat, used to vary current to the primary circuit of a step-down transformer to illuminate the filament of the x-ray tube (8) a filament amp meter (9) the primary coil of the step-down transformer (10) the step-down transformer and (11) the secondary coil of the step-down transformer. The secondary or high-voltage circuit consists of (12) the secondary of the step-up transformer, (13) an mA meter, (14) ground, (15) step-up transformer, (16) a rectification system, (17) the x-ray tube, (18) cathode of the x-ray tube, and (19) anode of the x-ray tube, including shock-proof grounded cables to conduct high voltage from the secondary of the step-up transformer to the tube. Solid state rectifiers are used to illustrate the rectification segment, since valve tubes (vacuum tubes with illuminated filaments) are no longer in common use. Valve tubes require step-down transformers. (B) Schematic representation of x-ray circuit components. (A) The filament ammeter is usually designated in the circuit by a circular meter containing the symbol A. (B) A voltmeter is similarly indicated by the symbol V. (C) A circuit breaker or a timer is often represented as an open-ended switch. (D) A rheostat is used to vary electrical current to the primary circuit of the step-down transformer. (E) The step-down transformer shown has more turns in the coils of the primary than in the secondary windings. (F) The universal symbol for ground. (G) Vacuum tubes that contain a filament and flat anode can be used for rectification of alternating current to direct current. (H) A solid-state rectifier. Three-phase transformers used for three-phase equipment generate a more homogenous x-ray beam. Three separate circuits are required in an x-ray machine using three-phase power, one for each phase. Each circuit requires its own transformer, rectifiers, line-voltage compensator, and so on. In the high-tension transformer, the primary coils are wound around separate arms of a core common to all three transformers. (1) A "delta" configuration depicts this arrangement of the coils. (J) The secondary high-tension coils have a common center, each coil radiating outward in a star ("Wye") pattern.

of the step-up transformer. Kilo voltage is determined by the amount of voltage supplied to the step-up transformer and is present only when the exposure is being made.

6. Timer and exposure switches: Timers are used for manual or automatic exposure control. The timer is situated between the autotransformer and the primary of the step-up transformer.
7. A filament circuit: Thermal energy is created from this circuit to heat the filament of the x-ray tube. High amperage is used to heat the filament for production of the thermionic emission. The heating of the filament is then controlled by the rheostats or resistors, which regulate the milliamperage delivered to the filament circuit and resultant heating.
8. A filament amp meter is used to measure the filament current.
9. The primary coil of the step-up transformer.

Components of the secondary or high-voltage circuit follow:

1. The secondary coil of the step-up transformer, which is "center-tapped" to allow an mA meter to be installed at ground potential.
2. The mA meter, used to measure tube current.
3. A milliampere-second (mAs) meter used to measure mAs values at short time intervals.
4. Rectifiers. The x-ray tube is most efficient when unidirectional high-voltage current is used. Current is made unidirectional for use by the x-ray tube by means of a rectification system that converts alternating current to DC.

5. Shockproof, grounded cables, which conduct high-voltage current from the secondary of the step-up transformer to the x-ray tube.
6. The x-ray tube.

BASIC COMPONENTS OF X-RAY TUBE

The basic components of the x-ray tube start with the filament. From the source of electrons or cathode side of the tube, the electron stream passes through a "focusing cup" or area and is directed into the anode. A rotating anode with a high positive potential is often used instead of a stationary anode in a fluoroscope (Figure 1-7).

This rotating anode allows for quicker dissipation of the heat generated. An extremely high-speed stator motor system is needed to keep the heat produced even and avoid damage to the anode. The x-ray is projected from the x-ray tube into the target area and gathered by the imager to be transformed into a radiographic image.

BASIC OPERATION OF X-RAY TUBE

Once the electrical signal is sent through the circuitry, the filament is energized to "boil off" electrons as a thermionic emission. As the increase of kVp passes through the filament, the creation of a higher potential difference results in the emission of electrons beyond the "cloud" of electrons that are found in the vicinity of the filament. The attraction of the electrons into the metal anode (+) surface and the following abrupt stopping of the electrons produces x-radiations and heat. Unfortunately, 99% of this energy is converted into undesired heat and less than 1% is converted into x-radiation.

The variation of the kilo voltage affects the speed of the electrons directed at the anode and generates various x-ray wavelengths. For example, a shorter wavelength makes the beam more penetrating. A longer wavelength x-ray is less energetic and less penetrating.

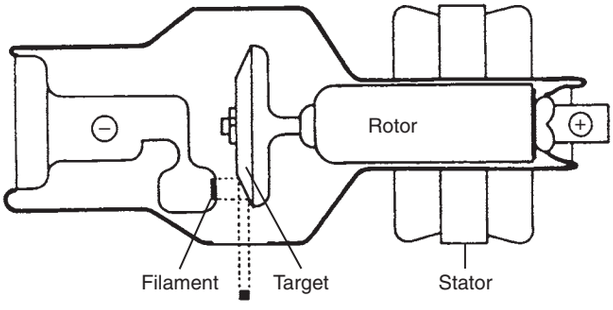

FIGURE 1-7

Basic components of an x-ray tube. A basic component of a radiographic tube is the filament, the source of electrons, at the cathode side of the tube. The cathode, a high negative potential, includes the focusing cup, which has a negative charge applied to it to "focus" the stream of electrons by the repulsion of like charges. The anode, a high positive potential, serves as a target for the focused electron stream. A stator rotor system that uses an induction motor rotates the anode at extremely high speed, usually 3000 rpm or 10,000 rpm. A Pyrex envelope houses the cathode and rotating anode and is placed in an oil-filled, lead-lined housing.

Control Panel

In clinical practice, the control panel is the most common interface of the fluoroscope and the radiographer (Figure 1-8). From this panel variations in power delivered through the x-ray tube can be controlled for improved images. The milliamperage (mA) determines the intensity of the x-ray beam. Kilo voltage determines the speed of the electrons and quality of the x-ray beam. The length of exposure is often measured in seconds and is the most obvious factor in measuring x-ray exposure.

The milliamperage is important in determining the quantity of x-rays produced. In combination with the length of exposure, the mA is important to the quality of the image produced. For a stop-motion situation, the operator may need to combine a high mA with a short exposure time.

Kilo voltage determines the penetrating ability and quality of the x-ray beam. The higher energy release of x-rays results in a greater number of photons to be captured by the imager. This allows for a more detailed and wider range of contrast of the gray scale.

Collimator buttons on the control panel are usually of two varieties, a circular shape and horizontal bars. Ideally the collimators should be used as much as possible to reduce the amount of radiation exposure. For this purpose, the radiographer may choose horizontal collimation for facet injections. The circular or "shutter" collimation is best used in techniques when a "tunneled approach" is used.

The timer is also located on the control panel. There are audible alerts set at 5-minute intervals to remind the fluoroscopist of the actual time of x-radiation exposure. Exposure is best limited by minimizing fluoroscopy time. To simplify the measurement of time, the timer should be reset prior to each new procedure.

Many of the other buttons available for manual control involve the orientation of the fluoroscopic image from left to right, inversion, or rotation. This function is important for the interventional physician in the performance of the procedure. A consistent habit provides continuity and accordingly limits risk and mistakes. For example, left sided procedures should always be correlated with left sided radiographic image to prevent accidentally performing the procedure on the wrong side.

IMAGING MODALITIES

PLAIN RADIOGRAPHY

Since the discovery of x-rays by Wilhelm Conrad Roentgen in 1895, plain films have been the mainstay of radiologic imaging. Even with the technological advances of recent decades, plain film radiography remains on the front line of imaging in the initial evaluation of musculoskeletal

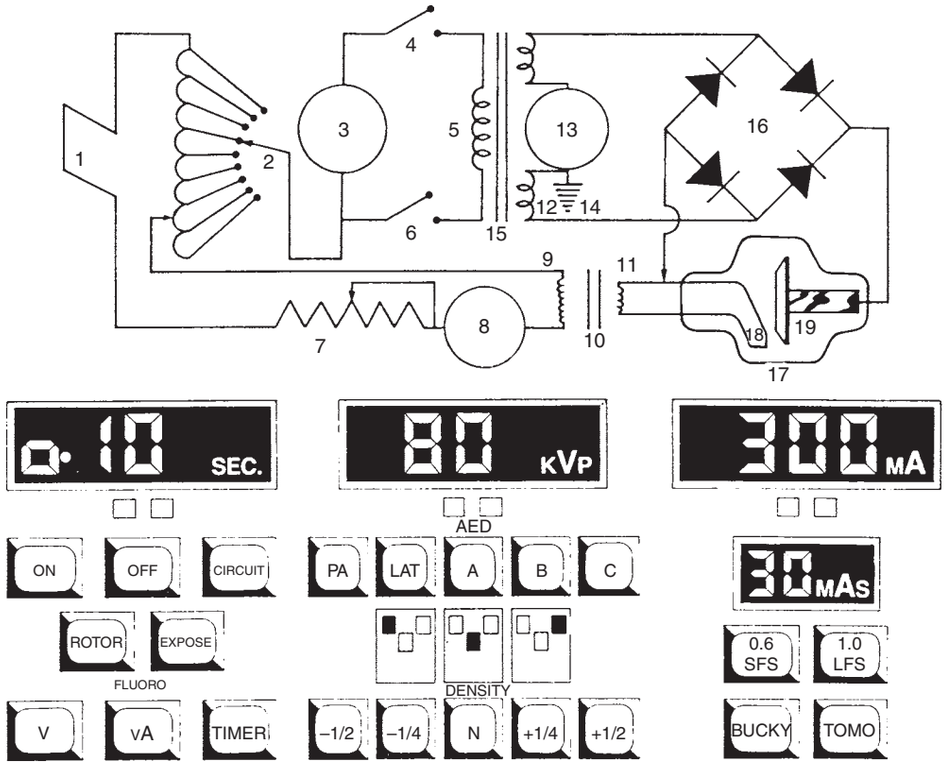

FIGURE 1-8

Operator controls of an x-ray machine. This representation of a control panel is divided into the following segments: kilovoltage and related circuits (center), milliampere settings and focal spot size selection (right), and timer control (left). A representative schematic of the x-ray circuit is shown above the control panel. Depending on the equipment design, these controls can be presented in many configurations. Additional meters such as tube load limits, heat displays, and a direct readout of the fluoroscopic examination time are often found on control panels. Kilovoltage can be raised or lowered (center) as required to adequately penetrate the part being examined. A power "on" and "off" button and a circuit breaker are shown. The rotor control, as well as the expose button, is on the left. At bottom are fluoroscopic kilovoltage and milliampere stations and a fluoroscopic timer that can be set to limit the length of the fluoroscopic procedure. The milliampere (mA) readout is shown on the right. Milliamperage can be raised or lowered depending on technical needs. A high mA value combined with a short exposure time is sometimes needed to overcome motion. The selection of a moderate mA value often permits the use of a small focal spot. The focal spots represented in this panel are 0.6 mm and 1.0 mm in size. Directly above the focal spot selection indicators is a mA meter. This device is required when extremely short exposures are used so that an accurate reading of the mAs used can be obtained. Below the focal spot size selection are the Bucky "on" and "off" buttons and tomographic selector control. At the top left of the control panel is the manual timing section. The time of exposure can be raised or lowered by the radiographer or an AED can be selected. Specific AED sensor indicators for the chest (posteroanterior or lateral) or other Bucky stations are shown. Table Bucky is represented by A, the upright Bucky by B, and the radiographic spot-film component of the fluoroscope by C. The darkened sensors (left, right, or center) indicate the sensors selected for the part under study. In the lower portion of the AED section on the control panel, density adjustment controls are shown. The center button, labeled *N*, is intended for use when a normal or preselected density is desired. The (–) control can be adjusted for a 1/4 or 1/2 decrease in density. The (+) control can be adjusted in a similar fashion for an increase in density. When an examination must be repeated, image density should be adjustable by the use of the (–) or (+) setting.

complaints, chest pathology, and the acute abdomen, including the preliminary search for the presence of radiopaque calculi in the kidneys, ureters, bladder, or gallbladder. Additionally, plain films are standard in the initial assessment in most cases of trauma. Plain films can frequently provide an accurate diagnosis and can do so in the most efficient and cost-effective manner possible.

Conventional radiographs are often used as an initial evaluation, especially in patients with musculoskeletal pain. Radiographs are readily available in nearly all medical facilities; they may be obtained and interpreted quickly, and cost is substantially less than other special modalities. Their noninvasive nature and short exposure times (a fraction of a second) make plain radiographs acceptable to most patients. Patients with moderate discomfort may be able to cooperate with radiographic positioning for a short duration, whereas the necessity of longer immobilization for magnetic resonance imaging, computed tomography (CT), or nuclear scanning may not be feasible.

Radiographs are indicated for evaluation of a number of skeletal abnormalities. Their obvious primary use is for diagnosis of fracture (including documentation of healing or complications), arthritis, and primary bone tumors. The fine anatomical resolution is not equaled by any other modality and maximizes precision of diagnosis in these types of disorders.

FLUOROSCOPY

Fluoroscopy is a technique for generating x-ray images and presenting them continuously as visible images during a diagnostic or interventional procedure. It is usually used to track the movement of a dye (contrast agent) or object through the body. Some examples of fluoroscopy include viewing contrast agents moving through the upper GI tract, examining blood flow to organs, or directing the placement of a catheter.

The two major risks associated with fluoroscopy are radiation-induced injuries to the skin and underlying tissues ("burns"), and the small possibility of developing a radiation-induced cancer later in life.

MYELOGRAPHY

Myelography has been performed since 1919, when Dandy introduced air contrast into the spinal canal.[5,6] Shortly thereafter, Mixter and Barr used the technique to investigate intervertebral disk protrusion and nerve root irritation.[7] In 1944, iophendylate (Pantopaque) was introduced into the subarachnoid space, and until recently myelography was most often performed with this agent (Figures 1-9 and 1-10).

Various water-soluble agents have been tried over the years, including methiodal sodium (Abrodil), meglumine iothalamate (Conray meglumine), and meglumine iocarmate (Dimer-X). Because of neurotoxicity, these agents have been discarded. Since 1978, metrizamide (Amipaque) has all but replaced Pantopaque for lumbar myelography (Figure 1-11).

Pantopaque remains the agent of choice for evaluating spinal block, for postherapeutic follow-up studies, for evaluating obese patients, and in cases where there are contraindications to metrizamide. The reader is referred to Sachett and Strother's excellent work on the techniques of using metrizamide.[8]

Myelography with Pantopaque clearly shows large extradural defects. The most laterally located or subtle lesions on the nerve root sleeve are easier to visualize with the less viscous metrizamide. When myelographic studies are done, one must keep in mind that disk protrusion may be seen in the asymptomatic patient.[9] This finding is not uncommon.

Myelography has been employed for more than 50 years as a safe, effective, and established method of imaging the spinal canal and thecal sac.[10–15] The procedure involves introducing a small amount of nonionic contrast into the thecal sac following a lumbar puncture. Imaging is then performed in multiple projections, allowing the contrast to delineate the subarachnoid space, spinal cord, and the nerve root sleeves.[10,11,15]

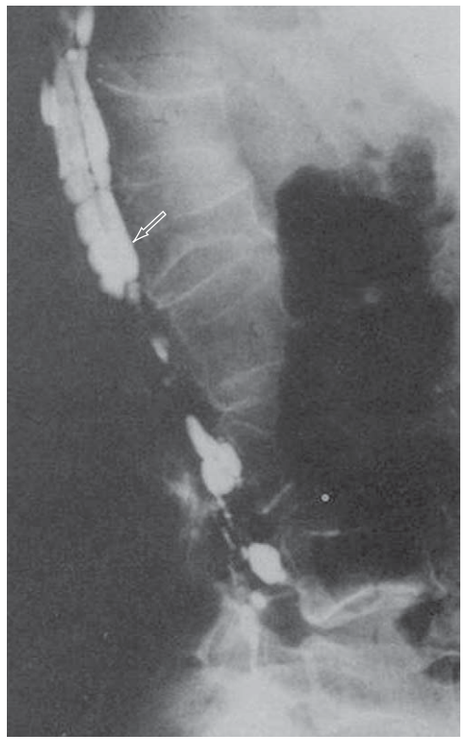

FIGURE 1-9

Pantopaque myelogram showing arachnoiditis (see arrow). (From Raj PP, editor: *Practical Management of Pain*. St. Louis, Mosby, 1986, figure 15B-6, p. 163, with permission.)

Myelography can be used alone or in conjunction with CT examination of the spine to evaluate intradural, extradural, or intramedullary lesions. Post myelography CT is of significant clinical value in many situations, such as in arachnoiditis or extradural abscess (Figure 1-12).

Myelography uses a contrast solution in conjunction with plain radiography to improve visualization of the spinal cord and intrathecal nerve roots. Water-soluble contrast agents (iohexol and iopamidol) are injected into the subarachnoid space. After injection, AP lateral and oblique views are obtained.

Myelography can be helpful in detecting a herniated disc above or below a segment that may be ambiguous or distorted on MRI secondary to metal placement. It is also useful in patients who are claustrophobic or have a pacemaker, or for whom MRI is otherwise contraindicated. A CT scan performed within 2 hours of completing myelography enhances the diagnostic quality and reliability of the imaging study by more accurately depicting osteophytes, disc herniations, and spinal cord contour.[16]

Myelography is an invasive technique and lacks diagnostic specificity. Recent advancements in technology have

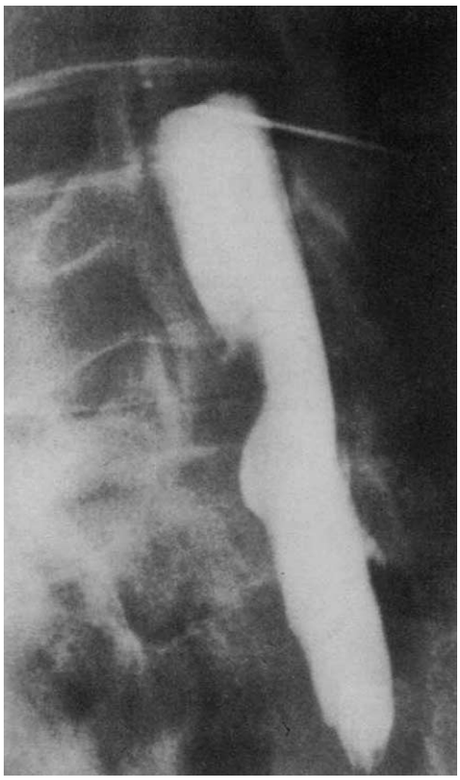

FIGURE 1-10

Pantopaque myelogram showing disk prolapse. (From Raj PP, editor: *Practical Management of Pain*. St. Louis, Mosby, 1986, figure 15B-7, p. 164, with permission.)

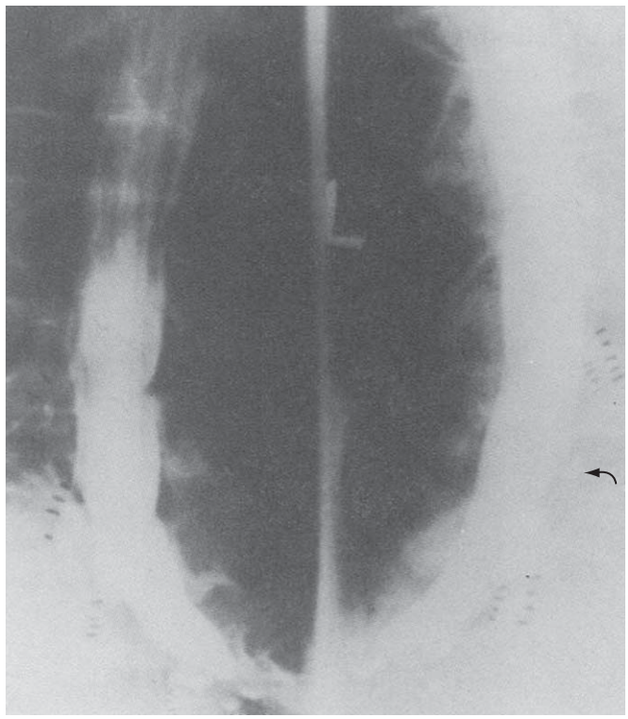

FIGURE 1-11

Metrizamide myelogram showing right L5 nerve root sleeve cutoff. (From Raj PP, editor: *Practical Management of Pain*. St. Louis, Mosby,(see arrow) 1986, figure 15B-8, p. 164, with permission.)

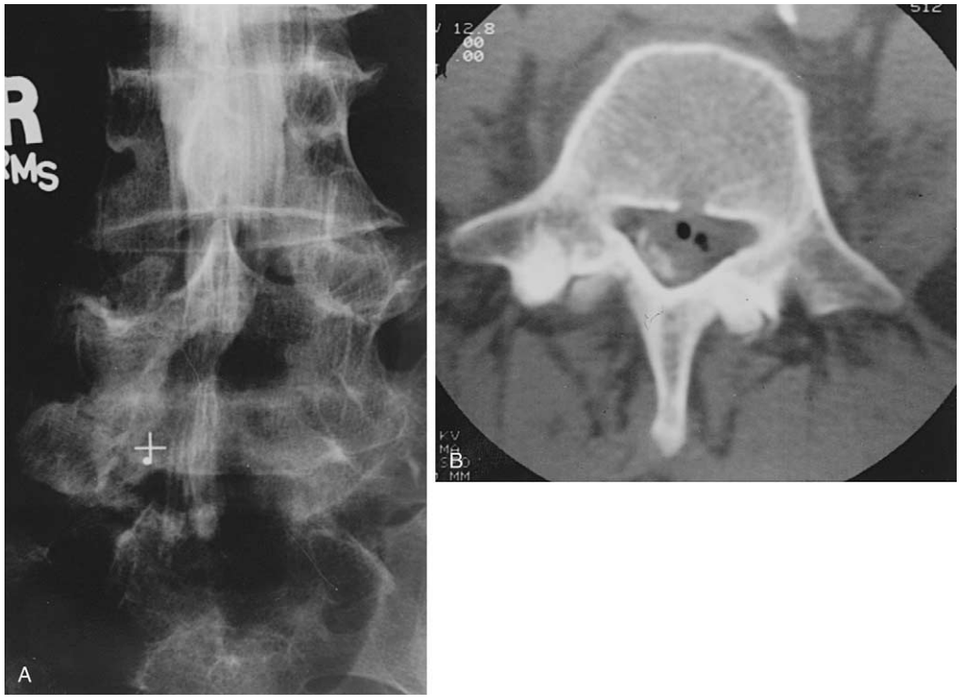

FIGURE 1-12

Epidural hematoma causing compression of the thecal sac. (A) Convention myelogram demonstrates narrowing of the thecal sac at L5 following intrathecal injection of water-soluble contrast. (B) Post-myelography CT shows both the epidural mass and impingement on the subarachnoid space and spinal cord. (From Raj PP, editor: *Practical Management of Pain*, 3rd ed. St. Louis, Mosby, 2000, figure 27-19, p. 397, with permission.)

allowed noninvasive procedures such as CT and MRI to equal the accuracy of myelography in detecting herniated lumbar discs.[17,18]

The most important limitation of myelography is its inability to visualize entrapment of the nerve root lateral to the termination of the nerve root sheath. It is thus unable to detect any far lateral disc herniations, which reportedly account for 1 to 12% of all lumbar disc herniations and occur most often at the L4-L5 and L3-L4 levels.[19,20]

Possible side effects of myelography include dural tear, which can cause headaches, nausea, vomiting, pain or tightness in the back or neck, dizziness, diplopia, photophobia, tinnitus, or blurred vision.[21,22] It is thought that a dural tear can result in a loss of cerebrospinal fluid volume, decreasing the brain's supporting cushion, so that when the patient is standing there is tension on the brain's anchoring structures.[23] A persistent postmyelography headache can be treated with an epidural blood patch, in which 10 to 20 ml of autologous blood is injected into the epidural space under sterile conditions.[24]

Myelography may have significant side effects and should not be undertaken without careful clinical evaluation. Lumbar puncture is obviously an invasive procedure. Pantopaque may induce aseptic inflammatory changes, central nervous system (CNS) hypersensitivity reactions, and arachnoiditis. Water-soluble media, which pass easily into the intracranial space, may induce acute cerebral cortical irritation with headache, seizures, and confusion. Meningeal and radicular irritation may also occur. Major adverse reactions are rare, however, occurring in approximately .02% of patients.[25] Newer nonionic agents (e.g., iohexol or iotrol) may have a decreased incidence of arachnoiditis or CNS side effects,[16,17] as well as lower cost than metrizamide.

Indications for myelography include (1) exclusion of a surgically correctable lesion when the working diagnosis is that of a degenerative process, and (2) localization of the exact level of a lesion before surgery. Evaluation of herniated disk falls into these categories and is a frequent indication for myelography. Characterization of a known lesion or evaluation for multiple lesions may also be performed.

Masses in the spinal cord may appear as local filling defects or obstruction of the opacified subarachnoid space. Herniated disk fragments produce extrinsic impression on the thecal sac, compression of a nerve root, or both of these findings (Figure 1-13). Adhesive arachnoiditis may result from agents introduced into the subarachnoid space, infection, hemorrhage or trauma, or may be idiopathic. Progressive, chronic, poorly localized signs and symptoms may develop. Myelography is an important diagnostic tool in arachnoiditis, demonstrating irregular filling defects, fusion of nerve roots, absent filling of nerve root sleeves, and constriction or obstruction of the thecal sac.[25]

CT is the appropriate study of choice in the evaluation of many intrathoracic and intra-abdominal processes.

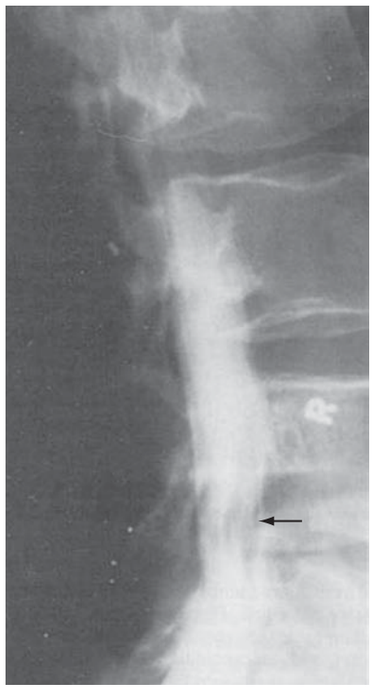

FIGURE 1-13

Metrizamide myelogram demonstrates localized impingement on the contrast filled thecal sac (arrow) by the herniated nucleus pulposus. (From Raj PP, editor: *Practical Management of Pain*, 2nd ed. St. Louis, Mosby, 1992, figure 12-3, p. 188, with permission.)

Other indications include the preliminary assessment for trauma, without the need for patient repositioning. Although coronal CT images of the head or an extremity can be acquired directly, this acquisition frequently comes at the cost of great discomfort to the patient.

The advent of MRI has seen a significant decrease in the number of myelograms performed in most institutions. MRI is considered to be superior in both specificity and sensitivity in the evaluation of the spinal cord. However, myelography is still advantageous when:

1. MRI or CT provides no diagnosis despite continued patient symptomatology.
2. The abnormality detected on MRI or CT does not correlate with the clinical picture.
3. The patient is unable to tolerate MRI or CT secondary to pain, claustrophobia, or body habitus.

CONVENTIONAL TOMOGRAPHY

Linear and complex motion tomography are other modalities that have been made somewhat obsolete by recent technological advances yet still have their place in patient evaluation as an adjunct to plain films. Tomography can be used in the evaluation of joint spaces where the anatomical features are complex and are often obscured by overlying structures. Examples include exclusion of an odontoid

fracture in the patient who is unable to cooperate for the open-mouth view or for more detailed examination of a tibial plateau fracture. Conventional tomography can further aid in the evaluation of solitary bone lesions, healing fractures, arthrodeses, and osteotomies.

COMPUTED TOMOGRAPHY

Computed tomography, first introduced in the 1970s, was utilized primarily in neuroradiology, having a dramatic impact on the fields of neurology and neurosurgery. It was not long afterward that CT had a similar impact on body imaging. CT is a computer-based, cross-sectional imaging technique that provides detailed images of virtually any region of the body. Although initially acquired in the axial plane, images can be reformatted in order to obtain sagittal and coronal planes as well. In patients with musculoskeletal problems, reformatted images can be particularly helpful in determining the three-dimensional extent of a fracture (Figure 1-14).

CT is used to complement information obtained from other diagnostic imaging studies such as radiography, myelography, and MRI. The principal value of CT is its ability to demonstrate the osseous structures of the lumbar spine and their relationship to the neural canal in an axial plane. A CT scan is helpful in diagnosing tumors, fractures, and partial or complete dislocations. In showing the relative position of one bony structure to another, CT scans are also helpful in diagnosing spondylolisthesis. They are not as useful as MRI in visualizing conditions of soft tissue structure, such as disc infection. The data used to generate the axial images are obtained in contiguous, overlapping slices of the target area. The axial image data can be reformatted to construct views of the scanned area in any desired plane.

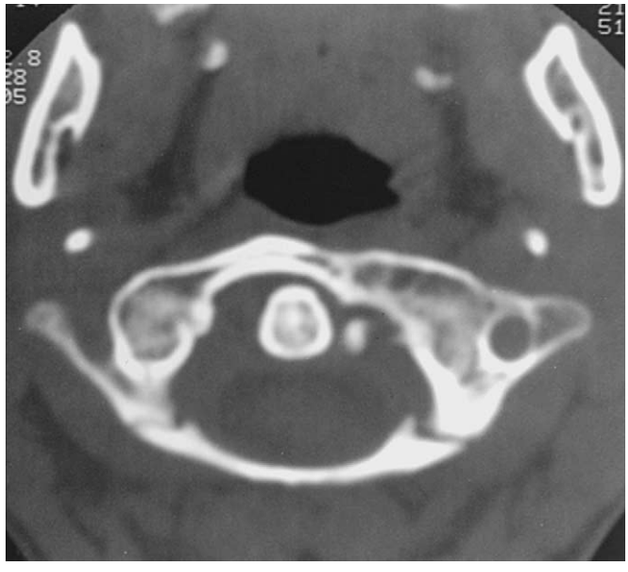

FIGURE 1-14

Jefferson fracture. Axial CT images showing fractores involving the posterior arch of C_1.

The limitations of CT include less-detailed images and the possibility of obscuring nondisplaced fractures or simulating false ones. In addition, radiation exposure limits the amount of lumbar spine that can be scanned, and results are adversely affected by patient motion; spiral CT addresses these weaknesses because it is more accurate and faster, which decreases a patient's exposure to radiation.

MAGNETIC RESONANCE IMAGING

Magnetic resonance imaging has been the premier development in imaging technology in the last two decades. Like CT, MRI is also a computer-based imaging modality that allows cross-sectional imaging with superb anatomic detail. Unlike CT, MRI has no spatial restriction; images can be generated in any plane.

The technology behind MRI takes into consideration the potential of hydrogen nuclei within the body to demonstrate a magnetic moment when placed in a large magnetic field. Electromagnetic radiation in the radiofrequency portion of the spectrum is then applied in order to change the direction of the net magnetization. MRI can produce cross-sectional images that provide similar anatomic information to those obtained through CT scanning. In CT, however, the gray scale spectrum, which represents tissue and body structures, does not change. The densest materials, such as bone and metal, are always white; less dense media, such as fat and air, are black; and soft tissue and fluids are the intermediate gray shades. Manipulation of the pulse sequences and the recording parameters causes differences in the appearance of tissues in MRI. The technology and physics of MRI are quite complex; however, one should have a basic understanding of the science behind MRI in order to fully comprehend the capabilities and applications of this modality. The description that follows is highly simplified. For a more detailed explanation of the principles and the physics of MR, many texts are available that can be consulted.[11,26,27]

The principles behind MRI take advantage of the abundance of hydrogen atoms within the tissues of the body and the effect that a large magnet has on their intrinsic magnetization. Any nucleus that possesses an odd number of protons can be forced to align within a magnetic field, analogous to a small bar magnet. After a new magnetization of the dipoles is established, the magnetic vector is manipulated with a series of radio pulses. This causes the magnetization to tip into a different plane. Upon cessation of the pulses, the nuclei then realign with the larger magnetic field. The time elapsed

until realignment is recorded, and through computer analysis, all the data collected are reconstructed into images.

The process of realignment with the larger magnetic field after the radio pulse has ended is termed *relaxation*.[27] These components are reached at varying rates, depending on intrinsic characteristic of the tissues; each tissue has its own characteristic T1 and T2 relaxation times. Additionally, images can be obtained that emphasize either the T1 or T2 properties. T1 relaxation, also known as spin-lattice relaxation, is dependent on the transfer of energy of the radio pulse from the protons back to the surrounding lattice configuration. Large molecules, such as proteins, and small molecules, such as water, do not exhibit efficient energy transfer; hence, their T1 relaxation times are long. These tissues appear relatively dark on T1-weighted images. Fat, however, has an efficient energy transfer and exhibits short T1 relaxation, causing it to appear bright on images.

As each substance possesses its own T1 and T2 relaxation times, imaging can be performed in such a way to enhance either characteristic—T1-weighted or T2-weighted images, depending on the task at hand.

In general, T1-weighted images illustrate anatomical detail and are well suited for localization of masses and demonstration of mass effect on adjacent structures (Figure 1-15). T2-weighted images, although less aesthetic than T1-weighted images, provide information on many disease processes, such as infection, neoplasm, infarction, and white matter disease, that would not otherwise be well visualized.

FUNCTIONAL MAGNETIC RESONANCE IMAGING

The recent discovery that magnetic resonance imaging can be used to map changes in brain hemodynamics that correspond to mental operations extends traditional anatomical imaging to include maps of human brain function. The ability to observe both the structures and also which structures participate in specific functions is due to a new technique called functional magnetic resonance imaging, fMRI, and provides high resolution, noninvasive reports of neural activity detected by a blood oxygen level–dependent signal.[28–33] This new ability to directly observe brain function opens an array of new opportunities to advance our understanding of brain organization, as well as a potential new standard for assessing neurological status and neurosurgical risk. The following briefly introduces the fundamental principles of fMRI, current applications at Columbia, and some potential future directions.

Functional MRI is based on the increase in blood flow to the local vasculature that accompanies neural activity in the brain. This results in a corresponding local reduction in deoxyhemoglobin because the increase in blood flow occurs without an increase of similar magnitude in oxygen extraction.[34–37] Since deoxyhemoglobin is paramagnetic, it alters the T2*-weighted magnetic resonance image signal.[28–33,38,39] Thus, deoxyhemoglobin is sometimes referred to as an endogenous contrast enhancing agent, and serves as the source of the signal for fMRI. Using an appropriate imaging sequence, human cortical functions can be observed without the use of exogenous contrast enhancing agents on a clinical strength (1.5 T) scanner.[40–44]

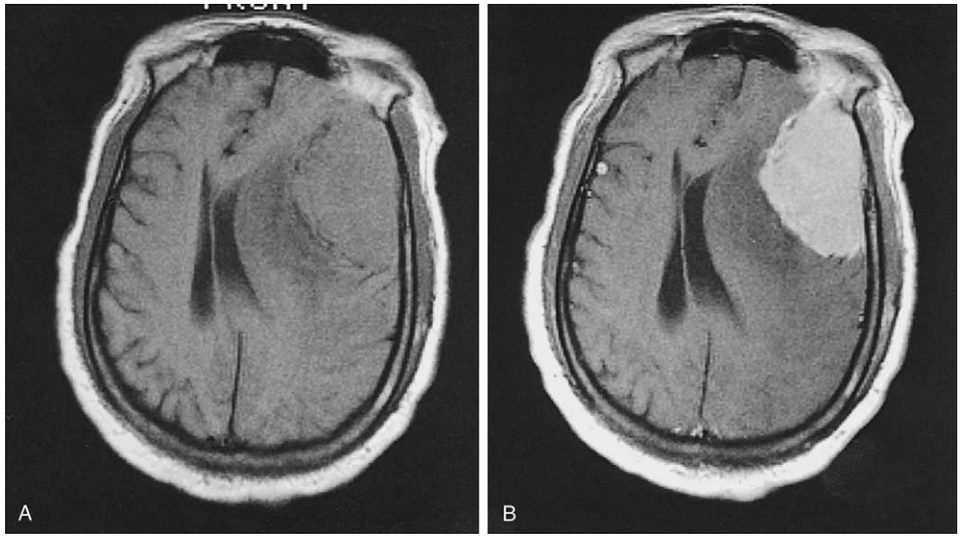

FIGURE 1-15

Meningioma. (A) Axial T1-weighted image shows a large isointense mass in the left frontoparietal region causing midline shift and effacement of the sulci and ventricles. (B) A T1-weighted post-contrast image shows intense enhancement of the meningioma. (From Raj PP, editor: *Practical Management of Pain*, 3rd ed. St. Louis, Mosby, 2000, figure 27-12, p. 393, with permission.)

FUTURE ROLE OF FUNCTIONAL MAGNETIC RESONANCE IMAGING IN PAIN MANAGEMENT

The experience of chronic and persistent pain is a debilitating condition for which the role of cortical processing is not well understood. We have focused on the identification of cortical areas that are modified by the reduction of pain following pain therapy. This novel approach to investigate the cortical representation associated with relief of pain has originated from our pilot studies where patients with chronic sympathetically maintained pain affecting one extremity (postherpetic neuralgia) were studied by comparing brain responses to light touch applied to the "now-affected" limb and to the "painful" limb before and after treatment.[45] These studies indicate that the cortical representation of sympathetically maintained pain involves specific and identifiable cortical activity, as well as does the relief of that pain achieved by a peripheral nerve block procedure. Continuing investigations will extend these findings to other pain treatments to determine the extent to which this finding is generalizable to other approaches using fMRI to investigate cortical representations of specific pain types, and therefore, new specific therapy options.

NUCLEAR MEDICINE

Bone Scanning

Radionuclide bone scanning has long been well known for its high degree of sensitivity in the detection of a variety of bone lesions. In recent years, applications of bone scanning to various orthopedic, traumatic, neoplastic, and infectious processes have proven the usefulness of this modality in the detection of clinically significant but often radiographically elusive problems.

The value of bone scanning, as in many nuclear imaging studies, lies in its ability to reflect physiological changes rather than anatomic detail. While other radiographic modalities excel in providing well-defined, precise anatomic information, nuclear scans by their inherent nature are not able to define small structures or provide high-resolution images. In fact, radiographs and bone scanning often provide complementary information in evaluation of skeletal pain. The greatest value of bone scanning is its high degree of sensitivity for detection of early or subtle bone abnormalities, and for this purpose the technique of bone scanning is not likely to become obsolete in the face of new anatomically oriented modalities.

Physiological Basis for Bone Scanning

Technetium-labeled, bone-seeking radiopharmaceuticals localize in bone by exchange or adsorption onto the hydroxyapatite component of bone. The amount of deposition of tracer is affected by two factors: (1) the rate of local repair and remodeling of bone[46] and (2) local skeletal blood flow, which delivers the tracer to the extracellular space, thus making it available for local exchange and adsorption.[47]

Any active process in bone that results in a local increase in bone turnover results in a larger surface area of bone available for tracer accumulation. Thus, a region of active bone turnover such as normal growth plate, fracture, tumor, infection or any other active process results in locally increased tracer deposition or a "hot spot." At the same time, any process that results in increased blood flow (e.g., cellulitis or sympathectomy) will locally increase tracer deposition based on increased tracer delivery to the site,[47] also resulting in a "hot" area on bone scan.

Technique

Bone scanning is performed following IV injection of 99m technetium-methylene diphosphonate (99mTc-MD) or similar diphosphonate compound. Approximately 2 to 3 hours after injection, scintigraphic images are obtained with a gamma camera to include the specific areas of interest or the entire skeleton.

In some cases, a "three-phase bone scan" may be utilized:

1. A radionuclide angiogram, or flow study, over the area of interest is obtained during the IV injection of tracer. As the tracer passes through the arterial circulation, rapid-sequence images are obtained to evaluate vascularity to the region being scanned.
2. The "blood pool" image, obtained immediately after the flow study, displays regional perfusion including that of soft tissues.
3. The routine, delayed "static" images, taken after 2 to 3 hours, demonstrate active bony abnormality, reflected as locally increased deposition of tracer in the skeleton.

Comparison of early (flow and blood pool) phases with the delayed (static) phase may yield useful information about inflammatory processes and other vascular changes such as those found in reflex sympathetic dystrophy (complex regional pain syndrome [CRPS]).

The radiation dose to the patient is relatively low, giving a whole body dose of approximately 0.02 rad per mCi administered.[48] Assuming a usual adult dose of 20 mCi, a total body dose of 0.4 rad per examination is obtained. This is less than the radiation dose from lumbar spine series.[49] Since iodinated contrast material is not used, side effects and allergic contrast reactions do not occur.

ULTRASONOGRAPHY

Diagnostic ultrasound is a widely used method of body imaging. A transducer converts an electrical pulse into a high frequency sound pulse, which is then transmitted through soft tissues of the body. Ultrasound is based on the amplitude of the refracted sound wave as it returns to the receiving transducer. The amplitude of the sound wave and, therefore, the echogenicity of a structure on images

are dependent on mass density and speed of sound through that particular substance. Because of the difference in the speed of sound between fluid and soft tissue, ultrasound provides accurate information regarding the cystic and solid nature of the lesion (Figure 1-16). Transducers employed in diagnostic imaging emit pulses within the 1- to 10-MHz range.

Ultrasound evaluation of the body can be severely hampered by very dense material, such as bone and gas containing organs (e.g., bowel). Sound waves do not conduct well through either medium.

Acquisition of good images in sonography is also largely a function of the ultrasonographer's technique, skill, and experience. Ultrasound is an excellent modality for evaluation of the liver, breast, soft tissue masses, and vascular structures of the neck and extremities. Doppler ultrasonography allows documentation of blood flow in the vessel, as well as direction and velocity of flow. This makes possible the evaluation of suspected carotid arterial stenosis or evaluation of DVT. Doppler examination of patency of venous structures of the thigh (84%) is much more sensitive than in the lower leg (25%).

Ultrasound is one of the most important and most rapidly progressive imaging modalities. By using an ultrasound beam that is transmitted and reflected from tissues, images are obtained without patient discomfort and without using ionizing radiation.

Ultrasound excels in characterizing tissues based on their echogenicity or reflective characteristics. Fluid can easily be distinguished from solid tissue in the kidney, liver, or thyroid, allowing the differentiation of cysts from solid masses. Fluid-containing structures (e.g., the common bile duct and renal collection systems) are likewise well delineated. Gallstones are identified clearly; ultrasound has replaced the oral cholecystogram in the evaluation of suspected gallbladder disease. Using the fluid-filled bladder as a "window," the uterus and adnexa are easily visualized. The lack of ionizing radiation makes ultrasound examination ideal for women of child-bearing age and children since no adverse effects have been demonstrated.

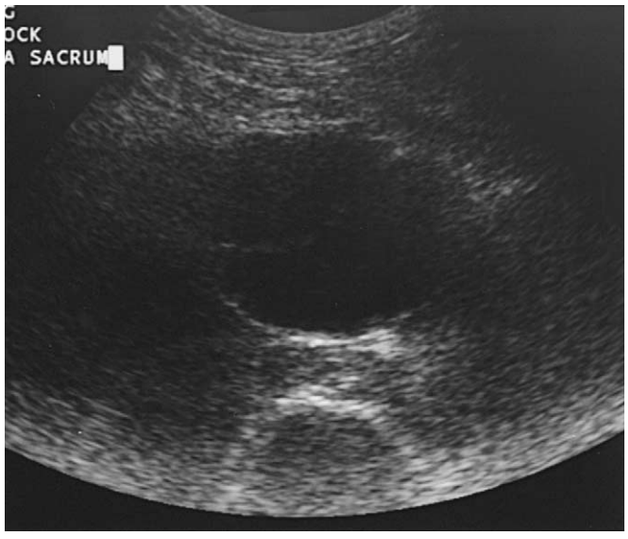

FIGURE 1-16

Soft tissue abscess. Grayscale ultrasound images show a complex cystic mass with septations and internal debris representing recurrent abscess in the soft tissues surrounding the sacrum. (From Raj PP, editor: *Practical Management of Pain*, 3rd ed. St. Louis, Mosby, 2000, figure 27-26, p. 403, with permission.)

Masses or abscesses may be visualized provided these are located in the pelvis or upper abdomen, where bowel gas does not interfere with transmission of the sound beam. Bone and air do not adequately transmit sound, precluding evaluation of the chest, mid abdomen, and musculoskeletal system.

Doppler flow scanning, in conjunction with ultrasound imaging of the vascular system, has become popular for the detection of arterial occlusions and venous thrombosis. New transcranial Doppler is being applied to cerebrovascular disease. Ultrasound has gained wide popularity with both patients and physicians and will certainly remain one of the most versatile and informative modalities in future years.

EPIDUROGRAPHY

Sanford and Doub introduced air into the epidural space in 1941.[50] Injection of contrast to improve visualization of the epidural space and nerve root sleeve to delineate the pressure change from disk protrusion was first reported in 1963.[51] Its use is suggested when myelography is equivocal or normal, especially when the L5-S1 epidural space is wide (Figure 1-17). Gupta correlated epidurograms with clinical and operative findings in 255 patients with spinal disorders.[52] In most cases, the caudal approach was used to facilitate the study of the sacral canal. The dispersion of the solution in the epidural space was directly proportional to the speed of injection. When 80 ml of contrast material was used, the cervical space was visualized without tilting the table. Contrast material dispersal was symmetric in 88.2% of cases and was not related to the direction of the tip of the needle. There was a negative correlation between epidurographic findings and surgical findings in 10% of patients. Gupta found epidurography useful for repeated visualization of the epidural space following surgery for spinal compression.

Some adverse reactions can occur on epidurography. In Gupta's study, 56% of patients complained of severe backache and 82 of 90 patients with prolapsed disk complained of sciatica. The pain lasted for 10 minutes and did not require treatment. Water-soluble agents such as meglumine iothalamate can produce CNS toxicity. Patients may experience muscle twitchings, difficulty breathing, and clonic convulsions. The treatment for such cases is intravenous diazepam with barbiturates for 24–48 hours. Usually patients recover fully after this period. Metrizamide has been shown to be the least toxic agent to the CNS and is now routinely recommended for epidurograms.

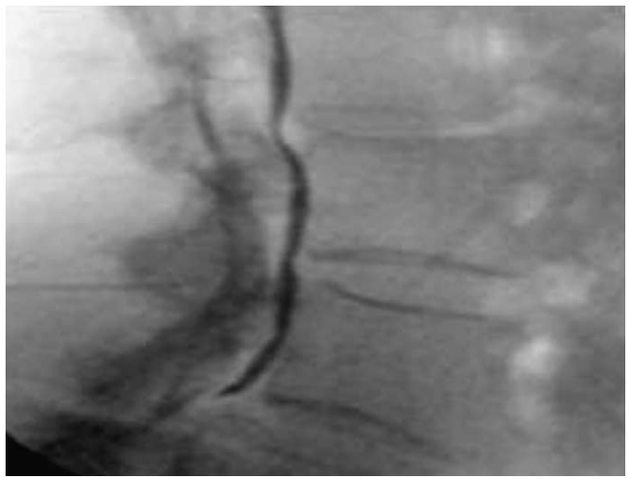

FIGURE 1-17

Lateral view lumber epidurogram showing contrast material in the epidural space.Note disc bolge at L_{3-4}.(From Raj PP, editor: *Practical Management of Pain*. St. Louis, Mosby, 1986, figure 15B-14, p. 167, with permission.)

UTILIZATION OF CONTRAST AGENTS

Up to a third of all radiological imaging utilizes contrast material either systemically or locally for improved visualization of vessels, organs, body cavities, or pathological processes.

Barium sulfate, an inert substance, is the standard gastrointestinal contrast agent used to visualize the esophagus, stomach, small and large bowel, and rectum in examinations such as barium enemas and enteroclysis, as well as for opacification of the gastrointestinal system during CT examinations. Double-contrast or single-contrast techniques can be employed for visualization of the mucosa, gross anatomy, and motility. Water-soluble contrast material can be substituted for barium for delineation of bowel perforation, fistulae, or swallowing abnormalities, leading to aspiration.

Intravenously injected iodinate contrast material is excreted by the kidneys and thus provides excellent visualization of the renal collecting system, ureter, and bladder, as well as the renal parenchyma. The excretory urogram, also known as an *intravenous pyelogram*, is frequently used for initial evaluation of the patient with suspected urolithiasis. Oral cholecystography once was the "gold standard" for the diagnosis of cholelithiasis. Orally ingested contrast excreted by the biliary system allowed visualization of the gallbladder and its contents. This technique has largely been made obsolete by ultrasonography.

Intravenously injected contrast medium is also employed in CT examinations. Enhancement of vessels and organs with contrast improves visualization; contrast material is used to assess tumor vascularity, organ and tissue perfusion, and the renal collecting system.

The U.S. Food and Drug Administration (FDA) has approved gadolinium-containing agents for the purpose of contrast-enhanced MRI; this has significantly increased the sensitivity and specificity of the modality.[10,11,27,53–56] Gadolinium is a paramagnetic contrast agent that causes focal irregularities in the magnetic field, with a resultant shortening of T1. Thus, enhancing tissues appear bright on T1-weighted, contrast-enhancing tissues appear bright on T1-weighted, contrast-enhanced images (Figure 1-18). These agents contain no iodine and are safe for use in patients with iodine allergy.[27,53] Additionally, gadolinium is not nephrotoxic and can be used in those patients with renal failure.

Scanning times are relatively short compared with those for CT, and the tight confines of the scanner itself can pose problems for obese patients or for patients with claustrophobia. Critically ill patients on life support systems cannot be accommodated unless the systems are nonferromagnetic. Technical limitations of MRI include the inability to detect bone detail and calcification.

CLINICAL APPLICATION OF VARIOUS IMAGING MODALITIES

IMAGING BASED ON TISSUE TYPE AND DIAGNOSIS

Given the multitude of imaging modalities available to the clinician and their differences in sensitivity and specificity, examination should be selected according to the type of

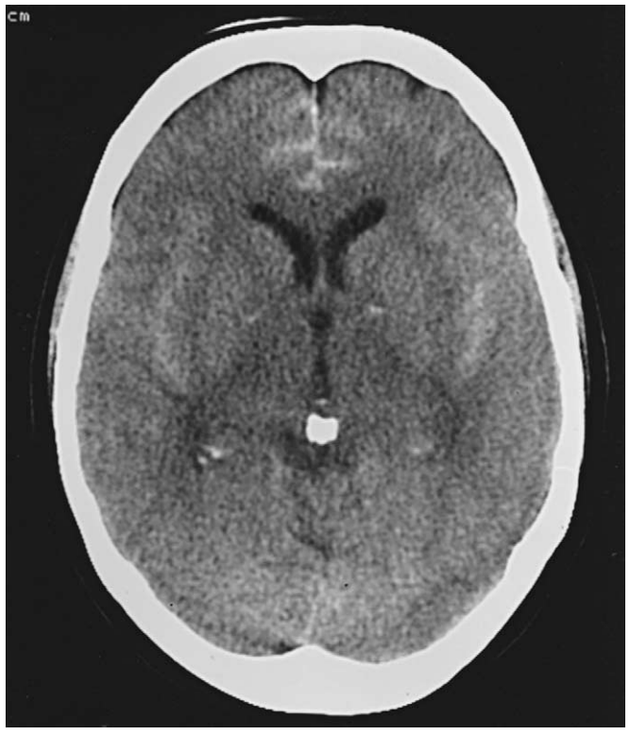

FIGURE 1-18

Subarachnoid hemorrhage. Axial CT demonstrates blood along the anterior falx and in the sulci along the convexities secondary to rupture of anterior communicating artery aneurysm (see also Figure 1-19). (From Raj PP, editor: *Practical Management of Pain*, 3rd ed. St. Louis, Mosby, 2000, figure 27-27, p. 403, with permission.)

tissue warranting the evaluation, as well as the presumptive diagnosis.

HEAD AND NECK

Evaluation of particular areas of the body, however, is better served by newer modalities such as CT and MRI.[27] Instead, plain films are most often ordered to resolve a question raised by CT or MRI. Additionally, facial bone films have become nearly obsolete because the anatomical definition of CT is superior.

Severe and sudden-onset headaches are most efficiently and effectively evaluated by CT to exclude subarachnoid hemorrhage (Figure 1-19) or other intracranial hemorrhage as the cause[11,27,57] for patients presenting with subacute, chronic, and unremitting headache, the most appropriate initial study is MRI.

Facial pain that is presumed to originate from sinusitis is best assessed with CT; facial bone fractures are also best evaluated in this manner. Panorex studies and MRI imaging of the temporomandibular joint are also appropriate for the evaluation of orofacial pain.[58,59]

NECK AND UPPER EXTREMITY

Causes of neck and upper extremity pain and discomfort are multiple. Pain can originate from soft tissues, spinal cord or nerve roots, or musculoskeletal structures, or it can be referred from viscera. Naturally, degenerative disease of the cervical spine and herniated or bulging disks are the culprits behind the majority of complaints, causing nerve root compression with resultant neck and upper extremity numbness, weakness, and pain.

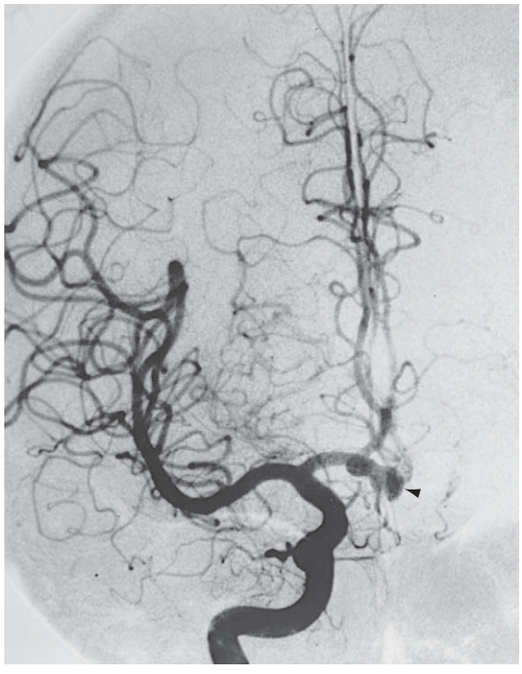

FIGURE 1-19

Cerebral angiography shows anterior communicating artery aneurysm (arrowhead) in a patient with subarachnoid hemorrhage. (From Raj PP, editor: *Practical Management of Pain*, 3rd ed. St. Louis, Mosby, 2000, figure 27-20, p. 398, with permission.)

Following initial plain films, MRI is the study of choice for further evaluation. It is superior in demonstrating not only disk disease and cervical spondylosis but also primary or metastatic lesions within the cord and spinal canal. Joint pain of the upper extremity is also best evaluated with MRI to follow up the initial plain films.

As already mentioned, arthrography can also be used alone or in conjunction with MRI for evaluation of entities such as rotator cuff tears, scapholunate dissociation, or glenoid labrum lesions.

CHEST AND ABDOMEN

Chest and abdominal pain is frequently addressed by the emergency department physician or primary care physician. Complaints due to gastroesophageal reflux disease, angina, peptic ulcer disease, pancreatic disease, prostate disease, or renal abnormalities are but a few examples from a host of disorders not seen by the pain physician until they have become chronic.

In the initial evaluation of chest and abdominal pain, plain films can be advantageous in ruling out processes such as pneumothorax, congestive heart failure, free intraperitoneal air from ruptured viscus, or calcifications in a distribution, which suggest cholelithiasis or urolithiasis. In the absence of findings on plain film or other diagnostic studies, such as echocardiography or electrocardiography (EGG), CT, ultrasound, or gastrointestinal contrast examinations are the next recommended course of action (Figure 1-20).

As previously noted, evaluation of the soft tissues with vascular structures is also accomplished with MRI. This includes visualization of the articular cartilage, ligaments and tendons, and bursal spaces. Hence, MRI is the study of choice for evaluation of joints and joint pathological processes (Figure 1-21).[26,60,61]

LOW BACK PAIN

The causes of low back and lower extremity pain are almost innumerable. Low back pain is a common complaint among patients of all ages and patients in both acute care and pain clinic settings. Causes include, but are not limited to, degenerative disease of the spine or hips, nerve root compression, referred visceral pain, pathology affecting the nerves, musculoskeletal pain, lower extremity joint disease, soft tissue pathological processes, or myofascial pain.

Low back pain is known to affect 80% of adults during their lifetime.[62] Spinal disorders represent the most common cause of disability among workers in the United States and account for the chief medical condition on which health care dollars are spent.[45]

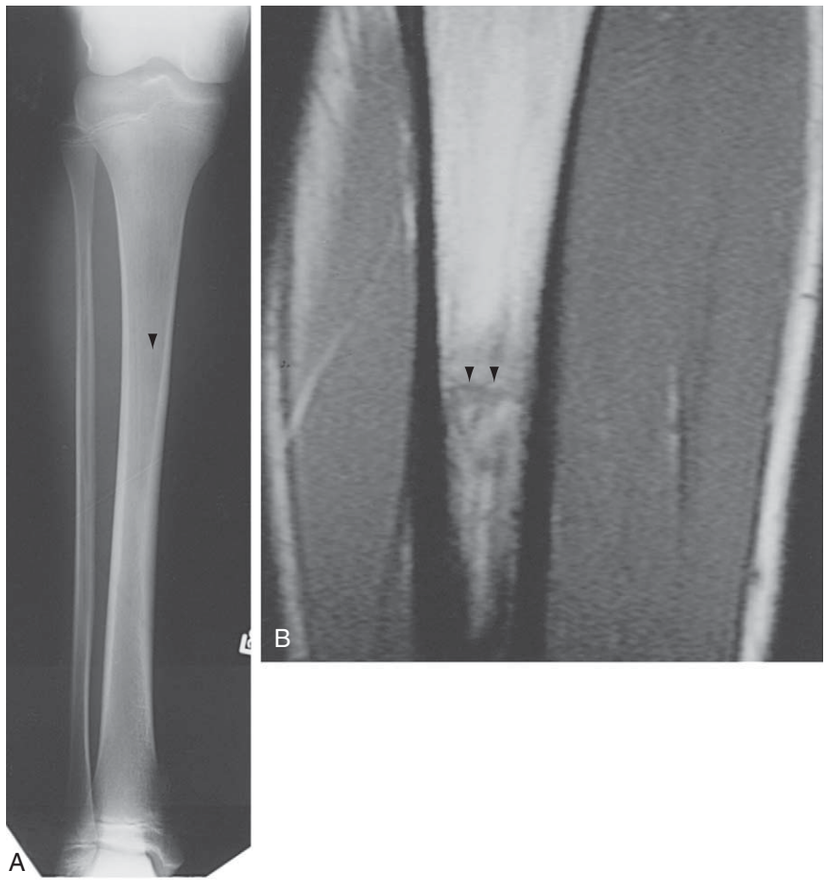

FIGURE 1-20

Stress fracture of the tibia. (A) Frontal area plain film of the tibia and fibula was nondiagnostic. A subtle linear area of increased density is seen in the midshaft of the tibia (arrowhead). (B) Coronal T1-weighted image shows a linear area of decreased signal intensity (arrowheads) consistent with stress fracture surrounding marrow edema. (From Raj PP, editor: *Practical Management of Pain*, 3rd ed. St. Louis, Mosby, 2000, figure 27-15, p. 395, with permission.)

Degenerative changes of the spine are the primary reason for spinal imaging. CT,[46] MRI,[10,11,53,55,64] and myelography[10,12,13,15] all provide valuable information in the evaluation of a patient with back pain. As in the assessment of intracranial lesions, the development of MRI has drastically improved the ease and accuracy of diagnosis and management of patients and has become the primary modality for diagnosis of low back pain.[10,11,53,55,64]

MRI has advantages over both CT and myelography in the evaluation of degenerative disk disease.[64] The multiple terms commonly used to describe degenerative disk disease are frequently poorly understood and used incorrectly. For example, the terms "bulging" disk and "herniated" (protruded, extruded, or sequestered) disk cannot be used interchangeably. Furthermore, the distinction has considerable effect on patient treatment, as follows:

- A bulging disk extends past the cortical margins of the adjacent vertebral bodies.[64]
- In a herniated disk, a defect in the annulus fibrosis allows extension of the nucleus pulposus through this defect producing a focal extension of the margin of the disk. The herniated nucleus pulposus is still attached posteriorly by some uninterrupted fibers.[64]
- In an extruded disk, no annular fibers remain intact. The nuclear material bulges into the spinal canal or intervertebral foramen (Figure 1-22).[64]
- A sequestered disk describes disk material that is no longer contiguous with the remaining nuclear material. This fragment can be located anterior or posterior to the posterior longitudinal ligament.[13]

Impingement on nerve roots or the spinal canal by bony structures is better evaluated with CT.

MRI can also be used to evaluate the extent of spinal trauma by an assessment of the condition of the ligaments and spinal cord and the presence or absence of hematoma (Figure 1-23).[65] MRI also demonstrates spinal alignment and the relationship of the vertebrae to the cord. MRI is superior to other modalities in detection of infection involving the cord, disks, or vertebral bodies.[56] It is also the

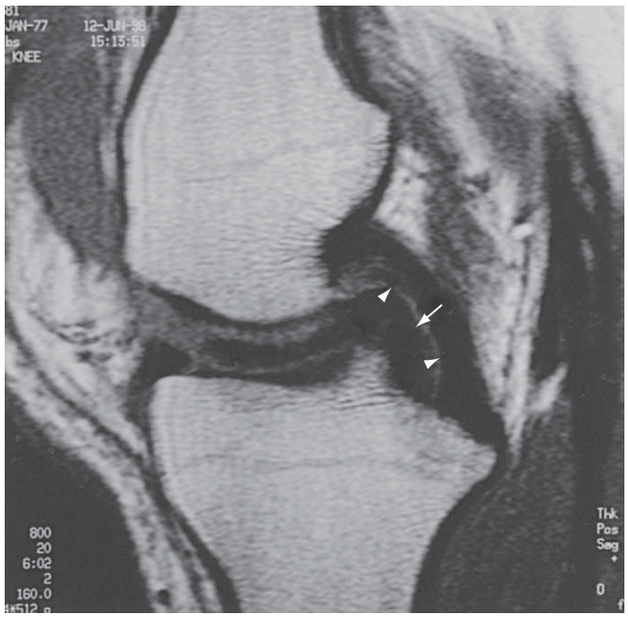

FIGURE 1-21

Bucket-handle meniscal tear. Sagittal T1-weighted image of the knee with the "double PCL sign." Normal posterior collateral ligament (PCL) (arrowheads) is intact; the torn and displaced meniscus is seen anteriorly (arrow). (From Raj PP, editor: *Practical Management of Pain*, 3rd ed. St. Louis, Mosby, 2000, figure 27-16, p. 396, with permission.)

method of choice for evaluation of intramedullar, intradural-extramedullary, and extradural lesions.[55,64]

In patients with facet syndrome, bone scans have not proven useful to determine levels at which facet blocks may be successful. Despite the good sensitivity of bone scanning, scan abnormalities in this clinical setting usually correspond to radiographically obvious degenerative changes and do not correlate with clinical outcome of facet blocks.[66]

Following lumbar spinal fusion, postoperative back pain may result from pseudoarthrosis or from other causes such as degenerative disease, musculoskeletal pain, or spinal stenosis. Radiographs may not demonstrate the failure of fusion, even with flexion-extension views, in which mobility may be precluded by metallic fixation. Bone scanning, augmented by single photon emission computed tomography, which provides tomographic views without superimposition of multiple structures, may be more sensitive than radiography in detecting focal areas of increase tracer uptake indicative of local bone reaction to pseudoarthrosis.[67]

Metastatic disease, fracture, and infection in the spine are frequent causes of back pain. Although routine bone scans may appear similar in all these entities, enhanced resolution by pinhole collimation may more precisely localize abnormal tracer uptake within the vertebra, allowing some differentiation between these conditions.[62]

Patients with hematogenous vertebral osteomyelitis may present with nonspecific back pain. Whereas radiographs may be normal or demonstrate only the advanced stages of vertebral and disc space destruction, bone scans

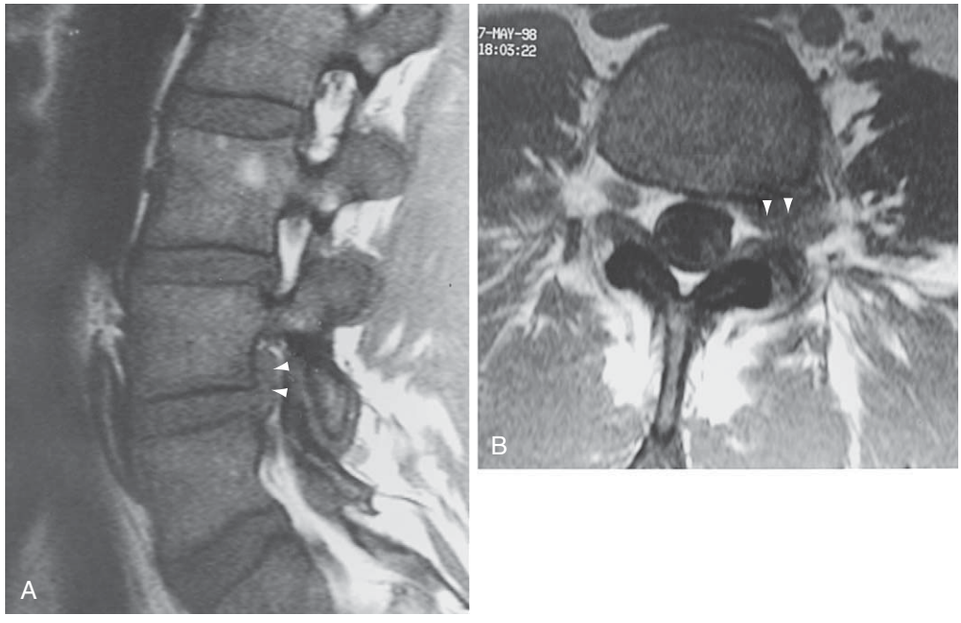

FIGURE 1-22

Left lateral disk herniation. (A) Sagittal T1-weighted images showing left lateral disk herniation into the neural foramen at L4-L5 with compression of the L4 nerve root (arrowheads). (B) Axial T1-weighted image illustrating left lateral disk herniation (arrowheads). (From Raj PP, editor: *Practical Management of Pain*, 3rd ed. St. Louis, Mosby, 2000, figure 27-28, p. 404, with permission.)

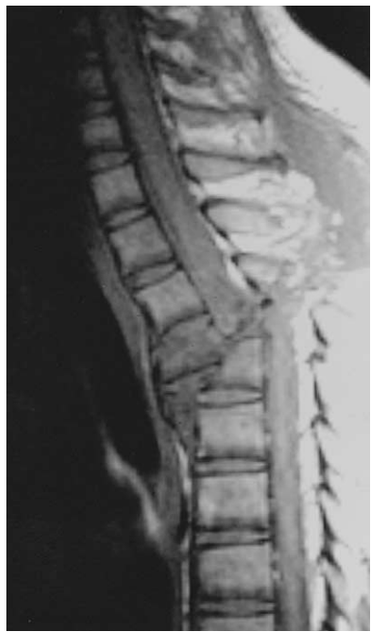

FIGURE 1-23

Traumatic spinal cord transsection. A T-1 weighted sagittal MR image demonstrates fracture dislocation at T3-T4 with complete transsection of the spinal cord. Decreased spinal intensity is noted in the involved vertebral bodies consistent with marrow edema. (From Raj PP, editor: *Practical Management of Pain*, 3rd ed. St. Louis, Mosby, 2000, figure 27-29, p. 405, with permission.)

have been reported to be 100% sensitive for this condition even in the early stages of the infection.[63]

FAILED BACK SURGERY SYNDROME

Failed back surgery syndrome (FBSS) is a large contributor to both medical and socioeconomic problems in the United States. More than two thirds of those patients enrolled in pain centers complain of FBSS. It is for that reason that additional consideration is given to this disorder.

By definition, FBSS is the persistence of back or leg symptoms following lumbar surgery. Any patient with multiple previous low back surgeries should be evaluated using a systematic and uniform approach to differentiate between low back pain and leg symptomatology.

First, there must be a differentiation between mechanical causes of FBSS. Mechanical lesions such as spinal stenosis, recurrent disk, or spinal instability can cause compression of the adjacent cord or nerve roots. These problems can often be corrected with an additional surgical procedure.[68] Nonmechanical lesions include epidural fibrosis or arachnoiditis, psychosomatic pain, and systemic medical illness; these entities are not amenable to treatment by additional surgery.[68]

Causes and prevalence of FBSS include the following:[69—72]

- Recurrent disk herniation (12–16%)
- Stenosis not identified in the preoperative period (central spinal stenosis, 7–40%)
- Lateral recess stenosis (50%)
- Epidural fibrosis (6–8%) (Figure 1-24)
- Arachnoiditis (6–16%)

Additional entities include postoperative bone overgrowth, postoperative nerve injury, pseudomeningocele, postoperative spondylolisthesis, operation at the wrong level, incomplete removal of herniated disk, or unsuccessful spinal fusion.[70] Pregadolinium and postgadolinium enhanced MRI is the method of choice for evaluating FBSS.[63,66]

PLAIN RADIOGRAPHY

Traditionally, the plain radiograph has been the first imaging test performed in the evaluation of low back pain because it is relatively inexpensive, widely available, reliable, quick, and

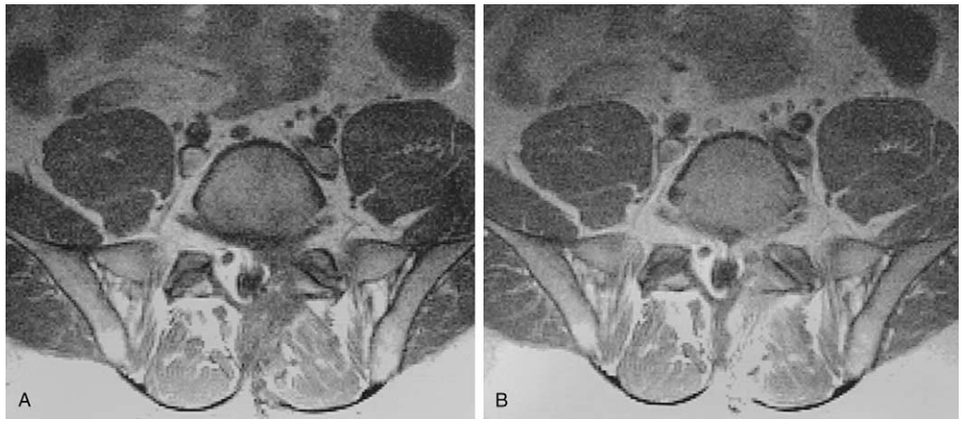

FIGURE 1-24

Epidural fibrosis. Pre-contrast (A) and post-contrast (B) enhanced axial T1-weighted images show significant epidural fibrosis involving the left lateral aspect of the spinal canal following lumbar spinal surgery. Postgadolinium images show characteristic enhancement of the scar tissue. (From Raj PP, editor: *Practical Management of Pain*, 3rd ed. St. Louis, Mosby, 2000, figure 27-30, p. 406, with permission.)

portable. Two major drawbacks to radiography are difficulty in interpretation and an unacceptably high rate of false-positive findings.[54] Plain radiographs are not required in the first month of symptoms unless the physical examination reveals specific signs of trauma or there is suspicion of tumor or infection.[73] It is important to obtain pictures that are free of motion or grid artifacts and that display soft tissue and osseous structures of the entire lumbar spine.

Having a standard approach to evaluating radiographs can help prevent a missed diagnosis; it is crucial to develop and maintain a specific sequence of observation. The traditional sequence includes anteroposterior (AP) and lateral views of the lumbar spine, primarily to detect tumors or spinal misalignments such as scoliosis. In the AP view, indicators of a normal spine include vertical alignment of the spinous processes, smooth undulating borders created by lateral masses, and uniformity among the disc spaces.

Misalignment of the spinous processes suggests a rotational injury such as unilateral facet dislocation. The AP view of the lumbar spine should include the whole pelvis; this allows for evaluation of the acetabulum and femoral heads and for the detection of possible degenerative changes to the pelvis.

The lateral view (Figure 1-25) provides a good image of the vertebral bodies, facet joints, lordotic curves, disc space height, and intervertebral foramen. Decreased disc space height can be indicative of disc degeneration, infection, and postsurgical condition. Unfortunately, there is poor correlation between decreased disc height and the etiology of low back pain.

The standard anteroposterior (AP), lateral, and oblique projections are the ones most commonly used in screening the cervical, thoracic, and lumbosacral spine and various joints of the body. Spot films coned to the area of pathology (e.g., angled AP and oblique views of the sacroiliac joints) may supplement the standard views, if clinically indicated.

Readily diagnosed by these studies are cases of ankylosing spondylitis with bamboo spine appearance, spondylolisthesis, a defect of the pars interarticularis, disk space narrowing, sclerosis of adjoining end-plates, fractures, anomalies, osteo-phytes, osteolytic neoplasms, scoliosis, abnormalities of joints, calcification, arthritis, and derangements of the joints (Figures 1-26 through 1-29).

Standard plain radiography may be supplemented by complex motion tomography to improve assessment of interosseous lesions or anatomical alignment of fractures.

Unfortunately, there are limitations to these studies. Limitations include significant radiation exposure, increased pain during the study (the patient must be in an uncomfortable difficult position), poor detail of the region under study, and absence of soft tissue for radiographic detail.[74–79]

Bone metastases normally appear as multiple foci of increased tracer uptake asymmetrically distributed (Figure 1-30). In extreme cases of bone metastases, diffusely increased uptake of tracer results in every bone being

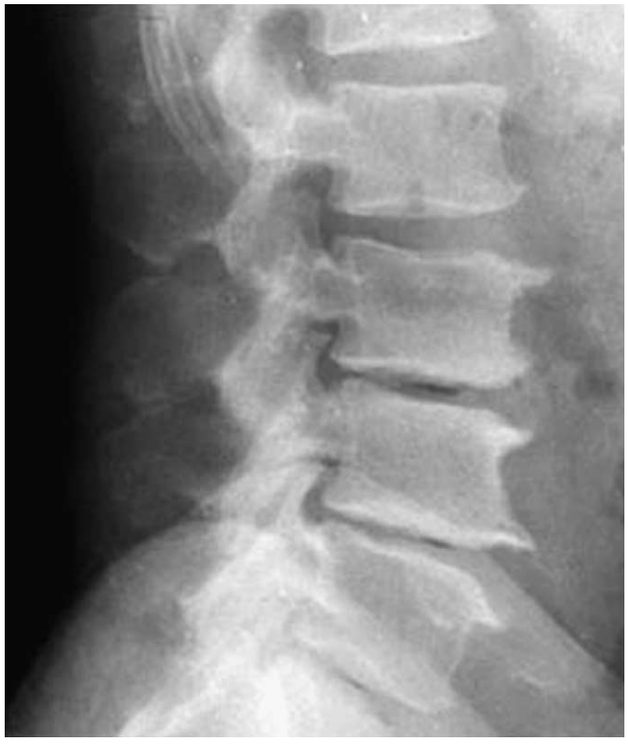

FIGURE 1-25

Sagittal lumbar spine radiograph showing degenerative changes (including decreased disc space heights and osteophyte formation) that are commonly found in patients of increased age. (From Humphreys SC, Eck JC, Hodges SD: Neuroimaging in low back pain. *Am Fam Physician* 65: 2217–2218, 2002, figure 3, with permission.)

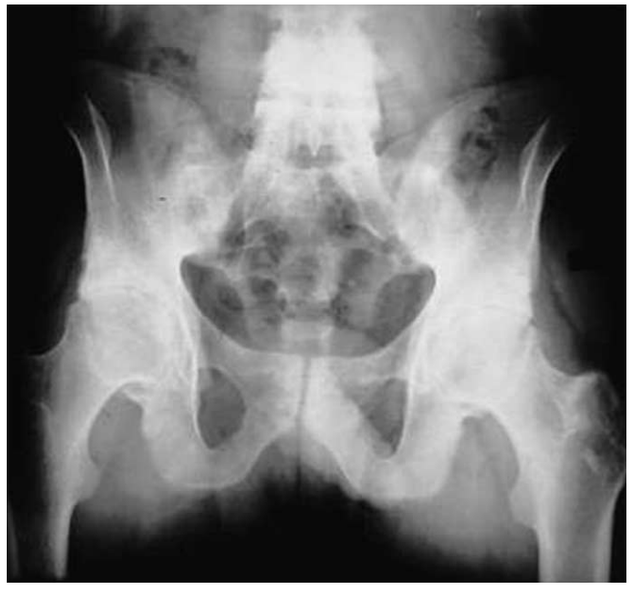

FIGURE 1-26

Plain radiograph showing a kylosing spondylitis in the sacroiliac joints. (From Raj PP, editor: *Practical Management of Pain*. St. Louis, Mosby, 1986, figure 15B-1, p. 161, with permission.)

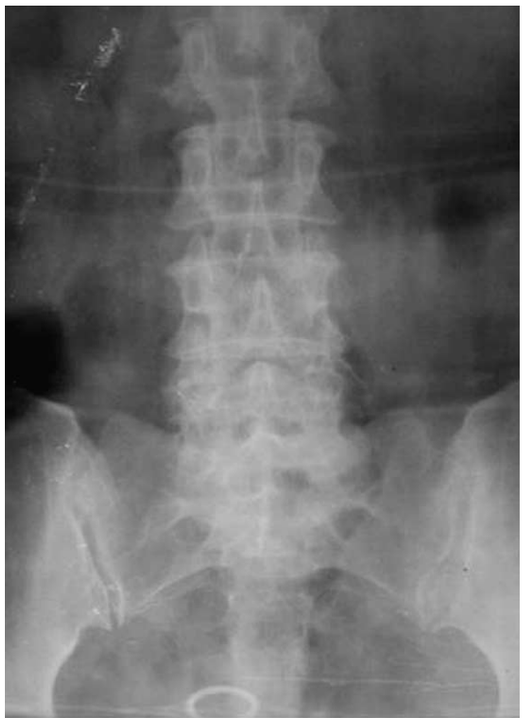

FIGURE 1-27

Plain radiograph showing severe degenerative arthritis of the lumbar spine at L4-L5 and L5-L1 levels. (From Raj PP, editor: *Practical Management of Pain*. St. Louis, Mosby, 1986, figure 15B-2, p. 161, with permission.)

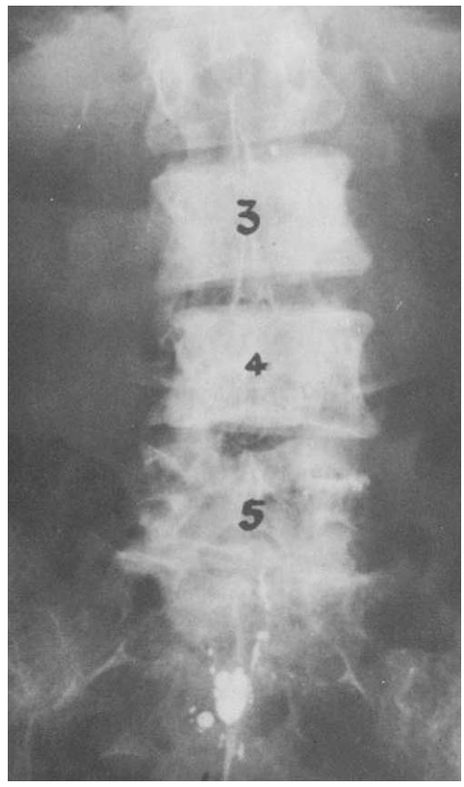

FIGURE 1-28

Plain radiograph showing osteoblastic lesion of the L_3-L_4 vertebra due to prostatic cancer with metastases. (From Raj PP, editor: *Practical Management of Pain*. St. Louis, Mosby, 1986, figure 15B-3, p. 162, with permission.)

uniformly illustrated and can be falsely interpreted as negative. Aggressive tumors that do not invoke an osteoblastic response, such as myeloma, can also yield a negative examination.

Primary spine tumors are usually benign. Osteoid osteoma, osteoblastoma, aneurismal bone cyst, and osteochondroma produce an active bone scan. These tumors generally affect the posterior elements of the spine. CT must be used to differentiate them and isolate their anatomic position.

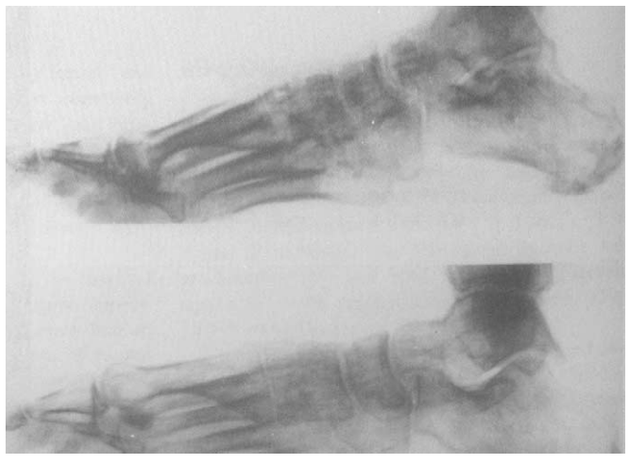

FIGURE 1-29

Plain radiographs showing Sudek's atrophy of the left foot (bottom). (From Raj PP, editor: *Practical Management of Pain*. St. Louis, Mosby, 1986, figure 15B-4, p. 162, with permission.)

Recent studies[80,81] have evaluated the ability of bone scans, with the addition of single-photon emission computed tomography (SPECT), to distinguish benign lesions from malignant lesions. SPECT scan differs from bone scan because it provides a three-dimensional image that enables physicians to locate the lesion more precisely. Lesions that affect the pedicles are a strong indicator of malignancy, while lesions of the facets are likely to be benign. Lesions of the vertebral body or spinous process are just as likely to be benign as malignant and, therefore, offer little diagnostic evidence.[80]

Gallium 67 is the most effective radioactive tracer in assessing infectious spondylitis. One study[82] compared bone scans using gallium 67 and Tc 99m with radiography and MRI. Gallium 67 had a sensitivity of 92%, a specificity of 100%, and an accuracy of 95%.[82] MRI was the second-best method of evaluation for infection, with a sensitivity of 96%, a specificity of 93%, and an accuracy of 94%.[82]

Beam-hardening artifacts or problems caused by filtration of low-energy photons, as occurs in the skull base in CT imaging, is not a concern with MRI. This modality

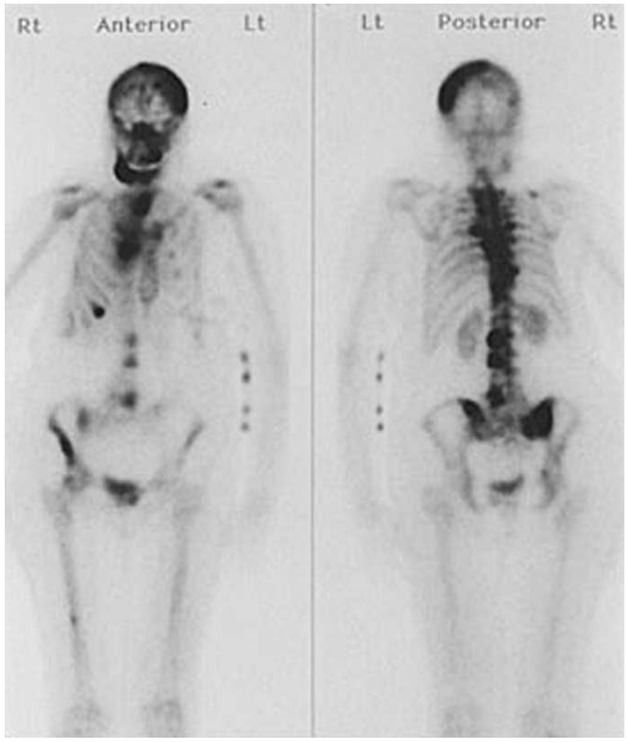

FIGURE 1-30

Bone scan showing metastatic disease of the spine. Areas of increased tracer uptake represent areas of active bone growth common in patients with osteoblastic cancer. (From Humphreys SC, Eck JC, Hodges SD: Neuroimaging in low back pain. *Am Fam Physician* 65:2217-2218, 2002, figure 7, with permission.)

allows clear visualization of the posterior and middle cranial fossae.

MRI is also better suited for defining and staging subacute and chronic hematomas, as well as for defining the contents of a cystic lesion.

Finally, patients with iodine allergy or those with acute renal failure can be evaluated safely with MRI because the contrast used contains no iodine and is not nephrotoxic.

MAGNETIC RESONANCE IMAGING

Magnetic resonance imaging (MRI) has emerged as the procedure of choice for diagnostic imaging of neurologic structures related to low back pain (Figure 1-31). Although a significant variation can exist in the quality of lumbar spine MRI images as a function of the imaging center and the image interpreter,[82] MRI is better than CT in showing the relationship of the disc to the nerve and at locating soft tissue and non-bony structures. For this reason, it is better than CT at detecting early osteomyelitis, discitis, and epidural-type infections or hematomas.

MRI provides high-resolution, multiaxial, multiplanar images of tissue with no known biohazard effects. The only contraindication to MRI is the presence of ferromagnetic

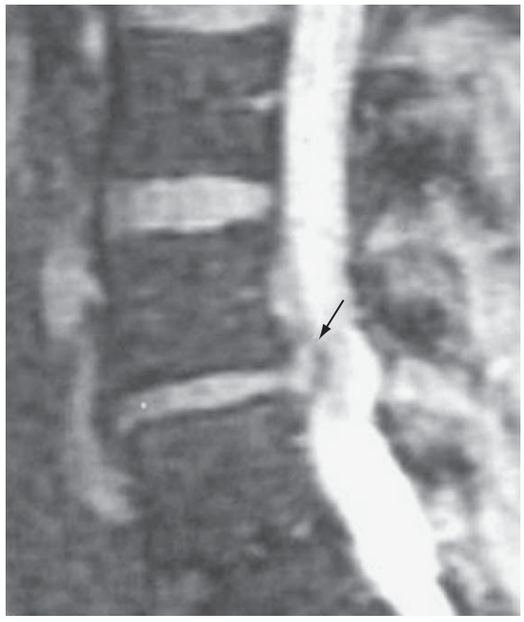

FIGURE 1-31

Lumbar herniated disc. T2 Sagittal view MRI shows disc material impingingon neighboring neural structures (arrow). (From Humphreys SC, Eck JC, Hodges SD: Neuroimaging in low back pain. *Am Fam Physician* 65:2217-2218, 2002, figure 6, with permission.)

implants, cardiac pacemakers, intracranial clips, or claustrophobia. Two different types of images are generally obtained using MRI: T1-weighted images in the sagittal plane and T2-weighted images in the axial and sagittal planes.

Spin echo is the standard pulse sequence when using T1-weighted images, which are commonly used to contrast tissues such as neural foramina and nerve roots. Spin echo provides good spatial resolution, allowing for confirmation of disc herniation, although the size of the herniation is difficult to determine. It can also detect metastatic disease by surveying the marrow signal intensity or by showing loss of fat.

T2-weighted spin echo images enhance the signal of the cerebrospinal fluid, making this more sensitive to spinal pathology (such as tumor, infection, osteomyelitis, and discitis), but it is often more time consuming with the pulse sequence.

As with other imaging techniques, MRI can identify abnormalities in asymptomatic persons. In one study, 21 MRIs of 67 asymptomatic persons 20–80 years of age were obtained. At least one herniated disc was identified in 20% of persons younger than 60 years and in 36% of persons older than 60 years.[83] Another study[84] discovered that 63% of asymptomatic persons had disc protrusion, and 13% had disc extrusion.

Many imaging centers use contrast-enhanced MRI to increase the visualization of herniated discs. Recent studies[85] have concluded that contrast enhancement in patients with previous lumbar spine surgery added limited diagnostic value and often resulted in more inaccurate interpretations.

Gadolinium is thought to enhance the appearance of nerve roots in viral or inflammatory conditions and can help distinguish recurrent disc herniation from scar tissue in the postoperative spine.[86]

MAGNETIC RESONANCE IMAGING OF THE SPINE

Magnetic resonance imaging has the distinct advantages of versatility and noninvasiveness. MRI may delineate normal spinal anatomy and a variety of pathological conditions. By using a combination of techniques, including T1 and a combination of techniques, including T1- and T2-weighted images, in sagittal and axial planes, most of the spinal structures can be well-delineated. For example, the spinal cord can be separated from cerebrospinal fluid and extradural structures by sagittal, T1-weighted sequence. A T2-weighted, spin-echo sequence may be used for evaluation of disk hydration and the cerebral spinal fluid (CSF)–extradural interface. Extradural defects are therefore detected, similar to those seen on contrast myelography (Figure 1-32). Lateral disk herniation and neural foramina are best visualized with transverse images.[87]

Because MR signals are based on T1 and T2 relaxation times and tissue characteristics other than electron density, greater tissue contrast can be achieved than with CT or radiography. Therefore, MR excels in evaluation of the intervertebral disk. Changes because of disk degeneration are manifested by changes in signal intensity within the disk. In addition, sequelae of disk degeneration, such as spinal stenosis, ligamentous hypertrophy, and facet disease can be demonstrated.[88]

MRI is applicable to a number of other spinal conditions including subluxation, vertebral osteomyelitis, diskitis, trauma, neoplasm, arteriovenous malformation, syringomyelia, and intramedullary neoplasms.

Since MRI excels in delineation of soft tissues, without the necessity of contrast or hospitalization, it has become the examination of choice in many spinal conditions. Disadvantages include long imaging time, discomfort for the patient, and sometimes a need for sedation. Magnetic hazards require that metalworkers and patients with intracranial aneurysm clips or cardiac pacemakers be excluded. Heating of metallic prostheses, or movement of other metallic clips, appears not to be a significant hazard.[89]

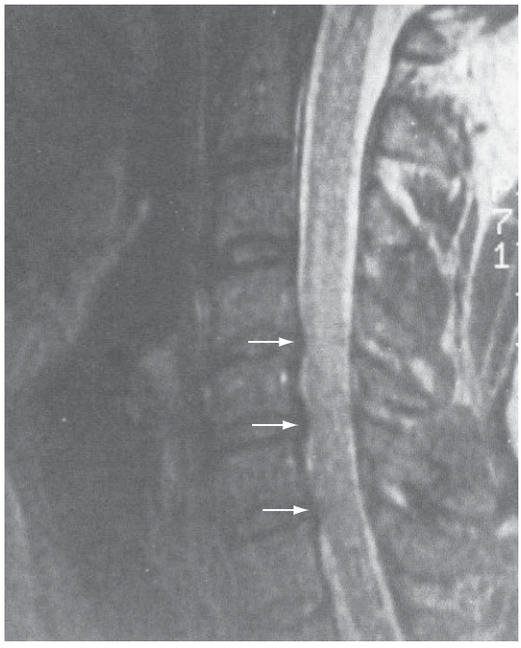

FIGURE 1-32

MRI. Extradural filling defects because of bulging and herniated disks at $C_{4–5}$,$C_{5–6}$ and $C_{6–7}$ ND (arrows) obliterate the CSF space at these levels. The CSF appears white on the sagittal, T2-weighted image. (From Raj PP, editor: *Practical Management of Pain*, 2nd ed. St. Louis, Mosby, 1992, figure 12-4, p. 189, with permission.)

SKELETAL SCINTIGRAPHY

The bone scan is the study with which referring physicians are the most familiar. The scan can be used either alone or in conjunction with other imaging procedures as either the initial or a follow-up examination. A bone scan is a sensitive and relatively inexpensive method of acquiring images of the skeletal system. Because of its method of localization, however, it is not highly specific, methylene diphosphonate (MDP) and hydroxy-methylene and diphosphonate (HDP), the agents most commonly used for skeletal scintigraphy today, absorb to the hydroxyapatite crystal of the surface bone.[90] Technetium 99m is the radioisotope used for labeling these biological markers. The labeled phosphate localizes at sites with active osteoblastic activity and increased blood flow. Hence, uptake occurs throughout the axial and appendicular skeleton. Areas of focally increased uptake are seen with both benign conditions, such as healing fractures, as well as malignant processes, such as osseous metastases (Figure 1-33).

Because minute differences in bone remodeling can be demonstrated, abnormalities and bone pathology can be uncovered prior to their visualization on plain film. Detection of a lytic lesion on plain radiographs requires loss of approximately 50% of the calcification, whereas scintigraphy can detect a lesion with as little as a 1% loss, much earlier in the disease process.[90]

There are many indications for skeletal scintigraphy:

- Assessment of bone or joint pain when plain films are nondiagnostic or normal
- Detection of osseous metastatic disease
- Ligamentous injury
- Detection of stress fractures or occult fractures
- Evaluation for osteomyelitis
- Evaluation of avascular necrosis
- Evaluation for suspected loose or injection joint prosthesis
- Primary bone tumor

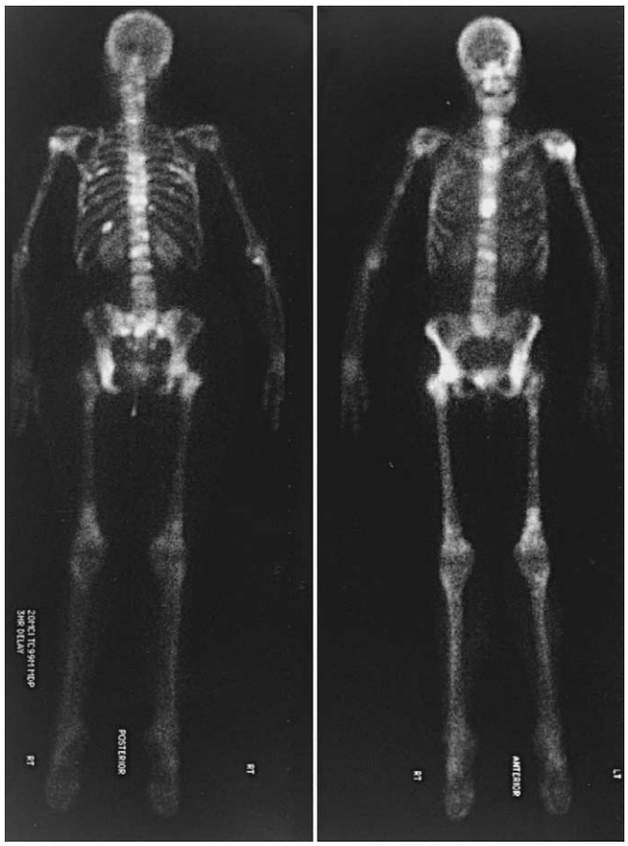

FIGURE 1-33

Metastatic breast carcinoma. This technetium 99m methylene diphosphonate (^{99m}Tc-MDP) bone scan in a patient with breast carcinoma shows multiple foci of increased activity throughout the appendicular and axial skeleton, representing widespread metastases. (From Raj PP, editor: *Practical Management of Pain*, 3rd ed. St. Louis, Mosby, 2000, figure 27-21, p. 399, with permission.)

- Diagnosis and assessment of Paget's disease
- Determination of biopsy sites
- Assessment of viability of bone graft

The most frequent clinical use of skeletal scintigraphy is in the evaluation of osseous metastases. It is used not only to detect but also to stage many malignancies and to monitor disease progression. Primary tumors most commonly metastasizing to bone include prostate, breast, renal cell, lung, and thyroid carcinomas.[12,13,59] Lymphomas and neuroblastomas are also monitored through the use of bone scanning.

Most fractures pose no serious diagnostic dilemma and can be easily identified on plain radiographs. Occasionally, however, a hairline fracture that is elusive on plain film can be easily detected on a bone scan. These are most notably fractures involving the femoral neck, scaphoid, spine, and pelvis.[90] The majority of adult patients and all pediatric patients demonstrate increased activity at the fracture site within 72 hours of injury. Some fractures in older patients, however, are not identified until up to 5 days after the injury.

Bone scintigraphy can also be used to estimate the age of a fracture, as with compression deformities of the vertebral bodies. Elderly, osteopenic patients often complain of back pain; plain films might show compression fractures of the spine but provide no clues as to the age of the fracture. In 95% of patients under 65 years of age, an increase in bone remodeling is evident by 48 hours; by 72 hours after injury, almost all patients show radionuclide uptake. Lack of uptake or normal activity in a collapsed vertebra is sufficient evidence that the fracture is not an acute event.[91] Additionally, approximately 60% of vertebral body compression deformities show normal uptake after 1 year, 90% after 2 years, and more than 95% 3 years after the initial trauma.[90]

Bone scanning is also helpful in the evaluation of a stress fracture (Figure 1-34). Plain radiograph findings in stress fractures can be extremely subtle, comprising a thin line or radio density, or they may not be apparent at all. Stress fractures may be the result of the overuse of normally mineralized bone, as with the classic March fracture of the third metatarsal described in military recruits, or they may be insufficiency fractures caused by normal use of inadequately mineralized bone.

Early detection of acute osteomyelitis is yet another indication for the use of bone scintigraphy. Changes due to osteomyelitis can be detected on a bone scan up to 7 to 10 days prior to their appearance on plain film. The bone usually demonstrates abnormal uptake within as little as 24 hours. However, because the radiolabeled phosphates localize to sites of increased blood flow, as well as those of

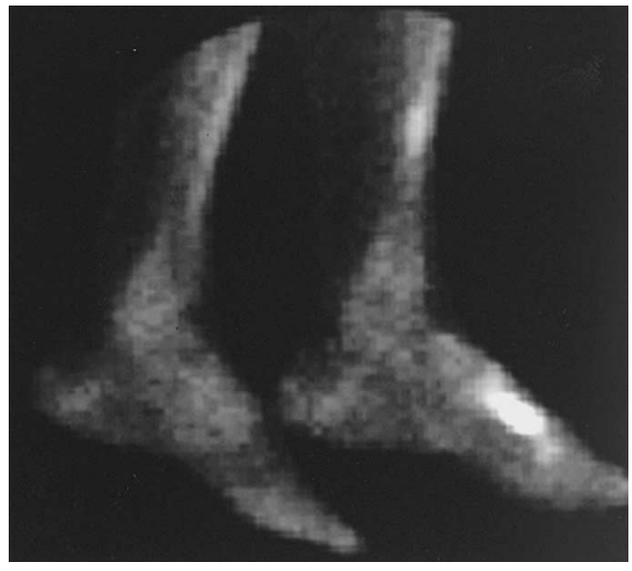

FIGURE 1-34

Stress fracture. Technetium 99m methylene diphosphonate (^{99m}Tc-MDP) scan demonstrating stress fracture of the second metatarsal in a female runner complaining of pain over the dorsum of the foot. This is a classic March fracture. There is also slightly increased uptake in the anterior cortices of the distal tibia, consistent with shin splints. (From Raj PP, editor: *Practical Management of Pain*, 3rd ed. St. Louis, Mosby, 2000, figure 27-22, p. 400, with permission.)

increased bone turnover, this can present a diagnostic dilemma in differentiating osteomyelitis from cellulites.

This is commonly seen in the diabetic patient with non-healing ulcers. The use of the dynamic, or three-phase, bone scan can aid in differentiation by acquiring early flow study and blood pool images, followed by the routine delayed, skeletal phase images. Osteomyelitis shows uptake on the flow study due to arterial hyperemia, followed by diffuse or focal uptake on the blood pool images. There is focal uptake within the involved segments of bone on the delayed images (Figure 1-35). Cellulitis, however, shows delayed activity owing to venous hyperemia on flow study after which intense and diffuse uptake occur on the blood pool images. Uptake does not appear on the delayed images secondary to the lack of bony involvement.

Gallium 67 citrate can also be employed in the attempt to diagnose osteomyelitis.[91-93] There are presently multiple theories on the mechanism of gallium 67 localization in tumors at sites of inflammation. Gallium is known to bind to transferrin, thereby localizing at sites of infection or inflammation secondary to the increase in vascular permeability. Gallium also binds to lactoferrin; the known affinity of gallium for leukocytes can be explained by the high concentration of lactoferrin therein. In addition, gallium may bind to the siderophores produced by bacteria living in low iron-containing environments, such as areas of inflammation (Figure 1-36).[94]

Occasionally, as frequently occurs in infants, bone scintigraphy may appear normal despite the presence of osteomyelitis.[95] Gallium 67 can be used for further evaluation, as it localizes to the site of infection. Differentiation of infection and loosening of orthopedic prostheses may also present a diagnostic problem. Use of gallium 67 in addition to MDP bone scanning is often sufficient to provide the answers.

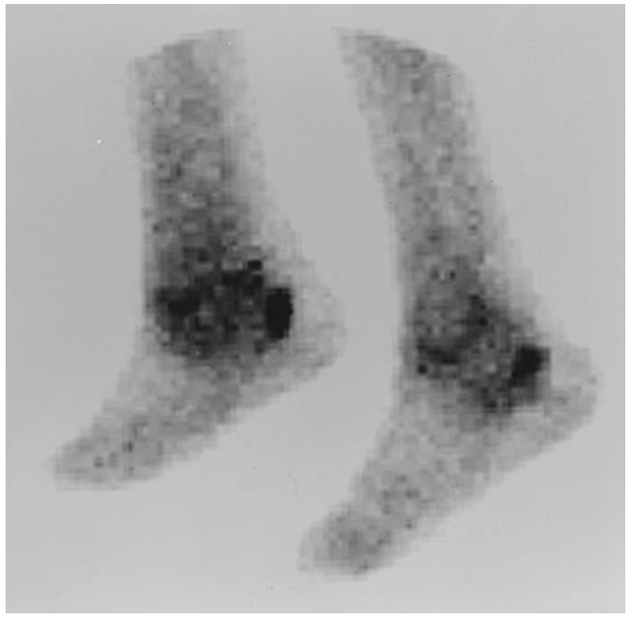

FIGURE 1-35

Osteomyelitis. Delayed images in three-phase bone scan with technetium 99m methylene diphosphonate (^{99m}Tc-MDP) showing increased activity about the tarsal bones, calcaneus, and tibiotalar joints bilaterally. (From Raj PP, editor: *Practical Management of Pain*, 3rd ed. St. Louis, Mosby, 2000, figure 27-23, p. 400, with permission.)

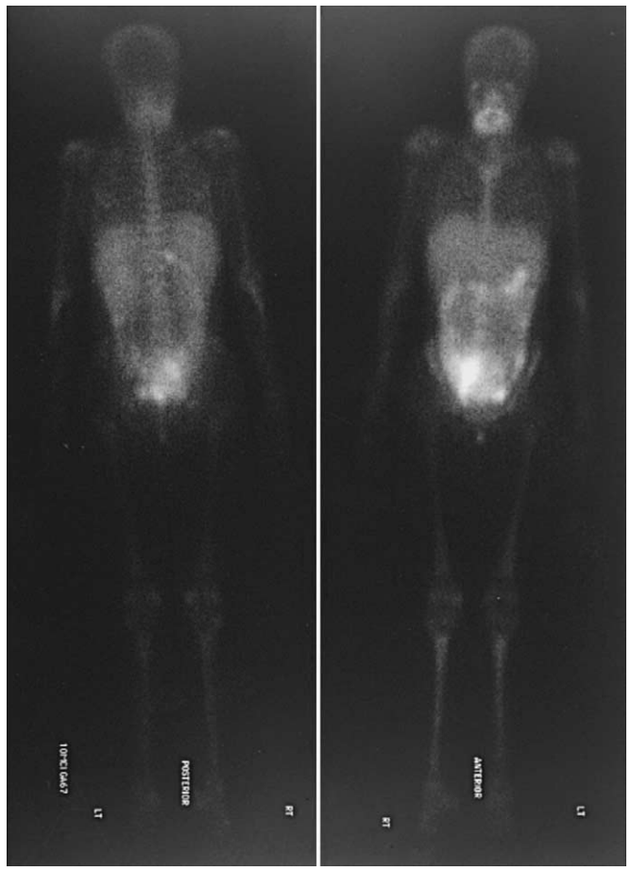

FIGURE 1-36

Seven-day gallium 67 scan. Planar images 7 days after injection with gallium 67 citrate in a 17-year-old patient with persistent abdominal pain. Images show increased uptake within the right lower quadrant. Follow-up CT examination revealed a large inflammatory mass in the region of the cecum. Periappendiceal abscess was confirmed at surgery. (From Raj PP, editor: *Practical Management of Pain*, 3rd ed. St. Louis, Mosby, 2000, figure 27-24, p. 401, with permission.)

Indium 111 is another tool in the arsenal for evaluation of osteomyelitis. When it is used together with technetium 99m MDP, the specificity and sensitivity of the combined examination are quite high.[96] Advantages of indium 111 over a gallium include absence of bowel activity, which can obscure sites of infection, particularly within the pelvis and shorter time to completion of the study. Indium scanning begins within 18 to 24 hours; most gallium imaging begins after 7 days. Indium 111–labeled white blood cells (WBCs) distribute to the site of active infection in any tissue. This method is often used in evaluation of postoperative patients with suspected sepsis. The tagged WBCs do not localize in noninfected granulation tissue, sites of osteoarthritis, bony non-unions, heterotopic bone formation, or inactive, chronic osteomyelitis. Disadvantages of

indium 111 WBC scanning in the diagnosis of osteomyelitis include:

1. Obscuration of the thoracolumbar junction due to high activity within both the liver and spleen.
2. Misinterpretation of uptake in an accessory spleen.
3. Activity in an additional area, such as decubitus ulcer or an area of bowel infarction.

Whereas a substantial loss of bone substance must occur before a destructive lesion or demineralization becomes radiographically visible, bone scanning does not rely on the actual amount of bone loss to demonstrate pathology. Consequently, destruction from metastases and osteomyelitis may be detected on bone scans much earlier than on radiographs. Similarly, subtle trauma sufficient to incite a local repair process, such as stress fracture, may also be obvious on bone scans but radiographically invisible.

The phrase "sensitive but nonspecific" is commonly applied to bone scanning. Although many different bone abnormalities result in so-called hot spots, careful attention to characteristics of the lesions usually reveals a specific diagnosis when interpreted in light of appropriate clinical information. The number, location, and distribution of lesions are important, as is clinical history (e.g., trauma or known primary malignancy). When radiographic correlation is obtained, even more precise and specific diagnosis is possible.

Since the advantage of bone scanning is its high sensitivity, certain painful conditions are more appropriately detected by bone scanning than by radiographs, in which findings may be subtle or even undetectable.

REFLEX SYMPATHETIC DYSTROPHY/COMPLEX REGIONAL PAIN SYNDROME

This syndrome is characterized clinically by pain, diminished function, joint stiffness, skin and soft tissue trophic changes, and vasomotor instability.

Bone scanning is highly sensitive and specific in establishing the diagnosis of reflex sympathetic dystrophy (RSD)[97] and in excluding other causes for significant extremity pain. Three-phase bone scanning usually shows hypervascularity to the affected extremity on early images, followed by diffusely increased uptake, in a periarticular distribution, on delayed images (Figure 1-37).

Recently, RSD has been found to yield varying scan appearances in different stages of evolution of the syndrome.[98] In early stages, there is the typical bone scan appearance of increased flow and increased delayed periarticular uptake. Later, as the syndrome progresses to stage II clinically, the flow normalizes but delayed views remain diffusely intense. In later, chronic (stage III) patients, flow is reduced, and delayed images return to a normal appearance. Therefore, in patients with a definite clinical diagnosis of RSD, the bone scan may be useful in staging the process. However, its usefulness in monitoring response to therapy is not well defined, since it may be difficult to distinguish true improvement from progression to a more advanced stage by bone scan alone. When the diagnosis of RSD is not firmly established, bone scanning is certainly helpful in ruling out other occult skeletal lesions that could be the cause for the patient's pain; osteomyelitis, occult or stress fracture, degenerative arthritis, bone infarction, malignancy, or benign bone lesion (e.g., osteoid osteoma) may be radiographically subtle or invisible but easily detected on scans. In these cases, the bone scan may detect the cause of otherwise "unexplained" pain and lead to definitive treatment. These other lesions are easily differentiated from RSD by their focal, rather than diffuse, appearance on the scan.

DISCOGRAPHY

Discography is used in conjunction with CT or MRI to localize disc herniation or fissure in the annulus fibrosis. A volume of contrast media is injected into the disc space to determine the integrity of the intervertebral disc. In the normal disc, the annulus fibrosis solidly encloses the nucleus pulposus and is only capable of accepting 1 to 1.5 ml of contrast media. If 2 ml or more of contrast media can be injected, there is likely to be a degenerative change in the disc.

In addition to determining the available volume of the disc, discography is used to reproduce the symptoms associated with a possible herniated disc. The patient's response to pain can help confirm the source of the symptoms. When saline or dye is injected, it pressurizes the disc, and the patient is able to confirm that this pain is the same as the pain he or she has been having.

Discography should be used cautiously because of the possibility of false-positive results. In one study,[99] lumbar discography was performed on 26 volunteers who were pain-free or had chronic cervical pain or primary somatization disorders without low back pain. Significant positive pain responses were reported in 10% of the pain-free group, 40% of the chronic cervical pain group, and 83% of the primary somatization disorder group.[99] Based on these results, the findings from discography should be interpreted cautiously. Discography is an invasive test that has an inherent risk of infection and neural injury. It should be used only to confirm an initial diagnosis, not as the primary diagnostic tool.

RADIATION SAFETY

Interventional radiology procedures can require substantial amounts of ionizing radiation and therefore necessitate particularly close attention to radiation management. This section reviews radiation units, regulations, and the fundamental principles of radiation management for patients and personnel and examines the procedures and devices

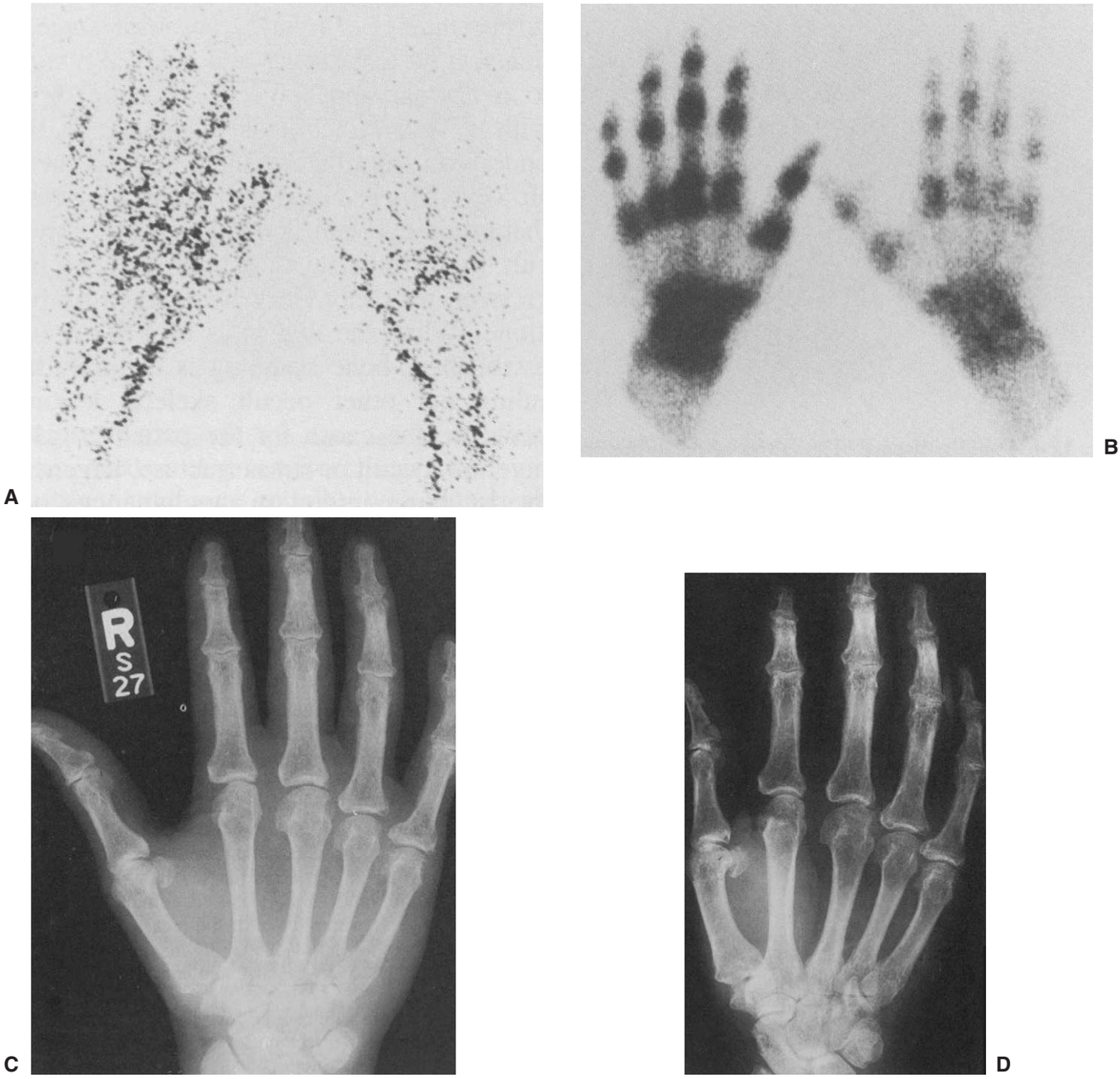

FIGURE 1-37

CRPSI (A) Flow study performed during bone scan shows increased vascularity to right hand. (B) Delayed bone scan shows periarticular uptake of tracer throughout the right hand, typical of RSD. (C) Initial radiograph at the time of the bone scan was normal. (D) One month later, osteoporosis has become radiographically evident. (From Raj PP, editor: *Practical Management of Pain*, 2nd ed. St. Louis, Mosby, 1992, figure 12-7, p. 194, with permission.)

designed to reduce patient and staff exposure in interventional radiology.

RADIATION UNITS

The fundamental interactions of x-rays with matter produce ion pairs via photoelectric absorption and Compton scattering.[100] The coulomb per kilogram (C/kg) is the unit used to measure the electrical charge produced by x- or gamma-radiation in a standard volume of air by ionization. Previously the roentgen unit (about 0.25 mC/kg) was used for this purpose. Radiation exposure is the formal term for the process of ion-pair productions.

The number of ion pairs produced in air does not directly measure the amount of energy deposited in another medium because of the differences in x-ray absorption by

different materials.[100] The gray (Gy) is used as a measure of the radiation absorbed dose (energy deposited per unit mass). A gray is equal to 1 J/kg. The older unit of the rad is equal to 0.01 Gy. These units are of fundamental importance in patient dosimetry.

Ionizing radiations other than x- and gamma rays, such as particles or neutrons, may induce a greater biologic effect for a given absorbed dose. To quantitate this observation, the sievert (Sv) is used to measure the dose equivalent. The sievert is equal to the number of grays multiplied by a quality factor ranging from 1 to 20 that expresses the degree of biologic insult for equal doses of different types of ionizing radiation. The quality factor for x and gamma radiation is equal to 1. The older unit of the rem is equal to 0.01 Sv. This unit is most often utilized in health physics and radiation-monitoring measures for personnel.

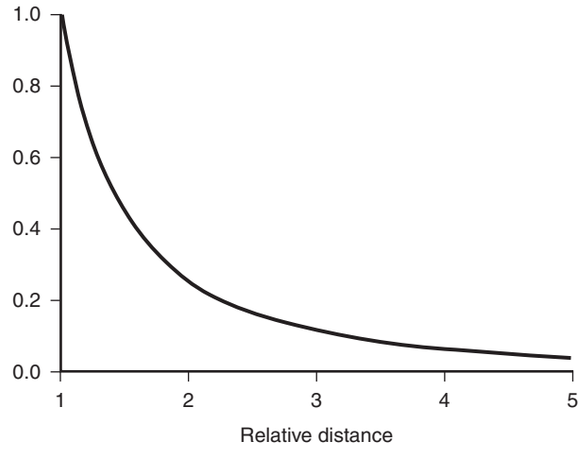

FIGURE 1-38
Reduction of radiation intensity (y axis) according to the inverse square of distance law.

RADIATION PROTECTION FUNDAMENTALS

In order to decrease the absorbed dose to the patient and the staff, the radiation protection principles of time, distance, and shielding must be considered. Radiation dose is directly related to exposure time, so by halving the exposure time, one halves the radiation dose. Personnel who do not need to be in the fluoroscopy suite during all or part of a procedure can reduce their exposure time by simply leaving the area. For other individuals the time is totally controlled by the fluoroscopist. Therefore, to reduce exposure time, the fluoroscopist should never depress the foot switch except while observing the fluoroscopic image.

Because an x-ray beam diverges as it passes through space, radiation intensity decreases as the inverse square of the distance from the radiation source:

$$I_2/I_1 = d_1^2/d_2^2$$

Hence, the distance from a radiation source is doubled; the radiation intensity decreases to one-fourth its original value (Figure 1-38). Although this relation holds strictly only for a point source, the distance principle is useful in reducing radiation dose to clinical personnel when the patient is the principal source of scattered radiation. Personnel who do not need to be in the immediate vicinity of the patient should always stay as far away as is reasonable from the portion of the patient that is being imaged.

The attenuation of an x-ray beam (loss of intensity as it passes through matter) is exponential, where I and I0 are the initial and transmitted radiation intensity, respectively; μ is the attenuation coefficient of the material (which depends on the atomic number and density of the material and on the energy of the photons); and x is the thickness of the attenuating material. Therefore, small amounts of attenuating (shielding) material can greatly reduce the intensity of an x-ray beam. For example, more than 90% reduction of a diagnostic x-ray beam is obtained by using material equivalent to 0.5 mm of lead (the nominal equivalent of a typical lead apron). Examples of exponential attenuation for diagnostic radiology x-ray beams are shown in Figure 1-39. Lead aprons should always be worn by anyone in a fluoroscopy suite. Because fluoroscopy is utilized extensively during some interventional radiology procedures, the continual observation of these fundamental principles is of far greater importance than in other areas of diagnostic radiology.

RADIATION PROTECTION REGULATIONS

Unlike other areas in medicine in which ionizing radiation is used to diagnose or treat disease (e.g., therapeutic radiology, nuclear medicine), x-ray use is not completely regulated at the federal level. No one federal body analogous to the Regulatory Commission exists to supervise x-rays. Instead regulations concerning equipment are handled by the Center for Devices and Radiology Health within the

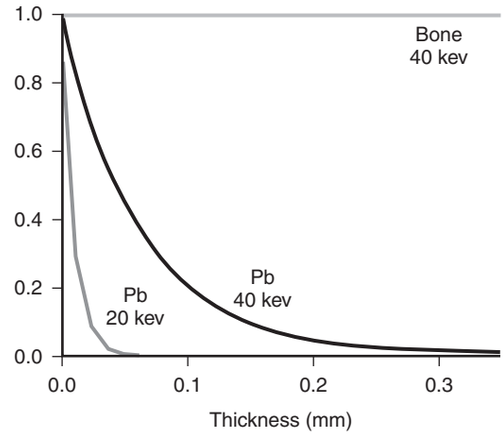

FIGURE 1-39
Reduction of radiation intensity with increasing thickness of lead (Pb) and bone at 60 kVp (20 kev) and 120 kVp (40 kev).

FDA[101]; the Occupational Safety and Health Administration (OSHA) places limits on the radiation doses of employees in the workplace; and individual states' departments of services place additional regulations on users of x-ray equipment. Although one might expect this decentralization of regulations to be confusing, the state-to-state variation actually is very minimal, since most states have patterned their regulations after the recommendations of the National Council on Radiation Protection and Measurements (NCRP). This body has developed an extensive set of regulatory guidelines that have become de facto standards for the safe and proper use of ionizing radiation (summarized in Tables 1-1 and 1-2). Other sources give further details of the general philosophy of radiation protection, as well as specific recommendations for particular situations.[102–108] Two other bodies also publish recommendations for radiation protection: the International Commission on Radiation Protection (ICRP) and the International Council on Protection and Units (ICRU).

The presence of these diverse recommendations is particularly important in interventional radiology, since the maximum quarterly dose to the eyes permitted by OSHA is one-third that recommended by other regulatory organizations. These quarterly allowances are intended for sporadic exposure, not continuous exposure. Doses should always be kept "as low as reasonably achievable" (ALARA).

Concern is often expressed about the absorbed dose to the eye of the fluoroscopist because of the risk of radiation-induced cataracts. This biologic effect appears to have a threshold, in that about 6 Gy of diagnostic x-irradiation over several weeks are necessary to produce cataracts in humans.[102,109,110] It may be that absorbed doses of about 15 Gy are necessary to induce cataracts in the diagnostic radiology setting.[105,109]

TABLE 1–1 Maximum Permissible Dose Equivalents (mSv)

Area	13 Weeks	Yearly	Cumulative
Total effective dose equivalent	12.5	50	Age × 10
Lens of eye	37.5	150	
Other organs (individually)	125	500	

TABLE 1–2 Maximum Number of Fluoroscopic Procedures in 3-Month Period without Exceeding Eye Exposure of 12.5 mSv/Quarter

Fluoroscopic Time per Procedure (hr)	Radiation Exposure at Eye Level (mSv/hr)					
	10	**25**	**50**	**100**	**200**	**300**
0.25	50.0	20.0	10.0	5.0	2.5	1.2
0.50	25.0	10.0	5.0	2.5	1.2	0.8
1.00	12.5	5.0	2.5	1.2	0.8	0.4
2.00	6.2	2.5	1.2	0.6	0.3	0.2

STAFF RADIATION DOSE MONITORING

In general, monitoring devices must be worn if it is reasonably likely that a person could receive 25% of the maximum permissible dose in the discharge of his or her duties. This rule most assuredly mandates dose monitoring of the interventional radiologist and anyone else routinely in the fluoroscopy suite during these procedures.

The radiation exposure of the fluoroscopist is heavily dependent on imaging geometry. Figure 1-40 shows typical ISO exposure lines for several imaging configurations; note the tremendous increase in operator exposure with configurations in which the x-ray tube is above the patient. This increase occurs for two reasons: the overall intensity of the scattered radiation beam is approximately 985 times greater at the entrance site on the skin compared to the exit site,[100] and there is less attenuating material (e.g., image intensifier) between the patient and the operator. As a rule of thumb, the maximum operator exposure at a given distance occurs when there is an unobstructed path between an object and the location at which the x-ray beam enters the patient.

In addition to time, distance, and shielding, another important radiation protection parameter is x-ray beam size (Figure 1-41). The amount of scattered radiation exposure is directly related to beam size. In addition, the patient dose and image quality are affected by changes in collimation. Hence, by limiting the beam size to the smallest necessary area, the fluoroscopist can decrease both personnel and patient doses while improving image quality.

RADIATION MANAGEMENT DURING IMAGE RECORDING

Because cine is an extension of fluoroscopy, all of the previous radiation protection considerations apply; however, radiation doses are significantly higher for the patient, as well as the staff. Typical patient skin entrance dose rates can range from 200 to 900 mGy/min skin entrance doses in fluoroscopy.[111–113]

MEASURING RADIATION DOSAGE

The scientific unit of measurement for radiation dose, commonly referred to as *effective dose*, is the millisievert (mSv). Other radiation dose measurement units include rad, rem, roentgen, and sievert.

Because different tissues and organs have varying sensitivity to radiation exposure, the actual dose to different parts of the body from an x-ray procedure varies. The term "effective dose" is used when referring to the dose averaged over the entire body.

The effective dose accounts for the relative sensitivities of the different tissues exposed. More importantly, it allows for quantification of risk and comparison to more familiar sources of exposure that range from natural background radiation to radiographic medical procedures.

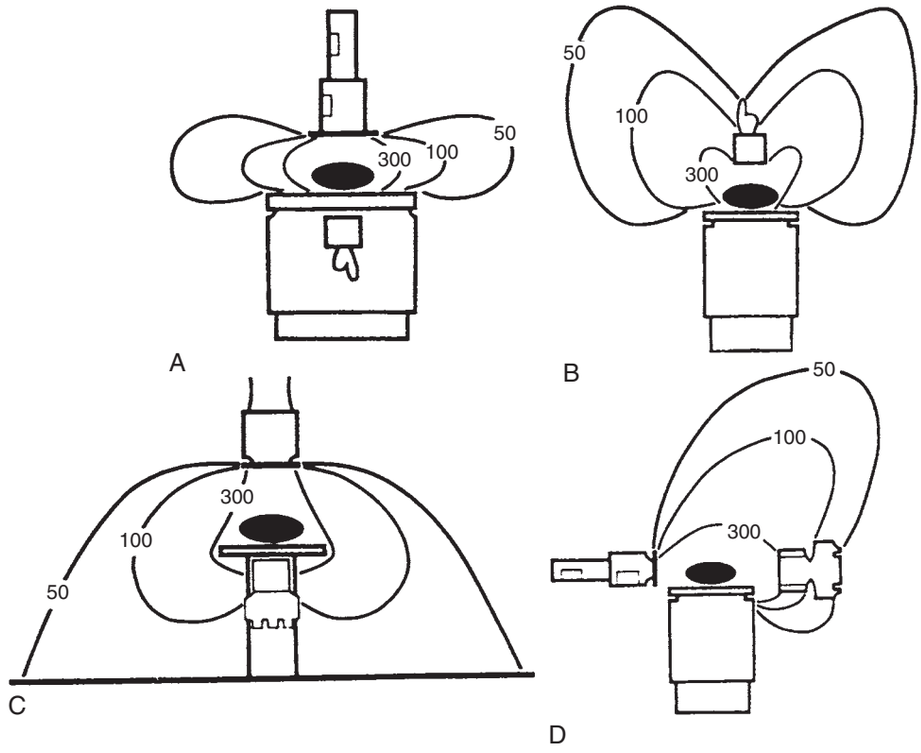

FIGURE 1-40

Scatter radiation from several equipment configurations. ISO exposure lines are given in mR/hr. (A) Conventional fluoroscopy. (B) Overhead tube. (C) Posteroanterior fluoroscopy with C-arm or U-arm. (D) Cross-table lateral fluoroscopy with C-arm or U-arm. (Courtesy of General Electric Medical Systems Division.)

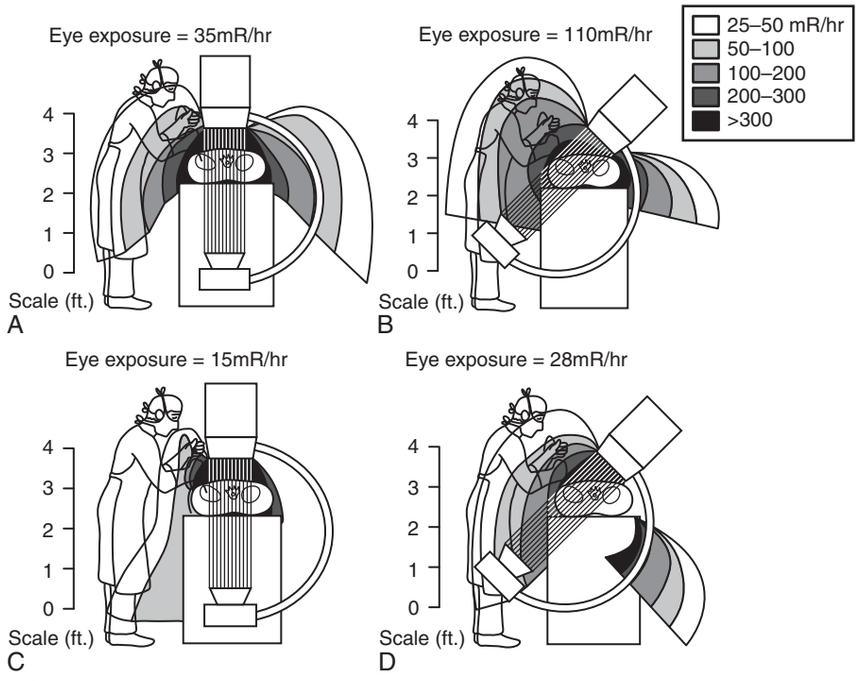

FIGURE 1-41

Scatter radiation reduction with surface shielding (2.8 R/min patient skin entrance exposure). (A) Vertical fluoroscopy without shielding. (B) Oblique (45°) fluoroscopy without shielding. (C) Vertical fluoroscopy with a 25 × 15 cm (0.75-mm lead equivalent) surface shield. (D) Oblique (45°) fluoroscopy with surface shielding in place. (From Young AT, Morin RL, Hunter DW, et al: Surface shield: device to reduce personnel radiation exposure. *Radiology* 159:801–803, 1986, with permission of the Radiological Society of North America, Inc.)

NATURALLY OCCURRING "BACKGROUND" RADIATION EXPOSURE

We are exposed to radiation from natural sources all the time. The average person in the United States receives an effective dose of about 3 mSv per year from naturally occurring radioactive materials and cosmic radiation from outer space. These natural "background" doses vary throughout the country.

People living in the plateaus of Colorado or New Mexico receive about 1.5 mSv more per year than those living near sea level. The added dose from cosmic rays during a coast-to-coast round-trip flight in a commercial airplane is about 0.03 mSv. Altitude plays a big role, but the largest source of background radiation comes from radon gas in our homes (about 2 mSv per year). Like other sources of background radiation, exposure to radon varies widely from one part of the country to another.

To explain it in simple terms, we can compare the radiation exposure from one chest x-ray as equivalent to the amount of radiation exposure one experiences from natural surroundings in 10 days.

X-RAY SAFETY

As with other medical procedures, x-rays are safe when used with care. Radiologists and x-ray technologists have been trained to use the minimum amount of radiation necessary to obtain the needed results. The amount of radiation used in most examinations is very small, and the benefits greatly outweigh the risk of harm.

X-rays are produced only when a switch is momentarily turned on. As with visible light, no radiation remains after the switch is turned off.

LIFETIME X-RAY EXPOSURE

The decision to have an x-ray exam is a medical one, based on the likelihood of benefit from the exam and the potential risk from radiation. For low-dose examinations, usually those that involve only films taken by a technologist, this is generally an easy decision. For higher-dose exams such as computed tomography (CT) scans and those involving the use of contrast materials (dyes) such as barium or iodine, the radiologist may want to consider your past history of exposure to x-rays. If you have had frequent x-ray exams and change health care providers, it is a good idea to keep a record of your x-ray history for yourself. This can help your doctor make an informed decision. It is also very important to tell your doctor if you are pregnant before having an exam that involves the abdomen or pelvic region.

PREGNANCY AND X-RAYS

As with any aspect of medical care, knowing that a patient is or could be pregnant is important information. Pregnancy, for example, might explain certain symptoms or medical findings. When a pregnant patient is ill or injured, the physician will carefully select medications to avoid potential risks to the developing child. This is also true of x-rays.

While the vast majority of medical x-rays do not pose a critical risk to a developing child, there may be a small likelihood of causing a serious illness or other complication. The actual risk depends on how far along the pregnancy is and on the type of x-ray. Ultrasound studies, for example, do not use x-rays and have never demonstrated any potential for risk to a pregnancy. X-ray studies of the head, arms, legs, and chest do not usually expose the fetus directly to x-rays, and typically the technologist who takes the x-rays will implement special precautions to ensure that the fetus of a pregnant patient is not directly exposed.

Sometimes patients need examinations of the abdomen or pelvis while they are pregnant. When studies of the abdomen or pelvis are required, the physician may prefer to order a different type of exam for a pregnant patient or reduce the number of x-rays from that which is normally acquired. Therefore it is important that you inform your physician or the x-ray technologist about your reproductive status before the x-ray study is performed.

Radionuclide exams, also known as nuclear medicine, also use x-ray–like radiation. But the method of use is quite different from x-rays, and they produce very different-looking images. The same advice for informing your physician or the nuclear medicine technologist about any possible pregnancy before the examination begins is important.

However, in nuclear medicine another precaution is advised for women who are breast-feeding. Some of the pharmaceuticals that are used for the study can pass into the mother's milk, and subsequently the child will consume them. To avoid this possibility, it is important that a nursing mother inform her physician and the nuclear medicine technologist about this before the examination begins. Usually, the mother will be asked to discontinue breast-feeding for a short while and pump her breast in the interim and discard the milk. Breast-feeding can often resume shortly afterwards.

RADIATION DOSE FROM INTERVENTIONAL RADIOLOGY PROCEDURES

Interventional radiologic procedures use diagnostic-type imaging equipment to assist a physician in the treatment of a patient's condition. These procedures frequently provide favorable medical results with minimal recovery time. In some cases, these procedures avoid the need for conventional surgery or improve the prospects for a favorable outcome from surgery. As with any medical procedure, there are associated risks and the nature of these risks depend on the procedure.

Use of interventional therapies has increased for hemodialysis access failure as it has for other vascular

diseases. Balloon angioplasty, stent angioplasty, thrombolysis, and thrombectomy are special techniques that provide great benefits to patients with access failure.

Potential health risks from occupational radiation exposure in many populations have been reported recently.[114,115] Schubauer-Berigan and Wenzl[114] reviewed the relationship between leukemia mortality and radiation exposure. Strong evidence existed that nuclear workers world wide have experienced slight but meaningful elevations in leukemia mortality that can be related to increases in low-linear-energy transfer radiation doses. Berrington et al.[115] also reported on 100 years of observations of British radiologists. The fact that cancer mortality increased in those radiologists who had job tenure of more than 40 years was attributed to the long-term effects of radiation exposure. Conversely, Niklason et al.[116] calculated the risk of fatal cancer in interventional radiologists based on annual radiation exposure to be less than one per 10,000 for almost the entire career of an interventional radiologist. However, it may be difficult to establish the potential risk of occupational exposure from image-guided intervention because only approximately 20 years have passed since the introduction of the techniques and recent improvements in devices, equipment, and procedures have accelerated the widespread use of many interventions. Operators and assistants must attempt to reduce occupational radiation exposure and optimize patient exposure doses.

Marx et al.[117] reported that the number of performances and the lead apron thickness were the primary determinants of total body dose in occupational radiation exposure. Although thicker lead coverage is effective in reducing radiation exposure, thicker lead aprons are heavy and uncomfortable to wear during the frequent performances and lengthy procedures of every intervention. Special procedure aprons may weigh as much as 8 kg[117] and are too heavy to wear for extended periods of time. Shielding devices near the scattering source should be developed instead of heavier and thicker lead coverage. Ito et al.[118] reported a radiation protection system for angiography of the cerebral, thoracic, abdominal, and lower extremity regions.

PERSONAL REPORT BY PAIN PHYSICIAN ON RADIATION INJURY TO HANDS AND FOREARM

In the early years, there was no serious recognition of hazards associated with radiation exposure by having the physician's hands visible on the fluoroscopic picture for minutes at a time. During a period of approximately seven years, the increased utilization of fluoroscopy resulted in incidentally noticed changes, such as opening an envelope would lead to stinging pain. Yet, there were no serious relationships between the changes that were the consequence of the cumulative radiation to the digits and the hand until 1986 (Figure 1-42A).

In 1986, a rather surprising finding was the wasting of the fingers in the terminal phalanges of the fifth, fourth, and third, and dorsum of the thumb and the terminal phalanges, index finger, and more so on the middle ring and fifth finger. Additionally, disappearance of the hair and wrinkles in the dorsum of the hand were noted. The thinness of the skin was so noticeable that a piece of paper placed in the pocket would cause the stinging and full thickness cut when the hand would be placed blindly to pick something out of the pocket. Also noted were upcurling and brittleness of the nails, pain of the joints of the hand, and light shining through the digits after closing the fingers.

Upon noting these changes, radiation protection measures were initiated in the form of wearing leaded gloves,

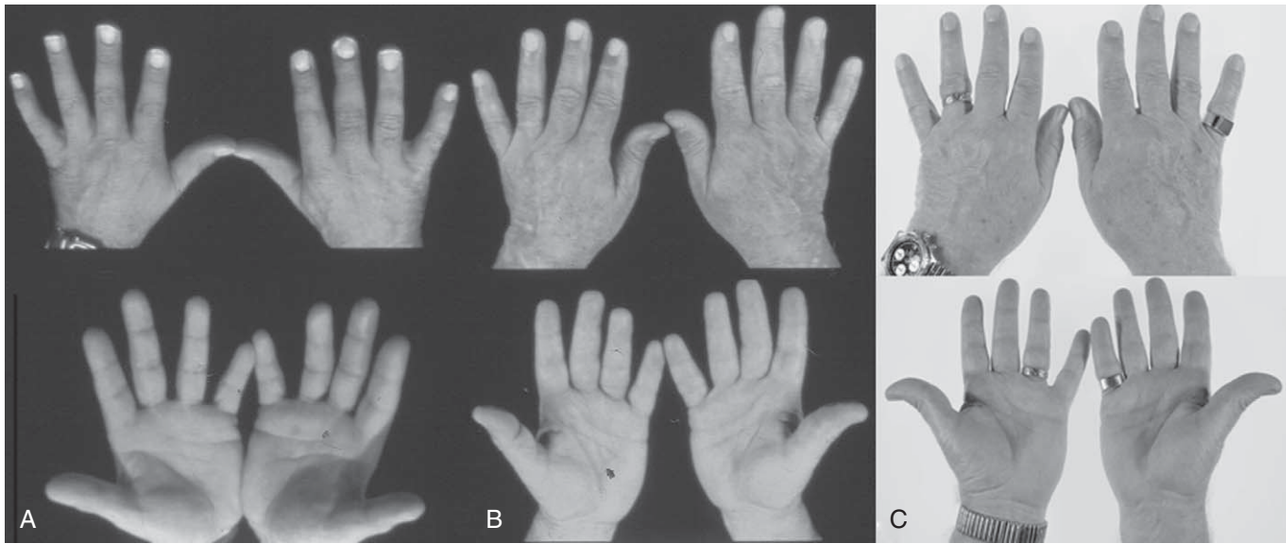

FIGURE 1-42

Appearance of a pain physician's hands due to radiation injury in 1986 (A), 2001 (B), and 2007 (C). (Courtesy of Gabor Racz, MD.)

and the changes gradually reversed. A conscious effort was made to avoid direct exposure to radiation and training the x-ray personnel to watch the operator's eyes and only turn the fluoroscopic unit on when requested. In the following 15 years to 2001, the digits regained connective tissue so that the fingers opposed to each other could keep the light out, the nails became less brittle, and the wrinkles returned to the dorsum of the hand together with some hair (Figure 1-42B and C). At this point, above the leaded glove line, forearm changes were still noted from the scatter radiation and this was seen as loss of hair and spotty hyperkeratosis without any pain or noted skin changes (Figure 1-43A).

To prevent further deterioration, forearm shields were constructed with Velcro to use for most repetitive procedures. The next set of pictures taken over the subsequent 6 years resulted in further normalization of the digits, hair, and skin, including the forearm (Figure 1-43B). At present, there are some achy feelings in the joints of the hand, but the skin has regained additional thickness (Figure 1-44). X-ray exposure is limited to accidental miscommunication,

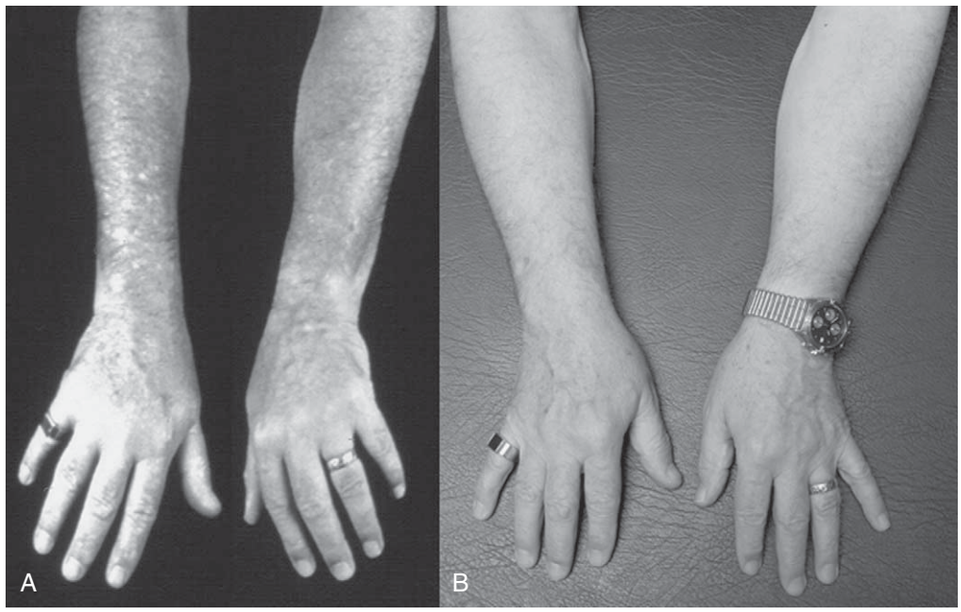

FIGURE 1-43
Radiation injury and recovery in the forearm of a pain physician in 2001 (A) and 2007 (B). (Courtesy of Gabor Racz, MD.)

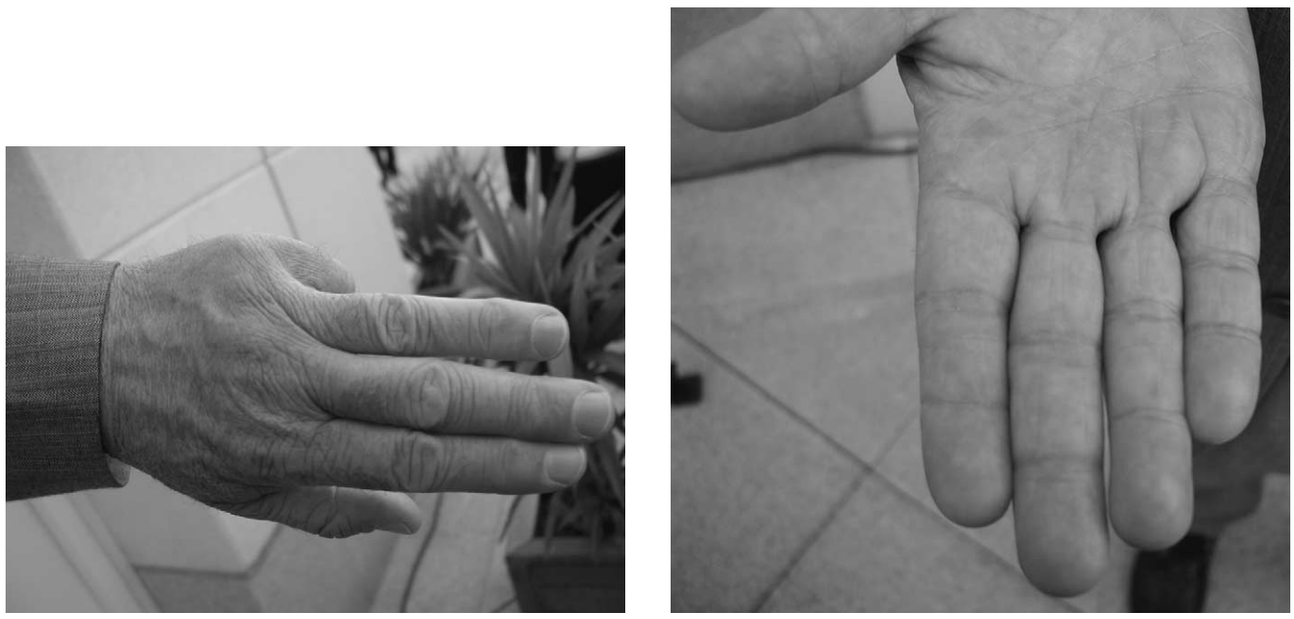

FIGURE 1-44
Appearance of pain physician's hands as seen from the dorsal side (A) and thenar side (B) in 2007. (Courtesy of Gabor Racz, MD.)

but never intentional or without protection unless it is for a very brief period of time. For comparative purposes, another senior pain physician who has been doing interventional pain procedures for many years with complete disregard for radiation protection with a rather similar attitude to that of the author was asked to compare hands in 2005 and found that the terminal parts of the digits were losing connective tissue, the skin was thin, and the hair was absent (Figure 1-45). Shortly after the picture in Figure 1-45B was taken in 2005, the physician reported a tendency of bleeding in the hand. Restorative measures have been initiated with early evidence of reversal of the radiation changes.

Radiation exposure indeed has cumulative effects; however, if protective measures are initiated early or when the changes are recognized, serious sequelae of radiation injury can be reversed.

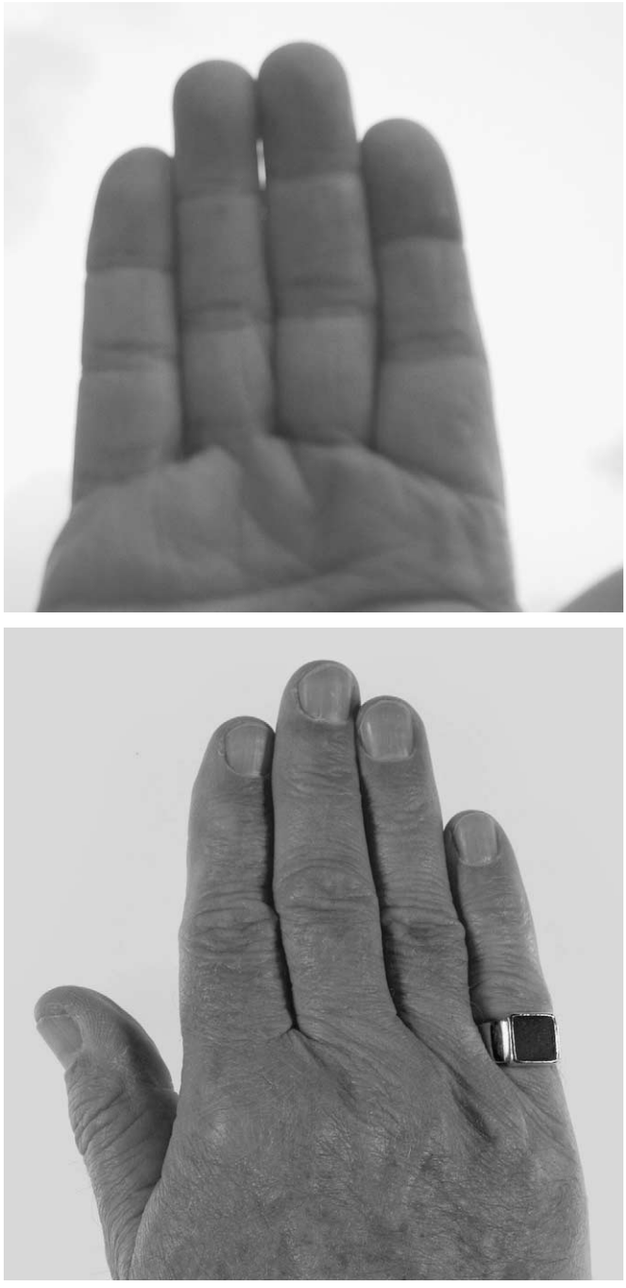

FIGURE 1-45

(A and B) Dorsum of hands of two physicians who sustained radiation injury. (Courtesy of Gabor Racz, MD.)

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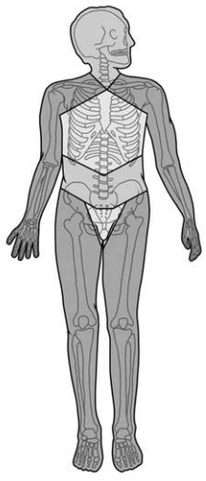
C H A P T E R



2 Drugs Used in Interventional Techniques

JAMES E. HEAVNER

LOCAL ANESTHETICS

Local anesthetics are used to prevent or treat acute pain (including procedure-related pain), to treat inflammatory, cancer, and chronic pain, and for diagnostic and prognostic purposes. Drugs classified as local anesthetics bind to a specific receptor site within the pore of the Na^+ channels in nerves and block ion movement through this pore. As a result, propagation of action potentials in nerve axons is blocked. Other actions of these drugs, such as anti-inflammatory by interaction with G-protein receptors,[1] also are thought to be relevant to their use to prevent or treat pain. Nociceptive pain, as well as neuropathic pain, is targeted with this group of drugs. Any part of the nervous system, from the periphery to the brain, may be where local anesthetics act to produce a desired anesthetic or analgesic effect.

A variety of formulations of local anesthetics, routes of administration, and methods of administration are used. They are injected as a single bolus, administered by constant infusion or by topical application and even orally. The drugs are formulated commercially or by medical personnel according to intended route of administration and/or to address specific concerns or needs. In general, their action is restricted to the site of application and rapidly reverses on diffusion from the site of action in the nerve. The chemical and pharmacologic properties of each drug determine its clinical use. Local anesthetics can be administered by a variety of routes, including topical, infiltration, field or nerve block, intravenous regional, spinal, or epidural, as dictated by clinical circumstances. Lidocaine, bupivacaine (racemic and levo forms), and ropivacaine probably are the local anesthetics most commonly used in interventional pain management.

EFFECTS ON SYSTEMS

As suggested above, local anesthetics have desirable effects on a number of body systems but they also have many undesirable effects. Local anesthetics interfere with the function of all organs in which conduction or transmission of electrical impulses occurs. Thus, they have important effects on the central nervous system (CNS), cardiovascular system (CVS), the autonomic ganglia, neuromuscular junction, and all forms of muscle.[2–4]

Central Nervous System

When local anesthetics enter the brain via systemic circulation, they may cause stimulation of the CNS, producing restlessness and tremor that may proceed to clonic convulsions. They may also produce depression manifested as sleepiness, loss of consciousness, or respiratory depression or arrest. These effects are dependent on the concentration of local anesthetic in the blood and other drugs the patient has received. Central stimulation is followed by depression; death is usually caused by respiratory or CVS failure.

Although drowsiness is the most frequent complaint that results from the CNS actions of local anesthetics, local anesthetics such as lidocaine and mepivacaine may produce dysphoria or euphoria and muscle twitching. Both lidocaine and procaine may produce loss of consciousness that is preceded only by symptoms of sedation.[5] Other local anesthetics also show the effect, but cocaine has a particularly prominent effect on mood and behavior.

Cardiovascular System

Local anesthetics, lidocaine in particular, are used to treat certain cardiac arrhythmias. However, if local anesthetic concentration in the blood reaches toxic concentration, life-threatening or lethal cardiovascular events may occur. The primary site of action is the myocardium, where electrical excitability, conduction rate, and force of contraction are altered. In addition, high concentrations of most local anesthetics cause arteriolar dilation. The cardiovascular effects usually are seen only after high systemic concentrations are attained and effects on the CNS are produced. However, on rare occasions lower doses cause cardiovascular collapse and death, probably due to either an action on

the pacemaker or the sudden onset of ventricular fibrillation. However, ventricular tachycardia and fibrillation are relatively uncommon consequences of local anesthetics other than bupivacaine.

Neuromuscular Junction and Ganglionic Synapse

Local anesthetics affect transmission at the neuromuscular junction. Procaine, for example, can block the response of skeletal muscle to maximal motor-nerve volleys and to acetylcholine at concentrations where the muscle responds normally to direct electrical stimulation. Similar effects occur at autonomic ganglia. These effects are due to blockade of the ion channel of the acetylcholine receptor.[6]

Smooth Muscle

Local anesthetics depress contractions in the intact bowel and in strips of isolated intestine.[7] They also relax vascular and bronchial smooth muscle, although low concentrations may initially produce contraction.[7] Spinal and epidural anesthesia, as well as instillation of local anesthetics into the peritoneal cavity, cause sympathetic nervous system paralysis, which can result in increased tone of gastrointestinal musculature. Local anesthetics may increase the resting tone and decrease the contractions of isolated human uterine muscle; however, uterine contractions seldom are depressed directly during intrapartum regional anesthesia.

BIOTRANSFORMATION OF LOCAL ANESTHETICS

The rate of local anesthetic biotransformation is of great practical importance because the toxicity of local anesthetics depends largely on the balance between their rates of absorption, biotransformation, and elimination. The rate of absorption of many local anesthetics can be reduced considerably by the incorporation of a vasoconstrictor agent in the anesthetic solution. However, the rate of biotransformation of local anesthetics varies greatly, and this is a major factor in determining the safety of a particular agent. Since toxicity is related to the free concentration of drug, binding of anesthetic to proteins in the serum and to tissues reduces the concentration of free drug in the systemic circulation and, consequently, reduces toxicity. For example, in intravenous regional anesthesia of an extremity, about half of the original anesthetic dose is still tissue bound 30 minutes after release of the tourniquet. The lungs also bind large quantities of local anesthetic.[8]

Aminoester-linked local anesthetics are hydrolyzed at the aminoester linkage in plasma-by-plasma pseudocholinesterase. This enzyme also hydrolyzes natural choline esters and the neuromuscular blocking agent, succinylcholine. The rate of hydrolysis of aminoester-linked local anesthetics depends on the type and location of the substitution in the aromatic ring. For example, 2-chloroprocaine is hydrolyzed about four times faster than procaine, which in turn is hydrolyzed about four times faster than tetracaine. In the case of 2-chloroprocaine, the half-life in the normal adult is 45 seconds to 1 minute. In individuals with atypical plasma pseudocholinesterase, the rate of hydrolysis of all the ester-linked local anesthetics is markedly decreased, and a prolonged half-life of these drugs results. Therefore, whereas the potential for toxicity from plasma accumulation of the ester-linked local anesthetics (e.g., 2-chloroprocaine) is extremely remote with repeated dosing of the drug in normal individuals, this likelihood should be considered with the administration of large doses or repeated doses to individuals with the atypical pseudocholinesterase enzyme.[9]

The hydrolysis of all aminoester-linked local anesthetics leads to the formation of para-aminobenzoic acid (PABA) or a substituted PABA. PABA and its derivatives are associated with a low but real potential for allergic reactions.[10] A history of an allergic reaction to a local anesthetic agent should be considered primarily as resulting from the presence of PABA or derived from aminoester-linked local anesthetics. Allergic reactions may also develop from the use of multidose vials of aminoamide-linked local anesthetics that contain PABA as a preservative. Allergic reactions to aminoamide-linked local anesthetics without preservatives are rare. However, Mackley and colleagues[11] concluded that contact type IV sensitivity to lidocaine may occur more frequently than previously thought.

The aminoamide-linked local anesthetics, in contrast to the aminoester-linked drugs, are biotransformed primarily in the liver by cytochrome P450 enzymes. Two major factors controlling the clearance of aminoamide-linked local anesthetics by the liver are (1) hepatic blood flow (delivery of the drug to the liver) and (2) hepatic function (drug extraction by the liver). Factors that decrease hepatic blood flow or hepatic drug extraction result in an increased elimination half-life.

Renal clearance of unchanged local anesthetics is a minor route of elimination. For example, the amount of unchanged lidocaine excretion in the urine in the adult is small, roughly 3% to 5% of the total drug administered. For bupivacaine, the renal excretion of unchanged drug is also small but somewhat higher, in the 10–16% range of the administered dose.

Lidocaine biotransformation occurs following uptake of the drug by the liver. The primary biotransformation step for lidocaine is a dealkylation reaction in which an ethyl group is cleaved from the tertiary amine (Figure 2-1). Interestingly, this primary step in lidocaine's biotransformation appears to be only slightly slower in the newborn than in the adult, indicating functional maturity of this particular enzyme system in the newborn. However, an increase in the elimination half-life of lidocaine in the newborn of about twofold is seen, which is believed to result not from enzymatic immaturity but, instead, to reflect the larger volume of distribution for lidocaine in the newborn. A larger volume of distribution means that a given dose of drug achieves a lower plasma concentration; thus, less drug would be delivered to the liver for metabolism per unit

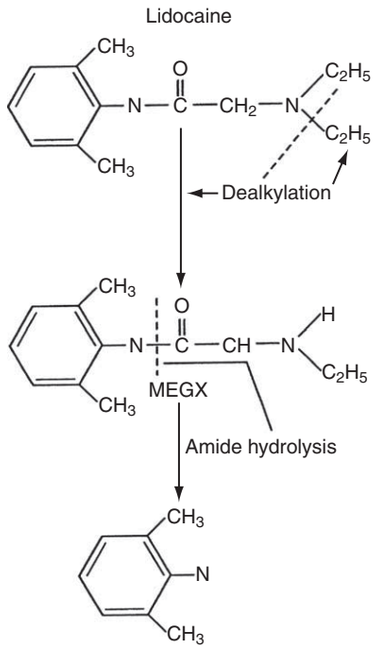


FIGURE 2-1

Metabolism of lidocaine illustrating a dealkylation reaction. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, figure 13-9, p. 188, with permission.)

time and to the kidney for excretion. Thus, it would take longer to clear a drug from the body when the drug has a larger volume of distribution.

As with the biotransformation of lidocaine, that of bupivacaine progresses with a dealkylation reaction as the primary step (Figure 2-2). Again, in the newborn an increased volume of distribution is present for bupivacaine and a longer half-life is thus anticipated compared with that expected in the adult. Other reactions in the biotransformation of amide-linked local anesthetics include hydrolysis of the amide link and oxidation of the benzene ring portion of the drug. The products of biotransformation can be cleared by the kidney as unchanged or conjugated compounds. For example, when hydroxy derivatives are formed from the oxidation of the benzene ring, they are conjugated and excreted as the glucuronide or sulfate conjugate.

With mepivacaine, the primary metabolic pathway is the oxidation of the benzene ring portion of the molecule, producing 3-hydroxy and 4-hydroxymepivacaine. Because this oxidation metabolic pathway is less well developed in the newborn, mepivacaine metabolism occurs much slower in the newborn than in the adult.

Ropivacaine metabolism in humans has been studied extensively (Figure 2-3). At low plasma concentrations, the drug is primarily metabolized by ring oxidation to 3-hydroxyropivacaine, which is conjugated and excreted in the urine.[12] Significantly less drug is metabolized by dealkylation at low concentrations to PPX. At high concentrations in vitro, dealkylation to PPX becomes an important pathway.[13] The metabolites formed are much less active than the parent compound ropivacaine. Renal clearance of ropivacaine also is relatively small, with only about 1% of the administered dose excreted unchanged in the urine.

The metabolism of local anesthetics, as well as that of many other drugs, occurs in the liver by the cytochrome P-450 enzymes. This enzyme family has been subdivided into a number of isoenzymes, with those predominantly involved in local anesthetic biotransformation reactions being CYP-1A2 and CYP-3A4. The predominant cytochrome P-450 isoenzyme present in the human liver is CYP-3A4. This isoenzyme accounts for approximately 30–60% of the total cytochrome P-450 content in the liver. It is primarily responsible for the dealkylation reaction in drug metabolism, which, in the case of lidocaine, produces MEGX; with bupivacaine and ropivacaine, PPX is produced.

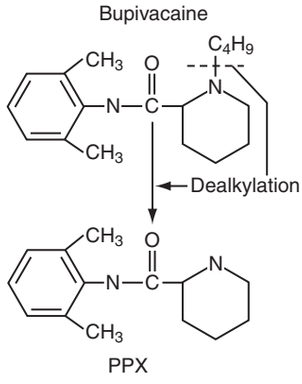


FIGURE 2-2

Metabolism of bupivacaine. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, figure 13-10, p. 188, with permission.)

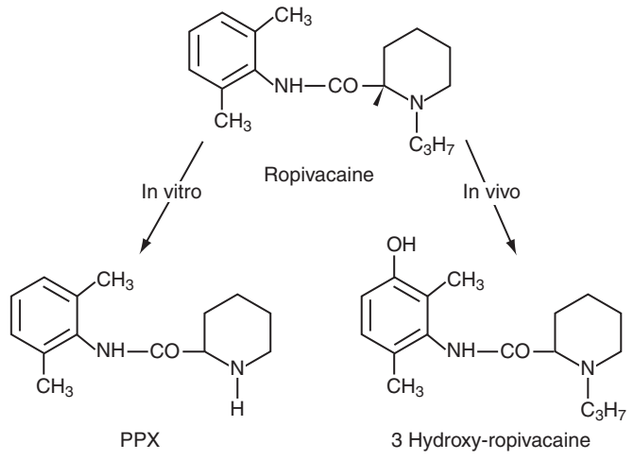


FIGURE 2-3

Schematic diagram showing the two major pathways of ropivacaine metabolism. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, figure 13-11, p. 189, with permission.)

SPECIFIC LOCAL ANESTHETICS

Local anesthetics are classified as either aminoester or aminoamide agents. Clinically useful ester agents are procaine, 2-chloroprocaine, and tetracaine.

Procaine

Procaine (Novocain), introduced in 1905 as the first synthetic local anesthetic, is an amino ester (Figure 2-4). Although it formerly was used widely, it is now confined to infiltration anesthesia and occasionally to diagnostic nerve blocks. This is because of its low potency, slow onset, and short duration of action. While its toxicity is fairly low, it is hydrolyzed in vivo to produce PABA, which inhibits the action of sulfonamides. Thus, large doses should not be administered to patients taking sulfonamide drugs.

2-chloroprocaine

2-chloroprocaine (Nesacaine), an ester local anesthetic introduced in 1952, is a chlorinated derivative of procaine (Figure 2-4). Its major assets are its rapid onset and short duration of action and its reduced acute toxicity due to its rapid metabolism (plasma half-life of approximately 25 seconds). Enthusiasm for its use has been tempered by reports of prolonged sensory and motor block after epidural or subarachnoid administration of large doses. This toxicity appears to have been a consequence of low pH and the use of sodium metabisulfite as a preservative in earlier formulations. There are no reports of neurotoxicity with newer preparations of chloroprocaine, which contain calcium EDTA as the preservative, although these preparations also are not recommended for intrathecal administration. A higher-than-expected incidence of muscular back pain following epidural anesthesia with 2-chloroprocaine also has been reported.[14] This back pain is thought to be due to tetany in the paraspinous muscles, which may be a consequence of Ca^{2+} binding by the EDTA included as a preservative; the incidence of back pain appears to be related to the volume of drug injected and its use for skin infiltration.

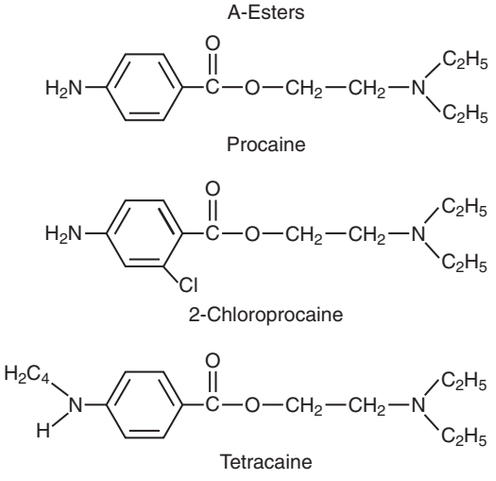


FIGURE 2-4

Local anesthetic chemical structures illustrating ester linkage. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, figure 13-12, p. 193, with permission.)

PHARMACOLOGY AND PHARMACODYNAMICS

2-chloroprocaine is procaine with the addition of a chlorine group to the benzene ring. This drug has a very rapid onset of action and a short duration of activity (30–60 minutes). Once absorbed into the circulation, the drug is rapidly metabolized. The approximate half-life in plasma in adults is 45 seconds to 1 minute; hence, it is the most rapidly metabolized local anesthetic currently used. Because of this extremely rapid breakdown in plasma, it has very low potential for systemic toxicity and has been particularly attractive to obstetric anesthesiologists for use when elevated maternal blood levels of local anesthetic can cause major problems for the fetus and mother. This drug is also frequently used for epidural and peripheral blocks in an ambulatory care setting when short duration of anesthesia is needed and rapid recovery is highly desirable.

The epidural use of this drug, however, has been limited because of several reported problems. Prolonged and profound motor and sensory deficits occurred with the unintentional subarachnoid injection of the original 2-chloroprocaine commercial preparation marketed with the preservative bisulfite. The classic work by Gissen and coworkers[15] and Wang and colleagues[16] demonstrated that bisulfite in the presence of a highly acidic solution releases sulfur dioxide (SO_2), which equilibrates in solution into sulfurous acid, which is neurotoxic. Gissen postulated that the injection of the highly acidic commercial 2-chloroprocaine (pH 3) solution into the spinal sac resulted in the slow formation of and prolonged exposure to sulfurous acid, causing spinal cord damage. More recently, a 2-chloroprocaine preparation was released in which the bisulfite was removed and EDTA was substituted as the preservative. This change, however, has not been totally satisfactory because there appears to be a significant occurrence of back muscle spasm after epidural application of this formulation.[17] It has been postulated that the EDTA in this commercial preparation binds calcium and causes spasm in the paraspinal muscles.

A new 2-chloroprocaine commercial preparation has been released in which all preservatives have been removed. Initial studies with this formulation appear to be promising. No preparations of 2-chloroprocaine are recommended for either spinal or intravenous regional anesthesia.

Aminoamide local anesthetics currently used are lidocaine, mepivacaine, bupivacaine, ropivacaine, and levobupivacaine.

Lidocaine

Lidocaine, introduced in 1948, is now the most widely used local anesthetic. The chemical structure of lidocaine is shown in Figure 2-5.

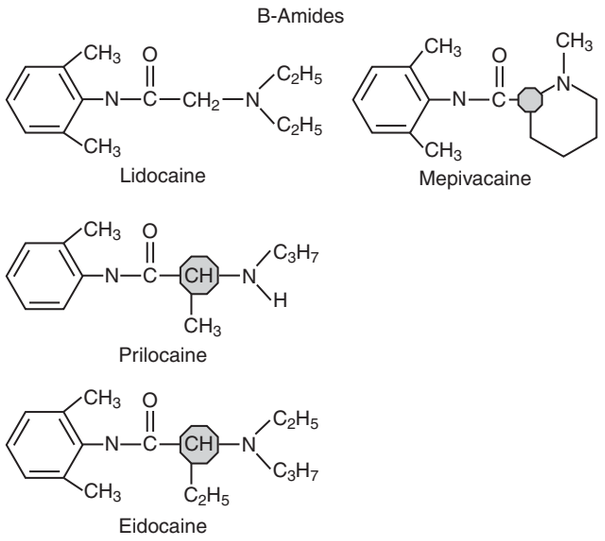


FIGURE 2-5

Local anesthetic chemical structures illustrating amide linkage and indicating an asymmetric carbon atom (asterisk) when present. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, figure 13-13, p. 194, with permission.)

Pharmacologic action: The pharmacologic actions that lidocaine shares with other local anesthetic drugs have been described widely. Lidocaine produces faster, more intense, longer lasting, and more extensive anesthesia than does an equal concentration of procaine. Unlike procaine, it is an aminoethylamide and is the prototypical member of this class of local anesthetics. It is a good choice for patients sensitive to ester-type local anesthetics.

Absorption rate and excretion: Lidocaine is absorbed rapidly after parenteral administration and from the gastrointestinal and respiratory tracts. Although it is effective when used without vasoconstrictor, in the presence of epinephrine the rate of absorption and the toxicity are decreased and the duration of action usually is prolonged. Lidocaine is dealkylated in the liver by mixed-function oxidases to monoethylglycine xylidide and glycine xylidide, which can be metabolized further to monoethylglycine and xylidide. Both monoethylglycine xylidide and glycine xylidide retain local anesthetic activity. In humans, about 75% of xylidide is excreted in the urine as the further metabolite, 4-hydroxy-2, 6-dimethylaniline.[8]

Toxicity: The side effects of lidocaine seen with increasing dose include drowsiness, tinnitus, dysphagia, dizziness, and twitching. As the dose increases, seizures, coma, and respiratory depression and arrest occur. Clinically significant cardiovascular depression usually occurs at serum lidocaine levels that produce marked CNS effects. The metabolite monoethylglycine xylidide and glycine xylidide may contribute to some of these side effects.

Clinical uses: Lidocaine has a wide range of clinical uses as a local anesthetic; it is useful in almost any application where a local anesthetic of intermediate duration is needed. Lidocaine also is used as an antiarrhythmic agent.

Mepivacaine

Mepivacaine, introduced in 1957, is an intermediate-acting aminoamide (see Figure 2-5). Its pharmacologic properties are similar to those of lidocaine. Mepivacaine, however, is more toxic to the neonate and thus is not used in obstetric anesthesia. The increased toxicity of mepivacaine in the neonate is related not to its slower metabolism in the neonate but to ion trapping of this agent because of the lower pH of neonatal blood and the pKa of mepivacaine. Despite its slow metabolism in the neonate, it appears to have a slightly higher therapeutic index in adults than lidocaine. Its onset of action is similar to that of lidocaine, and its duration slightly longer (about 20%) than that of lidocaine in the absence of a coadministered vasoconstrictor. Mepivacaine is not effective as a topical anesthetic.

Bupivacaine

Bupivacaine, introduced in 1963, is a widely used amide local anesthetic; its structure is similar to that of lidocaine, except the amine-containing group is a butyl piperidine (see Figure 2-5). It is a potent agent capable of producing prolonged anesthesia. Its long duration of action plus its tendency to provide more sensory than motor block has made it a popular drug for providing prolonged analgesia during labor or the postoperative period. By taking advantage of indwelling catheters and continuous infusions, bupivacaine can be used to provide several days of effective analgesia.

Bupivacaine was developed as a modification of mepivacaine. Its structural similarities with mepivacaine are readily apparent. Bupivacaine has a butyl (four-carbon substitution) group on the hydrophilic nitrogen.

Bupivacaine has made a contribution to regional anesthesia second in importance only to lidocaine. It is one of the first of the clinically used local anesthetic drugs that provides good separation of motor and sensory blockade after its administration. The onset of anesthesia and the duration of action are long and can be further prolonged by the addition of epinephrine in areas with a low fat content. Only small increases in duration are seen when bupivacaine is injected into areas with a high fat content. For example, a 50% increase in duration of brachial plexus blockade (an area of low fat content) follows the addition of epinephrine to bupivacaine solutions; in contrast, only a 10–15% increase in duration of epidural anesthesia results from the addition of epinephrine to bupivacaine solutions, since the epidural space has a high fat content.

Toxicity: Bupivacaine is more cardiotoxic than equieffective doses of lidocaine. Clinically, this is manifested by severe ventricular arrhythmias and myocardial depression after inadvertent intravascular administration of large doses of bupivacaine. The enhanced cardiotoxicity of bupivacaine probably is due to multiple factors. Lidocaine and bupivacaine both block cardiac Na^+ channels rapidly during systole. However, bupivacaine dissociates much more slowly than does lidocaine during diastole, so a significant fraction

of Na^+ channels remains blocked at the end of diastole (at physiologic heart rates) with bupivacaine.[18] Thus, the block by bupivacaine is cumulative and substantially more than would be predicted by its local anesthetic potency. At least a portion of the cardiac toxicity of bupivacaine may be mediated centrally, as direct injection of small quantities of bupivacaine into the medulla can produce malignant ventricular arrhythmias.[19] Bupivacaine-induced cardiac toxicity can be difficult to treat, and its severity is enhanced in the presence of acidosis, hypercarbia, and hypoxemia.

NEWER LOCAL ANESTHETICS

Chiral Forms

An area of newfound importance for anesthesiologists is in the use of stereoisomers of drugs to take advantage of differences in activity or toxicity of the isomers. For stereoisomerism to be present, an asymmetric carbon (a carbon atom in the molecule that has four distinctly different substitution groups) must be present in the molecule. Stereoisomers are possible for the local anesthetics etidocaine, mepivacaine, bupivacaine, prilocaine, and ropivacaine, and some of these drugs have differences in potency or toxicity for the isomers. For these local anesthetics, the asymmetric carbons are indicated in Figure 2-6 with an asterisk.

In the older literature, isomers were described as *L* and *D* on the basis of chemical configuration and as (+) or (–) on the basis of topical rotation, that is, *L* (+) or *D* (–). More recent literature describes isomers as *R* or S, and the optical rotation is still included in the parentheses as (+) and (–). *R* and *S* basically correspond to the *D* and L, respectively, in the older nomenclature.

As a rule, when differences between the activity of isomers are present for local anesthetics, the *S* form is less toxic and has a longer duration of anesthesia.[10,20] For instance, anesthesia produced by bupivacaine infiltration was of longer duration when the *S* isomer was used compared with the *R* isomer. Also, the *S* isomer had lower systemic toxicity. The mean convulsant dose of *R* bupivacaine was 57% of the *S* bupivacaine convulsant dose. When the isomers of ropivacaine were evaluated, the *S* isomer of the drug had a longer duration of blockade and a lower toxicity than its *R* isomer. Additionally, when cardiac electrophysiologic toxicity was evaluated in animal studies, ropivacaine (the commercial preparation is the *S* form of drug) at equipotent nerve blocking doses appears to have a safety margin that is almost twice that of commercial bupivacaine, which is a mixture of the *R* and *S* isomers.[20] Recent studies with the *R* and *S* bupivacaine isomers indicate that the *R* form is apparently more arrhythmogenic and more cardiotoxic.

Ropivacaine

The cardiac toxicity of bupivacaine stimulated interest in developing a less toxic, long-lasting local anesthetic. The result of that search was the development of a new amino ethylamine, ropivacaine (Figure 2-7), the S-enantiomer of

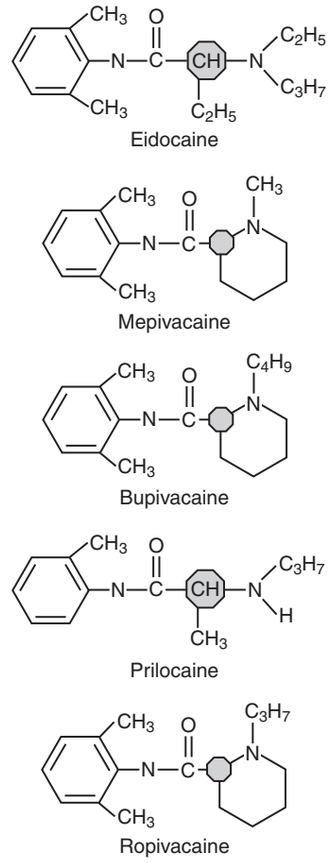


FIGURE 2-6

Stereoisomers are possible for local anesthetics: etidocaine, mepivacaine, bupivacaine, prilocaine, and ropivacaine. The asymmetric carbons are indicated with an asterisk. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, figure 13-14, p. 197, with permission.)

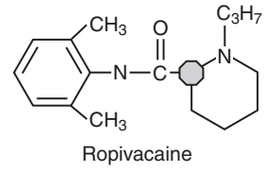


FIGURE 2-7

Ropivacaine. This chemical structure is the S-enantiomer of 1-propyl-2',6'-pipecoloxyclidide. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, figure 13-15, p. 197, with permission.)

1-propyl-2',6'-pipecolocylidide. The S-enantiomer, like most local anesthetics with a chiral center, was chosen because it has a lower toxicity than the *R* isomer. This is presumably due to slower uptake, resulting in lower blood levels for a given dose. Ropivacaine is slightly less potent than bupivacaine in producing anesthesia. In several animal models, it appears to be less cardiotoxic than equieffective doses of bupivacaine. In clinical studies, ropivacaine appears to be suitable for both epidural and regional anesthesia, with duration of action similar to that

of bupivacaine. Interestingly, it seems to be even more motor sparing than bupivacaine.

Ropivacaine is a long-acting, enantiomerically pure (*S*-enantiomer) amide local anesthetic with a high pKa and low lipid solubility that blocks nerve fibers involved in pain transmission (Aδ and C fibers) to a greater degree than those controlling motor function (Aβ fibers). The drug was less cardiotoxic than equal concentrations of racemic bupivacaine, but more so than lidocaine (lignocaine) in vitro, and had a significantly higher threshold for CNS toxicity than racemic bupivacaine in healthy volunteers (mean maximum tolerated unbound arterial plasma concentrations were 0.56 and 0.3 mg/l, respectively).

Extensive clinical data have shown that epidural ropivacaine 0.2% is effective for the initiation and maintenance of labor analgesia and provides pain relief after abdominal or orthopedic surgery, especially when given in conjunction with opioids (coadministration with opioids may also allow for lower concentrations of ropivacaine to be used). The drug had an efficacy generally similar to that of the same dose of bupivacaine with regard to pain relief but caused less motor blockade at low concentrations.

Levobupivacaine

Levobupivacaine injection contains a single enantiomer of bupivacaine hydrochloride that is chemically described as S-1-butyl-2-piperidylformo-2', 6'-xylidide hydrochloride and it is related chemically and pharmacologically to the amino amide class of local anesthetics (Figure 2-8).

Levobupivacaine hydrochloride, the S-enantiomer of bupivacaine, is a white crystalline powder with a molecular formula of $C_{18}H_{28}N_2O{\bullet}HCl$, a molecular weight of 324.9.

The solubility of levobupivacaine hydrochloride in water is about 100 mg/ml at 20°C, and the partition coefficient (oleyl alcohol/water) is 1624; the pKa of levobupivacaine hydrochloride is the same as that of bupivacaine hydrochloride, and the partition coefficient is very similar to that of bupivacaine hydrochloride (1565).

Levobupivacaine is a sterile, nonpyrogenic, colorless solution (pH 4.0–6.5) containing levobupivacaine hydrochloride equivalent to 2.5 mg/ml, 5.0 mg/ml, and 7.5 mg/ml of levobupivacaine, sodium chloride for isotonicity, and water for injection. Sodium hydroxide and/or hydrochloric acid may be added to adjust the pH. Levobupivacaine is preservative free and is available in 10- and 30-ml single-dose vials.

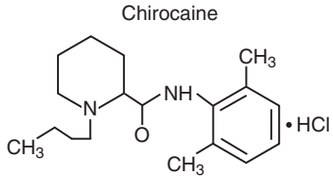


FIGURE 2-8

A single enantiomer of levobupivacaine hydrochloride (Chirocaine) chemically described as S-1-butyl-2-piperidylformo-2',6'-xylidide hydrochloride. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, figure 13-16, p. 200, with permission.)

Mechanism of action. Levobupivacaine is a member of the amino amide class of local anesthetics. Local anesthetics block the generation and the conduction of nerve impulses by increasing the threshold for electrical excitation in the nerve, by slowing propagation of the nerve impulse, and by reducing the rate of rise of the action potential. In general, the progression of anesthesia is related to the diameter, myelination, and conduction velocity of affected nerve fibers. Clinically, the order of loss of nerve function is as follows: (1) pain, (2) temperature, (3) touch, (4) proprioception, and (5) skeletal muscle tone.

Pharmacokinetics. After intravenous infusion of equivalent doses of levobupivacaine and bupivacaine, the mean clearance, volume of distribution, and terminal half-life values of levobupivacaine and bupivacaine were similar. No detectable levels of R(+)-bupivacaine were found after the administration of levobupivacaine.

Plasma-protein binding of levobupivacaine evaluated in vitro was found to be greater than 97% at concentrations between 0.1 and 1 μg/ml. The association of levobupivacaine with human blood cells was very low (0–2%) over the concentration range 0.01 to 1 μg/ml and increased to 32% at 10 μg/ml. The volume of distribution of levobupivacaine after intravenous administration was 67 liters.

Levobupivacaine is extensively metabolized with no unchanged levobupivacaine detected in urine or feces. In vitro studies using [^{14}C]levobupivacaine showed that CYP3A4 isoform and CYP1A2 isoform mediate the metabolism of levobupivacaine to desbutyl levobupivacaine and 3-hydroxy levobupivacaine, respectively. In vivo, the 3-hydroxy- levobupivacaine appears to undergo further transformation to glucuronide and sulfate conjugates. Metabolic inversion of levobupivacaine to R(+)-bupivacaine was not evident both in vitro and in vivo.

Following intravenous administration, recovery of the radiolabeled dose of levobupivacaine was essentially quantitative, with a mean total of about 95% being recovered in urine and feces in 48 hours. Of this 95%, about 71% was in urine, whereas 24% was in feces. The mean elimination half-life of total radioactivity in plasma was 3.3 hours. The mean clearance and terminal half-life of levobupivacaine after intravenous infusion were 39 l/hr and 1.3 hours, respectively.

Toxicity. Levobupivacaine can be expected to share the toxicity properties of other local anesthetics. Systemic absorption of local anesthetics can produce effects on the CNS and CVS. At blood concentrations achieved with therapeutic doses, changes in cardiac conduction such as excitability, refractoriness, contractility, and peripheral vascular resistance have been reported. Toxic blood concentrations depress cardiac conduction and excitability, which may lead to atrioventricular block, ventricular arrhythmias, and cardiac arrest, sometimes resulting in death. In addition, myocardial contractility is depressed and peripheral vasodilation occurs, leading to decreased cardiac output and arterial blood pressure.

Chiral local anesthetics, such as ropivacaine and levobupivacaine, have the potential advantage over racemic mixtures in showing reduced toxic side effects. However, these isomers also have reportedly lower potency than their optical antipode, possibly resulting in no advantage in therapeutic index. Potency for local anesthetics inhibiting Na^+ channels or action potentials depends on the pattern of membrane potential, and so also does the stereopotency ratio. Here the authors have quantitated the stereopotencies of *R*-, *S*-, and racemic bupivacaine, comparing several in vitro assays of neuronal Na^+ channels with those from in vivo functional nerve block, to establish relative potencies and to understand better the role of different modes of channel inhibition in overall functional anesthesia.

CHEMICAL NEUROLYTIC AGENTS

Prolonged interruption of painful pathways may be accomplished by the injection of neurolytic agents. This form of chemical neurolysis has been performed for many years. The first reported injection of a neurolytic solution in the treatment of pain was probably by Luton,[20] who in 1863 administered subcutaneous injections of irritant substances into painful areas. Levy and Baudouin (1906) were the first to administer the injection of neurolytic agents percutaneously.[21] Doppler, in 1925, was the first to report the use of phenol for neurolysis.[21] The first use of phenol for subarachnoid neurolysis was reported by Maher in 1955.[21,22] Today, phenol and ethyl alcohol (ethanol) are the most commonly used agents. It is indicated for patients with limited life expectancy and patients who have recurrent or intractable pain after a series of analgesic blocks.[26]

CONSIDERATIONS PRIOR TO USE OF NEUROLYTIC AGENTS

Diagnostic blocks are considered of prime importance due to the undesirable side effects of the neurolytic agents combined with a limited duration of analgesia. Potential side effects of neurolytic agents include neuritis and deafferentation pain, motor deficit when mixed nerves are ablated, and unintentional damage to nontargeted tissue.[23] Therefore, careful selection of patients combined with clinical expertise is of the essence. The following criteria should be considered before peripheral neurolysis is performed[23]:

- Determine and document that the pain is severe.
- Document that the pain will not be relieved by less invasive therapies.
- Document that the pain is well localized and in the distribution of an identifiable nerve.[24]
- Confirm that the pain is relieved with a diagnostic block performed with local anesthetic.
- Document the absence of undesirable deficits after the local anesthetic blocks.[23]

ETHYL ALCOHOL

Ethyl alcohol is commercially available in 1- or 50-ml ampules as a colorless solution that can be injected readily through small-bore needles.[23] It is hypobaric with respect to cerebrospinal fluid (CSF). However, specific gravity is not of concern when injecting on the peripheral nerve because injection takes place in a nonfluid medium.[23] It is usually used undiluted (absolute or >95% concentration). The perineural injection of alcohol is followed immediately by severe burning pain along the nerve's distribution, which lasts about a minute before giving way to a warm, numb sensation. Pain on injection may be diminished by the prior injection of a local anesthetic.[23] To precede the injection of any neurolytic drug with an injection of local anesthetic optimizes comfort and serves as a "test dose."[23] The alcohol spreads rapidly from the injection site. When injected in the CSF, only 10% of the initial dose remains at the site of the injection after 10 minutes and about 4% remains after 30 minutes.[25] Between 90% and 98% of the ethanol that enters the body is completely oxidized.[26] This occurs chiefly in the liver and is initiated principally by alcohol dehydrogenase.[27] Denervation and pain relief accrue over a few days after injection, usually after 1 week. If no pain relief is present in weeks, then the neurolysis is incomplete and needs repetition.[23]

Various concentrations and mixtures of alcohol have been studied in an attempt to determine selectivity for sensory nerves.[28,29] Schlosser[28] studied the effect of alcohol on somatic nerves. He reported that alcoholization was followed by degeneration and absorption of all the components of the nerve except the neurilemma. There is general agreement that with 95% absolute alcohol, the destruction involves the sympathetic, sensory, and motor components of a mixed somatic nerve, and therefore it is undesirable to block a mixed nerve with such concentrations of alcohol. However, there is a great discrepancy in determining the effects when the alcohol is placed on motor fibers at less than 80% concentration.

Despite the inconsistency in results for varying concentrations of alcohol, there is consensus regarding maximum and minimum concentrations. For complete paralysis, the concentration must be stronger than 95%. From Labat and Greene,[30] it may be concluded that a minimum concentration of 33% alcohol is necessary to obtain satisfactory analgesia without any motor paralysis.

Mechanism of Action

Histopathologic studies have shown that alcohol extracts cholesterol, phospholipids, and cerebrosides from the nerve tissue and causes precipitation of lipoproteins and mucoproteins.[31,32] This results in sclerosis of the nerve fibers and myelin sheath.[33,34] Alcohol produces nonselective destruction of nervous tissue by precipitating cell membrane proteins and extracting lipid compounds, resulting in demyelination and subsequent Wallerian degeneration. Because the basal lamina of the Schwann cell tube is often

spared, however, the axon often regenerates along its former course.[34] If injection is into a ganglion, it may produce cell body destruction without subsequent regeneration.[34] Topical application of alcohol to peripheral nerves produces changes typical of Wallerian degeneration. A subarachnoid injection of absolute alcohol causes similar changes in the rootlets.[32,35] Mild focal inflammation of meninges and patchy areas of demyelination are seen in posterior columns, Lissauer's tract, and dorsal roots and rootlets. Later, Wallerian degeneration is seen to extend into the dorsal horns. Injection of a larger volume can result in degeneration of the spinal cord.[32] When alcohol is injected near the sympathetic chain, it destroys the ganglion cells and thus blocks all postganglionic fibers to all effector organs.[36] A temporary and incomplete block results if the injection affects only the rami communicantes of preganglionic and postganglionic fibers. Histopathologically, Wallerian degeneration is evident in the sympathetic chain fibers.[32]

For subarachnoid block, concentrations between 50% and 100% are generally selected (Figure 2-9). Alcohol is hypobaric in nature relative to CSF. Therefore, the position of the patient must be in the lateral decubitus position with the painful site uppermost. Then, the patient must be rolled anteriorly approximately 45 degrees to place the dorsal (sensory) root uppermost.[37] The reported volumes required for neurolysis have ranged from 0.3 ml to a maximum of 0.7 ml of absolute alcohol per segment[26] to 0.5 to 1 ml to a maximum of 1.5 ml per segment.[29,33] For celiac plexus block, volumes of 10 to 20 ml of absolute alcohol bilaterally may be used.[23] Similar volumes have been reported for lumbar sympathetic block. Often, 100% alcohol is diluted 1:1 with a local anesthetic prior to injection.[29]

The most ominous complication associated with the use of alcohol is the possible occurrence of alcoholic neuritis. It has been postulated that alcoholic neuritis is due to incomplete destruction of somatic nerves. This seems plausible, in that neuritis has not been observed following the intraneural injection of a cranial or somatic nerve that produces a complete block.[29] Alcoholic neuritis occurs frequently following paravertebral block of the thoracic sympathetics. This may be due to the close proximity of the sympathetic ganglia to the intercostal nerves. The alcohol, which is intended for the ganglion, inadvertently bathes and partially destroys the somatic nerve.[29] During the period of regeneration, hyperesthesia and intense burning pain with occasional sharp, shooting pain occurs. These pains may be more intense than the original pain complaint. Fortunately, in most instances, these symptoms subside within a few weeks or a month. Occasionally, however, this complication persists for many months, requiring sedation, and in some instances, the performance of a subsequent rhizotomy or sympathectomy.[29] As a prophylactic measure, Mandl[38] recommends the injection of a local anesthetic during the insertion of the needle, at the site of injection before the alcohol is injected, and on withdrawing the needle. With this technique he has observed only two instances of alcoholic neuritis.

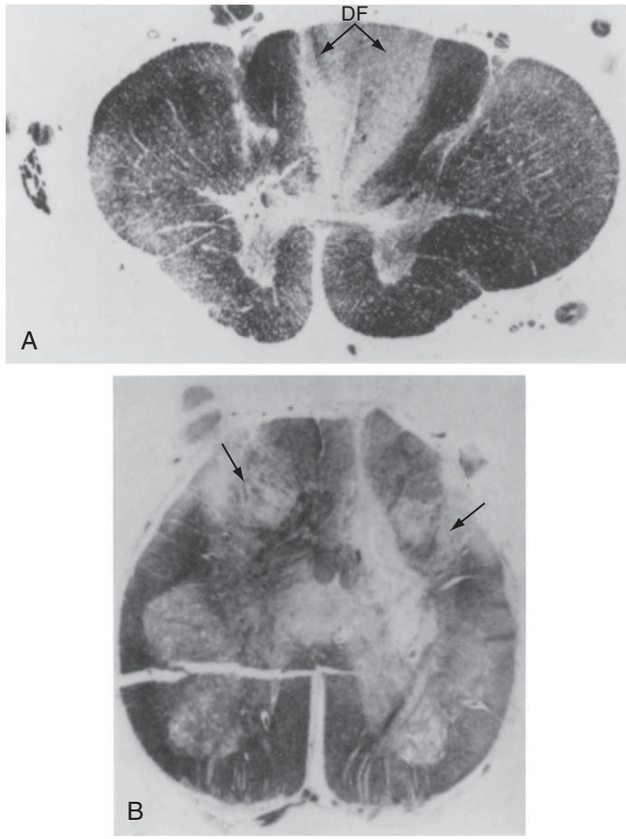


FIGURE 2-9

A, Effect of alcohol on the spinal cord 4 days after neurolytic block. A cross section through the spinal cord at T4 shows degeneration of the dorsal fascicularis (DF) after injection of 100% alcohol several interspaces lower. B, Effect of alcohol on the spinal cord 50 days after direct cord injection. Note the necrosis and degeneration (arrows) following accidental injection of 100% alcohol into the spinal cord. (From Gallagher HS, Yonexawa T, Hay RC et al: Subarachnoid alcohol block: II. Histologic changes in the central nervous system. *Am J Pathol* 35:679, 1961, with permission.)

Mild cases of alcoholic neuritis are treated conservatively with mild analgesics such as aspirin or with small doses of codeine.[29] Moderate cases of alcoholic neuritis may require more active therapy. In some cases, the administration of intravenous local anesthetics has been helpful. Bonica[39] determined that 250 mg of tetracaine dissolved in 500 ml of fluid was superior to procaine.[39] In one case in which intravenous procaine had been administered several times with only transient relief of pain, one infusion of tetracaine effected prolonged pain relief. In some cases, daily sympathetic blocks have been employed with excellent results.[29] In the case of lumbar nerve neuritis following lumbar sympathetic blocks, serial caudal blocks done at regular intervals can effect complete relief of pain.[29] Severe cases of alcoholic neuritis that do not respond to these conservative methods may require sympathectomy or rhizotomy.

De Takats[40] reported three such cases in which sympathectomy was required.

Another complication associated with alcohol nerve block includes hypoesthesia or anesthesia of the dermatomal distribution of the nerve roots treated with neurolysis. The lack of sensation can overshadow the pain relief obtained by the procedure. Fortunately, this complication is rare and recovery is relatively quick.[32] Loss of bowel or bladder sphincter tone, leading to bowel or urinary incontinence, has also been reported with intrathecal alcohol neurolysis in the lower lumbar and sacral areas.[32] To decrease the risk of this complication, it is recommended that during sacral neurolysis, only one side should be blocked at a time.[32] A complication of lumbar sympathetic neurolysis with alcohol is the development of genitofemoral neuralgia, which can cause severe groin pain. This is referred pain caused by the degeneration of the rami communicantes from the L2 nerve root to the genitofemoral nerve.[32–43] Paraplegia can result if injection of alcohol causes spasm of the artery of Adamkiewicz.[32]

PHENOL

Phenol is a combination of carbolic acid, phenic acid, phenylic acid, phenyl hydroxide, hydroxybenzene, and oxybenzene. It is not available commercially in the injectable form but can be prepared by the hospital pharmacy. One gram of phenol dissolves in about 15 ml of water (6.67%). It is very soluble in alcohol, glycerol, and a number of other organic substances. It is usually mixed with saline or glycerin. It may be mixed with sterile water or material used for contrast radiography.[23] Because it is highly soluble in glycerin, it diffuses from it slowly. This is an advantage when injecting intrathecally because it allows for limited spread and highly localized tissue fixation. This also makes it hyperbaric relative to CSF. When mixed with glycerin, it is so viscid that even when warmed, injection must be through at least a 20-gauge needle. This mixture must be free of water or the necrotizing effect will be much greater than anticipated.[23] When phenol is mixed in an aqueous mixture, it is a far more potent neurolytic.[32] Phenol oxidizes and turns red when exposed to air and light.[23] It has a shelf life that is said to exceed 1 year when preparations are refrigerated and not exposed to light. Phenol acts as a local anesthetic at lower concentrations and as a neurolytic agent in higher concentrations. It has an advantage over alcohol in that it causes minimal discomfort on injection.

Doppler was the first to use phenol to deliberately destroy nervous tissue in 1925.[44] After painting it on human ovarian vessels, he noted downstream vasodilation and flush. Later, he reported treating peripheral vascular disease in the lower extremity by exposing and painting the femoral arteries with a 7% aqueous solution. In 1933, Binet[44] in France reported painting ovarian vessels with 7% phenol. Both researchers attributed their good results to destruction of perivascular sympathetic fibers.[29] In 1933, Nechaev[45] reported the use of phenol as a local anesthetic. This was followed in 1936 by Putnam and Hampton,[46] who used an injection of phenol to perform a neurolysis of the gasserian ganglion.

In 1947, Mandl[47] suggested the injection of phenol to obtain permanent sympathectomy. In 1950, he reported its use in 15 patients without complications, suggesting that it was preferable to alcohol.[29,38] The paravertebral injection of phenol for peripheral vascular disease was also reported by Haxton[48] and Boyd and coworkers[49] in 1949. In 1955, Maher[50] introduced it as a hyperbaric solution for intrathecal use in intractable cancer pain, with the famous remark that "it is easier to lay a carpet than to paper a ceiling." Thereafter, he reported its epidural use as well.

By 1959, phenol was established as a neurolytic agent for the relief of chronic pain.[29] Then Kelly and Gautier-Smith[51] and Nathan[52] simultaneously reported the use of phenol for the relief of spasticity caused by upper motor neuron lesions. Phenol, in hyperbaric solution, was injected intrathecally with proper patient positioning to "fix" it on the anterior nerve roots, thus relieving the spasticity (Figure 2-10).

Maher studied varying concentrations (10% to 3.3%) of phenol in glycerin in the subarachnoid space in an effort to determine the ideal neurolytic strength solution.[50] There was a graduation of block according to the concentration. The stronger concentration produced motor damage. Pain sensation was blocked at lower concentrations (5%) than were touch and proprioception. The 3.3% concentration was ineffective. Iggo and Walsh[53] determined that 5% phenol in either Ringer's solution or oil contrast medium produced selective block of the smaller nerve fibers in cat spinal rootlets. The same conclusions were drawn from the investigations by Nathan and Sears.[54] For a long time thereafter, the idea prevailed that phenol caused selective destruction of smaller nerve fibers with slower conduction rates, the C afferents carrying slow pain, the Aδ afferents carrying fast pain, and the Aγ controlling muscle tone.[29]

Mechanism of Action

Histopathologic studies by Stewart and Lourie[55] demonstrated nonselective degeneration in cat rootlets, the severity being parallel to the concentration.[29] Nathan and associates[56] found evidence of Aα and Aβ damage in electrophysiologic experiments and confirmed the nonselectivity of damage by histologic examination.

At concentrations less than 5%, phenol produces protein denaturation. Concentrations greater than 5% cause protein coagulation and nonspecific segmental demyelination and orthograde degeneration (i.e., Wallerian degeneration).[34] Concentrations of 5% to 6% produce destruction of nociceptive fibers with minimum side effects. Higher concentrations result in axonal abnormalities, nerve root damage, spinal cord infarcts, and arachnoiditis or meningitis.[34,57] These characteristics may explain the long-lasting results of

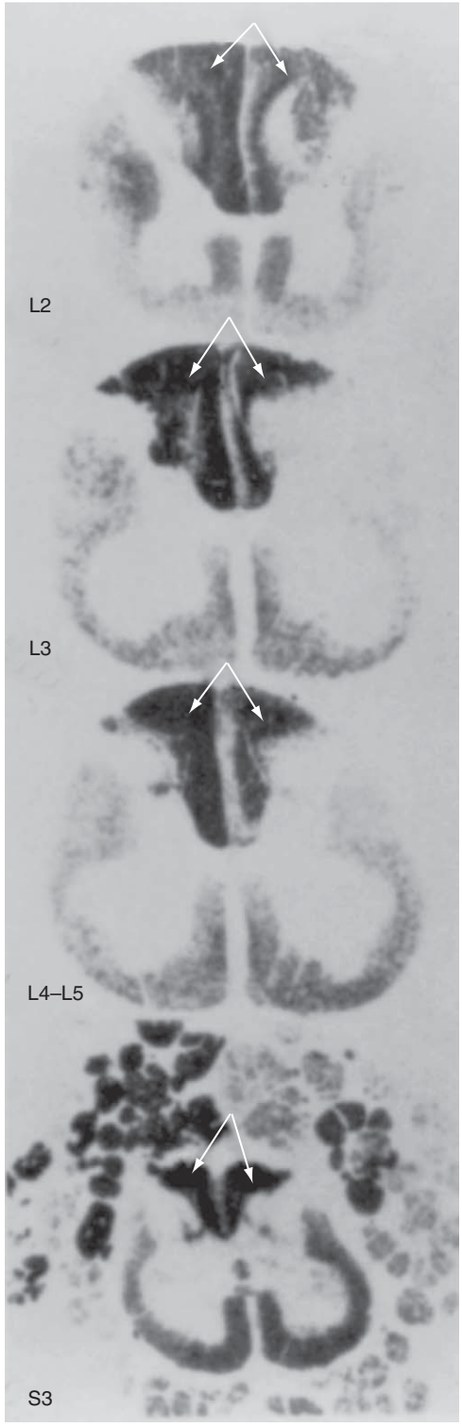


FIGURE 2-10
Effect of phenol on the spinal cord. Micrographs of transverse section at levels L2, L3, L4-5, and S3 show degeneration of the posterior column following subarachnoid injection of phenol at L3-L4. (From Smith MC: Histological findings following intrathecal injections of phenol solutions for relief of pain. *Br J Anaesth* 36:387, 1964, with permission.)

neurolytic blocks performed with 10% phenol in the sympathetic axis.[34]

The block produced by phenol tends to be less intense and of shorter duration than that produced by alcohol. Moller and associates[58] compared various concentrations of alcohol with phenol and concluded that 5% phenol equaled 40% alcohol in neurolytic potency. Axons of all sizes are affected by therapeutic concentrations and, as described by ethyl alcohol, appear edematous. The posterior root ganglia are unaffected by phenol.[59] Similar pathologic changes occur in peripheral nerves when exposed to phenol.[29] The process of degeneration takes about 14 days, and regeneration is completed in about 14 weeks. After an intrathecal injection of phenol, its concentration decreases rapidly to 30% of the original concentration in 60 seconds and to 0.1% within 15 minutes.[30,60]

Phenol is efficiently metabolized by liver enzymes. The principal pathways are conjugation to the glucuronides and oxidation to equinal compounds or to carbon dioxide and water. It is then excreted as a variety of conjugates via the kidney.[32]

A higher affinity for vascular tissue than for neuronal tissue has been suggested by Wood.[57] The interference with blood flow is believed to be the etiology for the observed neuropathy.[61,62] However, Racz and associates[63] studied the morphologic changes that occurred following epidural and subarachnoid injection. They found that massive tissue destruction was present after subarachnoid injection as compared with epidural injection despite intact vasculature in areas of spinal cord destruction.[63] These findings support a direct neurotoxic effect of phenol rather than an effect secondary to vascular destruction.[34,64]

Large systemic doses of phenol (>8.5 g) cause convulsions and then CNS depression and cardiovascular collapse. Chronic poisoning results in skin eruptions, gastrointestinal symptoms, and renal toxicity.[34] Clinical doses between 1 and 10 ml of 1–10% solutions (up to 1000 mg) are unlikely to cause serious toxicity.[34,65]

GLYCEROL

Glycerol is used mostly for neurolysis of the gasserian ganglion to treat idiopathic trigeminal neuralgia.[66] Considered a mild neurolytic, like other alcohols, it produces localized perineural damage, whereas intraneural injection results in Schwann cell edema, axolysis, and Wallerian degeneration.[34] In one histologic study, intraneural injection of glycerol was more damaging than topical application, although significant, localized, subperineural damage occurred after local application of a 50% glycerol solution.[34,67] Histologic changes included the presence of many inflammatory cells, extensive myelin swelling, and axolysis. Myelin disintegration occurs weeks after the injury along with ongoing axolysis during periods of myelin restitution, indicating an ongoing nerve fiber injury possibly caused by secondary events such as compression of

transperineal vessels and ischemia.[34,67,68] Electron microscopy shows evidence of Wallerian degeneration; with intraneural injection, all nerve fibers are destroyed.[33]

Mechanism of Action

The mechanism of action is not clear. Sweet and colleagues[69] suggested that glycerol affected primarily small myelinated and unmyelinated fibers.[34] Bennett and Lunsford,[70,71] using trigeminal evoked-potential studies, concluded that glycerol more specifically affects the damaged myelinated axons implicated in the pathogenesis of trigeminal neuralgia. Because there is no permanent injury to surrounding structures and facial sensation is preserved in most patients, Feldstein[72] thought that glycerol was superior to radiofrequency rhizotomy for the treatment of tic douloureux. However, potential spread to the subarachnoid space, the risk of neuropathy, and poor control of the spread of a fluid agent have made radiofrequency a continued attractive alternative. With the recent use of pulsed radiofrequency, the advantage of a discrete, controlled lesion remains without the concern for neuritis or loss of facial sensation. However, long-term follow-up on its effectiveness has not been reported.

HYPERTONIC AND HYPOTONIC SOLUTIONS

Hypertonic or hypotonic subarachnoid injections have been used for achieving neurolysis.[73] The intrathecal injection of cold (2–4°C) 0.9% NaCl is supposed to have a specific action on the pain-carrying C fibers, sparing the larger fibers that subserve sensory, motor, and autonomic functions.[74] The technique requires the spinal fluid to be withdrawn and replaced with cold saline as rapidly as possible.[29] Up to 40–60 ml of saline has been injected. Local anesthetic should be used concomitantly or the procedure can be quite painful. The pain relief is usually brief.[29]

Injections of hypertonic saline can be quite painful; therefore, local anesthetics are generally injected before the saline.[23] The intrathecal injection of hypertonic saline can produce a variety of complications.[75] Some degree of complications occurred in 11% and significant morbidity in 1% of patients. Two deaths have been reported secondary to myocardial infarction. During saline injection, sinus tachycardia or premature ventricular contraction have been seen,[76] and localized paresis lasting for many hours and paresthesia extending for weeks have been observed.[77] Other complications reported include hemiplegia, pulmonary edema, pain in the ear, vestibular disturbances, and loss of sphincter control with sacral anesthesia.[29,78,79]

Mechanism of Action

Pathologic changes due to hypertonic and hypotonic solutions have been extensively studied.[29,80,81] Microscopic changes seen on the peripheral nerves do not correlate with clinical effects of differential C fiber block.[79,80] However, application of distilled water on the dorsal root ganglia for 5 minutes produced a differential C fiber block similar to that seen with in vitro hypertonic saline. The mechanism of action seems to be the intracellular shifts of water with extracellular change in osmolarity.[29]

AMMONIUM SALTS

In 1935, Judovich used pitcher plant distillate for prolonged analgesia. The active component of the distillate was determined to be ammonium sulfate, ammonium chloride, or ammonium hydroxide, depending on the acid used to neutralize the distillate and on the pH.[29,82] Limited pathologic studies suggested that ammonium salts in concentrations of greater than 10% caused acute degenerative neuropathy. This degeneration is nonselective, affecting all types of nerve fibers.[29] More recent in vitro studies with pitcher plant distillate attributed the effects to benzyl alcohol contained in the vehicle.[29,83] Associated complications such as nausea and vomiting, headache, paresthesia, and spinal cord injury have led to the clinical abandonment of ammonium salt solutions, including pitcher plant distillate.[29]

The action of ammonium salts on nerve impulses produces obliteration of C fiber potentials with only a small effect on A fibers.[84,85] Limited pathologic studies suggest that injection of ammonium salts around a peripheral nerve causes an acute degenerative neuropathy affecting all fibers.[28]

Hand[86] reported the use of subarachnoid ammonium salts in 50 patients. Transient complications were nausea and headache, whereas paresthesias or burning sensation occurred in 30% of patients at doses of 500 mg of ammonium salt and lasted 2 to 14 days.[29]

SUMMARY

The use of chemical neurolytic agents for the interruption of painful pathways is one option for the treatment of intractable chronic pain. Owing to the undesirable side effects, it is imperative that this method be used by an experienced clinician. The use of fluoroscopic or radiographic guidance is strongly encouraged for accurate placement of the needle and the injection of the solution because the lesion created is not discrete. The patients must be carefully selected and give fully informed consent.

WATER-SOLUBLE RADIOPAQUE AGENTS

The first report describing a water-soluble contrast agent for myelography was published in 1931.[87] Its lower viscosity and density and improved miscibility with CSF allowed finer detail to be detected. Methiodal was spontaneously absorbed, which eliminated the need for removal following each investigation. Sodium methiodal was highly irritating.

TABLE 2–1 Recommended Concentration and Doses of Iohexol

Procedure	Formulations[a]	Concentration (mg/ml)	Volume (ml)	Dose (g)
Lumbar myelography (via lumbar injection)	Omnipaque 180	180	10–17	1.8–3.06
	Omnipaque 240	240	7–12.5	1.7–3
Thoracic myelography (via lumbar or cervical injection)	Omnipaque 240	240	6–12.5	1.7–3
	Omnipaque 300	300	6–10	1.8–3
Cervical myelography (via lumbar injection)	Omnipaque 240	240	6–12.5	1.4–3
	Omnipaque 300	300	6–10	1.8–3
Cervical myelography (via C1-C2 injection)	Omnipaque 180	180	7–10	1.3–1.8
	Omnipaque 240	240	6–12.5	1.4–3
	Omnipaque 300	300	4–10	1.2–3
Total columnar myelography (via lumbar injection)	Omnipaque 240	240	6–12.5	1.4–3
	Omnipaque 300	300	6–10	1.8–3

[a]Iohexol is the generic name for Omnipaque.

TABLE 2–2 Pharmacologic Properties of Iohexol

Concentration (mg Iodine/ml)	Osmolality[a] (mOsm/kg water)	Osmolarity (mOsm/L)	Absolute Viscosity (cp)		Specific Gravity at 37°C
			20°C	37°C	
140	322	273	2.3	1.5	1.164
180	408	661	3.1	2.0	1.209
210	460	362	4.2	2.5	1.244
300	672	465	11.8	6.3	1.349
350	844	541	20.4	10.4	1.406

[a]By vapor-pressure osmometry.

Meglumine iothalamate was too toxic for use above the lumbar region.[88] A major improvement in contrast agent design occurred in 1972 with the introduction of the first nonionic, water-soluble contrast medium, metrizamide (Amipaque). Metrizamide also proved to be far less neurotoxic than the ionic agents and was less likely to induce arachnoiditis.[89] It was the first water-soluble contrast agent used for investigation of the entire subarachnoid space.

The adverse reactions associated with metrizamide are minor, such as headache (reported in 21–68% of patients)[90] and nausea (reported in 25–40% of patients),[91,92] but there have also been a significant number of more serious reactions, such as mental disturbances, cortical blindness, aphasia, encephalopathy, and seizures.[93–99]

Ionexol is now a commonly used water-soluble nonionic contrast material. Table 2-1 shows recommended doses for each different region. Table 2-2 shows the pharmacologic properties of the various ionexol formulation.

GADOLINIUM CONTRAST AGENTS

Gadolinium-containing agents are used for contrast-enhanced MRI. This significantly increases the sensitivity and specificity of the imaging technique. Gadolinium is a paramagnetic contrast agent that causes focal irregularities in the magnetic field, with a resultant shortening of T1. The contrast between normal and abnormal tissue is enhanced on MRI following intravenous injection of gadolinium. These agents contain no iodine and are safe for use in patients with iodine allergy. Gadolinium is not nephrotoxic and can be used in patients with renal failure.

CORTICOSTEROIDS

Corticosteroids have numerous and widespread pharmacological effects. Historically, these substances were described as glucocorticoid (carbohydrate regulating) and mineralocorticoid (electrolyte balance regulating). They are grouped according to their relative potencies in Na^+ retention, effects on carbohydrate metabolism and anti-inflammatory effects. Corticosteriods are generally used in pain medicine for their anti-inflammatory action. Relative anti-inflammatory potency and Na^+-retaining potency are shown in Table 2-3.[100]

Corticosteroids (steroids) have immunosuppressive and anti-inflammatory actions. Inflammatory responses to radiant, mechanical, chemical, infectious, and immunological stimuli are suppressed by steroids. Immunosuppressive and anti-inflammatory actions of steroids are linked, as both involve inhibition of leukocyte function.

As a result of inhibition of the production of multiple cell factors involved in generating the inflammatory

TABLE 2–3 Relative Potencies and Equivalent Doses of Representative Corticosteroids

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Please refer to the printed publication.

[a]These dose relationships apply only to oral or intravenous administration, as glucocorticoid potencies may differ greatly following intramuscular or intraarticular administration.

[b]This agent is not used for glucocorticoid effects.

Source: Schimmer BP, Parker KL: Adrenocorticotropic hormone; adrenocortical steroids and their synthetic analogs; inhibitors of the synthesis and actions of adrenocortical hormones. In Hardman JG, Limbird LE, editors: *The Pharmacological Basis of Therapeutics*, 10th ed. New York, McGraw-Hill, 2001, p. 1657.

S, short (8- to 12-hour biological half-life); I, intermediate (12- to 36-hour biological half-life); L, long (36- to 72-hour biological half-life).

responses, steroids decrease release of vasoactive and chemoattractive factors, diminish secretion of lipolytic and proteolytic enzymes, decrease extravasation of leukocytes in areas of injury and ultimately decrease fibrosis.

Corticosteroids most commonly used for local injection for pain therapy include dexamethasone, betamethasone, methylprednisone acetate, and triamcinolone acetate. Numerous factors influence which steroid is used, including availability. In recent history, fear of embolic events associated with the use of corticosteroid formulations that contain particles, such as Aristocort®, Depo Medrol®, and Celestone®, has led some physicians to avoid using these preparations.

BOTULINUM TOXINS

Botulinum neurotoxins are potent neurotoxins produced by Clostridium botulinum. There are seven serotypes: A, B, C1, C, E, F, and G. In pain therapy, types A (Botox) and B (MyoBloc) are injected into trigger points. Once injected the toxin is taken up into nerve terminals and blocks acetylcholine release, thereby producing flaccid paralysis. Each neurotoxin consists of a 2 chain polypeptide linked by a disulfide bond. The chains are designated as A (light chain) and B (heavy chain), and each has a distinct role. The B chain binds to the surface of the target cell and facilitates endocytotic internalization of the toxin. After binding, the B chain mediates translocation of the A chain into the cytoplasm. Once in the cell, the A chain interferes with neurotransmitter release.

A series of proteins, including SNAP-25, VAMP, and syntoxin, are necessary for binding of synaptic vesicles containing neurotransmitter inside of nerve endings that precede neurotransmitter release. SNAP-25 is the target for botulinum toxin type A, and VAMP is the target for type B. Following injection of toxin into muscle, effects are felt in several days, maximum effect within about 2 weeks then gradually fade over the next 2–3 months. Recovery follows sprouting of new nerve terminals at the neuromuscular junction. Maximum recommended dose of Botox is 400–600 units, and maximum recommended dose of MyoBloc is 10,000–15,000 units. Actual dose used usually depends on muscle injected. Because the targets of A and B types differ, patients refractory to one may respond to the other.

There is evidence that botulinum toxin exerts direct sensory effects by preventing release of neurotransmitters such as substance P and calcitonin gene-related peptide in sensory pathways.

TOXICITY

Overdose can produce generalized muscle weakness and even paralysis. Dry mouth may be produced, especially following botulinum toxin B injection.

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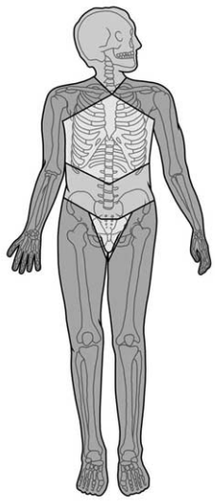
C H A P T E R



3 Thermal and Pulsed Radiofrequency

JAN VAN ZUNDERT, MENNO SLUIJTER, AND MAARTEN VAN KLEEF

HISTORY

UNTIL 1980

The use of electric current for pain management has a long history, but its popularity has waxed and waned over time because of concerns about safety and technical improvements. Already in the second half of the 19th century, brain lesions in animals were made with direct current application and empirical rules for quantifying lesion size based on current and time were developed.[1,2] One of the first uses in humans dates back to 1931, when direct current of 350 mA was delivered through a needle with a 10-mm uninsulated tip placed in the Gasserian ganglion under radiological control for the management of trigeminal neuralgia.[3] This technique produced lesions with unpredictable size, which resulted in complications.[4] Therefore, the use of high-frequency electric current was advocated to be more appropriate in obtaining lesions of predictable size.[5,6] Since high frequencies of 300–500 KHz were also used in radiotransmitters, the current was called radiofrequency (RF) current. Later, temperature monitoring was suggested to be the most important parameter in obtaining a standardized lesion size when performing stereotactic brain surgery with RF current.[7] The use of RF in pain management dates back to 1965 for percutaneous lateral cordotomy for unilateral pain in cancer patients.[6] A few years later RF treatment of trigeminal neuralgia was described.[8]

The first use of RF current for spinal pain was reported by Shealy,[9] who performed RF lesioning of the medial branch for lumbar zygapophyseal joint pain, using a 14-gauge (G) thermistor electrode introduced through a 12-G guide needle. This is a fairly large needle diameter that may produce mechanical lesions besides the desired thermolesions.[10] Another application in spinal pain was introduced by Uematsu,[11] who described the RF lesion of the dorsal root ganglion (DRG), using the same electrode as used by Shealy for medial branch block. The recommended tip temperature of 75°C, combined with the large electrode diameter, produced sizeable lesions causing deafferentation problems, and the technique was soon abandoned.[10]

1980–1995

At the end of the 1970s, percutaneous cordotomy and RF treatment of the Gasserian ganglion were the only widely accepted RF procedures. The use of RF for spinal pain was limited to a few enthusiasts who were regarded as eccentrics. A turning point came in 1980 when small-diameter electrodes, known as the Sluijter Metha Kit (SMK) system, were introduced for the treatment of spinal pain.[12] The system consists of a 22-G disposable cannula with a fine thermocouple probe inside for temperature measurement. The smaller electrode size resulted in a diminished discomfort during the procedure. Because there was now less risk for mechanical injury to major nerve trunks, targets in the anterior spinal compartment were no longer off limits and procedures such as the RF lesion adjacent to the DRG, the lesion of the communicating ramus,[13,14] and of the sympathetic chain became part of the armamentarium.

The RF lesion in the nucleus of the disc for discogenic pain dates back to 1991 and it was described in 1996.[15] This procedure was based on the idea that the low impedance inside the nucleus would cause a high power deposition. This was supposed to lead to indirect heating of the annulus fibrosus because the disc space is heat insulated cranially and caudally. The initial positive findings could not be substantiated in a randomized controlled trial, however.[16]

1996 TO PRESENT

Over the years the concept that the clinical effect of RF was caused by the formation of heat had not been challenged. Thermocoagulation of nerve fibers would interfere with the conduction of nociceptive stimuli, and pain

would be relieved when the nociceptive stimuli stopped reaching the spinal cord. A selective effect of heat on thin nerve fibers might or might not play a role,[17] but that was as far as the discussion went.

There were several reasons why the role of heat was finally questioned. First, the classical concept presupposes a strict configuration: the RF lesion must be made in between the nociceptive focus and the central nervous system. Yet RF lesions can be successfully used in very different situations. For example, in the treatment of acute radicular pain due to a herniated disc, the electrode is placed distally to the nociceptive focus.[18] Second, RF lesioning adjacent to the DRG induces only transient sensory loss in the relevant dermatome, which can be considered as heat related, while the pain relief may be of much longer duration.[19] And third, the role of heat was also questioned by the publication that no differences in outcome were noted when two different tip temperatures (40° C and 67° C) were applied to the cervical DRG in chronic cervical radicular pain.[20]

It is against this background that pulsed RF (PRF) was developed.[21] PRF aims to deliver strong electric fields, while the temperature effects are kept to a minimum. In PRF, radiofrequency current is applied in pulses instead of continuously. Two bursts of 20 milliseconds each are delivered in 1 second. Following the active phase of 20 milliseconds the silent period of 480 milliseconds allows for washout of the generated heat. The output is usually set at 45 V, which is much higher than the output used in continuous RF, which is 15–20 V.

Concerning pulsed RF, Cahana et al.[22] provide an extensive literature search. This group reviewed 58 reports on the clinical use of PRF in various applications, including 32 full publications and 26 abstracts. Because this is a new technique, a substantial part of these results are reported in the abstract collections of international scientific congresses. Reports are increasing, and every year more are published in peer-reviewed indexed journals.

RADIOFREQUENCY LESION GENERATOR SYSTEM

A modern RF lesion generator (Figure 3-1) has the following functions:

- Continuous on-line impedance measurement
- A nerve stimulator
- Monitoring of voltage, current, and wattage during the RF procedure
- Temperature monitoring
- Pulsed current delivery mode

These features are important for reasons described below.

Electrical impedance is measured to confirm the continuity of the electrical circuit and to detect any short circuits. The impedance signal can be converted to a varying audible pitch by the generator, which allows the various tissue interfaces to be "heard" while the operator concentrates on the procedure. The impedance will vary from about 300 Ω to 600 Ω in the extradural tissue. Furthermore, impedance monitoring is of special interest in cordotomies and in RF-disc lesions. In cordotomies the impedance increases above the level of 1000 Ω on entering the spinal cord,[23] thus indicating that the electrode is properly positioned. In RF-disc lesions, the impedance falls sharply, to less than 200 Ω, as the electrode enters into the disc.[15]

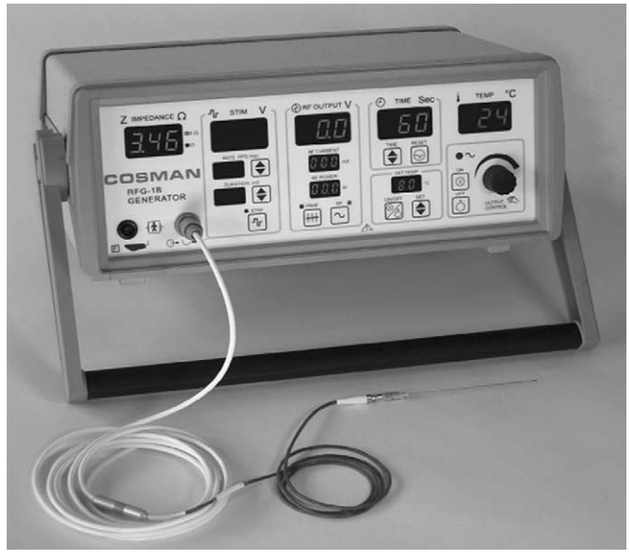


FIGURE 3-1
Cosman RFG-18 radiofrequency generator.

Nerve stimulator. Nerve stimulation is of great importance in RF procedures. After placement of the needle under fluoroscopic control, nerve stimulation is carried out to confirm the proper position of the electrode and to permit minor adjustments. Stimulation is carried out at 50 Hz to ensure the proximity of the electrode to the sensory fibers. Two hertz stimulations are performed to detect muscle contractions, which indicate that needle placement is too close to motor fibers.

Ford et al.[24] have shown that if an electrode is actually resting on the nerve, a minimum stimulation level required to produce a discharge is 0.25 V. At a distance of 1 cm from the nerve, 2 V would be required. In this manner, the stimulation threshold is an indicator for the electrode–nerve distance. But in our experience, the quality of the nerve also plays an important role. When the nerve is neuropathic, it is not uncommon to find stimulation thresholds greater than 1 V while there is clearly mechanical contact.

Temperature monitoring. Temperature measurement is performed by the thermocouple technique, which has the advantage that temperature can be measured in very small diameter electrodes. The thermocouple consists of a junction of two dissimilar metal elements, producing a thermodionic voltage, which is proportional to temperature (Figure 3-2). The thermocouple is placed at the tip of the electrode, which is in the hottest part of the lesion.

FIGURE 3-2
SluijterMathaKit thermocouple electrode.

THEORETICAL ASPECTS OF RADIOFREQUENCY LESIONING

CONTINUOUS RADIO FREQUENCY

The voltage of the generator is set up between the (active) electrode and the (dispersive) ground plate (Figure3-3). The body tissues complete the circuit and RF-current flows through the tissue, resulting in an electric field. This electric field creates an electric force on the ions in the tissue electrolytes, causing them to move back and forth at a high rate. Frictional dissipation of the ionic current within the fluid medium causes tissue heating. RF heat is therefore generated in the tissue, and the electrode is heated by the tissue.

The size of the lesion depends on the tip temperature (Figure 3-4), and the tip temperature depends on the power deposition. But there are other factors involved as well. Heat is also removed from the lesion area by conductive heat loss and blood circulation. This is referred to as heat "washout." The greater the heat washout, the smaller the lesion will be for a given tip temperature. Considerable variations of tissue factors influence heat washout. For example, bone is an effective heat insulator with little water content. For this reason, RF lesions close to bone will not have the same degree of heat washout as they might have in more conductive tissue. Similarly, segmental blood vessels, in relation to the dorsal root ganglion, may cause considerable heat washout, thereby reducing the size of the lesion.[25]

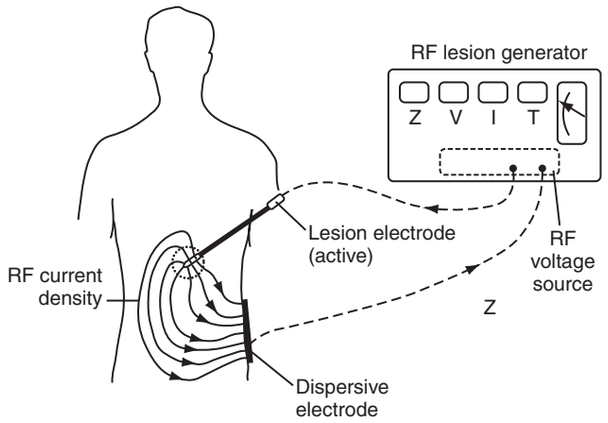


FIGURE 3-3
This figure shows fields of current from the generator through the body between active and dispersive sites. Note the pattern in the body.

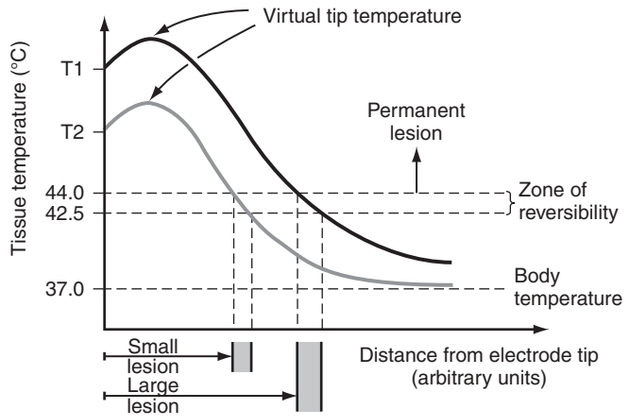


FIGURE 3-4
Tissue injury after radiofrequency lesioning. Note the shape of the lesion and the area of reversibility. (From Cosman ER, Nashold BS, Ovelman-Levitt J: Theoretical aspects of radiofrequency lesions in the dorsal root entry zone. *Neurosurgery* 15:945-950, 1984, with permission.)

The size of the lesion also depends on other parameters, such as the diameter of the electrode and the length of the uninsulated electrode tip. Cosman and Cosman[26] were the first to determine lesion size for a given electrode tip size and temperature. They made dorsal root entry zone lesions in cats and studied the relationship between temperature, size, and duration of lesioning. They concluded that at a tip temperature of 75°C the lesion size would only increase by about 20% beyond a lesion time of 30 seconds. The lesion size did not increase further after 60 seconds.

Moringlane et al.[27] studied experimental RF-coagulation with computer-based online monitoring of temperature and power. They concluded that the maximum "volume" of a lesion is effectively obtained after 40 seconds, and that the lesion size strongly depends on tip temperature and on probe diameter. Bogduk et al.[28] also studied the shape and size of lesions made by RF electrodes. Experimental lesions were made in egg white and in fresh meat. They concluded that RF lesions do not extend distally to the tip of the electrode, and that they extend radially around the electrode tip in the shape of an oblate spheroid.

When an RF lesion is made with continuous RF, the output of the lesion generator is adjusted to the tip temperature. Parameters such as impedance and voltage are not taken into account. Many brands of generators have the option of automatic temperature control, where the adjustment of the output to the desired tip temperature is automatic.

PULSED RADIO FREQUENCY

Pulsed RF (Figure 3-5) is based on the dual effect of exposure of the tissue to RF fields. Besides the ionic friction that causes the production of heat, there is an independent, nonthermal effect that has the potential of producing

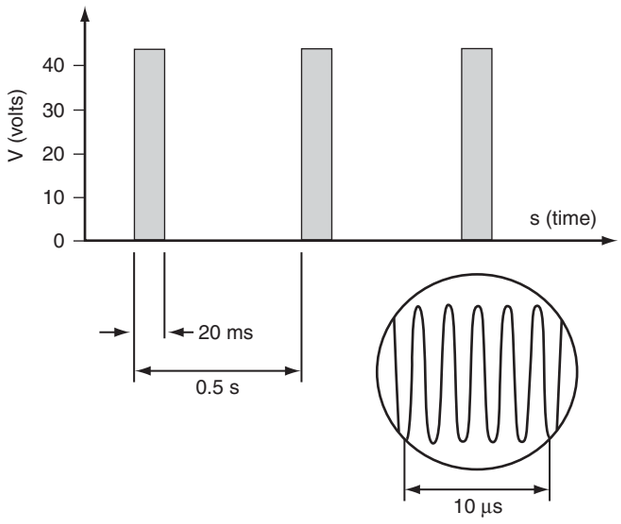


FIGURE 3-5

Schematic drawing of the duty cycle during pulsed radiofrequency. There are two active cycles per second of 20 milliseconds each. During the active phase, radiofrequency is delivered at the normal frequency of 500,000 Hz. (Adapted from Sluijter ME: *Radiofrequency, Part I.* Meggen, Switzerland, Flivopress, 2001, with permission.)

modification of neural structures and neuronal behavior. Thermal and nonthermal effects must therefore be discussed separately.

Thermal Effects of PRF

Temperature spikes. During the 20-millisecond active phase of PRF, heat is produced and there is a very brief rise in temperature around the needle tip. These brief elevations have been named "temperature spikes." The height of these spikes can be calculated, and they have indeed been measured with a very fast thermocouple[25] (Figure 3-6). Because it is a fast phenomenon, and because the thermocouples that are used for RF treatment are slow, the spikes do not show up on the display of a lesion generator.

The height of a heat spike is entirely dependent on the power deposition during the active phase. Because PRF is delivered with a fixed voltage, the power deposition is strongly dependent on the current, and therefore on the impedance. Calculated values vary from 4.30°C for 800 ohm to 13.80°C for 250 ohm, at the start of the procedure. During the later phases of the procedure, the spikes are superimposed on the mean tip temperature.[25]

It is presently not known if these brief elevations of temperature have a biological effect. A mild ablative effect of PRF has been described,[29,30] but these changes may equally have been caused by the nonthermal effect of strong electric fields.

The heat mostly spreads ahead of the tip of the electrode, because this is where the strongest electric field is. But the penetration into the tissue is minimal; the rise in temperature beyond a distance of 0.2 mm from the electrode is irrelevant (Figure 3-7).

Mean tip temperature. Once the heat has been generated near the electrode tip, it spreads into the tissue like the ripples in water after a stone is tossed in. The farther away from the electrode, the slower and less pronounced the rise in temperature (Figure 3-8). After a number of pulses, the mean tip temperature will rise, and this can be read on the display of the lesion generator.

As in continuous RF, the mean tip temperature depends on the power deposition on the one hand and on the

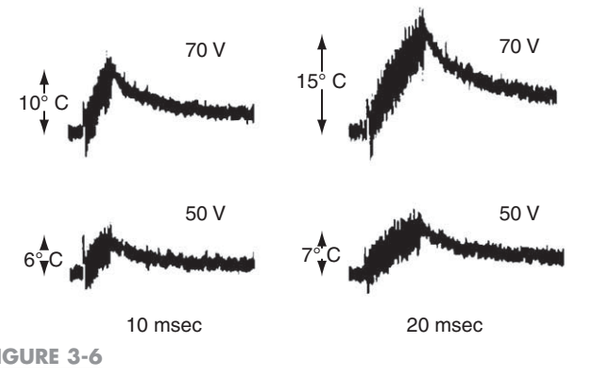


FIGURE 3-6

Measured temperature bursts during pulsed radiofrequency pulses in liver at 70 V and 50 V (peak) settings: on the left for duration of 10 milliseconds and on the right for duration of 20 milliseconds. (Adapted from Cosman ER Jr, Cosman ER Sr: Electrical and thermal field effects in tissue around radiofrequency electrodes. *Pain Med* 6(6):405–424, 2005, with permission.)

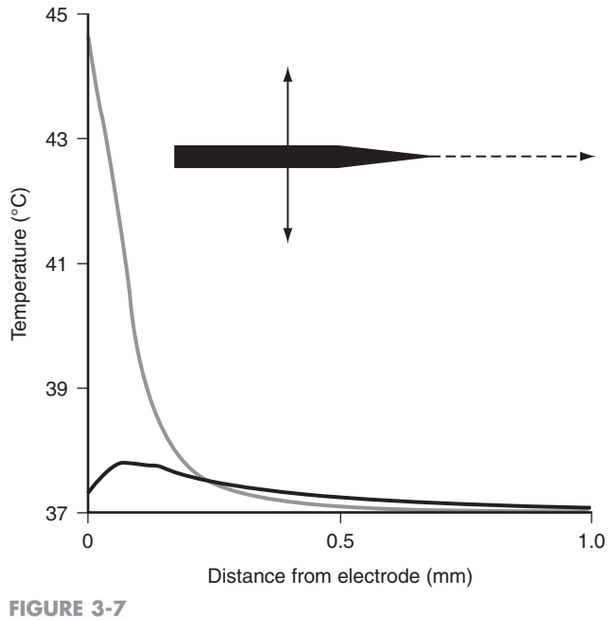


FIGURE 3-7

The temperature field magnitudes as a function of distance from the radiofrequency electrode (SMK) along two different directions for pulsed radiofrequency with V (peak) of 45 V and duration of 20 milliseconds. (Adapted from Cosman ER Jr, Cosman ER Sr: Electrical and thermal field effects in tissue around radiofrequency electrodes. *Pain Med* 6(6):405–424, 2005, with permission.)

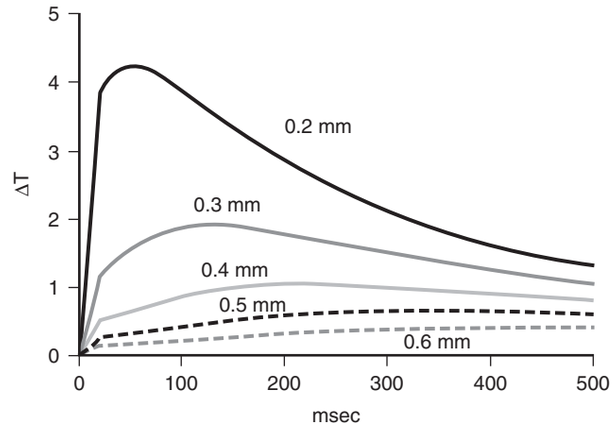


FIGURE 3-8

Calculated temperature curves during the first pulse of a pulsed radiofrequency procedure at various distances from the electrode tip. Impedance = 250 ohm.

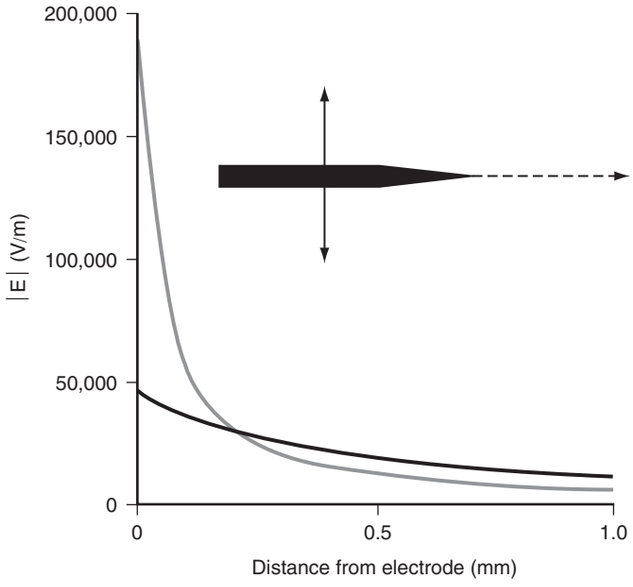


FIGURE 3-9

The electric field magnitudes as a function of distance from the radiofrequency electrode (SMK) along two different directions for pulsed radiofrequency with V (peak) of 45 V and duration of 20 milliseconds. (Adapted from Cosman ER Jr, Cosman ER Sr: Electrical and thermal field effects in tissue around radiofrequency electrodes. *Pain Med* 6(6):405–424, 2005, with permission.)

heat washout on the other, and the same considerations are valid for the factors determining heat washout.

Nonthermal Effects of Pulsed Radio Frequency

There is extensive knowledge on the effect of constant electric fields on cells because cell biologists use such fields to modify cells. The effects may vary from an effect on the functioning of voltage- or transmitter-gated ion channels or ion pumps in the membrane that control the conduction of Na+, K+, Ca++, to a reversible disruption of the cell membrane known as electroporation,[31] to cell death. Recovery following the exposure is in the milliseconds range, but some recovery components last several minutes.[32] This is relevant for PRF because it may explain why continuous RF at a very low voltage has a more pronounced effect on cell functioning than PRF at 45 V.[29] Obviously the silent period of the PRF duty cycle is not only important for the removal of heat, but for functional recovery following the active cycle as well.

Much less is known on the effects of alternating electric fields such as in PRF. It is therefore uncertain at which level the strength of the RF field becomes significant. A value of 5000 V/m seems to be a reasonable assumption. The distribution of the electric fields can again be calculated[25] (Figure 3-9). The pattern is comparable to the distribution of temperature, but there is a significant difference. The electric field at a reasonable distance from the electrode is still greater than 5000 V/m, whereas the rise in temperature during a temperature spike is insignificant at a distance of more than 0.2 mm.

PRACTICAL CONSIDERATIONS

When making a heat lesion with continuous RF adjacent to the DRG, it is customary to observe a minimum value of the stimulation threshold of 0.4 V, in order to avoid denervation sequelae. This rule does not apply to PRF because despite the microscopic evidence for destruction, no alterations in nerve function have been reported in a clinical setting. Yet it may be wise to avoid the ultra-low thresholds (<0.05 V) because such values may reflect intraneural electrode placement. A very small area of necrosis around the needle tip does occur,[25] which is not desirable in this location.

In a small proportion of procedures the mean tip temperature exceeds 43°C at the end of the procedure. In this case, as a precaution, the power deposition should be decreased. This can be done by lowering the voltage, or by decreasing either the duration of the active cycle or the cycle frequency.

It is undesirable to adjust the voltage during a PRF procedure to the mean tip temperature. The mean tip temperature does not affect the outcome of the procedure,[10] and because there is a large variation in heat washout, such a practice will cause large and unpredictable variations in voltage.

MODE OF ACTION OF PULSED RADIO FREQUENCY

Pulsed RF was initiated as a method to explore the mode of action of RF, not as a discovery de novo. It is therefore not surprising that the mode of action is not yet clear. Much has been learned about the physical events around the electrode,[25] but it is not known yet how these events

cause the clinical effect. There are presently two theories. First, there may be a mild but significant ablative effect, mainly affecting thin nerve fibers. This would not be in contradiction with the absence of sensory changes following PRF because, in fact, the situation is the same after application of continuous RF. Following a heat lesion, manifest sensory changes only occur during the period of postprocedural discomfort, but they are absent once the period of pain relief has set in.

Second, there might be an effect on the dorsal horn, where trans-synaptal induction of gene expression has been found, both short[33] and long[34] term. If this is the case, it is not yet clear how these changes are caused because the frequency of RF is far above the physiological range.

INDICATIONS AND CONTRAINDICATIONS FOR (PULSED) RADIOFREQUENCY

The available information on RF treatment indicates that it may be a useful tool, but the evidence varies from one indication to another. The technique has a low invasive character and a target-selective approach, and can be performed as outpatient treatment. The following conditions should be fulfilled: patients are carefully selected with attention to both somatic and psychosocial factors, and the technique is performed by a trained clinician in the optimal environment.

The most frequently described indication for RF treatment is trigeminal neuralgia, an indication for which there is extensive experience.[35] A review of 25 years of experience with 1600 patients receiving percutaneous RF trigeminal rhizotomy for idiopathic neuralgia indicates acute pain relief in 97.6% of the patients and continued complete pain relief at the 5-year follow-up in 57.7%.[36] Complications in this series were diminished corneal reflex, masseter weakness and paralysis, dysesthesia, anesthesia dolorosa, keratitis, and transient paralysis of cranial nerves II and VI. Comparisons with other techniques are mainly based on retrospective evaluations.[37–45] PRF treatment for this indication has been reported.[46] Recently, a RCT comparing PRF with RF showed longer pain relief with RF.[47]

Cluster headache is a neurovascular form of headache. Attacks of cluster headache can be relieved by anesthetizing the sphenopalatine ganglion.[48] Promising results have been reported of RF treatment[49] as well as of PRF treatment of the ganglion sphenopalatinum.[50]

Chronic cervical pain can arise from several structures in the cervical region, including zygapophyseal joints, discs, nerve roots, ligaments, and myofascial structures.[51] The prevalence of cervical pain is judged to be as frequent as low back pain. The cervical pain syndromes, which are accessible for invasive RF treatment, are cervical pain, cervicobrachialgia, and cervicogenic headache.[52] Each pain syndrome may have more than one nociceptive source. As a consequence, more than one RF treatment modality may be needed in relieving patients' pain. Cervicobrachialgia is described as pain originating from the cervical spine radiating from the neck beyond the gleno-humeral joint into the upper limb with referral to a particular spinal segment.[12] Cervicogenic headache could originate from structures in the neck. The cardinal feature delineating cervicogenic headache from the other headache syndromes is the concept that the pain originates from a structural abnormality in the cervical spine.[53] Various structures in the cervical spine, such as the facet joints, segmental nerves, intervertebral discs, muscles, and ligaments, are capable of causing neck pain and headache.

The management of cervicobrachialgia with RF treatment adjacent to the cervical DRG was described in one open and two randomized controlled trials (RCTs).[19,20,54] One of the RCTs compared RF treatment at 67°C with RF treatment at 40°C.[20] The clinical efficacy varies from one trial to another, and in a recent review Geurts et al.[55] concluded that there is limited evidence for RF facet denervation in chronic cervical pain after whiplash, and there is limited evidence that RF dorsal root ganglion (DRG) is more effective than placebo in chronic cervicobrachialgia. The first RCT on pulsed radiofrequency adjacent to the cervical DRG in patients with chronic cervical radicular pain was recently published. At 3 months, the PRF group showed a significantly better outcome with regard to the global perceived effect (>50% improvement) and visual analogue scale (20-point pain reduction). The need for pain medication was significantly reduced in the pulsed radiofrequency group after 6 months. No complications were observed during the study period.[56]

The value of RF of the medial branch for chronic cervical zygapophyseal joint pain has been demonstrated in one RCT with excellent results for RF compared to sham.[57]

Radiofrequency of the medial branch for cervicogenic headache suggested initially some chance of benefit,[58,59] but due to the absence of a consensus about the diagnostic classification of cervicogenic headache and the uncontrolled study setup, those results are not compelling. A prospective study showed a significant improvement in patients selected on the basis of the diagnostic criteria described by Sjaastad et al.[53,60] However, in a recently published RCT no evidence was found indicating that RF treatment of cervical zygapophyseal joints and upper dorsal root ganglions is better than the infiltration of the greater occipital nerve followed by TENS for patients with cervicogenic headache.[61]

Pain syndromes originating from the thoracic spine occur in 5–7% of the patients seen in a pain clinic. In this type of patient, diagnostic evaluations should exclude underlying pathology such as herniations, aneurysms, tumors, old fractures, or infections. One should distinguish thoracic pain, which can be described as pain originating from the zygapophyseal joints, and/or thoracic disc and thoracic segmental pain with referral into one or more particular spinal segments due to involvement of the segmental nerve in the pain syndrome, related to vertebral collapse, 12th rib syndrome, and segmental peripheral neuralgia.[62,63]

Documentation on the use of RF treatment in the thoracic region is relatively scarce and restricted to open studies.[64] After thoracic facet denervation, more than 80% of the patients experienced good pain reduction for at least 2 months.[63,65] RF treatment adjacent to the thoracic DRG is a difficult technique due to the need of drilling through bone.[66] Potential complications are segmental nerve injury, spinal cord injury, pneumothorax, and thoracic neuritis. Two open studies using RF DRG at the thoracic level report good short- and long-term results.[67,68]

Radiofrequency treatment of low back pain is most frequently used and most often described.[69] This phenomenon is partly due to the fact that incapacitating chronic low back pain develops in more than 14% of the patients. The vast majority of those patients suffer nonspecific low back pain that may be of discogenic origin, from the facet (or zygapophyseal) joints, or from the sacroiliac joint. Low back pain is frequently divided into two components: neurogenic radicular pain and mechanical low back pain.[70] RF lesioning of the DRG is developed as an alternative to the surgical rhizotomy; use is based on the principle that nociceptive input at the level of the primary sensory neuron might be reduced by coagulation of a small part of the DRG without causing sensory deficit.[71] One prospective and four retrospective studies have reported beneficial effects of lumbosacral RF DRG.[71–75] One randomized controlled study failed to show advantage over sham treatment with local anesthetic.[76]

Pulsed radiofrequency treatment adjacent to the DRG in the lumbar region may be indicated for chronic radicular pain.[77,78] PRF treatment has also been advocated for acute pain due to a herniated disc,[18] with good results and a remarkably low tendency for recurrence of pain. This may be a useful replacement for periradicular steroid infiltration because this widely adopted treatment does not reduce the need for surgery[79] and is not without serious risk.[80]

Lumbar percutaneous facet denervation (PFD) by means of RF treatment is based on the premise that neurolyzing the medial branches of the distal portions of the spinal posterior rami nerves that supply painful lumbar facet joints will result in alleviation of back pain and a return of function. Technically, there are two prerequisites for success of RF PFD: the identification of the painful joint using diagnostic blocks and the precise localization of the nerve supply to the targeted joints.[69] The clinical outcome of this technique has been evaluated in four randomized clinical trials.[81–84] Results of the RCTs are somewhat contradictory, although comparing them is not possible because of differences in patient selection criteria, use of diagnostic blocks, and efficacy parameters followed by each study. Currently, two different techniques are commonly used in clinical practice: temperature- and voltage-controlled lesioning. In a combined in vivo and in vitro study, the electrophysiological consequences and the effect on lesion size were determined. Temperature-controlled radiofrequency lesioning is preferred to create reproducible lesion size.[85]

It seems that RF neurotomy is an effective but temporary management of lumbar facet pain. When pain recurs, RF neurotomy is usually repeated. The outcome and duration of relief for repeat interventions was investigated by means of a retrospective chart review. It was concluded that the frequency of success and duration of relief remained consistent after each subsequent procedure.[86]

Considering the safety of the technique, the incidence of complications associated with fluoroscopically guided percutaneous RF denervation of lumbar facet joints was retrospectively assessed in 92 patients receiving 616 lesions during 116 procedures. The technique was associated with an overall 1% incidence of minor complications per lesion site.[87]

Discogenic back pain can be treated by heating the annulus fibrosus or the nuclear/annular interface through a catheter.[88,89] Effectiveness has been reported in one RCT,[90] but this could not be confirmed in another one.[91] The method is not free of serious complications.[92,93] Patients who do not respond to IDET-treatment may benefit from RF treatment of the communicating ramus.[14] A new method using an intensive duty cycle of PRF through a centrally placed electrode had good initial results.[94]

Radiofrequency has been used for interruption of the sympathetic chain to treat intractable pain in the sacral-pelvic region[95] or for the management of visceral pain[96] and/or complex regional pain syndrome (CRPS).[97] The application of RF current in this indication differs from its use for other targets such as sensory nerve tissue because no sensory threshold can be achieved in the sympathetic nerves. The use of RF treatment has the advantage over surgical resection and phenol or alcohol neurolysis, in that it is more selective and may cause fewer complications.[98,99] RF treatment of CRPS was compared with phenol neurolysis. The efficacy of RF treatment seems to be comparable to phenol neurolysis, but the incidence of complications was lower.[97] One should be aware of the potential injury of the genitofemoral nerve, especially if multiple RF lesions are performed, but no controlled trials are available. Visceral pain due to chronic pancreatitis, pancreatic cancer, liver cancer, or postabdominal surgery pain that is not or no longer responding to pharmacological treatment can be managed by RF lesioning of the splanchnic nerves. From the available experience, retrospectively analyzed, we can deduct that this technique is more selective and causes fewer complications.[96]

Neuroablative procedures have been used frequently in the past for the management of intractable cancer pain. At present the percutaneous cervical cordotomy represents the most important neuroablative technique in cancer pain treatment.[100–105] Success rates have been reported to be high (54/62 patients).[106] Considering the potential for major permanent complications—urinary retention hemiparesis and mirror image pain—percutaneous cordotomy should only be used for unilateral pain.[106] Recent pharmacological developments in sustained release of opioids have resulted in increasing degrees of pain relief, thus restricting the number of patients for an intervention.

CLINICAL DECISION MAKING

The available documentation on the RF treatment in various pain syndromes indicates that this option will only be considered when conservative causative and symptomatic treatment has been used to its full extent and fails to provide satisfactory pain relief. For the well-documented indications, authors mention patient selection criteria, consisting of clinical signs, medical imaging, and identification of the causative nerve structure—if possible by means of diagnostic blocks—and psychological assessment. The optimal environment for applying RF treatment is a multidisciplinary setting facilitating diagnosis, treatment, and guidance in terms of expectations and coping with the rest pain.

The application of RF in the management of chronic pain may be a useful tool because of its low invasive character, the target-selective approach, the possibility of outpatient treatment, and safety if done by a well-trained pain physician in the right setting. In line with the World Health Organization treatment ladder for the management of chronic cancer pain, we propose an integrated treatment algorithm, including pharmacological, interventional, and multidisciplinary management as illustrated in Figure 3-10.[107]

MINIMAL STANDARDS AND RECOMMENDATIONS

Among the invasive pain management options, RF treatment is probably the most described. RF treatment as part of a multimodal and multidisciplinary approach may avoid the use of more invasive and often more expensive treatment options. We recommend the use of RF techniques under the following conditions:

- Multidisciplinary patient selection, using validated selection criteria.
- Informed consent is (redundant)
- Where indicated, use of diagnostic blocks.
- Use of fluoroscopy.
- Standardized report on the intervention including: impedance, volt, temperature, time, and radiographic photos.
- Standardized patient follow-up with validated outcome evaluation tools.
- Physicians should receive accurate training on the anatomy, technical aspects, hands-on experience, and radiation protection.

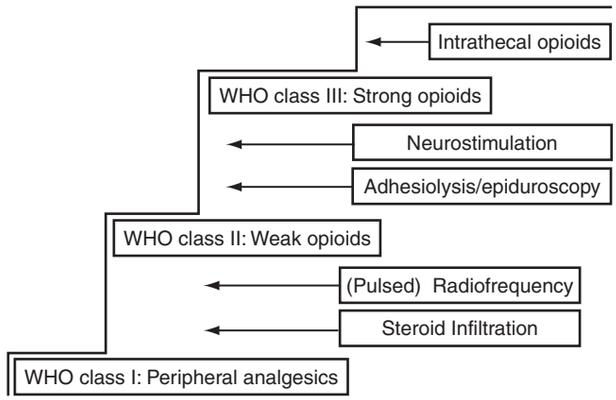


FIGURE 3-10

Schematic representation of the stepwise approach of chronic pain.

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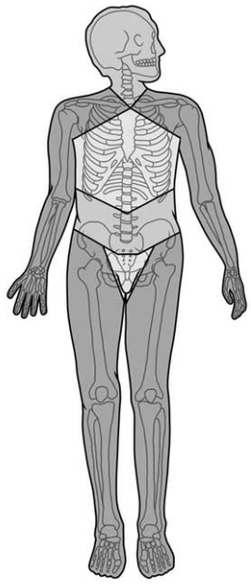
C H A P T E R



4 Principles and Mechanisms of Cryoneurolysis

LELAND LOU

Cryoneurolysis is a technique in which the application of low temperatures produced by cryosurgical equipment achieves anesthesia or analgesia by blocking peripheral nerves or destroying nerve endings.

HISTORY

The analgesic effect of low temperatures has been recorded since Hippocrates (460-377 BC).[1–4] Avicenna of Persia (980–1070) and Severino of Naples (1580–1656) recorded their use of cold for preoperative analgesia.[5,6] James Arnott (1797–1883) advocated local cooling during surgery and in the treatment of headache and cancer pains.[7] In 1777, John Hunter studied the reversible destructive effects of freezing on animal tissues. More practical and portable methods of cooling used ether spray and ethyl chloride to reduce temperatures locally as low as –12°C.[5,6] In 1917, Trendelenburg[7] demonstrated that freezing caused severe but reversible damage to nerves without scar or neuroma formation.

Interest in cryotherapy was revived in 1939, when Smith and Fay[8] reported evidence of tumor regression after localized freezing.[9] Cooper developed the first cryoprobe in 1961.[10] He was able to produce a temperature of –196°C by using liquid nitrogen. Amoils[11] introduced the enclosed gas expansion cryoprobe in which carbon dioxide was used. Since then, nitrous oxide has also been used as the refrigerant. Lloyd and colleagues[12] introduced the technique termed *cryoanalgesia*, with which prolonged analgesia could be obtained after a single freezing of a peripheral nerve. They reported that this was a safe procedure, nerve function always returned, and neuroma formation did not occur. The present generation of thin, long probes incorporates thermocouples and stimulators.

PHYSICS OF CRYOANALGESIA

Expansion of gas enclosed in the cryoprobe results in Joule-Thompson or Kelvin effects; that is, gas under pressure escaping through a small orifice expands and cools (Fig. 4-1). The probes are made of stainless steel insulated with a coating of polytetrafluoroethylene (Teflon) and are of coaxial design. A thin outer tube carries the gas under pressures between 4000 and 6000 kPa to the tip, where it passes through a narrow orifice, leading to a pressure drop to 50 to 75 kPa. The gas subsequently expands and cools, achieving temperatures between –50° and –70°C at the tip, and returns through an inner tube. The inner tube acts as an exhaust conduit to vent the gas. Modern probes use either nitrous oxide or carbon dioxide. The shaft diameter has now been reduced to 1.3 mm and the length increased to 120 mm. The tip can be trocar shaped or hemispherical. Thermocouples and stimulators with variable voltages and frequencies are built into the exposed tip surface, and by using a console with a variable flow control it is possible to achieve a wide range of subzero temperatures (Fig. 4-2).

The ice ball encompasses the end of the probe and is about 3.5 mm in diameter for a 1.3-mm tipped probe. The variables involved in ice ball size include probe size, freeze time, tip temperature, tissue thermal conductivity, tissue permeability to water, and presence or absence of vascular structure (i.e., a heat sink). When thermal equilibrium between the probe and tissues is achieved, there is no further increase in the size of the ice ball; however, repetition of the freeze-thaw cycle increases the size of the cryolesion.[13]

When the probe is used percutaneously, it is difficult to ensure close proximity to the nerve, and large ice balls have a greater chance of producing the desired lesion. For myelinated fibers, a direct lesion 3 mm in diameter with a freeze time of 1 minute produces a conduction block.[14]

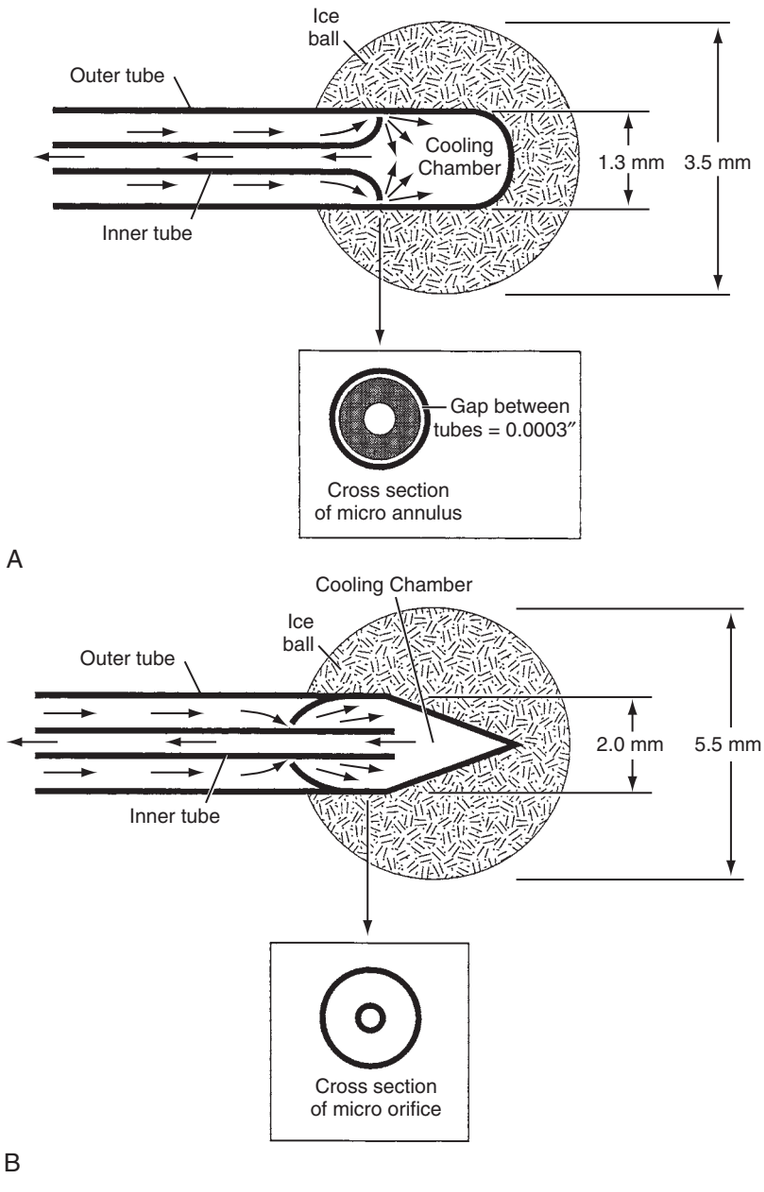


FIGURE 4-1

(A and B) Two typical cryoprobe designs. High-pressure gas flows through the outer tube and expands after passing through the orifice. The gas is vented through the center tube.

Where the nerve is frozen amid other tissues, the duration of exposure should be approximately 90 to 120 seconds. Rapid defrosting aids removal of the probe from tissues.

PATHOLOGY OF THE LESION

Freezing involves removal of pure water from solution and its isolation into biologically inert ice crystals. The extent of the lesion depends primarily on the rates of freezing and thawing.[15] When cooling is slow, ice crystal nucleation occurs in the extracellular fluid. When freezing is rapid, crystal nuclei develop uniformly throughout the tissue. The central zone close to the probe tip cools rapidly compared with the peripheral zone, which is influenced by heat generated by the surrounding tissues. Intracellular ice is formed at the center of the lesion, and extracellular crystals are formed at the periphery.[16,17] Tissue destruction is more complete at the center of a cryolesion. It is also likely that the areas at the edge of the cryolesion undergo ischemic necrosis.

Application of cold to peripheral nerves induces a reversible block of conduction similar to that produced by local anesthesia. The extent and duration of the effect depend on the temperature attained in the tissue and the duration of exposure. Large myelinated fibers are initially affected with relative sparing of smaller sensory nerves.

A prolonged conduction block occurs when the nerve is frozen at temperatures between –5 and 20°C.[18,19] This

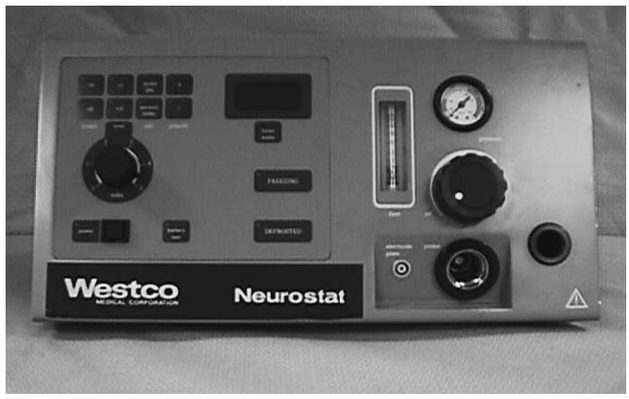


FIGURE 4-2
Typical cryodenervation incorporating a variable flow control, a thermocouple, and a nerve stimulator.

causes axonal disintegration and breakdown of myelin sheaths. Wallerian degeneration occurs with the perineurium and epineurium remaining intact. The absence of external damage to the nerve and the minimal inflammatory reaction following freezing ensure that regeneration is accurate and complete. Recovery depends on the rate of axonal regeneration and the distance of the cryolesion from the end organ. All elements of the nerve are involved. The rate of axonal regrowth is 1 to 3 mm per day. Histologic sectioning of nerve suggests that regeneration is still occurring in functionally intact nerves.

TECHNIQUE

The cryolesion is attempted only after successful temporary reduction of symptoms by a diagnostic block. After a small skin wheal is raised with local anesthetic, a 1.3- or 2-mm probe is passed via a 16- or 12-gauge catheter, respectively, depending on the nerve size (Fig. 4-3). Larger probes counteract arterial warmth where heat sinks are expected. Localization is facilitated with stimulation between 50 and 100 Hz at less than 0.5 V for sensory nerves or at 2 to 5 Hz for motor nerves. Two or three 2-minute cycles are usually sufficient. During the freezing, care is taken to prevent frostbite if the probe comes in direct contact with the skin. Continuous irrigation with 0.9% saline solution at room temperature reduces the possibility of skin injury.

FIGURE 4-3
Lloyd probe.

COMMON PROCEDURES

HEAD AND NECK

Supraorbital nerve. Irritation of the nerve occurs primarily at the supraorbital notch. Supraorbital neuralgia may be secondary to blunt trauma, entrapment neuropathy, acute herpetic infection, Paget's disease, or neoplasm.

Cryoneurolysis can be accomplished via an open operative technique or percutaneously. The importance of cosmesis should be considered in avoiding thermal damage to the sensitive skin around the eye. Entry of the catheter and probe should be below or above the eyebrow line to avoid damage to the brow follicles. Potential risks include nerve trauma after insertion of the probe, hematoma, infection, and skin necrosis.

Infraorbital nerve. The infraorbital nerve is a terminal branch of the second division of the trigeminal nerve as it exits through the infraorbital foramen. It is in the same vertical plane as the pupil when the eye is in a forward gaze. It is a sensory nerve to the lower eyelid, cheek, lateral aspect of the nose, upper lip, and part of the temple.

Infraorbital neuralgia is typically characterized by maxillary pain worsened by smiling or laughing. Patients sometimes experience referred pain in the teeth.

Cryoneurolysis can be accomplished via an open operative technique or percutaneously. It can also be accomplished by an intraoral approach to minimize cosmetic damage. The same introducer and probe are inserted through the superior buccal-labial fold. The probe is then advanced until it lies over the infraorbital foramen.

Mandibular nerve. After emerging from the foramen ovale, the mandibular nerve runs through the infratemporal fossa posterior to the posterior border of the pterygoid plate. It provides motor supply to lateral pterygoid, masseter, and temporalis muscles and sensory supply to the skin and buccal mucosa of the cheek and gingiva. The auriculotemporal and lingual nerves constitute a posterior division.

Neuropathy of the mandibular nerve may result from muscular hypertrophy of the pterygoids caused by chronic bruxism and loss of vertical dimension of the oral cavity with loss of posterior dentition.

An insulated needle is introduced through the mandibular notch and advanced through the infratemporal fossa until it encounters the lateral pterygoid plate. It is then walked back off the lateral pterygoid plate, maintaining the same depth, until paresthesia or stimulation with a nerve stimulator is achieved. The depth of the needle is noted, and then it is removed. The cryoprobe is advanced to the same depth using stimulation to localize the nerve.

Mental nerve. The mental nerve emerges from the mental foramen. The foramen becomes progressively cephalad with advancing age.

Irritative peripheral neuropathy occurs principally at the mental foramen. The pain of mental neuralgia is typically manifested in the chin, lower lip, and gum line. The nerve may also become entrapped in surgical scars.

Closed extraoral or intraoral cryoneurolysis can be performed. Intraorally, the probe is advanced through the gingivobuccal reflection at the level of the premolar tooth and makes contact with the mandible. Care must be taken not to enter the foramen because of the risk of nerve injury.

Greater occipital nerve. The greater occipital nerve is a branch of the cervical plexus located halfway between the mastoid process and the greater occipital protuberance at the crest of the occipital bone and lies adjacent to the occipital artery.

Cryoneurolysis is performed for relief of occipital neuralgia and relief of occipital muscle tension headaches. The procedure is often performed bilaterally.

Spinal accessory nerve. The spinal part of the 11th cranial nerve emerges from the posterior border of the sternocleidomastoid muscle at the junction of the lower and middle thirds to cross the neck and supply the trapezius muscle.

Cryoneurolysis is used for severe tonic or clonic spasms of the trapezius muscle, spasmodic torticollis, and certain whiplash injuries.

The nerve is identified by motor stimulation and can be frozen either at its exit from the sternocleidomastoid or close to its entry into the trapezius.

SPINE

Although it is possible to cryodenervate cervical, thoracic, and lumbar facet joints, denervation is best performed with radiofrequency probes because of the smaller probe size and better maneuverability. Radiofrequency is also preferred for sacral nerve root pain. Coccygodynia is amenable to cryoanalgesia at the sacral hiatus.

ABDOMEN/PELVIS

Iliohypogastric nerve. The iliohypogastric nerve arises from the Tl1 and Tl2 nerve roots and passes anteriorly to the rectus sheath. Neuropathy results in an upper quadrant pain, which may mimic that of cholecystitis or pancreatitis or may be caused by the surgical treatment of upper abdominal pathology.

Ilioinguinal nerve. The ilioinguinal nerve arises from the T12 and L1 nerve roots. It is often injured at the lateral rectus sheath, approximately 5 cm from the midline, 10 cm inferior to the umbilicus. At this point, the nerve perforates the superior crus of the superficial inguinal ring. The nerve may be injured during inguinal herniorrhaphy; by compression resulting from bladder retraction during abdominal surgery; or, rarely, by tight-fitting garments.

Genitofemoral nerve. The genitofemoral nerve arises from the L1 and L2 nerve roots. The genital branch of the nerve passes under the inguinal ligament and over the symphysis pubis immediately lateral to the pubic tubercle. This sensory nerve then travels to the labia or scrotum. It can be injured as the result of surgical trauma during abdominal surgery and inguinal herniorrhaphy.

The clinical presentation of genitofemoral neuralgia and ilioinguinal pathologic conditions consists of dull, aching pain in the lower quadrants of the abdomen. Pain worsens with Valsalva's maneuver, cough, bowel movement, and lifting. Patients often experience increased pain intensity and frequency with menstruation and sexual intercourse. Irritation of either nerve can result in referred pain to the testicle or vulva, interior thigh, or upper lumbar region.

The abdominal wall nerves can be localized percutaneously or with laparoscopic guidance. In the latter procedure, lower-than-usual intra-abdominal insufflation pressures are used with *minimal* sedation to permit active feedback from the patient during nerve localization. The internal inguinal ring can be identified, nerve entrapment isolated, and the nerve released. A cryoprobe can be inserted percutaneously, and the nerve lesion made under direct vision.

UPPER EXTREMITY

Suprascapular nerve. The supraclavicular nerve passes through the suprascapular notch and provides innervation to the supraspinatus, infraspinatus, and shoulder joint. Clinically, the patient complains of a poorly localized upper shoulder pain. Tenderness is elicited by palpation of the suprascapular notch. Fluoroscopic guidance is helpful in locating the superior scapular border during cryoneurolysis.

Radial nerve. Cryoneurolysis can be performed at the elbow and wrist. The radial nerve passes over the anterior aspect of the lateral epicondyle. Probe entry is 2 cm lateral to the biceps tendon on the intercondylar line. Localization is facilitated by stimulation. At the wrist, branches of the radial nerve are located in the anatomic "snuff box" close to the exterior pollicis longus and extensor pollicis brevis tendons.

Ulnar nerve. Cryoneurolysis can be achieved at the elbow 2 to 3 cm proximal to the ulnar groove in the medial epicondyle. Similarly, at the wrist the nerve lies medial to the ulnar artery and beneath the flexor carpi ulnaris. The nerve is approached from the ulnar side of the tendon to block the cutaneous branches.

Median nerve. The median nerve lies medial to the brachial artery along the intercondylar line at the elbow. At the wrist, the nerve is approached 2 cm proximal to the distal wrist crease beneath the palmaris tendon. If the tendon is absent, the point of entry is 1 cm to the flexor carpi radialis tendon.

Digital nerves. The volar and dorsal digital nerves can be frozen at each side by insertion of the cryoprobe at the dorsolateral aspect of the base of the involved finger. Cryoneurolysis of the common volar digital nerve can also be done in the web space.

LOWER EXTREMITY

Lateral femoral cutaneous nerve. The lateral femoral cutaneous nerve passes under the inguinal ligament near the anterior superior iliac spine. It is amenable to cryoneurolysis

for the treatment of meralgia paresthesia. The procedure can be performed after surgical exposure or percutaneously, medial to the anterior superior iliac spine.

Superior gluteal nerve. The superior gluteal nerve is a branch of the sciatic nerve. After exiting the sciatic notch, it passes cauda1 to the inferior border of the gluteus minimus and penetrates the gluteus medius. It is injured as a result of shearing between the gluteal muscles with forced externa1 rotation of the leg extension of the hip. The neuralgia presents as pain in the lower back, dull pain in the buttock, vague pain in the popliteal fossa, and occasionally pain extending to the foot, mimicking radiculopathy. Patients describe a "giving way" of the leg and sit with the weight on the contralateral buttock.

Saphenous nerve. Neuralgia caused by irritation of the infrapatellar branch of the saphenous nerve is seen weeks to years after blunt injury to the tibial plateau, varicose vein surgery, or knee replacement. The nerve is vulnerable as it passes superficially to the tibial collateral ligament, piercing the sartorius tendon and fascia lata, inferior to the medial tibial condyle. The clinical presentation consists of dull pain in the knee joint and aching below the knee. Patients have trouble localizing the pain and tend to walk in a way that minimizes flexion of the knee.

Cryotherapy may be performed posteromedia1ly to the patella at the level of the knee or more distally superior to the medial malleolus.

Peroneal nerves. Neuralgia caused by irritation of the deep peroneal and superficial peroneal nerves can be seen weeks to years after injury to the knee, ankle, and foot. These superficial sensory nerves pass through strong ligamentous structures and are vulnerable to stretch injury with innervation of the ankle, compression injury resulting from edema, and sharp trauma caused by bone fragmentation.

The course of the superficial peroneal nerve is superficial and medial to the lateral malleolus and superficial to the inferior extensor retinaculum, terminating in the fourth and fifth toes. The clinical presentation consists of dull ankle pain aggravated by passive inversion of the ankle.

The deep peroneal nerve runs beneath the tendon of the extensor ha1lucis brevis, superficial to the dorsal interosseous muscle, in between the first and second metatarsal heads, terminating in the first and second toes. Patients with diabetes and women seem most vulnerable to this injury, but it is also seen occasionally after blunt injury to the dorsum of the foot. The clinical presentation consists of dull pain in the great toe that is often worse after prolonged standing. There may also be pain in the ball of the foot that is poorly localized and occasionally burning.

Cryotherapy of these nerves is best performed as far distally as possible. Lesions of the common peroneal nerve may cause significant motor weakness.

Interdigital nerve. Entrapment neuropathy at the metatarsal head presents as Morton's neuroma. Cryoanalgesia is performed at the apex of the metatarsal bones.

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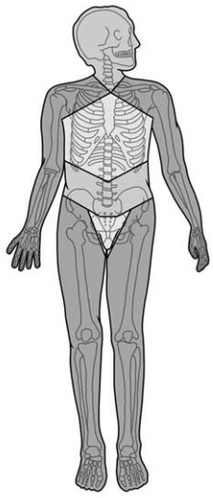
CHAPTER



5

Basic Risk Management for Interventional Procedures

KEN BRAXTON

Many of the procedures discussed in this book involve cutting-edge approaches to interventional pain management of patients in which other traditional therapies have failed. When such advanced techniques are attempted on patients in constant pain, claims of poor result and professional negligence are too often the result. These claims have always been a fear of physicians practicing in the area of interventional pain management because of the emotional and financial drain on their practice. How can you as an interventional pain management anesthesiologist avoid this turmoil? Follow a few simple guidelines by proactively reviewing each of the procedures that you perform and adjusting your individual practice with the risk management tools presented in this chapter. Risk management in your practice is often dictated by hospital policies, federal and state laws including those on mandatory risks to be discussed with your patients, billing and compliance laws, privacy concerns, and standards of care within your practice area. You must become familiar with all of these guidelines before performing interventional techniques. In this chapter, I will discuss areas to review that have been the focus of my representation of anesthesiologists over the past 20 years that are aimed at proactively helping to avoid legal problems with patients. A risk management checklist shown in Table 5-1 serves as a quick reference to the major areas to consider prior to any interventional procedure.

PREOPERATIVE GUIDELINES

KNOW YOUR STRENGTHS AND WEAKNESSES

All physicians who perform interventional procedures have specific techniques, instruments, anatomical landmarks, drugs, or procedures that they feel particularly proficient in performing based on their training and experience. This proficiency is generally the result of extensive training as a resident and a fellow. It is also likely combined with years of experience in performing a particular technique. Congratulations on accomplishing a level of proficiency that allows you to concentrate on other areas of risk management.

For those practitioners who have not reached a level of proficiency where you are comfortable in performing a particular procedure, know your limitations. Failure to do so gets many physicians in trouble both in terms of poor performance of the particular technique or procedure, and the ability to react to complications. For example, if you do not know the specific anatomy prior to performing a trigeminal ganglion block, although you are comfortable with performing somatic blocks generally, either refer to a review course or assist in the procedure prior to any attempts as the primary physician. This axiom seems very basic, but the number of lawsuits involving physicians who were performing a procedure in which they had general knowledge, but were in "a little too deep" for their experience base, is voluminous. You will get no sympathy from your peers by attempting a risky procedure for which you have little or no training and experience.

Because many of the techniques in this book are innovative or evolving in the particular drug or equipment used, you must constantly be vigilant of your knowledge base. Remember that in any claims involving professional negligence, another physician in the same area as your practice must have an expert criticism of you for the claim to proceed in the legal arena. Some of the harshest criticism from these experts in interventional pain management is of practitioners attempting to perform procedures for which they are minimally qualified. Juries and judges pay close attention to any evidence that a physician was practicing in an area for which he or she was not fully qualified, privileged, and certified. This is especially true for physicians using "off-label" drugs in their pain practice, unless the physician can demonstrate peer-reviewed clinical trials to support the therapy.

TABLE 5–1 Pain Practice Risk Management Assessment Guide

Strengths and weaknesses	Qualifications and experience with drugs/equipment.
	Technique has support of "peer-reviewed" trials.
	Your credentials withstand critique by pain management MD expert.
Patient	Past medical history verified.
	Physical, neurologic, and mental assessment.
	MD—-patient discussion of treatment plan.
	Always ask, *"What questions do you have?"*
	Patient dishonesty results in termination of care.
Other medical providers	Exchange all information regarding patient.
	Do not forget pharmacy records.
Consent	Standard is *"what* patient *needs to know to make an informed decision."*
	Duty to discuss with patient is MD's responsibility.
	Document on consent form *and* progress note.
Operative policies and staffing	Facility policies and procedures followed.
	Monitor team member expertise and workload.
	Announce the procedure as routine practice.
Instruments, sharps, drugs	All instruments maintained and certified sterile and free from any defects.
	Correct drugs and anatomy identified by team.
	Crash cart available.
Vital signs and patient discharge	Vital signs taken and documented are *mandatory.*
	Written instructions to patient including caution of *"if symptoms worsen return to ER."*
	No patient leaves without "discharge vitals."
Documentation	Absolutely necessary to defend any claims.
	Lack of documentation is the biggest mistake made by physicians.
	Just do it. More is better.

KNOW YOUR PATIENT

Pain management practice is known for a population of patients who attempt to abuse the health care system. In review of a physician's office practice, I always begin with a review of the patient charts to check for how thorough the history and physical (H&P) information is. I think that a good risk management tool is to place responsibility for past history on the patient. The patient should realize from the first visit that he/she is a critical part of the health care team, which includes both the patient and all health care providers. I suggest that you make patient information forms available for patients to complete prior to their first visit, either via the internet or sending the forms to them by mail. You must identify *all* the patient's other medical providers and pharmacies in order for you to properly take care of the patient *and* communicate with the other providers. Too many times I have seen patients with multiple pain specialists providing care at the same time, and none of the providers has any knowledge of the others!

Sit down with the patient during the initial visit and reinforce with each patient that she or he is a critical part of the medical team. Stress that any dishonesty in medical history provided by the patient will result in termination of care. Have the patient sign a form that he/she acknowledges responsibility for providing an accurate history and following the pain management regimen set up by you and your pain management team. This forms a "contract" with the patient that sets out the patient's responsibilities. In my representation of pain management physicians, I have always encouraged a policy of immediate termination of any patient who violates the practitioner's guidelines for pain therapy.

Conduct a full physical examination of the patient in order to ascertain a full picture of the patient's pain concerns. A patient may be emphatic that his or her only issue is headaches without other problems, but a full physical examination may reveal underlying issues impacting on your pain management decisions to include any contraindications for certain techniques or drug therapies. I do not know of another area of medicine in which the practitioner must have a more well-defined knowledge of the mental, neurological, and physical status of the patient.

COMMUNICATION WITH OTHER HEALTH CARE PROVIDERS

This is an easy way to avoid problems with drug dependence or malingering issues with pain management patients. You will need to obtain "disclosure of information" and release forms in compliance with the regulations where you practice to allow for communication between you and all of the patients' other health care providers. From the list of providers given to you at the initial visit, provide each of the other providers a summary of each visit along with working diagnoses and prescriptions. Have your staff contact other providers to obtain

pertinent medical records for inclusion in your office chart. If you find conflicting or duplicative therapies, confront your patient at the next visit regarding these issues. If you do not, you will be criticized by experts during any lawsuit regarding these unresolved issues. These communications should be used as a screening for any comorbidities that could be a potential risk in your pain management of the patient. Additionally, you may be surprised at what your patient provides other providers regarding their pain history and therapies.

CONSENT

Consent is such an easy way to avoid problems. Always remember that the information that you are to provide a patient regarding any procedure is basically what "a reasonable patient under the same or similar circumstances" would want to know about both the risk and benefits of the procedure. It is *not* what you as the physician think the patient should be told—it is what the patient needs to know to make an informed decision as a patient. Most states have specific requirements for particular procedures that you must be aware of prior to any discussion with the patient. The responsibility to obtain informed consent from the patient is yours, *not* the responsibility of your staff or the hospital staff! Never provide guarantees, but do provide the objectives of the procedure along with side effects and complications. You should explain the steps of the procedure, especially if the patient is going to be conscious during the procedure.

I always advise physicians to routinely end the discussion regarding consent with the open-ended question, "What questions do you have?" In taking patient depositions in a subsequent lawsuit, I am then able to ask the patient, "Did the physician end the discussion asking what questions you had?" Since this is quickly becoming an established practice of physicians with every patient, I usually get a positive response. This shifts the burden to the patient to disprove that they did not get all of their consent questions answered.

If the forum where you are practicing requires a specific form regarding informed consent, fill it out with the patient and also document your discussion in your progress notes. Documentation regarding consent is specific to the procedure and can be as long as 20 pages or more for experimental procedures, or as simple as a progress note stating that "the risks and benefits of the procedure have been discussed with the patient and the patient understands them." Just remember that you will be judged not by what you think the patient should be told, but what the reasonable patient would want to know in order to make an informed decision. Applied to buying a car, it is not what the salesman thinks you should know about the vehicle, but what you as the purchaser/consumer need to know to make an informed decision about the particular vehicle before you make your purchase.

OPERATIVE GUIDELINES

OPERATIVE TECHNIQUE, POLICIES, AND STAFFING

Now that you have (1) decided on a specific procedure based on your background, training, and experience; (2) obtained a thorough knowledge of the patient's medical, neurological, and physical history; (3) reviewed previous and current medical providers' records; and (4) discussed the procedure with the patient to include complications, risks, and benefits, the next step in the risk management process is to review your operative policies. Basically, is the team ready for the patient to undergo the specific procedure? As the physician, you have to ensure that the team consists of personnel knowledgeable in their duties to be performed during the procedure. It is just as important for the nurse assisting you to understand the procedure objectives, approach, equipment, drugs, and risks, as it is for you. This includes making sure that the correct instruments, drugs, and equipment are available. Protocols should be in place for every aspect of the procedure from patient positioning and sedation of the patient through reversal of sedation at the conclusion of the procedure. Each member of the team must know the objectives of the procedure. Excessive workloads among the team members can lead to inattention to details including wrong medications or dosage, and wrong instrument counts at the conclusion of the procedure. Miscommunication between team members is a common factor in operative errors, including failure to communicate abnormal laboratory or radiological results, failure to communicate the operative goals and postoperative plan of care, and failing to provide each other with continuing updates of the patient's status.

As a last safeguard prior to the procedure, a good practice for the physician to follow is to "announce" to the team the specific procedure, approach, and objectives of the procedure prior to beginning the procedure. This repetitive approach to any procedure alleviates possible mistakes in last-minute staffing, instrumentation, and drug issues that could arise.

Proper operative technique is an area that should be routinely addressed in facility policies and protocols. Sterile equipment, needle/sponge counts, personnel training, and crash cart stock are sample areas to be covered in written policies before any procedures are performed. By addressing these concerns through quality assurance policies and checklists, the possibility for any iatrogenic events is decreased.

INSTRUMENTATION, DRUGS, AND EMERGENCIES

Intraoperative mistakes are the most litigious area of interventional pain management. Equipment failure and improper use of equipment rank highest in the number of these misadventures. The physician must not become complacent in her or his review of the quality control of

each piece of equipment to be used on a patient. Examples of equipment-related lawsuits include simple items such as (1) hoses that can wear out over time resulting in failure intraoperatively when placed under pressure, (2) items that become infected due to overuse or improper cleaning resulting in postoperative infection/sepsis in the patient, and (3) nondisposable catheters in which pieces can either microscopically shear or crack while inside the patient producing catastrophic results. You must check on the quality of all of your equipment and have a quality assurance protocol in place for proper testing of all surgical equipment on a regular basis.

Drug misidentification, drug interactions and allergies, and improper dosing have recently become very hot topics for discussion by the medical community and news media. At least once a week, a headline news item talks about a drug error in a hospital, or a new Food and Drug Administration alert seems to be published warning practitioners to be vigilant of these medication errors. Many of the drugs used in pain management must be carefully injected, and the patient must be vigilantly monitored for side effects. There are numerous cases in the medical and legal literature of experimental drugs being incorrectly administered to patients with catastrophic results. Examples of these errors include (1) injection of caustic drugs into the subdural space causing paralysis, (2) sedation with Norcuron or similar paralytic agents without intubation causing death, and (3) incorrect dosage of narcotics causing both oversedation and death.

An extremely important aspect of interventional pain practice is the physician's ability to identify potential emergent situations and properly respond to any emergencies that may arise. Because a great many procedures occur in the outpatient setting, it is critical for the physician to proactively ensure that both personnel and equipment are available to react instantly. This includes training of personnel in advanced life support and the availability of a crash cart with appropriate resuscitative drugs and equipment. There are many horror stories in quality assurance and risk management periodicals of outpatient facilities being incapable of handling an emergency cardiac or neurological event. An office policy of calling 911 as the primary reaction to a cardiac event during an interventional procedure is not going to be considered "standard of care" by your peers. Similarly, not having Narcan or another drug for reversal of narcotic drug effects available in any procedure in which sedation is administered would raise red flags to any expert reviewing a potential claim of negligence.

POSTOPERATIVE GUIDELINES

VITAL SIGNS AND PATIENT INSTRUCTIONS

From the legal perspective, the second most critical step behind documentation in the interventional pain practice is recording vital signs. Vital signs being taken at every step from beginning to end of a procedure are not just to be considered, they are *mandatory* in any subsequent defense of the anesthesiologist's care. Whenever I as an attorney discuss any case regarding interventional procedures, the first area any expert reviewer asks me about is, "How were the patient's vital signs during and after the procedure?" No matter what procedure, what drugs, type of facility, patient history, or physician experience, vital signs are at the base of reconstructing what happened in any particular procedure. I cannot stress enough how important the documentation of vital signs is in subsequent litigation involving interventional procedures. It is imperative that not only vital signs be recorded during the procedure, but they must be taken postoperatively to establish patient stability before discharge from your facility. It is much more difficult to defend any negligence claims if I am unable to establish that the vital signs of the patient were normal "at the time the patient went home." The physician has to establish a practice by his staff that no patient is allowed to leave the facility without getting the patient's vital signs at the time of discharge!

Prior to discharge following any interventional procedure, the patient must be provided with written instructions for them to follow at home. Verbal instructions will not suffice as patients do not typically remember conversations following interventional techniques, either because of sedation or the white coat syndrome. Written instructions must be provided to each patient regarding their follow-up, medication orders, and possible side effects. All instructions should include a statement similar to the following: "If symptoms worsen, go immediately to the nearest emergency room."

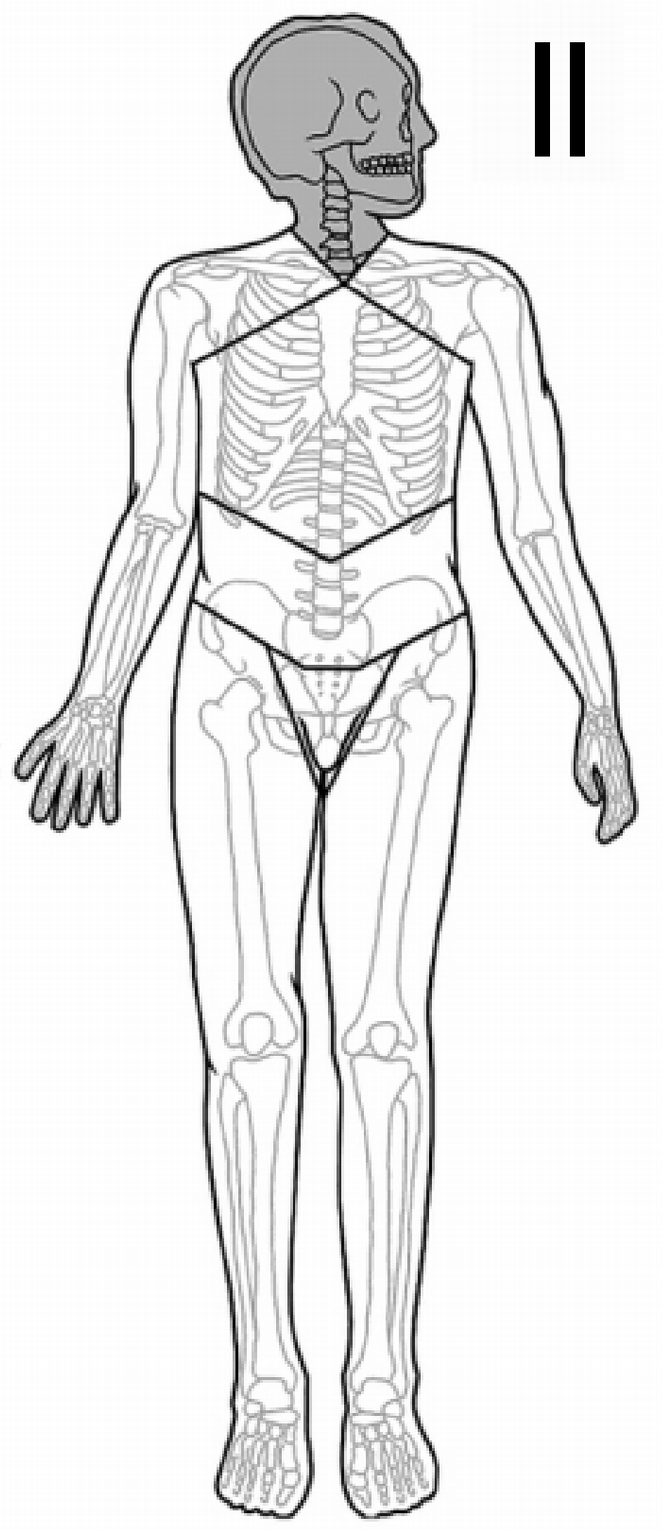
DOCUMENTATION

No basic risk management checklist can be created without emphasizing the number one principle for interventional procedures, which is documentation. Documentation is the core defense of any subsequent claim or lawsuit. To protect yourself at every step in your delivery of pain management intervention to the patient, you must document your thoughts and actions. Documentation is the simplest and fastest way for any risk manager to defend claims of negligence. As important as "location" is to selling real estate, "documentation" is to the defense of any professional negligence claims brought against a physician. Remember that any negligence claim or lawsuit will not proceed without review by your peers. If the reviewing physician is unable to determine the rationale for your actions through your documentation, you are inviting the reviewer to be critical of you. Of the thousands of cases I have defended on behalf of physicians, by far the easiest to defend have great documentation, and the most difficult to defend are those with nonexistent documentation.

In conclusion, I have provided the attached checklist for you to review your risk management philosophy and to help in structuring a process for successful interventional techniques in your pain practice.

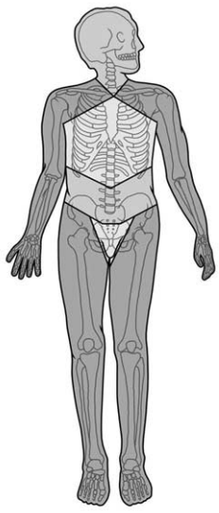
SECTION

II



Head and Neck

C H A P T E R



6

Somatic Blocks of the Head and Neck

SERDAR ERDINE, GABOR B. RACZ, AND CARL E. NOE

BLOCK AND NEUROLYSIS OF TRIGEMINAL GANGLION AND BRANCHES

HISTORY

Trigeminal neuralgia was treated for the first time by alcohol injection into the nerve by Pitres in 1902.[1] He was followed by other authors who gave this technique a great deal of publicity. By 1905, Schlosser[2] had reported 68 cases of severe trigeminal neuralgia successfully treated by alcohol nerve block. According to Cushing,[3] the percutaneous transforamen ovale approach to the trigeminal (gasserian) ganglion using absolute alcohol was first described by Hartel in 1912.[4]

In the early 1930s, Kirschner[5] began to use radiofrequency neurolysis. Using diathermy, it produced high-current lesions of the trigeminal ganglion for relief of trigeminal neuralgia, being the first report in medical literature to use radiofrequency for the treatment of chronic intractable pain.

Putnam and Hamptom,[6] who reported 18 cases of trigeminal neuralgia and four cases of carcinoma of the mouth, recommended x-ray control during the procedure, using 0.5 mil of 5% phenol, and were the first to publish the use of phenol as a neurolytic agent for the treatment of this condition.

In the evolution of the treatment, radiofrequency (RF) lesioning for this ganglion was described by Sweet and Wepsic in 1965,[7] retrogasserian glycerol injection by Hakanson in 1981,[8] and percutaneous balloon compression by Mullan and Lichtor in 1978 and published in 1983.[9]

ANATOMY

The ganglion lies within the cranium in an area called *Meckel's cave* or *Meckel's cavity*, close to the apex of the petrous part of the temporal bone (Figure 6-1A). Medially, the trigeminal ganglion is bounded by the cavernous sinus, superiorly by the inferior surface of the temporal lobe of the brain, and posteriorly by the brain stem. The ganglion is shaped like a crescent moon. The convex side is aimed anterolaterally. It is bounded medially by the internal carotid artery and trochlear and optic nerves. The posterior border of the ganglion includes the dura of Meckel's cave and cerebrospinal fluid (CSF). Anteriorly, the ganglion gives off three branches intracranially: ophthalmic, maxillary, and mandibular.

Sensation of the oral mucosa, anterior and middle cranial fossa, tooth pulp, surrounding gingiva, and periodontal membrane is innervated by the trigeminal nerve. Proprioceptive information from the muscles of mastication and extraoccular muscles also terminates in the trigeminal ganglion. The trigeminal ganglion is named after a Viennese anatomist, Johann Laurentius Gasser (Figure 6-1B). The two medial (ophthalmic and maxillary) are sensory, whereas the lateral most mandibular branch is partly motor. The trigeminal ganglion is somatotropically located. The ophthalmic branch is located dorsally, the maxillary branch is intermediate, and the mandibular branch is located ventrally. These nerves and their branches provide the cutaneous and dermatomal innervation of the head and face as shown in Figure 6-2.

Trigeminal ganglion links with the autonomic nervous system via the ciliary, sphenopalatine, otic and submaxillary ganglia, and communicates with the oculomotor, facial, and glossopharyngeal nerves.[10]

INDICATIONS

Approaches to the trigeminal ganglion by various methods aim to relieve the pain transmitted through the trigeminal nerve. In the past, trigeminal ganglion block has been extensively used in the treatment of trigeminal neuralgia or tic douloureux. With the introduction of thermogangliolysis, the trigeminal ganglion block is rarely used, except for intraoperative or postoperative pain. In addition

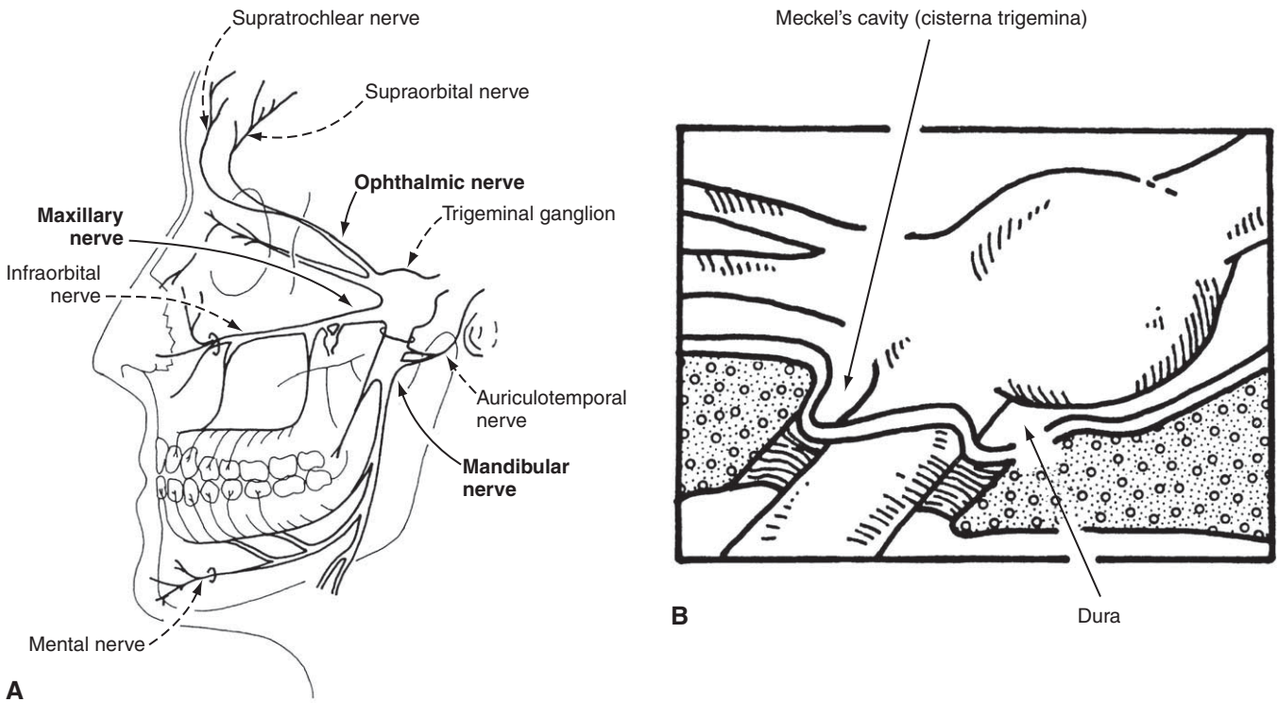


FIGURE 6–1

(A) The figure shows the location of the trigeminal ganglion in the middle cranial fossa and the course of its three branches: (1) ophthalmic, (2) maxillary, and (3) mandibular. (B) The relationship of the trigeminal ganglion in Meckel's cavity. CSF, cerebrospinal fluid.

to idiopathic trigeminal neuralgia, secondary neuralgic pain due to facial pain resulting from terminal cancer or multiple sclerosis may also be treated with these approaches. These techniques are to be used only when conventional medical treatment is inadequate or causes undesirable side effects. Table 6-1 enumerates the indications and contraindications.

EQUIPMENT

Trigeminal Block

- 25-gauge needle (for skin infiltration)
- 5-ml syringe (for local anesthetic solution)
- 22-gauge, B-bevel, 8- to 10-cm needle (for injection of local anesthetic for a block)

Radiofrequency Lesioning

- RF thermocoagulation (RFTC) lesion generator and cables
- 25-gauge needle (for skin infiltration)
- 5-ml syringe (for local anesthetic solution)
- 16-gauge intravenous catheter (for introducing the RF needle)
- RF needles, 10 cm in length; 2-mm or 5-mm RF tip (depending on the branch to be lesioned)

Balloon Compression

- 25-gauge needle (for skin infiltration)
- 5-ml syringe (for local anesthetic solution)
- 2-ml syringe (for iohexol [Omnipaque] injection)
- 14-gauge, 10-cm needle (for initial insertion prior to Fogarty catheter)
- Fogarty catheter (4-French)

DRUGS

Block

- 1% lidocaine for infiltration
- 0.25% bupivacaine or 0.2% ropivacaine
- Methylprednisolone, optional

Balloon Compression

- 1% lidocaine for infiltration
- Iohexol

Neurolytic Block

- Alcohol 97%–1-ml vial or
- Phenol in saline or glycerin 6%–1 ml or
- Phenol in iohexol 6 to 10%–1 ml
- Glycerol 40 to 50%–1 ml

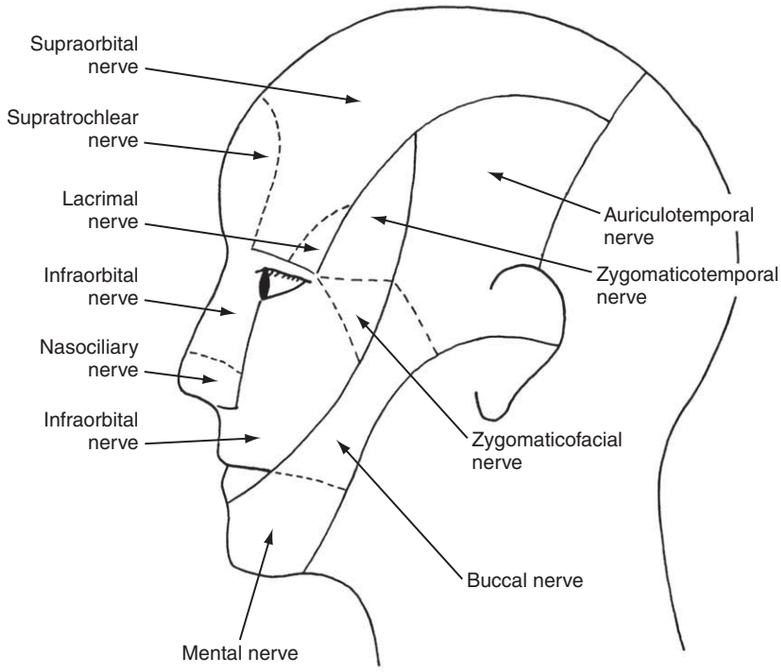


FIGURE 6–2
This drawing illustrates the innervation of the skin and the face by the peripheral branches of the trigeminal nerve.

TRIGEMINAL GANGLION BLOCK PROCEDURE

Preparation of Patient

Comfort should be provided to the patient during percutaneous procedures. The patient should be alert enough to respond to the testing, for example, with electrical stimulation. Generally, intravenous fentanyl (average dose 0.1–0.16 mg), midazolam (average dose 3.0–5.5 mg), and methohexital (average dose 51.4 mg) are used. In a study comparing several regimens it was concluded that high-dose fentanyl and midazolam together with droperidol improved the comfort of the patient during the prodecure.[11]

Technique of Needle Insertion

The procedure should be performed under fluoroscopic control. Landmarks follow: (1) entry point is 2 to 3 cm lateral to the commissura labialis (angle of the mouth) (Figure 6-3); (2) needle should be directed 3 cm anterior to the external auditory meatus when seen from the side (Figure 6-4B); and (3) needle should be directed toward the pupil when seen from the front of the face (Figure 6-4A). Cannula insertion should be performed following the bisector (45°C) of the sagittal plane, which passes through the pupil and the frontal-mentonian plane.

TABLE 6–1 Use of Trigeminal Ganglion Nerve Block

Indications	Contraindications
Trigeminal neuralgia	Local infection
Cluster headaches	Sepsis
Intractable ocular pain	Coagulopathy
Cancer pain	Increased intracranial pressure
Surgical anesthesia	Major psychopathology

Position of Patient

The patient is supine on the table with the head in an extended position. The C-arm is placed at the head of the table for posteroanterior (PA), lateral, and submental views. The direction of the needle is toward the pupil when one looks from the front and midpoint of the zygomatic arch when one looks from the side.

X-Ray Technique

1. Oblique projection. Lateral inclination of approximately 30 degrees toward the side of the lesion, with caudal inclination of approximately 30 degrees. The mentonian arch must be seen and, in the upper-internal quadrant to it, the foramen ovale.
2. Lateral projection. Performed when the cannula has already been inserted into the foramen ovale. Its usefulness is to calculate the insertion of the cannula into the bony tunnel of the foramen ovale. The tip of the cannula must not exceed 2 mm in distance from the plane of clivus.

A finger may be placed inside the mouth. This helps guide the needle and prevents penetration of the oral

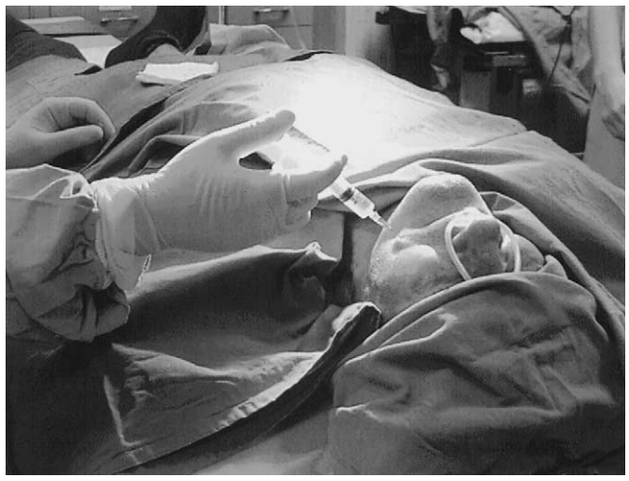


FIGURE 6–3
The needle entry point is 3 cm lateral to the angle of the mouth.

mucosa (Figure 6-5). There is a definite risk of meningitis if the needle enters the mucosa.

The direction of the needle should be verified under fluoroscopy in submental, lateral, and PA views (Figure 6-6). To obtain the submental view, the C-arm of the fluoroscopy is first placed in the PA direction. In this view, the orbital line, the petrous ridge may be visualized through the orbits. The target site in this dimension is a point approximately 9 mm to 1 cm medial to the lateral rim of the internal auditory meatus. This usually coincides with the medial extent of a dip that occurs in the petrous ridge.

Then the C-arm is moved slightly lateral and oblique submentally to see the foramen ovale (Figure 6-7). In many patients, it is possible to see the foramen ovale. When the foramen ovale is seen, the needle is directed toward the foramen through the entrance point (Figure 6-8). Note anatomically, the mandibular nerve is on the lateral part of the foramen ovale, whereas the maxillary and ophthalmic divisions are more medial.

When the needle enters the foramen ovale, the fluoroscope is turned laterally (Figure 6-9). The lateral image should reveal that the needle is directed toward the direct angle produced by the clivus and the petrous ridge of the temporal bone (Figures 6-10 to 6-12). The lateral view is important to verify the depth of the needle inside Meckel's cave. The aspiration test is mandatory. A 0.5-ml iohexol solution helps determine that the needle has not penetrated the dura.

Diagnostic Block

For confirming that the pain generator is the trigeminal ganglion, after negative aspirations, up to 1 ml of local anesthetic (lidocaine, bupivacaine, or ropivacaine) is injected. The patient should have pain relief if the pain generator is present. The physician should monitor that the solution has not entered the cranial CSF. The brain stem function should be evaluated to determine if the local anesthetic solution has not reached it. Brain stem function is affected if the patient complains of bilateral headache or fourth or sixth nerve palsy, or if pupillary changes occur.

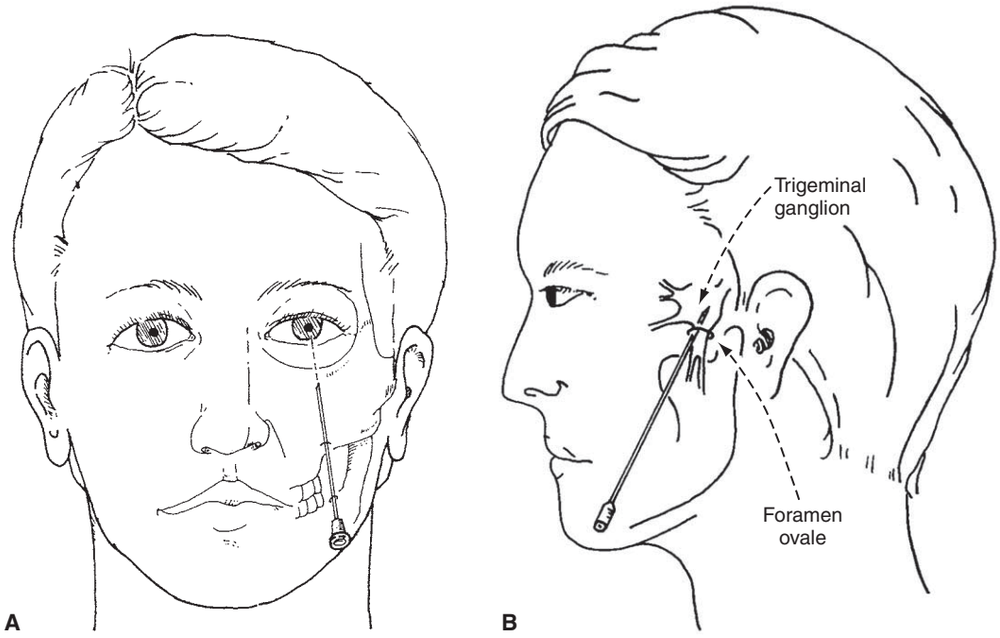


FIGURE 6–4
(A) The drawing shows the needle penetration toward the pupil in the anterior view. (B) This illustration shows the needle direction 3 cm anterior to the external auditory meatus on the zygoma.

FIGURE 6–5
To prevent the needle from penetrating the cheek and the oral cavity, one can put a finger in the mouth, as the needle is advanced toward the foramen ovale.

NEUROLYSIS OF TRIGEMINAL GANGLION

The amount of the neurolytic solution should not exceed 1 ml given in smaller aliquots. Otherwise, it may spread to the brain stem and cause severe complications. Phenol and alcohol have been used commonly in the past but are not recommended currently.

Three neurolytic agents used in neurolysis are alcohol, phenol, or glycerol.

1. Alcohol is the most spreadable solution and hence should be used with caution. A maximum of 1 ml alcohol is used in divided doses watching for signs of bilateral spread.
2. Phenol is a viscous solution. Consequently, it will spread less and have more contact time with the target tissues. The most commonly used neurolytic agent is 6% phenol in glycerol. Recently some clinicians are using 6–10% phenol in contrast (Omnipaque) instead.
3. Glycerol may be directly injected like other neurolytic agents, or the retrogasserian glycerol injection technique may be used as described below.

Technique for Glycerol Injection

After correct needle placement on the trigeminal ganglion, the patient is kept in a supine position. The needle should pierce the foramen ovale just anterior to its geometric center to place the needle into the trigeminal cistern. The needle is advanced until free flow of the CSF is observed. The patient is then placed in the semi-sitting position, and the neck is flexed. Contrast solution, iohexol 0.1 to 0.5 ml, is injected at this position in the cistern.

Failure of visualization or diffusion of the dye indicates a wrong placement of the needle and the needle should be repositioned. When the cistern is visualized, the contrast material is drawn back by free flow. The flow of

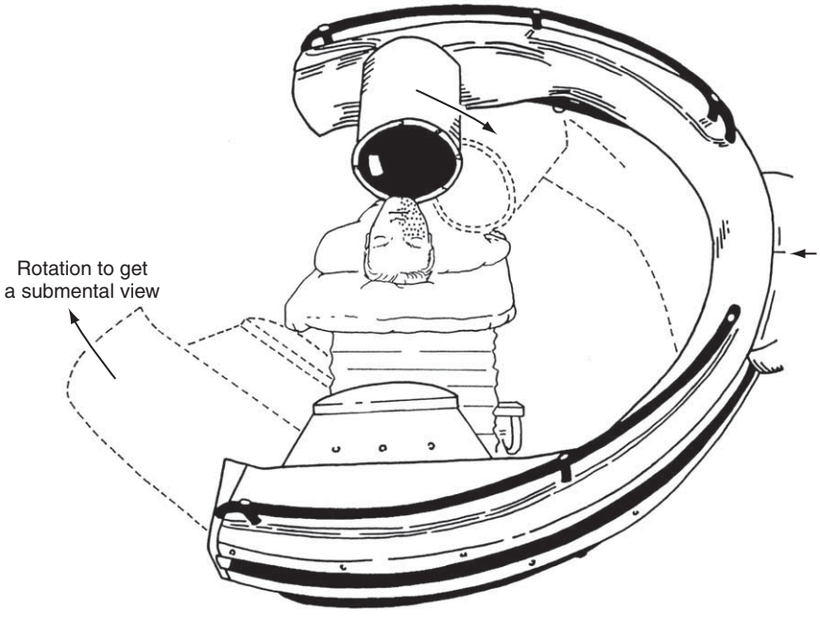


FIGURE 6–6
The fluoroscopic position to obtain the submental view of foramen ovale.

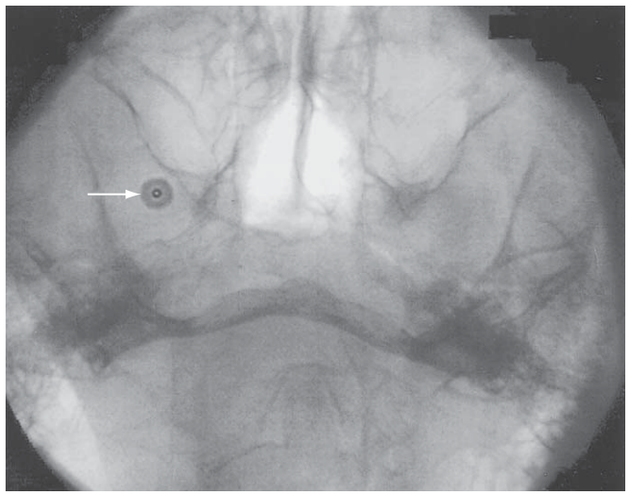


FIGURE 6–7

The submental view of the face with the needle in the foramen ovale. Note the "tunnel view" of the hub of the needle. The arrow indicates the rim of the foramen ovale.

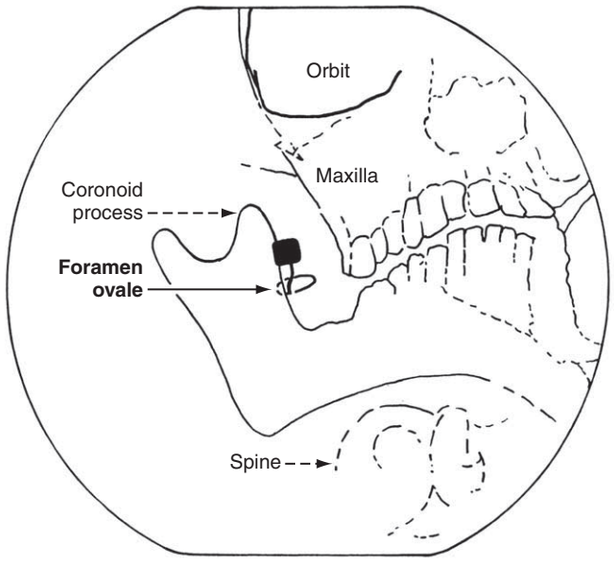


FIGURE 6–8

When the submental view is obtained, the foramen ovale is seen to appear medial to the medial edge of the mandible. Depending on the lateral rotation of the C-arm, the foramen ovale visualization can move more medially toward the maxilla.

the dye is slower than the CSF itself. The same amount of glycerol is injected in the cistern. The patient is kept at the same semi-sitting position for the next 2 hours.

During this injection, severe headache or dysesthesia may occur, and the patient should be warned about this result prior to the injection. Some patients may get benefit immediately, whereas some patients may experience relief within the next 2 weeks.

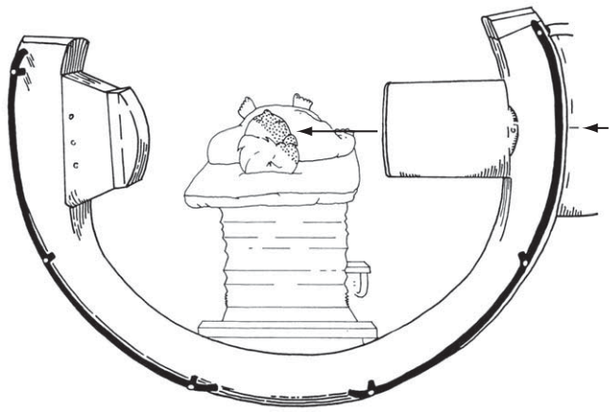


FIGURE 6–9

The lateral C-arm placement for viewing the lateral view of the base of the skull.

Complications

Complications of retrogasserian glycerol injection are paresthesia, dysesthesia, anesthesia dolorosa, corneal hypoesthesia or anesthesia, diminished corneal reflex, keratitis, and masticatory weakness.

TECHNIQUE OF TRIGEMINAL GANGLION STIMULATION AND RADIOFREQUENCY LESIONING

Stimulation

A test stimulation is mandatory before radiofrequency lesioning. Apart from other techniques like glycerol, neurolytic solution injection, or balloon compression, the lesion is more precise with radiofrequency lesioning and should be limited to the affected nerve. Thus, during the sensorial stimulation the patient should be awake enough to respond to the test with sensorial stimulation for proper localization of the tip of the electrode.

The mandibular nerve has some motor fibers. If the nerve is stimulated at 2 Hz with 0.1 to 1.5 V, the muscle contraction of the lower mandible is observed. This is also a way of verifying that the needle is passed through the foramen ovale and is on the retrogasserian rootlets. If the first and second divisions are affected, there should be no motor response.

The second step is to seek for paresthesia in the proper localization. A stimulation at 50 to 100 Hz is given with 0.1 to 0.5 V. If the needle is properly located, there will be a tingling-like sensation or electric-like paresthesias in the innervation of that branch in the face. If this sensation is obtained after 0.5-V stimulation, then the needle should be redirected to get the same response at a lower voltage. However, it should be kept in mind that there might be residual sensorial deficits from previous lesioning.

When the electrode is adjusted for localization, it should also be remembered that the gasserian ganglion

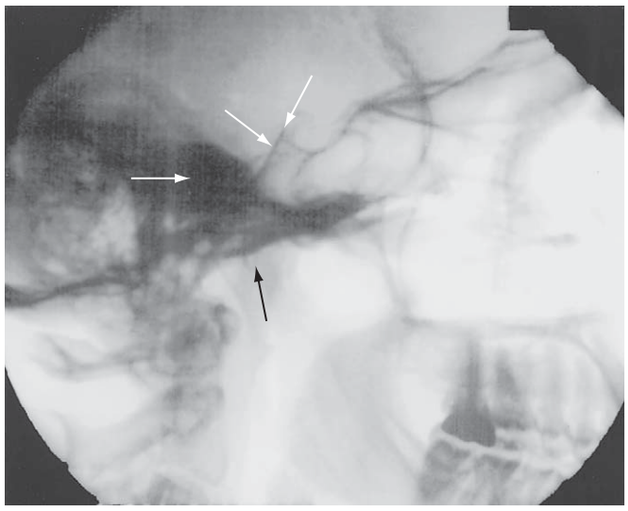


FIGURE 6–10

Lateral view radiographic imaging showing the anterior clinoid process (A), posterior clinoid process (B), clivus (C), temporal bone (D), and line drawn from the point clivus meets the temporal ridge perpendicularly towards the base of the skull (D) to (E). At the base of the skull, this locates the foramen ovale.

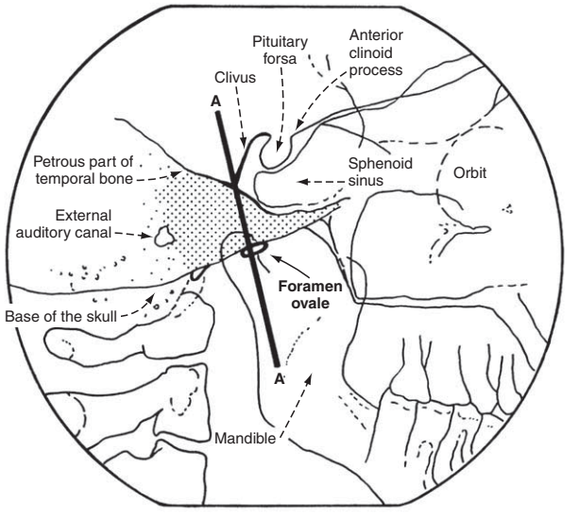


FIGURE 6–11

A line drawn perpendicularly (D–E) through where the intersection of the clivus and petrous part of the temporal bone meet identifies the foramen ovale at the base of the skull.

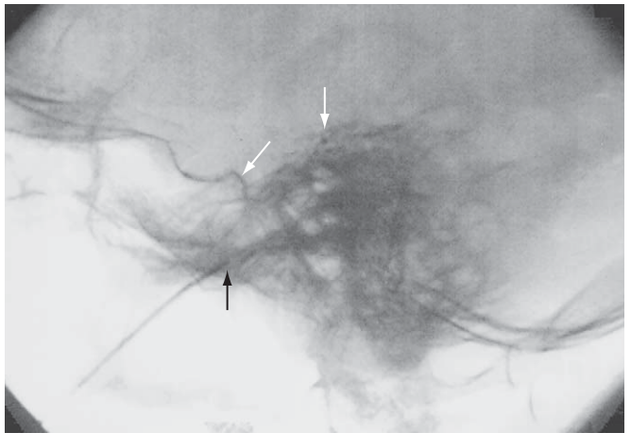


FIGURE 6–12

Another lateral view (see Figure 6-10) of the cranium. (A) Clivus. (B) Petrous part of the temporal bone. (C) Foramen ovale with needle entering it.

and its retrogasserian rootlets lie on a plane running from a superomedial to inferolateral direction. If there is a motor response, it means that the needle is too lateral, and for a better response, it should be more medial.

After stimulation is completed, the physician should again rule out if the needle is in a vessel or not. If blood is aspirated, the needle position should be adjusted. If blood is still aspirated, the procedure should be terminated and a second attempt should be made another day. Impedance monitoring is not essential for trigeminal ganglion lesioning, but if used, it should be 150 to 350 O for rootlets bathing in the CSF and 1000 O if it is in a non-neural tissue.

Lesioning

Several types of electrodes may be used for lesioning, such as cordotomy-type electrodes and trigeminal electrodes with the Tew needle and the Racz-Finch curved-blunt needle. In order to prevent inadvertent puncture of vessels in the region, it is preferable to use the curved blunt needle. If the needle is properly placed and stimulated, the patient is then ready for lesioning (Figures 6-13 to 6-16).

Stimulation Parameters

Voltage is 0–1 volts, and sensory, 50 Hz. Paresthesia between 0.2 and 0.5 V must be noted in the painful zone. Motor is 2 Hz. Motor contraction of the masseter muscle is sought with 0.7–1 V. If no motor contraction happens, the tip of the needle is positioned in the I or II branch of V cranial nerve.

Lesion Parameters

First lesion: 60 seconds at 65°C. When the lesion is induced, check the bilateral corneal reflex and pain sensitivity in the neuralgic and contralateral zones. Second lesion: 60 seconds at 70°C. Proceed in a similar manner. Third lesion: 60 seconds at 72–75°C. Proceed in a similar manner. A fourth lesion may be assessed at 75°C if pain involves two branches of the V cranial nerve.

The patient can either be sedated by midazolam and fentanyl or 0.5 ml of 0.25% bupivacaine, or 0.2% ropivacaine may be injected. One should wait at least 30 seconds prior to RF lesioning. RF lesioning is done at 60°C for 60 seconds. If the patient cannot tolerate the

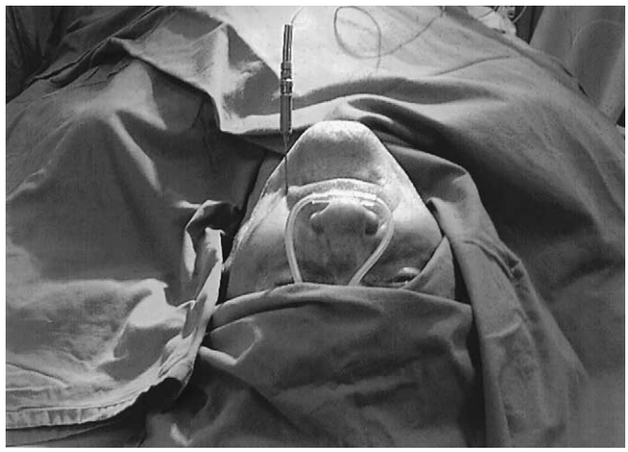


FIGURE 6–13

Radiofrequency needle entering the facial skin without a catheter. Note the draping of the patient with the area of entry exposed and an O2 cannula in place for trigeminal ganglion radiofrequency.

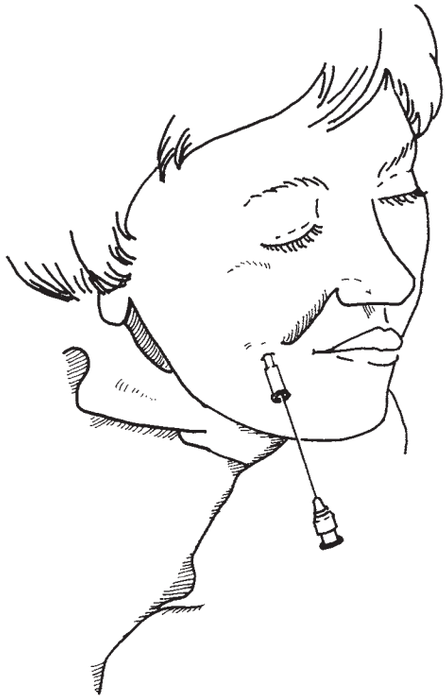


FIGURE 6–14

The alternative technique of introducing the curved-blunt Racz-Finch radiofrequency needle is shown in this drawing. Initially, an angiocatheter is introduced at the entry site toward the foramen ovale. Following that, the RF needle is inserted through the angiocatheter.

lesioning, stop and wait another 30 seconds, and try again or add another 0.5 ml of local anesthetic prior to RF lesioning.

If more than one branch of the trigeminal nerve is affected, several lesions by repositioning of the needle should be performed. After each repositioning, the stimulation test should be repeated to seek paresthesia at the desired site.

For the first division lesioning, corneal reflex should be preserved at each lesion, and lesioning should begin at lesser degrees than 60°C to preserve the corneal reflex. After the lesioning is completed, the needle is removed. The patient is instructed to watch for swelling of the face and to put ice on the face to reduce any swelling that may occur.

Patient Follow-Up

Immediate and later follow-up of the patient are important. Some authors prefer to do the lesioning on an outpatient basis, and some hospitalize the patient for a day. In some patients there is immediate pain relief, but the next day or within the first week the pain may return. In such patients, lesioning may be repeated. The patient should be monitored for an additional month to determine if side effects appear.

PERCUTANEOUS TRIGEMINAL GANGLION BALLOON COMPRESSION

The percutaneous trigeminal ganglion balloon compression procedure is performed under light general anesthesia. The position of the patient is the same as it is with RF lesioning. The needle is introduced, as described earlier, through the foramen ovale. A 4-French Fogarty catheter is advanced through the needle into Meckel's cavity. The balloon of the catheter is inflated by injecting contrast solution. The shape of the balloon inside the cavity in the lateral position resembles a pear (Figure 6-17). The inflated balloon is left there for 60 seconds or more, although there is no agreement on the duration.

The procedure should be done with vital sign monitoring because bradycardia and hypertension may be observed.

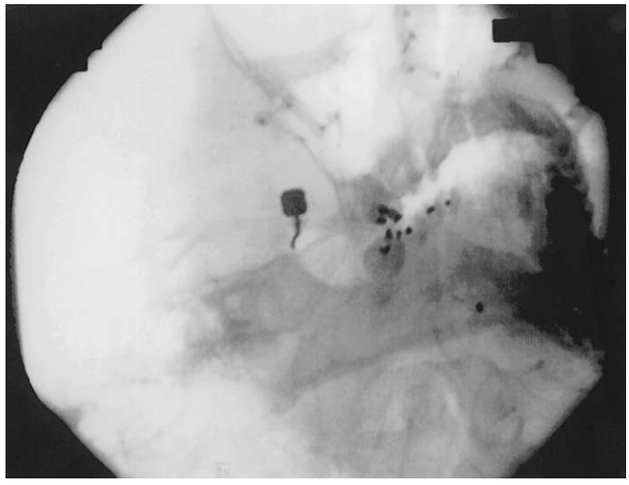


FIGURE 6-15

Submental view with a fluoroscope. Note the curved-blunt Racz-Finch radiofrequency needle entering the foramen ovale in its lateral aspect.

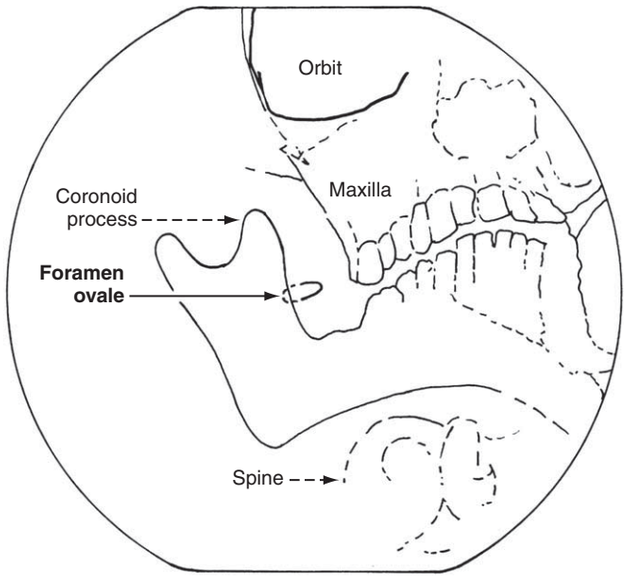


FIGURE 6-16

This drawing of the submental view of the face illustrates the relationship of the foramen ovale and the needle entry at the medial border of the mandible and maxilla. The needle entry is shown in the lateral aspect of the foramen ovale.

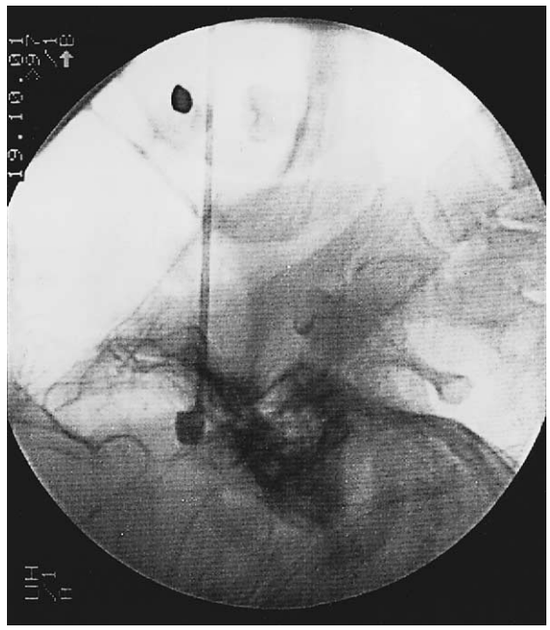


FIGURE 6-17

The lateral view of the balloon during trigeminal ganglion neurolysis.

Complications

Significant masseter weakness is a common complication, especially in the initial period. This weakness generally disappears within the first 3 months. Hypoesthesia, dysesthesia, anesthesia dolorosa, balloon failure, and hematoma on the cheek may also be observed (Table 6-2).

COMPLICATIONS

Percutaneous interventions of the trigeminal ganglion are not free of complications. In selected series, Taha and Tew[12] compared the results and complications of percutaneous techniques. The total number of patients was 6205 for RF rhizotomy, 1217 for glycerol rhizotomy, and 759 for balloon compression. Facial numbness occurred in 98% of the patients after RF rhizotomy, in 72% after balloon compression, and in 60% after glycerol injection. Taha and Tew found that anesthesia dolorosa occurred in 1.5%, 1.8%, and 0.1%, respectively.[12] Anesthesia dolorosa occurred at a rate of 0.3% to 4% in RF lesioning.[13–15] For glycerol injection, anesthesia dolorosa occurs in 0–2% of cases.[16–20] For balloon compression, ipsilateral masticatory weakness, hypoesthesia, dysesthesia, and anesthesia dolorosa may occur in 3–5% of cases.[21–23]

Loss of Corneal Reflex

The overall incidence of corneal reflex loss and neurolytic keratitis is 0.6–1.8%, depending on the technique used. Corneal anesthesia was the highest for RF rhizotomy with 7%; it was less for glycerol with 3.7% and balloon compression with 1.5% occurrence rates. It is lowest for balloon compression and highest for RF lesioning. This is not a desirable condition, but in some patients, because of the intolerable pain, it may be preferred.[12]

Motor Deficit

Motor deficit occurs during the lesioning of the third branch, the mandibular nerve. The incidence is the highest, 66%, with balloon compression. For RF rhizotomy it is 24%, and for glycerol injection it is 1.7%. The motor deficit improves within 1 year.

Carotid Artery Puncture

Carotid artery puncture occurs when the radiographic landmarks are not employed and the needle is too inferior and medial. Blind technique is not recommended.

Retrobulbar Hematoma and Hematoma in Cheek

If the needle is advanced to the retrobulbar space, retrobulbar hematoma may develop. This is a dramatic complication to the patient, although it is relieved by

TABLE 6–2 Complications of Trigeminal Ganglion Block or Neurolysis

Annoying dysesthesia and anesthesia dolorosa, loss of corneal reflex
Neurolytic keratitis
Visual loss
Retrobulbar hematoma
Hematoma in the cheek
Significant motor root deficit
Carotid puncture
Meningitis
Inadvertent intracranial placement of electrode resulting in intracranial hemorrhaging, penetration through wrong foramen causing defects in other cranial nerves

conservative methods without any sequelae. The eyeball is pushed from the retrobulbar space and exophthalmus develops. Compression over the eye stops the bleeding, and the swelling subsides during the following days. Hematoma in the cheek may develop if the needle passes through a vessel while it is introduced. Compression over the cheek by cold pack after the needle is withdrawn may be helpful.

Infection

One of the main concerns is infection and the incidence of infection. In the series by Sweet, there were 24 cases of meningitis in 7000 cases. One of these patients died.[24] Ocular motor paralysis and cavernous sinus fistula[25] is a possibility. An intracranial hemorrhage[26] has been reported to be fatal. Misplacement of needles into incorrect skull base foramina can lead to vascular damage and secondary hypertension that, in turn, can lead to bleeding.[27] The most common problem from neurodestructive procedures is altered sensation or numbness that has been reported to range from 6 to 26% of patients undergoing RF-type procedures.

CLINICAL PEARLS

With edentulous patients, the needle's point of introduction sometimes needs to be a little more posterior than for the patients with a full set of teeth; the needle will strike the foramen ovale at too acute an angle. This may be prevented if the procedure is done under fluoroscopy.

Because this is an uncomfortable procedure, some form of intravenous sedation given immediately before the procedure often affords satisfactory analgesia for the procedure without obtunding the patient's ability to cooperate and provide necessary feedback. The patient must be conscious between each coagulation application so that sensory testing of the face can take place.

The placement of the needle should be confirmed by the lateral view. In case of deep needle placement, one can enter the brain stem and cause hemorrhage.

The aspiration test is mandatory because the posterior part of the trigeminal ganglion is surrounded by an invagination of cranial dura mater containing CSF in Meckel's cavity. Inadvertent injection of therapeutic agents into this cul-de-sac can spread to other intracranial structures, producing profound and rapid loss of consciousness and collapse. This is obviously an eminently reversible situation when local anesthetic agents are used, but in the event that such a catastrophe occurred with neurolytic agents, inadvertent neurolysis of adjacent cranial nerves could occur.

If slight liquorrhage occurs during the procedure, it should be considered to be a consequence of the ganglion puncture, with the risk of CSF fistula being minimal.

Irritation of the dura may cause persistent headache, and in some patients, nausea and vomiting lasting for days may also be observed. If blood is aspirated, the needle should be replaced, and if bleeding continues, the procedure should be stopped.

During repeated lesioning, the aspiration test should be repeated and impedance should be monitored to verify the position of the needle before each RFTC application. If the needle is in the nerve, the impedance is generally between 300 and 450 O.

The endpoint is reached when the desired division of the trigeminal nerve has become slightly analgesic but not anesthetic. Usually at about 70°C, analgesia occurs and further coagulations are made at the same temperature until some analgesia is produced in the required division. At this stage, the time for each coagulation can be increased or decreased; however, if the temperature is increased without first trying extra time, anesthesia will suddenly develop. Analgesia produced by this method tends to increase over the first 2 hours.

Sequential throbbing of the cannula may occasionally be observed during the early seconds of the lesion. This is due to the fact that in conventional RF, current is emitted every 0.66 seconds, but every 20 milliseconds in pulsatile RF.

To prevent hematoma in the cheek, ice compression after the needle is withdrawn should be done in every instance. Hemifacial numbness that develops after chemical neurolysis or extensive RF lesioning, especially if three branches of the trigeminal nerve are involved, is a distressing experience for patients.

Weakness of the homolateral masseter muscle may occur during the postoperative period.

Because of the subsequent analgesia of the conjunctiva, the eye must be protected from chronic inflammatory processes that would go undetected because of the altered sensation. Therefore, it is usually necessary to approximate the upper and lower eyelids surgically to reduce the area of conjunctiva exposed to dust and other environmental sources of contamination. Protective spectacles with side shields can also help reduce the introduction of foreign bodies into the numb eye.

Another difficulty with long-term hemifacial analgesia is saliva dribbling from the anesthetized half of the mouth; this can sometimes be alleviated by an antisialagogue such as diphenhydramine, 25 mg tid.

EFFICACY

The three most popular techniques are RF rhizotomy, retrogasserian glycerol injection, and percutaneous compression of the gasserian ganglion. All the techniques have several advantages and several disadvantages. The advantages of RF lesioning are a high pain relief rate, a low relapse rate, and a high degree of effectiveness.

There is a light sensory deficit after retrogasserian glycerol injection. Shorter duration of pain relief, higher recurrence rates, and development of fibrosis at the foramen ovale are the main disadvantages. Slight sensory deficit and

moderate rate of recurrence may be the advantages of gasserian ganglion compression. However, it cannot be connected to a single branch, and the gauge of the needle entering the foramen ovale is larger than the ones used in other percutaneous methods, which may damage the nerve.

Technical Success

The technical success rate varies between 97.4 and 100% for RF lesioning at the initial phase. The success rate is 94% for glycerol and 99% for balloon compression. In another study, technical failure for glycerol was reported to be as high as 15%.[14,19] However, there is no general agreement on these results.

Pain Recurrence

Evaluating pain recurrence is not easy because of the heterogeneity of the follow-up reported. The highest rate of recurrence is 54% for glycerol rhizotomy, with a mean follow-up of 4 years.[15] In several series, this result varied. Retrogasserian glycerol injection is also an effective method, but the initial pain relief and duration of pain relief are less than RF lesioning. It may easily be applied when RF facilities are absent. Partial sensorial loss may also develop with this technique. Fibrosis may develop at the entrance of foramen ovale, enhancing further injections.

Percutaneous balloon compression causes mild sensory loss in most cases. However, it is not possible to restrict compression to a single division. It is not used as commonly as other techniques.

All these techniques are less morbid and more cost effective than open surgical techniques. However, each technique must be applied in precise indications and in well-equipped centers with experienced personnel.

Pulsed RF is not useful in the treatment of V cranial nerve neuralgia and could only be indicated in postherpetic V par neuralgia, together with other pharmacological therapies and in the painful sequelae of "anesthesia dolorosa" in the V cranial nerve territory by conventional RF, with variable results.

Comparison of Techniques

Apfelbaum compared 20 years of data on 702 patients who had microvascular decompression (MVD), percutaneous neurolytic procedures (PTN), radiofrequency lesioning (RFL), or glycerol.[28] MVD initially produced 91% excellent results, 6% good results, and failed in 3%. On long-term follow-up, 66% were excellent and 15% good for an 81% success rate. PTN using RFL initially produced 87% excellent results, 6% good, and failed in 7%, while glycerol produced 83% excellent and 9% good results with 8% failures. Thus, both achieved 92–93% initial success. In long-term follow-up, RFL had 71% excellent and 10% good for an 81% success rate. Glycerol had 52% excellent and 12% good results for a 64% long-term success rate. The average time for recurrence with either procedure was 18–19 months.

In conclusion, the initial success rate with all three approaches is similar—91–93%. The long-term success rate for RFL and MVD are also equal (81%), while glycerol has a 64% success rate, indicating more frequent recurrences. Complications with MVD could be serious or even life threatening (1%), such as cerebellar hemorrhage or edema. A number of transient cranial nerve deficits were also seen with a 2% chance of permanent ipsilateral hearing loss. These complications were not seen with PTN, but meningitis and intracerebral hemorrhage occurred in rare cases. Being destructive, PTN procedures intentionally reduced fifth nerve function. RFL was associated with annoying dysesthesia in 22%, anesthesia dolorosa in 2%, and corneal anesthesia in 1.2% of the patients. Glycerol produced only 2–4% annoying dysesthesia and 0.3% anesthesia dolorosa. Both procedures are effective ways to treat trigeminal neuralgia.[28] MVD is recommended for younger, better-risk patients, and PTN for patients who are medically infirm or older (over age 65).

CONCLUSION

Procedures involving the trigeminal ganglion and its branches are occasionally carried out to facilitate acute facial pain relief during surgery. However, much more frequently the indications are chronic, debilitating painful conditions. Clearly, the use of fluoroscopy and additional training led to better outcome and reduction of potentially devastating complications. All three percutaneous techniques may be used to block the trigeminal nerve in the treatment of neuralgic pain of the face. There are advantages and disadvantages of each of the techniques.

MAXILLARY NERVE BLOCK

HISTORY

There are four different approaches to the maxillary nerve. Of these approaches, an oral approach is commonly used by dentists. An orbital approach described originally by Rudolph Matas involves inserting a needle through the orbital cavity and exiting the infraorbital fissure.[29] Schlosser[30] in 1907 described an anterolateral approach with skin entry anterior to the coronoid process of the mandible and inferior to the zygomatic arch. The more commonly used lateral approach described by Levy and Baudoin[31] in 1906 is described and preferred by the authors.

ANATOMY

The maxillary nerve is the second division of the trigeminal nerve and is also known as the V2 division of the trigeminal ganglion. The maxillary nerve is a purely sensory nerve that begins at the gasserian ganglion and travels anteriorly and inferiorly along the cavernous sinus through the foramen rotundum (Figure 6-18).[32] It extends to the

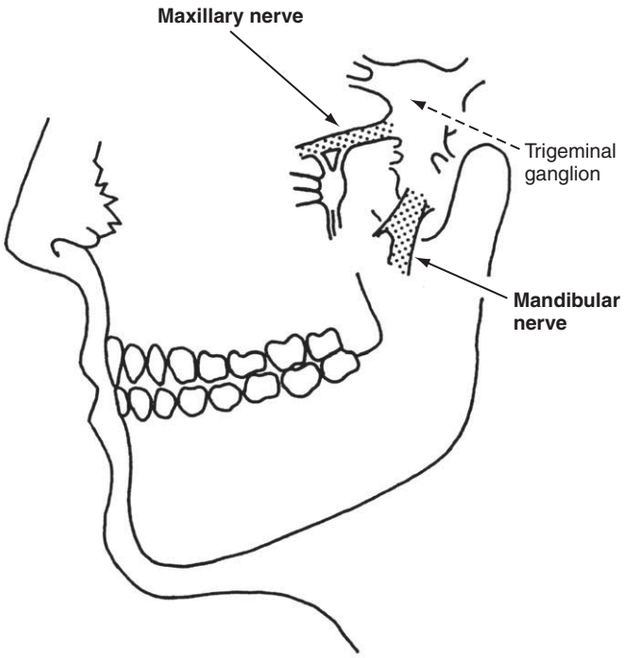


FIGURE 6–18
Maxillary nerve anatomy.

superior aspect of the pterygopalatine fossa along the inferior portion of the orbit in the infraorbital fissure and exits through the infraorbital foramen in the face.

The nerve innervates the maxillary sinus, as well as the anterior teeth of the upper jaw via the anterior and middle superior alveolar nerves. The branch that leaves the infraorbital foramen innervates the skin of the face, the underlying mucosa from the lower eyelid to the upper lip. While the nerve is at the pterygopalatine fossa, it is connected to the pterygopalatine ganglion, through which it gives the branches to the nasal cavity, pharynx, and palate. The zygomatic branch supplies the lateral portion of the face and posterior superior alveolar branch supplies the upper molar region.

The branches of the maxillary nerve are divided into four regional groups: (1) the intracranial group, including the middle meningeal nerve, which innervates the dura mater of the medial cranial fossa; (2) the pterygopalatine group including zygomatic nerve, which provides sensory innervation to the temporal and lateral zygomatic region, and sphenopalatine branches to innervate the mucosa of the maxillary sinus, upper gums, upper molars, and mucous membranes of the cheek; (3) the infraorbital canal group, comprising the anterosuperior alveolar branch innervating the incisors and canines, the anterior wall of the maxillary antrum, the floor of the nasal cavity, and the middle superior branch, supplying the premolars; and (4) the infraorbital facial group, consisting of the inferior palpebral branch, which innervates the conjunctiva and the skin of the lower eyelid, the external nasal branch, which supplies the side of the nose, and the superior labial branch, which supplies the skin of the upper lip and part of oral mucosa.

The 10 branches of the maxillary nerve supply sensation to the dura, upper jaw, teeth, gums, hard and soft palates, and cheek, as well as carry parasympathetic fibers. The maxillary artery and five terminal branches are also contained within the pterygopalatine fossa. Also within this space are emissary veins from the orbit.

The main part of the maxillary nerve, which constitutes the second division of the trigeminal nerve, can be anesthetized in the pterygopalatine fossa. Its branches can be anesthetized at the posterior and lateral borders of the maxilla, and its terminal branch can be anesthetized as it emerges through the infraorbital foramen on the front of the face 1 cm below the orbital margin in the same vertical plane as the pupil (Figure 6-19).

INDICATIONS

The maxillary nerve block is usually performed for regional analgesia of the upper jaw and can be used for acute intraoperative pain during maxillofacial surgery. It can also be used for surgical procedures on the teeth of the upper jaw. It provides excellent postoperative pain relief for such surgical maneuvers, and it is also used to treat chronic pain, most frequently for diagnostic and therapeutic blocks involving painful tumors of the maxillary antrum that are unresponsive to more conventional methods.

CONTRAINDICATIONS

- *Absolute*
- Local infection
- Coagulopathies
- *Relative*
- Altered anatomy

EQUIPMENT

Nerve Block

- 25-gauge, 3/4-inch needle
- 22-gauge, 3-1/4-inch spinal needle
- 3-ml syringe
- 5-ml syringe
- IV T-piece extension

Neurolytic Block/Pulsed Radiofrequency

- 16-gauge, 1-1/2-inch angiocatheter
- 10-cm curved radiofrequency thermocoagulation needle (RFK) with 5-mm active tip

DRUGS

Local Anesthetic Block

- 1.5% lidocaine for skin infiltration
- 2% lidocaine

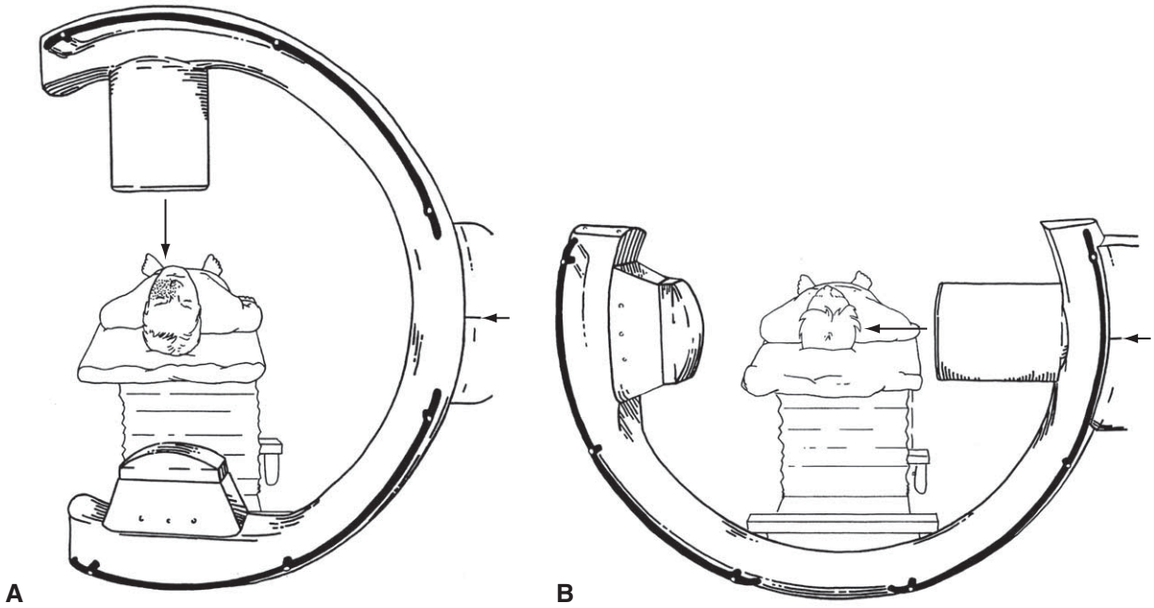


FIGURE 6–19
The patient is supine with the C-arm positioned for the AP view (A) and lateral view (B).

- 0.5% bupivacaine/ropivacaine
- Steroids (optional)

Neurolytics

- 6% phenol with or without contrast agent
- 40–50% glycerol with or without contrast agent

PREPARATION OF PATIENT

For preoperative medication, use the standard recommendations for conscious sedation by the American Society of Anesthesiologists.

PROCEDURE

The patient is placed supine with the head straight (Figure 6-19). Landmarks are assessed as follows:

1. Midpoint of the zygomatic arch of the temporal bone
2. Condyle of the mandibular head
3. Coronoid process of the mandible
4. Mandibular notch between the condyle and coronoid process

Extraoral Approach

The mandibular notch is identified, which is most easily done by having the patient open and close the mouth. A 22-gauge, 3-1/4-inch needle is then placed perpendicular to the skin at the posterior and inferior aspects of the notch, which should be close to the middle of the zygoma. The needle is advanced until it encounters the lateral pterygoid plate (4–5 cm). The needle is then withdrawn and redirected anteriorly and superiorly at about a 45-degree angle toward the upper root of the nose (Figure 6-20). The needle is again advanced with the pterygopalatine fossa until a paresthesia is obtained. It is important to obtain a paresthesia, otherwise the block will have a high rate of failure (Figure 6-21).

Three to 5 ml of local anesthetic is injected, although some authors advocate the use of as much as 10 ml. Neurolytic procedures can be done with 6% phenol or absolute alcohol. A maximum volume of 1–1.5 ml delivered in 0.1-ml divided doses is recommended.

Pulsed radiofrequency. Placement of the radiofrequency (RF) needle is the same as described previously (Figure 6-22). Confirmation of proper needle placement is with sensory stimulation (50 Hz, 0.3–0.6 V) and motor stimulation (2 Hz, 0.6–-1.2 V) . Once satisfactory placement is obtained, pulsed radiofrequency for 120–-180 seconds at 42°C for two cycles is performed. A local anesthetic does not need to be injected prior to removal of the needle.

Intraoral Approach

Three technique variations when performing intraoral maxillary block follow:

1. A retractor or left index finger retracts the cheek at the angle of the mouth upward and backwards until the first upper molar tooth is seen. The needle is introduced through the mucosa over the tooth and advanced backward,

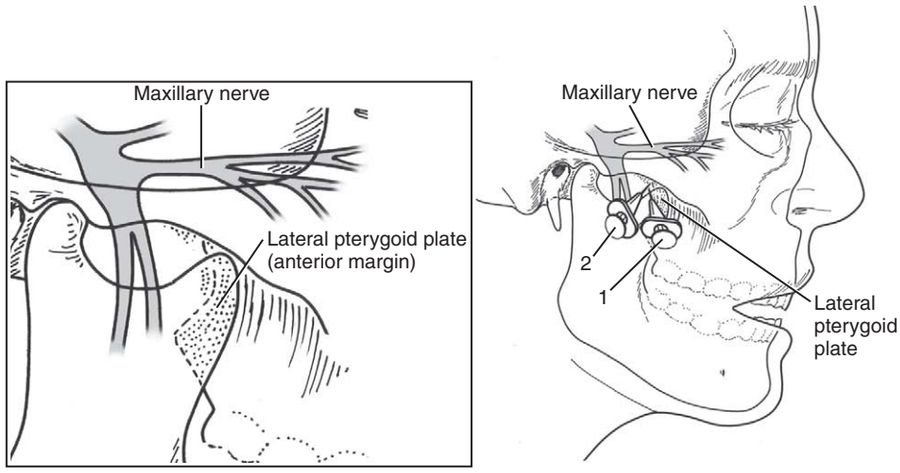


FIGURE 6–20

Maxillary nerve block in the lateral view. Initial needle direction (1) and redirection (2) after it encounters the pterygoid plate are shown. Inset shows detailed anatomy. (From Raj PP, editor: *Practical Management of Pain*, 3rd ed. St. Louis, Mosby, 2000, figure 41-8, p. 586, with permission.)

inward, and upward, making a 40-degree angle with the sagittal plane of the skull passing tangential to the maxillary tuberosity. When the contact with the bone is lost at a depth of 3–4 cm from the point of entrance, the needle is then advanced 0.5 cm more and 2 ml of 1% lidocaine is injected.

2. 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 maxilla and the pterygoid process at a depth of 3.5–4 cm and reaches the sphenomaxillary fossa; 2 ml of 1% lidocaine is injected after aspiration test.
3. Posterior palatinal approach: The same technique by the pterygomaxillary route is employed through the posterior palatinal foramen into the canal until the needle tip reaches the sphenomaxillary fossa, and 2 ml of 1% lidocaine is administered.

Infraorbital Block

The infraorbital nerve is the terminal branch of the maxillary nerve. In some cases with trigeminal neuralgia, in spite of radiofrequency lesioning or other percutaneous techniques of the gasserian ganglion, the pain in the area of innervation of the infraorbital nerve continues and infraorbital block may be useful at that instance.

The infraorbital foramen is situated 0.5–1 cm below the lower margin of the orbit, at the uppermost part of the canina fossa. The infraorbital canal is directed 45 degrees backward and upward and 20–25 degrees outward and varies from 1 to 1.5 cm in length.

The needle is introduced through a point on the cheek 0.5–1 cm lateral to the midportion of the ala of the nose. As soon as there is contact with 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 more than 1 cm and only a small amount of glycerol, 0.2–0.3 ml, may be given to the area. If a larger volume is used, there is the risk of compression neuropathy. Pulsed RF may also be applied.

Complications

In the extraoral approach, it is essential that the needle be introduced in a horizontal fashion, and it certainly should not enter the pterygomaxillary fissure in a cephalad direction or advance too deeply, because anesthetic injections here are rapidly spread to the posterior aspect of the orbit and the optic nerve, producing temporary blindness with reversible agents or, more seriously, permanent blindness with neurolytic agents. Because of the exceedingly vascular nature of the compartment in which the maxillary nerve lies (the pterygomaxillary fissure is a veritable network of small vessels), intravascular injection is quite possible, and meticulous aspiration tests are essential. Hematoma may develop. If the direction of the needle is too backwards, penetration to the pharnyx is possible. If this happens, air can be aspirated in the syringe.

Toxic reaction to local anesthetics may also develop.

Inadvertent puncture of the dura is possible if the needle is advanced too deep. During aspiration CSF may come. In such cases, the block should immediately be ceased.

Careful aspiration can help prevent vascular and subarachnoid injection. The close proximity of the orbit to this nerve makes it likely to be involved in a complication. Orbital swelling, anesthesia of the orbital tissues, ophthalmoplegia, loss of visual acuity, or diplopia can occur if the local anesthetic or neurolytic solution enters the infraorbital fissure. Damage to vascular structures can cause hemorrhage into the orbit, and blindness can occur.

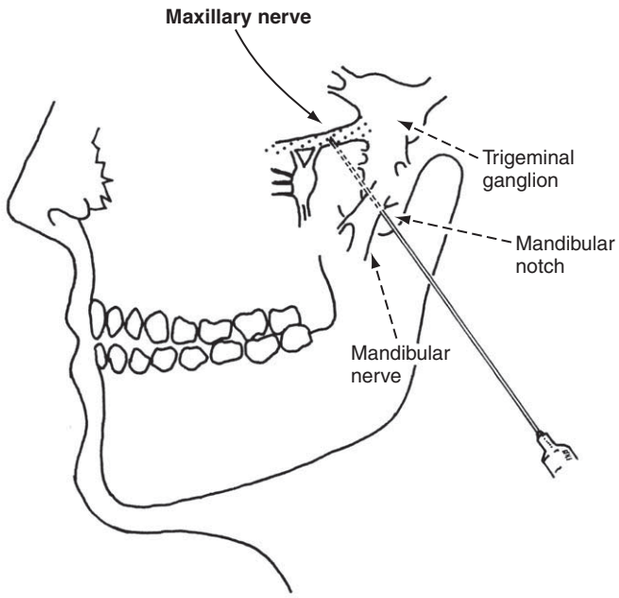


FIGURE 6–21

A patient with the needle on the maxillary nerve entering through the mandibular notch.

CLINICAL PEARLS

Because the maxillary nerve injection site is quite vascular, hematoma formation is common. An intravascular injection can also occur despite negative aspiration if the maxillary or mandibular artery or vein is injured during the performance of the block. Aspiration of air usually indicates that the needle has been placed too far posteriorly and the pharynx has been entered. If this occurs, it is prudent to change the needle before proceeding.

Seeking paresthesia is important for the precise localization of the needle. However, when the tip of the needle contacts with the lateral pterygoid lamina, the patient perceives this as a paresthesia. The paresthesia should be felt in the whole area; where the nerve innervates, the pain of the periosteum is more localized.

EFFICACY

On an individual patient basis, maxillary nerve block has been helpful in managing upper and midfacial pain, but no reliable data can be found for efficacy and prolonged relief.

MANDIBULAR NERVE BLOCK

HISTORY

There is no specific history on who first described the mandibular nerve block. It followed soon after the first description of the trigeminal ganglion block was described.

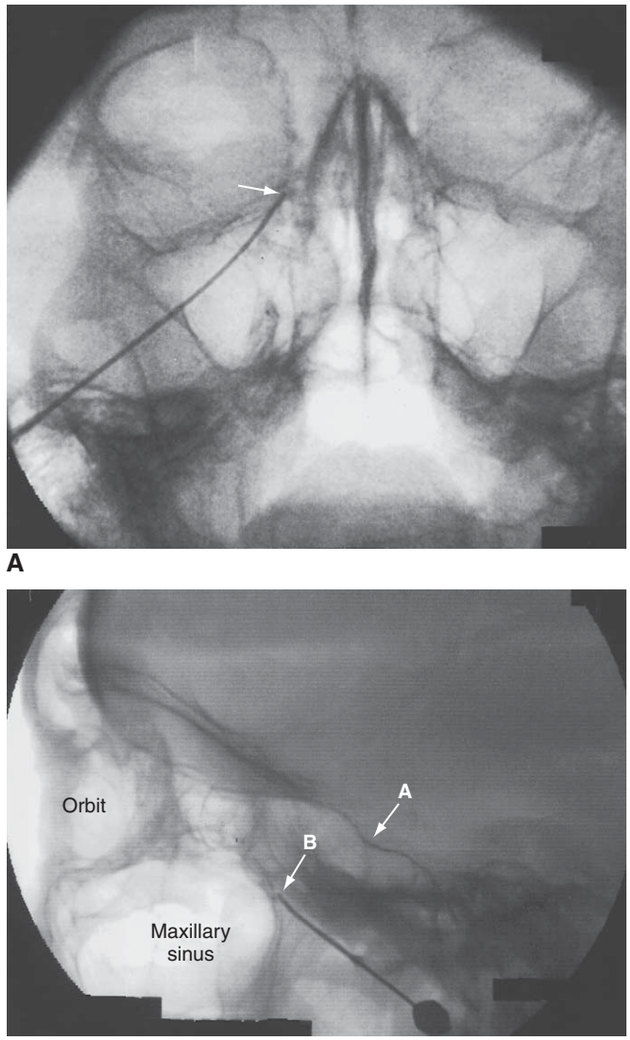


FIGURE 6–22

(A) The needle is in the pterygopalatine fossa (arrow in A–P view). (B) Confirmation of the needle in the lateral view. Arrow (A) shows the base of the skull. Arrow (B) shows the needle in pterygopalative.

ANATOMY

The mandibular nerve is the third largest nerve. It is the only mixed division of the trigeminal ganglion, being formed by the union of a large sensory root and a small motor root (Figure 6-23). The sensory fibers arise from the anterolateral portion of the gasserian ganglion, whereas the motor fibers are the same motor nerve mentioned in connection with the trigeminal ganglion, which arises from the pons and passes beneath the gasserian ganglion to reach the foramen ovale, through which, together with the sensory root, it leaves the cranial cavity. Within or immediately outside the foramen ovale, the two roots fuse into a single trunk. The formed nerve traverses anteriorly and inferiorly deep in the infratemporal fossa just anterior to the middle meningeal artery; lateral to the otic ganglion and internal

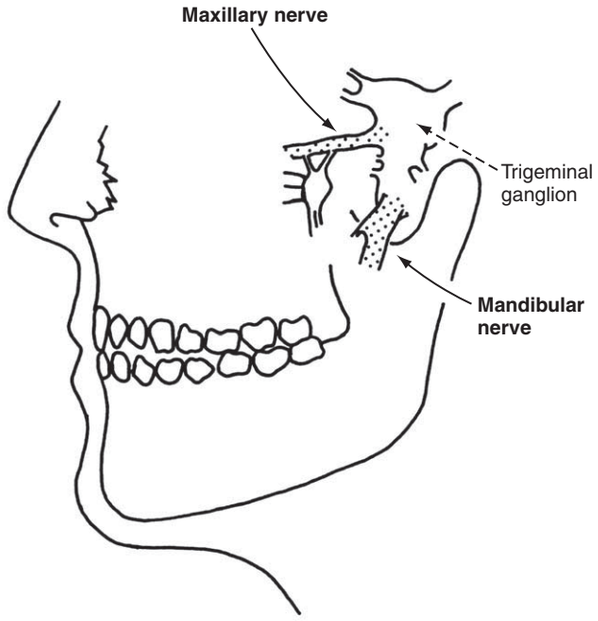


FIGURE 6–23
The drawing shows the anatomic location of the trigeminal ganglion and its mandibular and maxillary branches (lateral view).

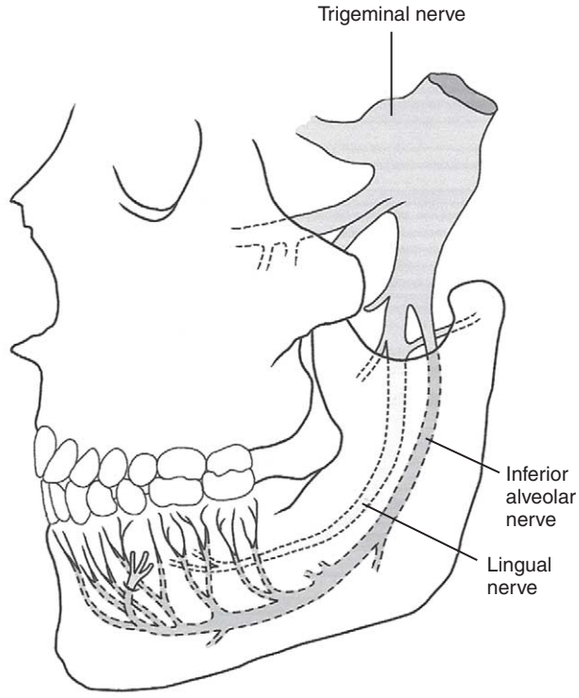


FIGURE 6–24
This line drawing shows the course of lingual and inferior alveolar nerve.

pterygoid muscle; and medial to the external pterygoid, the masseter and the temporal muscles, and the ramus of the mandible.

Soon after it is formed, the mandibular nerve gives off two small branches: the nervus spinosus, which enters the cranial cavity with the middle meningeal artery to supply the dura, and the nerve to the internal pterygoid muscle. It then divides into a small anterior and large posterior trunk. The small anterior trunk, which is composed mostly of motor fibers, then promptly divides into the masseteric, the anterior and posterior deep temporal, and the external pterygoid nerves that supply the muscles of mastication and also give off a small sensory branch, the buccinator, which supplies the mucous membrane and skin over this muscle. The large posterior trunk, on the other hand, is composed mostly of sensory fibers. After a short course it also divides into the auriculotemporal, the lingual, and inferior alveolar nerves. The auriculotemporal nerve arises from the posterior aspect of this trunk and immediately runs posterolaterally beneath the external pterygoid muscle to reach the medial side of the neck of the mandible, where it turns sharply cephalad to ascend between the anterior border of the auricle and the condyle of the mandible under cover of the parotid gland, finally reaching the subcutaneous tissue overlying the zygomatic arch, where it divides into the anterior auricular, the external meatal, articular, parotid, and superficial temporal branches. The lingual and inferior alveolar nerves proceed in an inferolateral direction to reach the medial side of the ramus of the mandible and to be distributed to the anterior two thirds of the tongue and inferior jaw, respectively (Figure 6-24).

The terminal branch of the inferior alveolar nerve is the mental nerve, which exits the mandible via the mental foramen and provides sensory innervation to the chin and to the skin and mucous membrane of the lower lip.

INDICATIONS

The mandibular nerve block is excellent for intraoperative or postoperative pain control after surgical reduction of a fractured mandible. It is also useful for chronic pain states, such as carcinoma of the tongue, lower jaw, or floor of the mouth.

CONTRAINDICATIONS

- *Absolute*
- Local infection
- Coagulopathies
- *Relative*
- Distorted anatomy

EQUIPMENT

Local Nerve Block

- 22-gauge, 3-1/2-inch spinal needle
- 25-gauge, 3/4-inch infiltration needle
- 3-ml syringe

- 5-ml syringe
- IV T-piece extension

Pulsed Radiofrequency

- 10-cm Racz-Finch radiofrequency thermocoagulation needle (RTK)
- 5-cm RFTC needle may be acceptable
- 16-gauge, 1-1/4-inch angiocatheter

DRUGS

Local Nerve Block

- 1.5% lidocaine for skin infiltration
- 0.5% bupivacaine/ropivacaine
- 2% lidocaine
- Steroids (optional)
- Iohexol (Omnipaque 240) contrast medium

Neurolytics

- 6% phenol
- Absolute alcohol
- 50% glycerol

PREPROCEDURE PREPARATION

Physical Examination

Examine for anatomic anomalies and local infections that may interfere with performance of the block. Also confirm that the jaw can be opened and closed.

Preoperative Medication

For preoperative medication, use the standard recommendations for conscious sedation by the American Society of Anesthesiologists.

PROCEDURE

Position of Patient

The patient is placed supine on the table. The C-arm is initially placed in an anteroposterior and lateral position to locate needle entry (Figure 6-25).

Extraoral Approach

The approach for blocking this nerve is identical to that for blocking the maxillary nerve, that is, the needle is introduced through the mandibular notch of the mandible, and advanced through the infratemporal fossa, with the lateral pterygoid plate serving as a bony endpoint (Figures 6-26, 6-27). However, in this instance, the needle is walked backward off the lateral pterygoid plate, maintaining the same depth as the plate until paresthesia

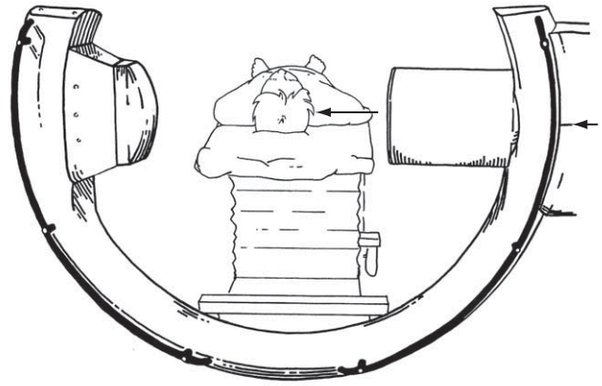


FIGURE 6–25
The position of the patient and C-arm for an external approach to a mandibular nerve block (lateral view).

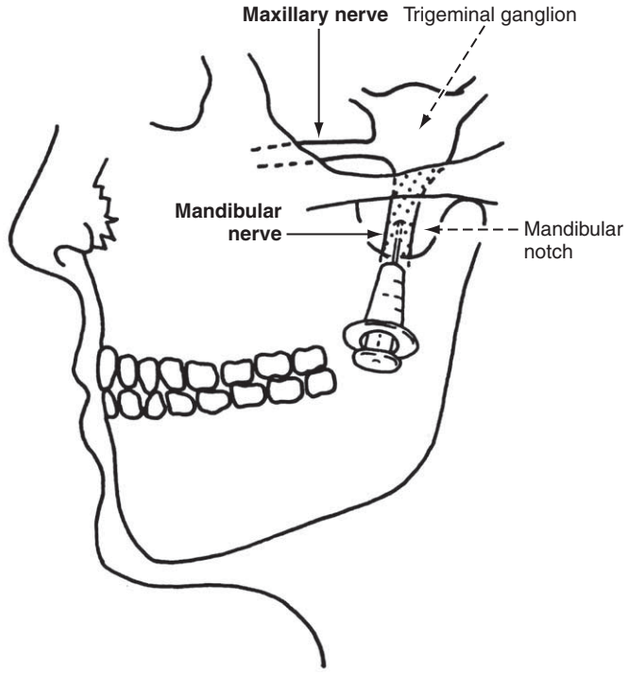


FIGURE 6–26
Point of needle entry in the mandibular notch for extraoral mandibular nerve block.

of the lower lip, lower jaw, or ipsilateral tongue or ear is obtained (Figure 6-28).

For best results, paresthesia should be elicited before 2 to 4 ml of anesthetic solution is injected.

Intraoral Approach

The cheek is retracted by the index finger or retractor until the second upper molar tooth is seen. A 5-inch needle is inserted into the mucous reflection above mucosa on the tooth, directed backward, upward, and inward

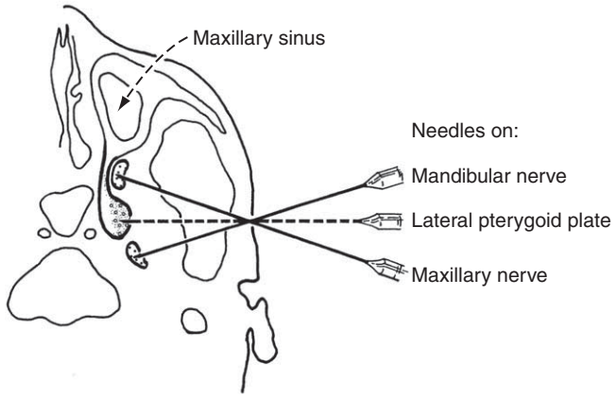


FIGURE 6–27

Transverse section of the head and face at the level of the mandibular notch showing needle placement on the mandibular nerve, on the lateral pterygoid plate, and on the maxillary nerve. After the pterygoid plate is touched, the needle is slightly withdrawn and pushed posterior until it slips off the pterygoid plate.

toward the infratemporal plate. The direction of the needle from lateral view should be toward the midpoint of the zygomatic arch and from the frontal view 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 ml of 1% lidocaine is injected slowly.

Mental Nerve Block

In some cases in spite of blocking the gasserian ganglion, the peripheric branches of the trigeminal nerve are blocked. The mental nerve is one of them. Also, in some cases with trigeminal neuralgia, only the mental nerve is affected and mental nerve block may be adequate.

Mental block by extraoral route (by Labat). A line is drawn from the two lower bicuspid teeth perpendicular to the lower margin of the mandible. The distance between the gingival margin of mandible and lower margin of the mandible is bisected. Through this bisecting point a line is drawn parallel to the lower margin of the mandible. These two lines cross each other at right angles and their intersection marks the position of the mental foramen. The quadrant in which the second bicuspid lies is bisected and a point is taken on the bisector. A 5-cm needle is introduced until it contacts the bone 1.5 cm from the point of intersection of the lines. The needle is then inclined slightly inward and passed through the foramen. In some cases the foramen can also be palpated.

The needle should not be introduced too deep in the foramen and the solution should be given in very few amounts in order to prevent compression over the nerve, which may cause neuropathy. Glycerol, 0.2–0.3 ml, may be injected or pulsed. RF may be applied (Figure 6-29).

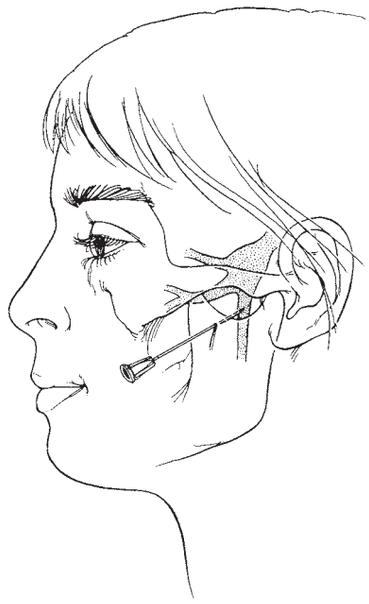


FIGURE 6–28

The technique for the extraoral block of the mandibular nerve is essentially the same as that for the maxillary block, except that the needle is directed upward and posteriorly; thus, the mandibular nerve is contacted as it exits from the foramen ovale. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, figure 20-17, p. 338, with permission.)

COMPLICATIONS

Mandibular nerve block, a relatively straightforward block, is associated with a high degree of success. However, there is always the risk of complications. As the needle is walked posteriorly off the lateral pterygoid plate, it comes to lie on the superior constrictor muscle of the pharynx, which is attached to the border of the lateral pterygoid plate. If the needle is advanced deeper at this stage, it can enter the pharynx. If the tip of the needle enters the pharynx, air bubbles will be seen during aspiration.

A very close posterolateral relation of the mandibular nerve at this site is the middle meningeal artery, which enters the cranial cavity through the spinous foramen, thus making meticulous aspiration tests necessary.

Hemorrhage in the cheek often occurs during and following the block by the anterolateral extraoral route. Hematoma of the face and subscleral hematoma of the eye may occur.

CLINICAL PEARLS

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 before it is reintroduced.

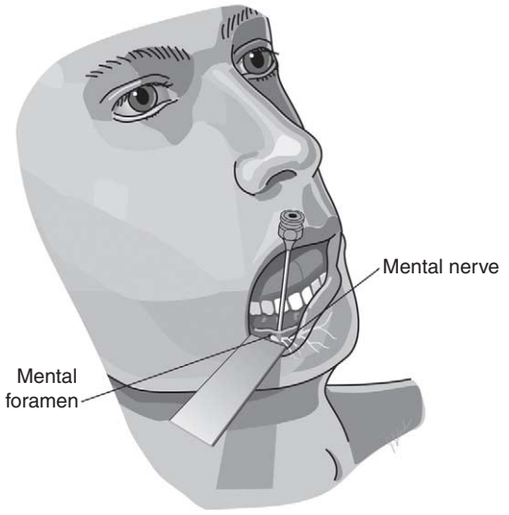


FIGURE 6–29

Injection technique for mental nerve block. (From Waldman SD: *Atlas of Interventional Pain Management*, 2nd ed. Philadelphia, Saunders, 2003, p. 54, with permission.)

EFFICACY

No efficacy studies are available. The efficacy is determined by the patient's successful pain relief after the nerve block.

GLOSSOPHARYNGEAL NERVE BLOCK

HISTORY

The early use of glossopharyngeal nerve block in pain management centered around two applications: (1) the treatment of glossopharyngeal neuralgia, and (2) the palliation of pain secondary to head and neck malignancies. In the late 1950s, the clinical use of the glossopharyngeal nerve block as an adjunct to awake endotracheal intubation was documented.

Weisenburg first described pain in the distribution of the glossopharyngeal nerve in a patient with a cerebellopontine angle tumor in 1910.[33] In 1921, Harris reported the first idiopathic case and coined the term *glossopharyngeal neuralgia*.[34] He suggested that blockade of the glossopharyngeal nerve might be useful in palliating this painful condition.

Early attempts at permanent treatment of glossopharyngeal neuralgia and cancer pain in the distribution of the glossopharyngeal nerve consisted principally of extracranial surgical section or alcohol neurolysis of the glossopharyngeal nerve.[35] These approaches met with limited success in the treatment of glossopharyngeal neuralgia, but were useful in some patients suffering from cancer pain mediated by the glossopharyngeal nerve. Intracranial section of the glossopharyngeal nerve was first performed by Adson in 1925 and was subsequently refined by Dandy. The intracranial approach to section of the glossopharyngeal nerve appeared to yield better results for both glossopharyngeal neuralgia and cancer pain but was a much riskier procedure.[36] Recently, interest in extracranial destruction of the glossopharyngeal nerve by glycerol or by creation of a radiofrequency lesion has been renewed.[37]

ANATOMY

The glossopharyngeal nerve is the ninth cranial nerve. It contains both motor and sensory fibers.[38] The motor fibers innervate the stylopharyngeus muscle. The sensory portion of the nerve innervates the posterior third of the tongue, the palatine tonsil, and the mucous membranes of the mouth and pharynx. Special visceral afferent sensory fibers transmit information from the taste buds of the posterior third of the tongue. Information from the carotid sinus and body, which help control the blood pressure, pulse, and respiration, are carried via the carotid sinus nerve, a branch of the glossopharyngeal nerve.[38] Parasympathetic fibers pass via the glossopharyngeal nerve to the otic ganglion. Postganglionic fibers from the ganglion carry secretory information to the parotid gland (Figure 6-30A).[39]

The glossopharyngeal nerve exits the jugular foramen near the vagus and accessory nerves and the internal jugular vein.[40] All three nerves lie in the groove between the internal jugular vein and internal carotid artery (Figure 6-30B).

A significant landmark for glossopharyngeal nerve block is the styloid process of the temporal bone. This structure is the calcification of the cephalad end of the stylohyoid ligament. Although usually easy to identify, when ossification is limited, it may be difficult to locate with the exploring needle.

INDICATIONS

Indications for glossopharyngeal nerve block are summarized in Table 6-3. In addition to application for surgical anesthesia, glossopharyngeal nerve block with local anesthetics can be used as a diagnostic tool when performing differential neural blockade in the evaluation of head and facial pain.[41] Glossopharyngeal nerve block is used to help differentiate geniculate ganglion neuralgia from glossopharyngeal neuralgia. If destruction of glossopharyngeal nerve is being considered, this technique is useful as an indicator of the extent of motor and sensory impairment that the patient will likely experience.[42] Glossopharyngeal nerve block with local anesthetic may be used to palliate acute pain emergencies, including glossopharyngeal neuralgia and cancer pain until pharmacologic, surgical, and

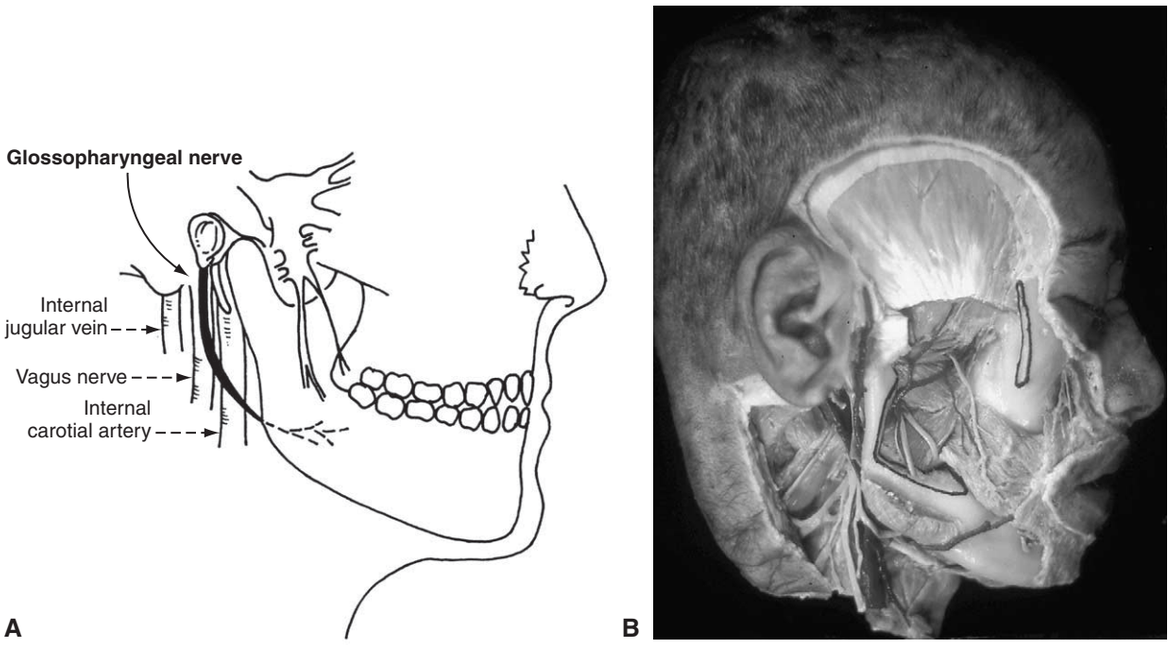


FIGURE 6–30

(A) The anatomy of the glossopharyngeal nerve as it exits the jugular foramen. Note the close relationship of the vagus nerve. (B) This is an anatomical dissection of the region where the glossopharyngeal nerve is traversing below the jugular foramen close to vagus, accessory nerves and internal carotid artery, and internal jugular vein. (Courtesy of U. Pai, MD.)

antiblastic methods take effect.[43] This technique is also useful for atypical facial pain in the distribution of the glossopharyngeal nerve[44] and as an adjunct for awake endotracheal intubation.[45]

Destruction of the glossopharyngeal nerve is indicated in the palliation of cancer pain, including invasive tumors of the posterior tongue, hypopharynx, and tonsils.[38] This technique is useful in the management of the pain of glossopharyngeal neuralgia for those patients who have failed to respond to medical management or who are not candidates for surgical microvascular decompression.[46]

CONTRAINDICATIONS

Contraindications to the blockade of the glossopharyngeal nerve are summarized in Table 6-4. Local infection and sepsis are absolute contraindications to all procedures. Coagulopathy is a strong contraindication to glossopharyngeal nerve block, but owing to the desperate nature of many patients' suffering from invasive head and face malignancies, ethical and humanitarian considerations dictate its use, despite the risk of bleeding.

When clinical indications are compelling, blockade of the glossopharyngeal nerve using a 25-gauge needle may be carried out in the presence of coagulopathy, albeit with increased risk of ecchymosis and hematoma formation.

EQUIPMENT

Local Nerve Block

- 25-gauge, 3/4-inch needle for infiltration
- 22-gauge, 1-1/2-inch needle for injection at the site
- 3-ml syringe
- IV T-piece extension

Pulsed Radiofrequency

- 16-gauge, 1-1/4-inch catheter
- 5-cm radiofrequency thermocoagulation (RFTC) needle with 5-mm active-tip Racz-Finch Kit Needle

TABLE 6–3 Indications for Glossopharyngeal Nerve Block

Local Anesthetic Block
Surgical anesthesia
Differential neural blockade
Prognostic nerve block prior to neurodestructive procedures
Acute pain emergencies (palliation)
Adjunct to awake intubation
Neurolytic Block or Neurodestructive Procedure
Cancer pain (palliation)
Management of glossopharyngeal neuralgia

TABLE 6-4 Contraindications to Glossopharyngeal Nerve Block

Local infection
Sepsis
Coagulopathy
Significant behavioral abnormalities
Anatomical anomaly

DRUGS

Local Nerve Block

- 1.5% lidocaine for skin infiltration
- 0.5% ropivacaine/bupivacaine mixture
- 2% lidocaine
- Steroids (optional)
- Iohexol (Omnipaque 240)

Neurolysis

- 6% phenol in glycerin/iohexol
- Absolute alcohol (97%)

PREPROCEDURE PREPARATION

Physical Examination

It is customary to obtain a full history and physical examination. The physical examination should include an assessment of the ability to move the neck and inspection for normal landmarks at the site of the needle insertion.

Preoperative Medication

For preoperative medication, use the standard recommendations for conscious sedation by the American Society of Anesthesiologists.

PROCEDURE

Position of Patient and Physician

The patient is placed in the supine position. The landmarks are (1) ipsilateral mastoid process; (2) angle of the mandible, anteriorly; and (3) feel the styloid process of the temporal bone, in the middle between the two landmarks. An imaginary line is visualized or drawn running from the mastoid process to the angle of the mandible.[47] The fluoroscope should be placed in an oblique position and directed toward the area of the mandible and the mastoid process (Figure 6-31). The styloid process should lie just below the midpoint of this line.

Extraoral Approach

The skin is prepared with antiseptic solution. After a local infiltration with a 25-gauge needle, a 22-gauge, 1.5-inch needle attached to a 3–5-ml syringe is advanced at this midpoint location in a plane perpendicular to the skin. The styloid process should be encountered within 3 cm. After contact is made, the needle is withdrawn and walked off the styloid process posteriorly. As soon as bony contact is lost and careful aspiration reveals no blood or CSF, 7 ml of 0.5% preservative-free lidocaine combined with 80 mg of methylprednisolone is injected in incremental doses.

Subsequently, daily nerve blocks are performed in the same manner, but 40 mg of methylprednisolone are substituted for the first 80-mg dose. This approach may also be used for breakthrough pain in patients who previously experienced adequate pain control with oral medications (Figures 6-32 and 6-33).[46]

Pulsed Radiofrequency

Informed consent and intravenous access were obtained. The patient was placed supine on the fluoroscopy table. Oxygen was administered by nasal cannula, and vital signs were monitored noninvasively. The right mastoid, lateral neck, and mandible were prepped and draped in a sterile fashion. A lateral fluoroscopic image was obtained. The styloid process, mastoid, and angle of the mandibular ramus were visible. An intracutaneous skin wheal with 1% lidocaine was raised at a point overlying the distal tip of the styloid process. A 16-gauge angiocatheter was placed about 1.5 cm through the skin, aiming for the styloid process. An anteroposterior view confirmed that the tip of the needle was at the level of the mandibular ramus. A 20-gauge blunt curved radiofrequency needle (RFK), 10 cm in length, 10-mm active tip is advanced through the angiocatheter until bony contact with the styloid process is made. The needle is then walked off posteriorly and advanced another 1–1.5 cm (Figure 6-34). Intermittent dual rotation C-arm fluoroscopy was used during needle advancement. Aspiration with a 1-cc syringe was negative for blood and CSF. One to 2 milliliters of Omnipaque 240 mg/dl, iodinated, nonionic contrast demonstrated local filling, inferior spread, and absence of vascular runoff on a lateral view (Figure 6-34). A line drawing of this fluoroscopic projection is displayed (Figure 6-35). Sensory stimulation up to 1 volt at 50 Hz reproduced concordant pain at the base of the tongue, pharynx, and tonsils. Motor stimulation up to 2.5 volts at 2 Hz reproduced local muscular contractions. Contractions of the muscles innervated by the phrenic and spinal accessory nerves were absent. The patient remained hemodynamically stable without any bradycardic or hypotensive episodes. Impedance was approximately 220 ohms but dropped to 113–140 ohms following instillation of 3 cc of a 1:1:1 mixture of lidocaine 2%, ropivacaine 0.2%, and 4 mg dexamethasone. Pulsed radiofrequency lesioning was performed for three cycles of 120 seconds at a constant temperature of 42°C. The rate was 2 Hz, and the pulse width was 20 milliseconds. The patient was monitored for 1 hour postprocedure, andvital signs remained stable.

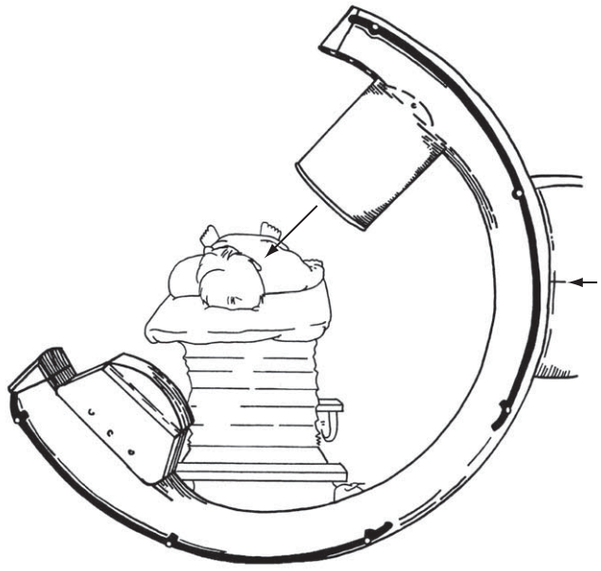


FIGURE 6–31
The C-arm is turned obliquely toward the mandible to visualize the styloid process to create a lateral radiographic image.

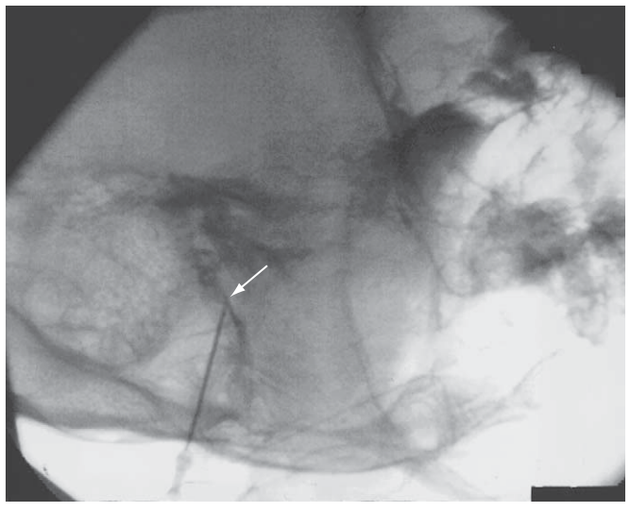


FIGURE 6–33
The lateral radiographic view shows the tip of the needle on the styloid process (arrow). This position ensures that the needle tip is close to the glossopharyngeal nerve.

EFFICACY

The patient's pain intensity reduced to 0/10, and this pain relief persisted for 8-1/2 months. Thereafter, her pain recurred and gabapentin 200 mg/day was started. This was not helpful and she went to the emergency room for intravenous analgesics on two occasions. Several analgesics were prescribed: zonisamide, hydrocodone 5 mg with acetaminophen 500 mg, sodium valproate, and nonsteroidal anti-inflammatory drugs. An outside physician started the patient on OxyContin 20 mg bid. The patient still had no relief. A repeat glossopharyngeal pulsed radiofrequency was performed, but this offered minimal relief for the first 2 weeks. Remarkably, there was a gradual improvement in pain and by the 6th week the patient was pain free. The patient was weaned off of all analgesics except gabapentin, and this pain relief lasted for 6 months. Pulsed radiofrequency lesioning was repeated, and the patient reported complete pain relief at 8 months. However, she had a syncopal episode during this period and required a pacemaker. In total, pulsed mode RF lesioning of the glossopharyngeal nerve was performed three times over a 24-month period.

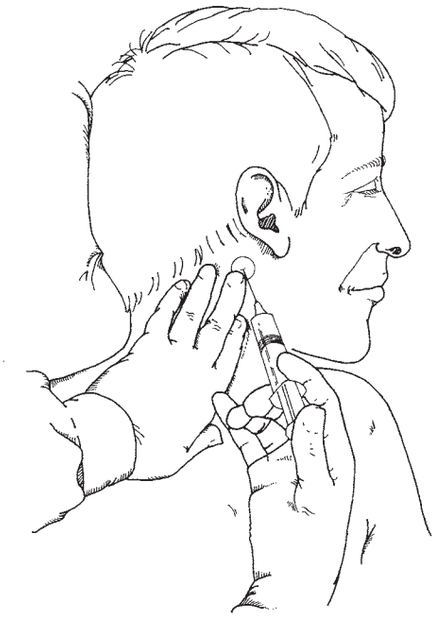


FIGURE 6–32
The site of entry for a glossopharyngeal nerve block is between the mastoid process and the angle of the mandible.

Treatments for glossopharyngeal neuralgia can be divided into surgical versus nonsurgical. Several classes of drugs are used empirically with anecdotal success: carbamazepine, phenytoin, diazepam, amitriptyline, phenobarbital, ketamine, and baclofen.[49,50] Nonetheless, intolerable side effects and difficulty with oral intake impede patient compliance. Surgical methods include peripheral neurectomy, rhizotomy, styloidectomy, microvascular decompression (MVD), and motor cortex stimulation.[49,51,52] Initially introduced by Jannetta,[53,54] MVD has been refined from a technical standpoint to reduce complication rates.[52] MVD continues to demonstrate the most successful and reproducible long-term outcomes.[52,53] Peripheral neurectomy and surgical rhizotomy have poorer outcomes and higher rates of recurrent pain and morbidity.[55] Several authors suggest that MVD alone should be performed for glossopharyngeal neuralgia,[53,55,56] but these series were limited to patients with primary glossopharyngeal neuralgia. MVD is not applicable to secondary glossopharyngeal neuralgia.

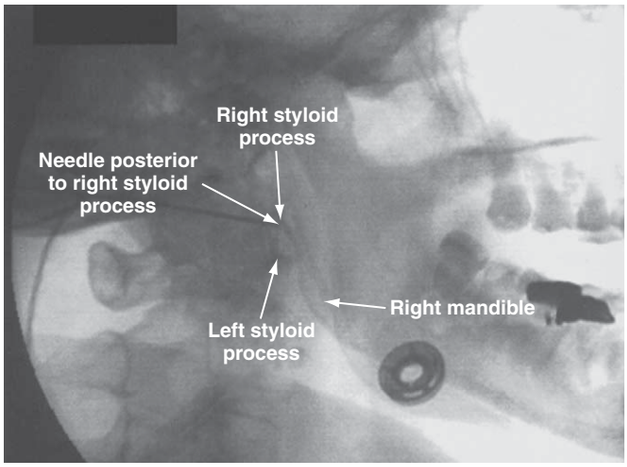


FIGURE 6–34

Lateral fluoroscopic image displaying needle tip posterior to right styloid process. (From Shah RV, Racz GB: Case conference: pulsed mode radiofrequency lesioning to treat chronic post-tonsillectomy pain. *Pain Pract* 3:233, 2003, with permission.)

Styloidectomy is recommended as a treatment for chronic post-tonsillectomy pain in the otolaryngology literature.[51] The role of styloidectomy, in the absence of peristyloid pathology or an elongated styloid process, is unclear.[57] Styloidectomy, moreover, is not a benign procedure. We are aware of one death due to iatrogenic vascular injury following styloidectomy. This case is undergoing litigation and is not reportable.

Percutaneous radiofrequency thermocoagulation of the glossopharyngeal nerve has been successful in treating primary and secondary glossopharyngeal neuralgia.[58–60] Percutaneous thermocoagulation of peripheral nerves, however, carries the risks of neuritis, deafferentation pain, and neurovascular injury.[61] Percutaneous thermocoagulation of the glossopharyngeal nerve, in particular, carries the hazard of damage to the vagus nerve.[58] Vagal nerve damage or stimulation can cause severe hemodynamic problems, such as syncope, asystole, or bradycardia.[49] Due to these concerns and the success of MVD, glossopharyngeal nerve RF has not gained widespread acceptance.[52,53,55] MVD is a surgical procedure requiring a craniectomy[53] that has a 5% risk of mortality in the most experienced centers.[39] These figures are sobering, since untreated primary glossopharyngeal neuralgia is typically a nonterminal illness.

Arias[58] modified the RF technique by using low-temperature lesioning. This avoids iatrogenic injury to the vagus nerve.[58] Pulsed RF is a newer, nondestructive neural lesioning method that provides relief of experimental and clinical neuropathic pain.[62,63] Short pulses of radiofrequency energy, delivered at a constant temperature, produce central and peripheral neuromodulatory effects.[62] The precise mechanisms of pain relief are unknown but may involve alterations in the expression of genes such as c-fos.[64]

Temperatures in pulsed RF, unlike conventional RF, typically do not exceed 42°C. Temperatures below 45°C do not irreversibly harm neural tissues.[64,65] The risks of neuritis, deafferentation pain, and neuroma formation are minimal with pulsed RF. Furthermore, even if identical temperatures are used, pulsed RF demonstrates better efficacy than conventional RF.[64] This implies that the electrical field rather than the heat lesion may be responsible

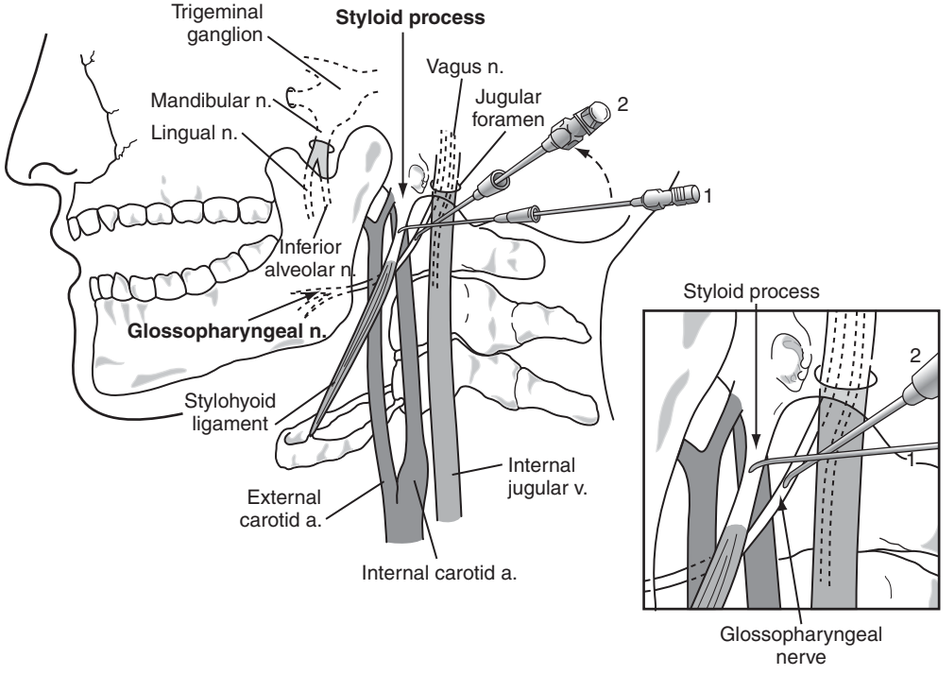


FIGURE 6–35

Drawing detailing relevant anatomic structures, initial needle position contacting styloid process (1), and final position at glossopharyngeal nerve (2). (Adapted from Shah RV, Racz GB: Case conference: pulsed mode radiofrequency lesioning to treat chronic post-tonsillectomy pain. *Pain Pract* 3:233, 2003, with permission.)

for the clinical effect of RF.[66] Pulsed RF may provide long-term pain relief, reduce analgesic consumption, and provide patient satisfaction.[66] Even when the pain recurs, the procedure is easily repeatable.

Technically, there are several percutaneous methods to target the glossopharyngeal nerve. An intraoral approach is often used for preemptive analgesia,[67] but this method caries the risk of infection and iatrogenic injury to several neurovascular structures, including internal carotid artery, vagus nerve, brainstem, vertebral artery, and upper cervical spinal nerves. Two extraoral approaches can be performed with fluoroscopic guidance. One approach, similar to that used for trigeminal ganglion blockade, uses Hartel's projection.[58] Instead of aiming for the foramen ovale, the operator aims for the medial part of the jugular foramen. This approach, however, can cause severe damage to the vital neurovascular structures mentioned earlier. The technique of Shah and Racz can be safely performed, especially when curved blunt needles, contrast fluoroscopy, preprocedure motor and sensory electrical stimulation, hemodynamic monitoring, and pulsed mode RF are used. These technical refinements may dispel concerns about this procedure's safety and permit its gradual re-introduction as a treatment for glossopharyngeal neuralgia. Larger studies are needed to further substantiate claims of safety and efficacy.

COMPLICATIONS

Inadvertent puncture of either vessel during glossopharyngeal nerve block can result in intravascular injection or hematoma formation. Even small amounts of local anesthetic injected into the carotid artery at this site can produce profound local anesthetic toxicity.[36]

Because extraoral blocks of the glossopharyngeal nerve can readily spread to the vagus and accessory nerves, neurolytic blocks often produce analgesia of the hemilarynx and/or trapezius muscle, and sternocleidomastoid paralysis on the ipsilateral side. Both these complications may be well tolerated by patients with terminal cancer pain.

The major complications associated with glossopharyngeal nerve block are related to trauma to the internal jugular vein and carotid artery.[38] Hematoma formation and intravascular injection of local anesthetic with subsequent toxicity are significant problems for the patient. Blockade of the motor portion of the glossopharyngeal nerve can result in dysphagia secondary to weakness of the stylopharyngeus muscle.[43] If the vagus nerve is inadvertently blocked, as it often is during glossopharyngeal nerve block, dysphonia secondary to paralysis of the ipsilateral vocal cord may occur. Reflex tachycardia secondary to vagal nerve block is also observed in some patients.[38] Inadvertent block of the hypoglossal and spinal accessory nerves during glossopharyngeal nerve block results in weakness of the tongue and trapezius muscle.[68]

A small percentage of patients who undergo chemical neurolysis or neurodestructive procedures of the glossopharyngeal nerve experience postprocedure dysesthesias in the area of the nerve.[69] These symptoms range from a mildly uncomfortable burning or pulling sensation to severe pain. Such severe postprocedure pain is called *anesthesia dolorosa*. Anesthesia dolorosa can be worse than the patient's original pain and is often harder to treat. Sloughing of skin and subcutaneous tissue has been associated with anesthesia dolorosa.

The glossopharyngeal nerve is susceptible to trauma from needle, hematoma, or compression during injection procedures. Such complications, although usually transitory, can be quite upsetting for the patient.

Even though risk of infection is uncommon, it is ever present, especially in patients with cancer who are immunocompromised.[44] Early detection of infection is crucial to avoid potentially life-threatening sequelae.

CLINICAL PEARLS

Patients with pharyngeal cancer will often have undergone radical neck dissection and the sternocleidomastoid muscle will have been removed. This makes identification of the styloid process much easier, since this particular bony landmark is now almost subcutaneous, allowing this block to be performed easily.

Because of the proximity of the large vascular conduits of the internal carotid artery and the internal jugular vein, the risks of intravascular injection are always significant, demanding meticulous aspiration tests. With the temporary and perhaps permanent analgesia produced by this block, a degree of incoordination of swallowing, with the accompanying potential risk of aspiration, must be appreciate d by patients and attendants alike. With numbness of half of the pharynx and the larynx, ingestion and swallowing are often severely compromised.

EFFICACY

No data are available to establish the efficacy of the block. Pain relief by the patient is a good indication of success and purely anecdotal.

GREATER AND LESSER OCCIPITAL NERVE BLOCKS

HISTORY

The term occipital neuralgia was first used in 1821, when Beruta y Lentijo and Ramos made reference to an occipital neuralgic syndrome.[70] The technique of occipital

nerve block seems to be first described by Bonica in 1953.[71]

ANATOMY

The greater occipital nerve gets fibers from the dorsal primary ramus of the second cervical nerve and to a lesser extent from the third cervical nerve. The lesser occipital nerve arises from the ventral primary rami of the second and third cervical nerves (Figure 6-36).

The greater occipital nerve ascends in the posterior neck over the dorsal surface of the rectus capitis posterior major muscle, at the midpoint of this muscle; turns dorsally to pierce the semispinalis capitis; and then runs a short distance rostrolaterally, lying deep to the trapezius. The nerve becomes superficial below the superior nuchal line, along with the occipital artery.

It supplies the medial portion of the posterior scalp as far anterior as the vertex. The lesser occipital nerve passes superiorly along the posterior border of the sternocleidomastoid muscle, innervating the lateral portion of the posterior scalp and the cranial surface of the pinna of the ear.[72–74]

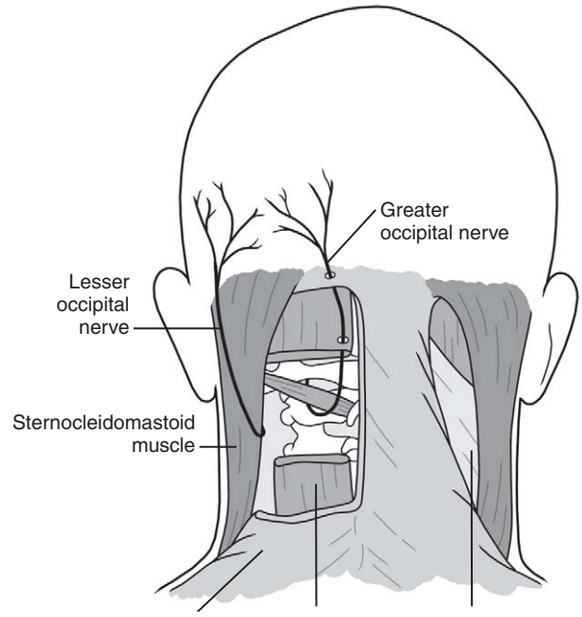


FIGURE 6–36

Anatomy and technique of injection of the greater occipital nerve. (A) The third occipital nerve, which is not shown, is usually located medial to the greater occipital nerve. The lesser occipital nerve (B) can be blocked at a point 2.5 cm lateral to the site of the injection for greater occipital nerve block.

INDICATIONS

- Diagnosis of occipital neuralgia
- Management of occipital neuralgia
- Treatment of cancer pain in region
- Headache associated with muscular tension or spasm
- Cervigonenic headache
- Anesthesia of posterior part of the scalp

CONTRAINDICATIONS

- Local infections
- Coagulopathies
- Metastasis in region
- Significant behavioral abnormalities

EQUIPMENT

Local Anesthetic Nerve Block

- 22-gauge, 1-1/2-inch needle
- 5 ml syringe

Radiofrequency Lesioning

- 25-gauge, 3/4-inch needle
- 16-mm or 14-mm catheter
- 3 ml syringe
- 5-cm radiofrequency thermocoagulation needle with 5-mm active tip

DRUGS

- 1.5% lidocaine for skin infiltration
- 2% lidocaine for nerve block
- Steroids (optional)

Neurolysis

- 2 ml 6% phenol in glycerine or in Omnipaque

PROCEDURE

With the patient seated and the head flexed slightly forward, both the greater and lesser occipital nerves can be blocked. Alternatively, the patient can lie prone with the head hyperflexed on a pillow.

There are three landmarks for locating the greater occipital nerve: (1) the occipital artery, (2) the mastoid process, and (3) the greater occipital protuberance. An imaginary line is passed through these landmarks, and the occipital artery is generally found at a point approximately one-third the distance from the occipital protuberance on the superior nuchal line. The lesser occipital nerve is found at a two-thirds distance from the occipital protuberance on the superior nuchal line (Figure 6-37).

The artery is palpated and a short (1–1/2-inch), 25-gauge needle is inserted through the skin at the level of the superior nuchal line. The nerve is often medial to the

artery at this level. However, the anatomy varies and it may also be lateral to the artery.

The needle is advanced until a paresthesia or bone is encountered and then withdrawn 2 mm. Local anesthetic solution of 2–5 ml is injected after negative aspiration. A paresthesia is not necessary for a successful block. If the artery is not identified, the medication is injected in a fan-like fashion (medially and laterally) and 5 ml of local anesthetic is injected.

The lesser occipital nerve is blocked by introducing the needle medial to the origin of the sternocleidomastoid muscle at the mastoid process. The needle is aimed in a cephalad and medial direction until it contacts the skull. The needle is withdrawn 2 mm and aspirated, after which approximately 3 ml of local anesthetic should be injected. To prolong effectivity, 80 mg of depot steroids may be added.

Neurolytic Block

The needle is advanced until a paresthesia is encountered. It will be better to seek for paresthesia with a stimulator. When the paresthesia is met, 1 ml 6% phenol in glycerine or Omnipaque is slowly injected after negative aspiration.

Pulsed Radiofrequency

A 5-cm radiofrequency needle with 5-mm active tip is advanced to make contact, through a previously introduced catheter, with the bone in the close vicinity of the nerve. Sensorial stimulation with a frequency of 50 Hz is the next step. The stimulation should be felt below 0.5 V. When the patient feels the paresthesia, 1 ml of 2% lidocaine is injected. Ten minutes later, pulsed RF at 42°C for two or three cycles of 120 seconds is performed (Figure 6-38).

COMPLICATIONS

1. Due to the high vascularity of the scalp, ecchymosis or hematoma formation can occur. This is usually transient.
2. Although very rare, intravascular injection of the local anesthetic can occur. A small volume (1–2 ml) of local anesthetic has the capability of developing CNS toxicity.
3. Nerve injury due to the direct trauma from the needle or compression of nerves with large volume of local anesthetic can occur.
4. If the needle is introduced too deeply when trying to achieve paresthesia, inadvertent placement of the needle into the foramen magnum can occur. Administration of local anesthetics in this situation can result in total spinal block and respiratory depression.

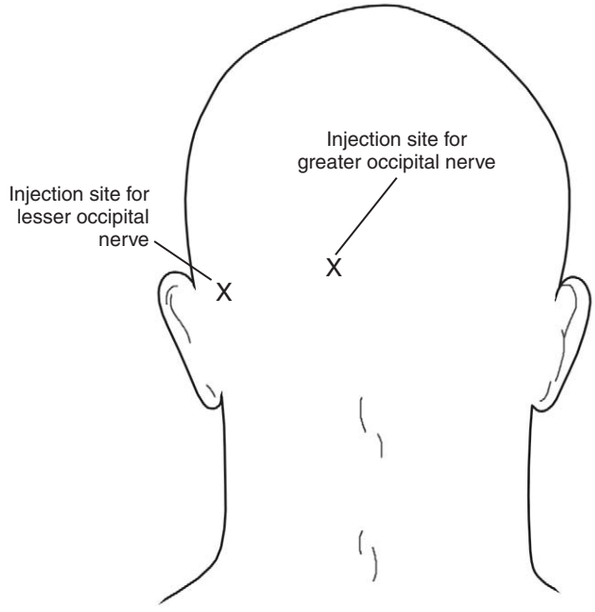


FIGURE 6–37
Anatomic landmarks for skin entry points of the greater occipital nerve (A) and lesser occipital nerve (B).

CLINICAL PEARLS

The principal role of occipital nerve block is for the diagnosis of occipital neuralgia. If a diagnostic block is planned, the dose should be limited to 1–2 ml to minimize confusion

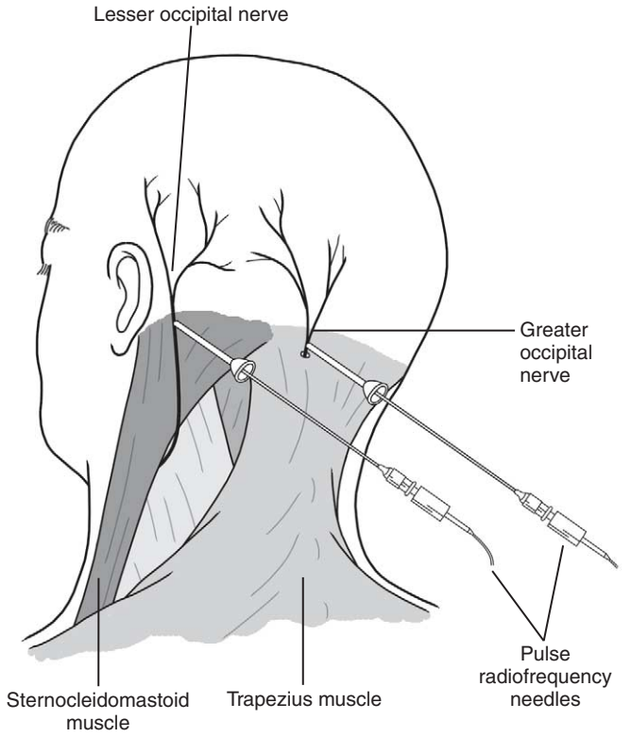


FIGURE 6–38
Technique of pulsed radiofrequency.

with relief of myofascial pain when larger volumes are injected. Failure to obtain successful block can be due to an anatomic variation.

The vascularity and the proximity to the arterial supply give rise to an increased incidence of postblock ecchymosis and hematoma formation. These complications can be decreased if manual pressure is applied to the area of the block immediately after the injection. Application of cold packs for 20-minute periods after the block will also decrease the amount of postprocedure pain and bleeding.

Strict care must be taken to avoid inadvertent needle placement into the foramen magnum, as the subarachnoid administration of local anesthetic in this region will result in an immediate total spinal block.

EFFICACY

No good data are available to evaluate the efficacy of the block. Success is anecdotal.

SUBOCCIPITAL COMPARTMENT DECOMPRESSION

HISTORY

In 1980, a 76-year-old woman complaining of severe occipital neuralgic type pain was evaluated. Upon examination there was tenderness at the C1-C2 occipital area. Following review of literature and anatomy, the entrapment of the greater occipital nerve in the suboccipital compartment was assumed. Ten milliliters of local anesthetic and steroid mixtures were injected from just below the nuchal line bilaterally, through the deep fascia trapezius and semispinalis muscle layers into the suboccipital compartment. The pain relief was very rapid and lasted 3 years, the rest of her life. The needle used was 1/2-inch, 22-gauge B bevel. The technique of the procedure, virtually unchanged, was repeated several thousand times in numerous cases in subsequent years.

In a 1994 presentation in Perth, Australia, Umberto Rossi, in a patient with the same condition, dissected down to the C1-C2 lamina to cut the inferior oblique muscle with prompt relief of the pain on recovery. He noted the greater occipital nerve to be flattened. He also observed that while the pain would stop, these patients would develop similar pain on the opposite side.

The suboccipital compartmental injection technique from the beginning has been a bilateral injection. In 2004, similar neurosurgical observations were made where the sectioning of the inferior oblique muscle was recommended.[75]

The longest follow-up observation following the injection technique has been of the mother of one of the authors, where one injection gave 13 years of pain relief from 1982 to 1995, at which point the severe pains returned and repeat injection needed to be carried out. The analgesic effect lasted the rest of her life.

From the clinical experience of many users, a pattern of problems has become evident. The injection technique coming from just below the nuchal line through the facial layers is clearly a very safe technique; however, if the tip of a sharp needle enters the greater occipital nerve, retrograde longitudinal spread may give rise to a "locked-in phenomenon" where the patient stops breathing and stares with dilated pupils. Airway ventilation must be initiated. One of the patients had this occurrence; after approximately 30 minutes of ventilation, the patient made a full and uneventful recovery. A similar event occurred in the practice of one of the trainees after 6–8 years of practice and many procedures with similar good outcomes. Intraneural injection–related problems have been reported.[76]

Eight additional cases have been reported in the medicolegal literature, where the worst complication was permanent brain damage because of absence of ventilator support. Several cases of infarction of the brain stem were reported in which glossopharyngeal nerve impairment and swallowing difficulty were the consequences as predicted by Seelander.[76] During the last 6-8 years, the technique changed to the use of fluoroscopy and a bullet-tipped, side-ported stealth needle (Epimed International) and injection of contrast to verify a lack of intraneural spread prior to the large-volume injection.

Lessons learned are that it is a bilateral disease and repeat injections are safe and effective. Younger patients need to be trained in physical therapy exercises and relaxation technique (Figures 6-39 and 6-40).

Cadaver studies confirm the suboccipital compartment and the spread of the injected contrast along the greater occipital nerve (Figures 6-41 and 6-42).

ANATOMY

The suboccipital triangle, not to be confused with the occipital triangle, is bounded by rectus capitus posterior major muscle laterally and above. The muscle originates on the spinous process of the axis and inserts on the lateral

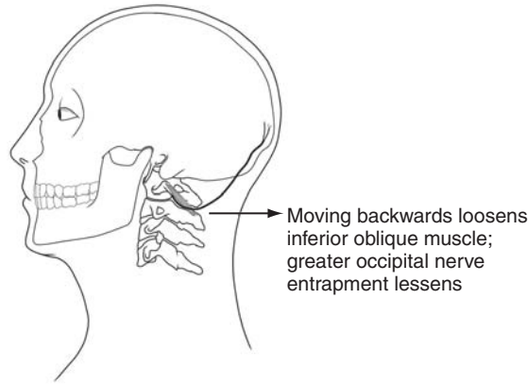


FIGURE 6–39

Moving backwards loosens inferior oblique muscle; greater occipital nerve entrapment lessens.

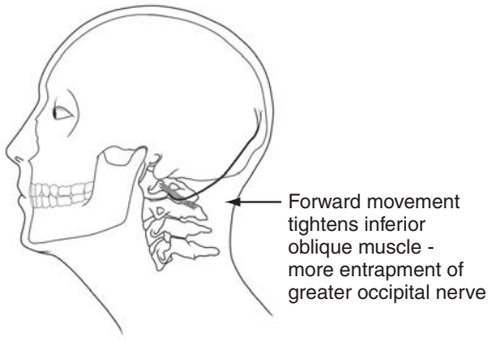


FIGURE 6–40

Forward movement tightens inferior oblique muscle. More entrapment of greater occipital nerve.

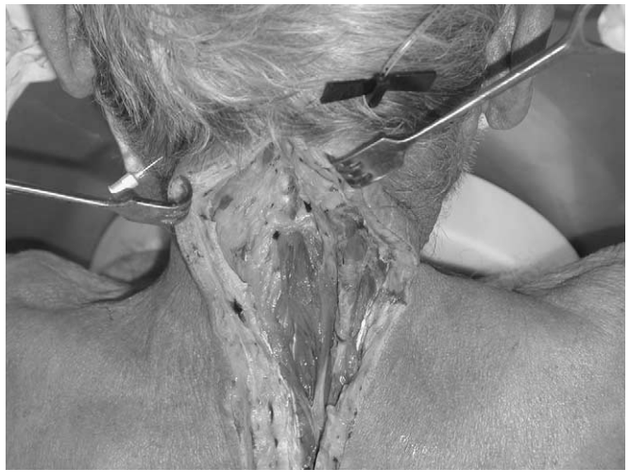


FIGURE 6–41

Stealth needle placement in a cadaver is shown on the right side, while conventional approach is shown on the left side.

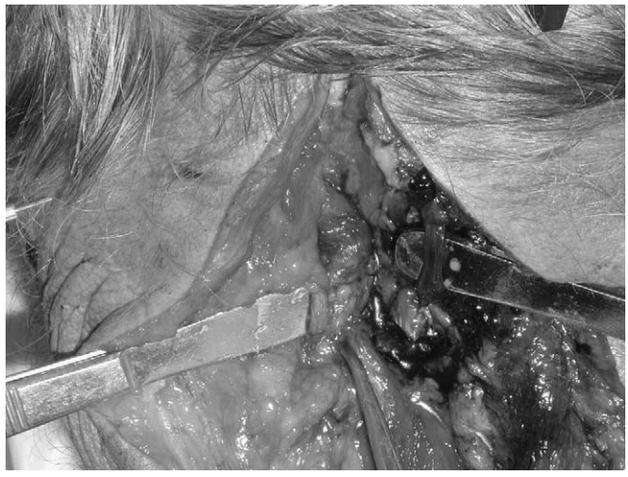


FIGURE 6–42

Methylene blue injected bilaterally shows suboccipital compartment spread on right. Occipital nerves are retracted.

aspect of the inferior nuchal line of the occiput. It rotates the skull ipsilaterally.

The obliquus capitus superior muscle is the lateral, upper border. It originates from the transverse process of the atlas and inserts on the occipital bone between the superior and inferior nuchal lines lateral to the semispinalis capitus. The obliquus capitus superior pulls the head backward to the ipsilateral side.

The obliquus capitus inferior is the lateral boundary below, and it originates from the spinous process of the axis and inserts on the transverse process of the atlas. It rotates the atlas and occiput. The roof of the space is a tough layer of connective tissue beneath the semispinalis capitus, and the floor is the occipito-atlantal membrane and posterior arch of the atlas.

The posterior branch of the first occipital nerve, the suboccipital nerve, exits posteriorly between the occiput and the posterior arch of the atlas. It supplies the muscles bounding the suboccipital triangle and communicates with the greater and lesser occipital nerves. Entrapment can occur at the obliquus capitus inferior (inferior oblique) or semi-spinalis or trapezius (Figures 6-43 and 6-44).

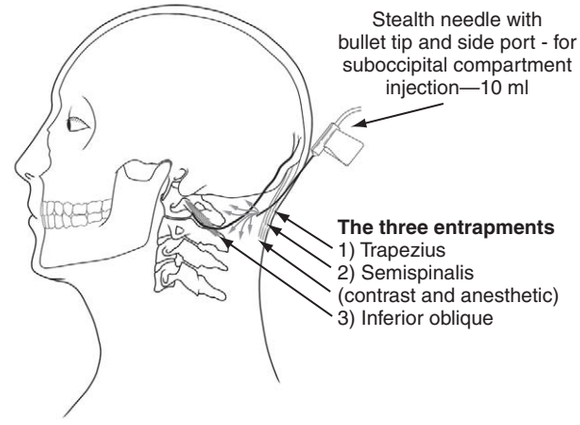


FIGURE 6–43

Lateral view suboccipital entrapment.

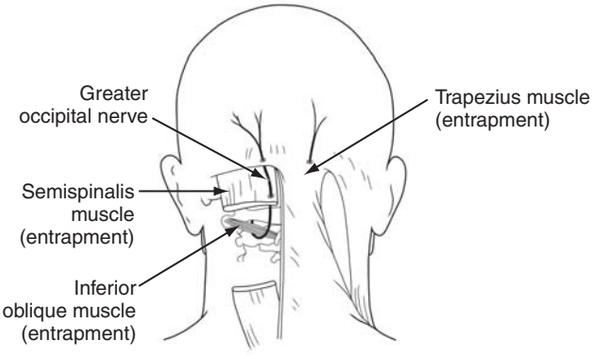


FIGURE 6–44

Posterior view suboccipital entrapment.

INDICATIONS

- Diagnostic therapeutic
- Suboccipital tenderness dorsolateral C1-C2 area
- Occipital frontal headache

CONTRAINDICATIONS

- Infection
- Previous local surgery unless contrast is used

EQUIPMENT

- Stealth needle
- Fluoroscopy
- Small-bore tubing
- Syringe

DRUGS

- Corticosteroid
- Omnipaque 240
- 0.2% ropivacaine

TECHNIQUE

The patient is positioned in the prone position with the neck in flexion. The nuchal line is palpated. Skin entry is made 1/2 inch paramedial. Aim and advance the stealth needle through fascial layers (Figure 6-45).

On the lateral fluoroscopic view, the direction should be toward the arch of C1. Inject contrast, which should spread around needle tip and not within the nerve. Ten milliliters of 0.2% ropivacaine and 20 mg of depomedrol are used for the block.

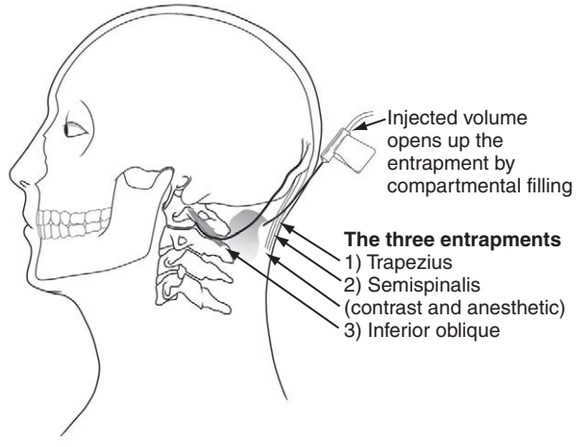


FIGURE 6–45
Stealth needle placement.

The suboccipital compartment opens and injectate spreads in the perineural space of greater occipital nerve (Figures 6-46 and 6-47).

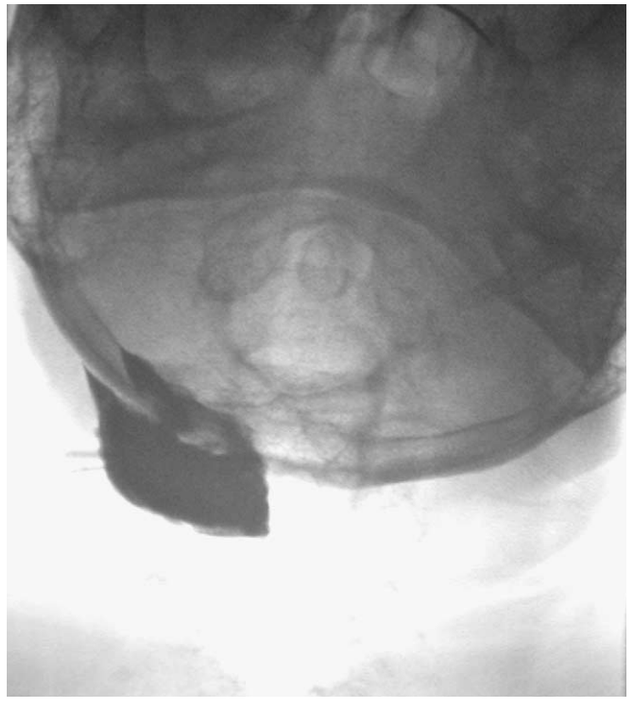


FIGURE 6–46
Anteroposterior view with contrast.

FIGURE 6–47
Lateral view with contrast.

COMPLICATIONS

All complications have followed the use of a sharp needle and intraneural injection leading to respiratory arrest and brain stem infarction in settings where the occurrence of these problems is not appreciated such as the office setting where the personnel are not trained or ready to administer airway support and ventilation. However, these complications are extremely rare and preventable.

EFFICACY

Reports of successful treatment with single injections have been described.[77,78] A randomized controlled trial of injections of local anesthetic with and without corticosteroid showed superior benefit of steroids in patients with cluster headache.[79]

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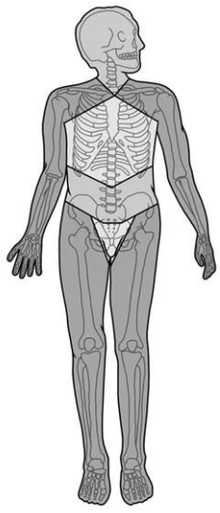
C H A P T E R



7

Sympathetic Blocks of the Head and Neck

SERDAR ERDINE

SPHENOPALATINE GANGLION BLOCK AND NEUROLYSIS

HISTORY

Sphenopalatine ganglion (SPG) involvement in the pathogenesis of pain has been understood since Sluder first described sphenopalatine neuralgia in 1908 and treated it with an Sphenopalatine ganglion block (SPGB).[1] Over the past century, physicians have performed SPGB for pain syndromes ranging from headache and facial pain to sciatica and dysmenorrhea.[1] In the medical literature on SPGB, large gaps—spanning decades—reflect physicians' varying interest in and skepticism about the efficacy of SPGB.

ANATOMY

The SPG is the largest group of neurons outside the cranial cavity (Figure 7-1). It lies in the pterygopalatine fossa, which is approximately 1 cm wide and 2 cm high, and resembles a "vase" on a lateral fluoroscopic view. The pterygopalatine fossa is bordered anteriorly by the posterior wall of the maxillary sinus, posteriorly by the medial plate of the pterygoid process, and medially by the perpendicular plate of the palatine bone and superiorly by the sphenoid sinus, and laterally it communicates with the infratemporal fossa.[2]

The foramen rotundum, through which the maxillary branch of the trigeminal nerve passes, is located on the superolateral aspect of the pterygopalatine fossa; the opening to the pterygoid canal, which houses the vidian nerve, is located on the inferomedial portion of the fossa.

The ganglion within the fossa is located posterior to the middle turbinate of the nose and lies a few millimeters deep to the lateral nasal mucosa. Also contained in the fossa are the maxillary artery and its multiple branches.

The Sphenopalatine ganglion has a complex neural center and has multiple connections. It is "suspended" from the maxillary branch of the trigeminal nerve at the pterygopalatine fossa via the pterygopalatine nerves, and lies medial to the maxillary branch when viewed in the sagittal plane. Posteriorly, it is connected to the vidian nerve, also known as the *nerve of the pterygoid canal*, which is formed by the greater petrosal and the deep petrosal nerves. The ganglion itself has efferent branches and forms the superior posterior lateral nasal and pharyngeal nerves. Caudally, the ganglion is in direct connection with the greater and lesser palatine nerves.

As a neural center, the ganglion has sensory, motor, and autonomic components. The sensory fibers arise from the maxillary nerve, pass through the SPG, and are distributed to the nasal membranes, the soft palate, and some parts of the pharynx.[3] A few motor nerves are also believed to be carried with the sensory trunks.

The autonomic innervation is more complex. The sympathetic component begins with preganglionic sympathetic fibers originating in the upper thoracic spinal cord, forming the white rami communicantes, and coursing through the sympathetic ganglion, where the preganglionic fibers synapse with the postganglionic ones. The postganglionic fibers then join the carotid nerves before branching off and traveling through the deep petrosal and vidian nerves. The postganglionic sympathetic nerves continue their path through the SPG on their way to the lacrimal gland and the nasal and palatine mucosa.

The parasympathetic component has its preganglionic origin in the superior salivatory nucleus then travels through a portion of the facial nerve (cranial nerve VII) before forming the greater petrosal nerve. The greater petrosal nerve in turn joins the deep petrosal nerve to form the vidian nerve, which ends in the SPG.

Within the ganglion, the preganglionic fibers synapse with their postganglionic cells and continue on to the nasal

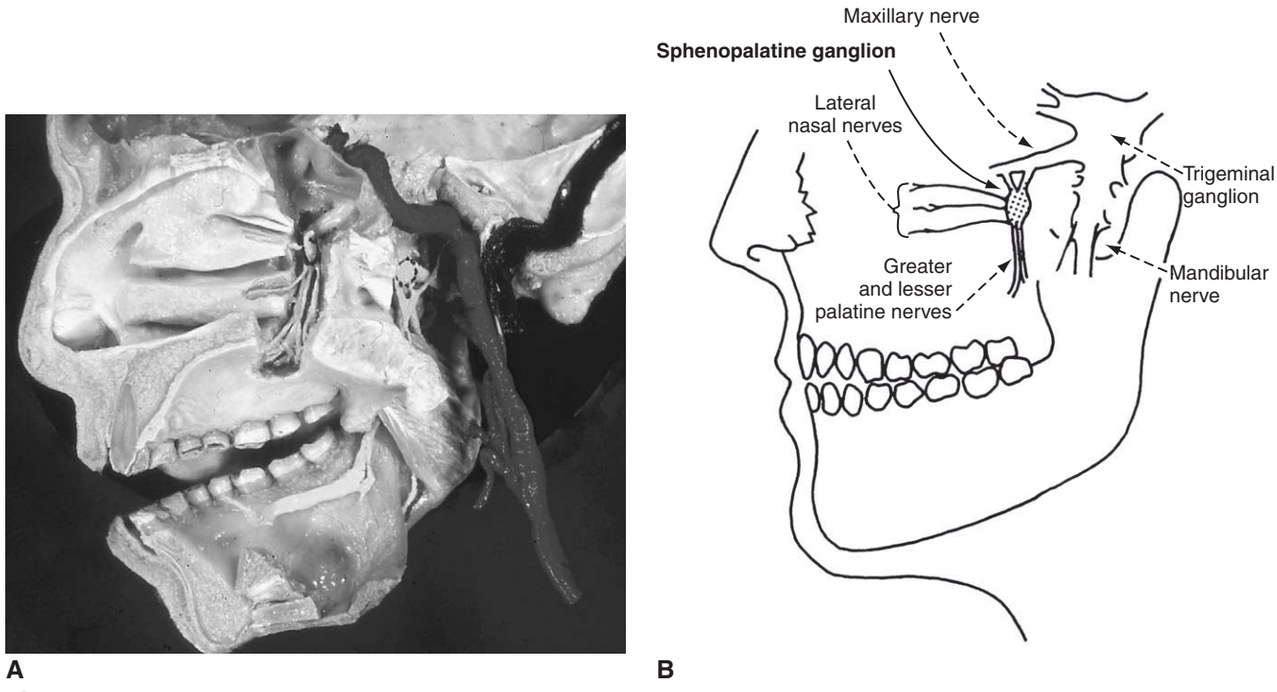


FIGURE 7–1

(A) A cadaver dissection of the face showing the course of the sphenopalatine ganglion and its branches to the nose and the palate. Note the location of the spg in the pterygopalatine fossa. (With permission from U. Pai, MD.) (B) Anatomy of the sphenopalatine ganglion and its immediate connections.

mucosa, and one branch travels with the maxillary nerve to the lacrimal gland.

INDICATIONS

Currently, SPGB is used for relief of facial pain and headache (Table 7-1). The indications supported by current literature include sphenopalatine and trigeminal neuralgia, cluster and migraine headaches, and atypical facial pain. SPGB has been used to treat many painful medical syndromes.

Percutaneous sphenopalatine ganglionolysis should only be considered in patients with intractable facial pain or cluster headache who failed or cannot tolerate pharmacological management.

Sluder,[4] who is credited as the first physician to describe SPGB for the treatment of sphenopalatine neuralgia, described a unilateral facial pain at the root of the nose that sometimes spread toward the zygoma and extended back to the mastoid and occiput. This pain is typically associated with the parasympathetic features such as lacrimation, rhinorrhea, or mucosal congestion. Sluder believed the cause of this pain was the spread of infection from the paranasal sinuses that irritated the SPG. This was initially accepted as a possible cause but came into question when other syndromes, such as low back pain, sciatica, and dysmenorrhea, were attributed to irritation of the SPG.

In the early 1940s, Eagle[3] sought to revive interest in sphenopalatine neuralgia when he presented his thesis to the American Laryngological, Rhinological, and Otological Society. He agreed with Sluder on the existence of sphenopalatine neuralgia but disagreed on its cause. Eagle believed that intranasal deformities, such as deviated septum, septal spurs or ledges, and prominent turbinates, were responsible for irritation of the ganglion, which caused the pain.

Others attribute it to a reflex vasomotor change or possibly a vasomotor syndrome.[5] Regardless of the cause, sphenopalatine neuralgia is an indication for SPGB.

Trigeminal neuralgia is also an indication for SPGB. In 1925, Ruskin[6] disagreed with Sluder on the indication for SPGB and suggested involvement of the SPG in the pathogenesis of trigeminal neuralgia. The SPG is directly connected to the maxillary branch of the trigeminal nerve via the pterygopalatine nerves. He believed that blockade of the SPG would in turn relieve the symptoms associated with trigeminal neuralgia. Few case reports in the current literature support this theory.[7]

Although new medications for the treatment of migraine and cluster headache are introduced every year, a certain small subset of patients fail to respond to oral and parenteral dosing and are forced to seek alternative methods for pain control. In recent years, blockade of the SPG has been used in such cases, with varying success.[8–10]

Another indication for SPGB is atypical facial pain. Such pain is usually unilateral, described as constant, aching, and burning, and is not confined to the distribution of a cranial nerve.[11] It may involve the entire face, scalp, and neck. The pain may have a sympathetic component, which makes the SPGB ideal, because the postganglionic sympathetic nerves pass through the ganglion.

TABLE 7-1 Use of Sphenopalatine Ganglion Block

Indications
Sphenopalatine neuralgia
Trigeminal neuralgia
Headaches (cluster, migraine)
Atypical facial pain
Herpes zoster ophthalmicus
Contraindications
Back pain
Sciatica
Angina
Arthritis

Other reported indications for SPGB include back pain, sciatica, angina, arthritis, herpes zoster ophthalmicus, and pain from cancer of the tongue and floor of the mouth.[12–14] These are not "true" indications for SPGB; instead, they reveal its broad applications in situations when conventional therapies are ineffective. The author does not recommend this indication.

CONTRAINDICATIONS

- *Absolute*
- Local infection
- Coagulopathy
- *Relative*
- Where anatomy has been altered secondary to surgery, infection, or genetic variations
- Patient refusal to undergo the procedure or inability to obtain informed consent
- Inability by the patient to determine a cause-and-effect relationship to symptoms

EQUIPMENT

- 25-gauge, 3/4-inch infiltration needle
- 16-gauge, 1-1/4-inch angiocatheter
- 22-gauge, 3-1/2-inch spinal needle for ganglion block
- 10-cm curved, blunt radiofrequency thermocoagulation (RFTC) needle with a 10-mm active tip for RF lesioning

DRUGS

- Iohexol (Omnipaque) contrast solution
- Preservative-free normal saline (0.9%)
- 1.5% lidocaine for infiltration
- 0.5% bupivacaine or ropivacaine, preservative free, for diagnostic block
- 2% lidocaine, preservative free, for diagnostic block
- Water-soluble steroids: methylprednisolone or triamcinolone diacetate (optional)
- Triple-antibiotic ointment for skin after procedure

PREPARATION OF PATIENT

Rule out paranasal sinus infections, which can cause irritation of the ganglion resulting in pain. Nasal deformities can be responsible for the irritation of the ganglion and thus the pain.[3,4] Trigeminal neuralgia may be the cause of this disorder.[6] There may be dysequilibrium between sympathetic and parasympathetic tone in the ganglion that results in release of substance P or blockade of local enkephalins.[15]

PREOPERATIVE MEDICATION

For preoperative medication, use the standard recommended conscious sedation by the American Society of Anesthesiologists (ASA).

MONITORING

For monitoring, use the standard ASA-recommended monitors, such as electrocardiograph, sphygmomanometer, and pulse oximetry.

PROCEDURE

The SPG can be blocked by several techniques. The drugs frequently used are local anesthetics (4% cocaine, 2% to 4% lidocaine, or 0.5% bupivacaine/ropivacaine/levobupivacaine); depot steroids (methylprednisolone or triamcinolone diacetate), with or without 6% phenol to prolong the blockade; radiofrequency thermocoagulation; or pulsed RF.

Technique One: Intranasal Topical Application of Local Anesthetic

The intranasal topical application of local anesthetic is relatively easy to perform and can be taught to the patient if effective. A 3.5-inch cotton tip applicator is dipped in the anesthetic solution (cocaine or lidocaine).[16,17] The applicator is inserted through the ipsilateral nare on the affected side while a parallel line is maintained with the zygomatic arch, which corresponds to the level of the middle turbinate. A slow advance is made while the applicator is pushed laterally toward the back of the nasal pharynx. The ganglion lies a few millimeters beneath the lateral nasal mucosa (Figure 7-2). Once the first applicator is in place, a second applicator is inserted in the same fashion, except that it is placed slightly superior and posterior to the first. The applicators are left in for approximately 30 to 45 minutes. If additional medication is needed, the local anesthetic can be trickled down the shaft of the applicator. Because of the connections with the lacrimal gland, blockade of the SPG results in ipsilateral tearing because of unopposed parasympathetic activity. If the block is effective, it can be repeated or a radiofrequency can be performed for prolonged analgesia. The authors do not recommend the use of phenol for neurolysis with this technique.

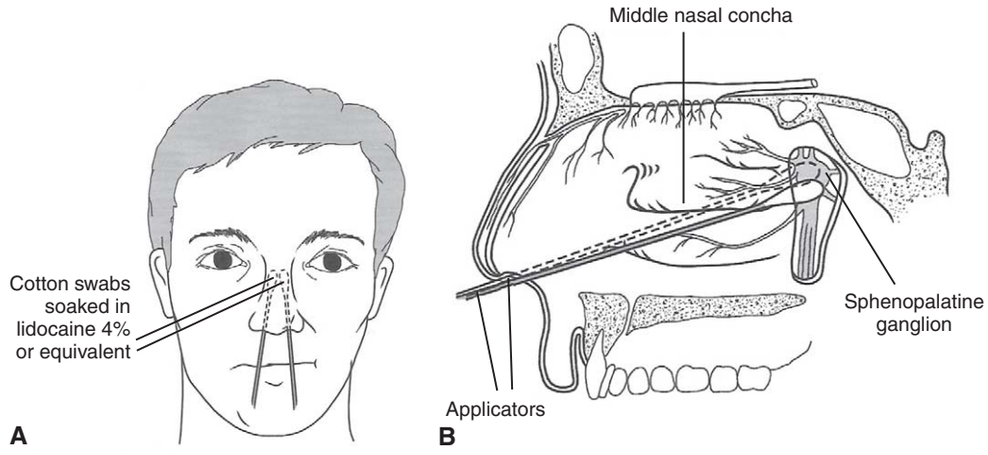


FIGURE 7–2

(A) An anterior view of two applicators placed through the nose at the level of the middle concha for sphenopalatine block (nasal approach). (B) Lateral view of applicators placed intranasally adjacent to sphenopalatine in the lateral wall of the nose.

Spencer[18] developed a variation of this approach at Concord Hospital in Concord, New Hampshire. Specific hollow-lumen, cotton-tipped applicators (Hardwood Products, Guilford, Maine) are placed as described earlier. The white plastic disposable spray nozzle from a bottle of 10% Oral Spray (Astra, USA, Westborough, Massachusetts) is cut to a length of 4 cm. A sterile 2.5-mm i.d. uncuffed tracheal tube is also cut to 4 cm and used to connect the spray nozzle to the hollow applicator. Each actuation of the metered-dose valve delivers 10 mg of lidocaine. The hollow lumen of the applicator is primed with two to three doses (not counted toward the total dose of lidocaine). Additional doses are administered and disconnected from the applicator. The applicators are left in place for at least 30 minutes. The applicators can be recharged with more local anesthetic during this time as long as the dose does not exceed 4 mg/kg.

Technique Two: Greater Palatine Foramen Approach

The patient is placed in the supine position with the neck slightly extended. The greater palatine foramen is located just medial to the gum line of the third molar. Sometimes, a dimple can be seen, which signifies the foramen. A dental needle with a 120-degree angle is inserted through the mucosa and into the foramen. This procedure can be performed with or without fluoroscopic guidance. The needle is advanced approximately 2.5 cm in a superior and slightly posterior direction (Figure 7-3).

A paresthesia may be elicited because the maxillary nerve is just cephalad to the ganglion. If fluoroscopic guidance is used, 1 ml of a nonionic, water-soluble contrast is injected. The spread of the contrast into the pterygopalatine fossa should be visible. Cocaine or lidocaine, 2 ml, is injected after negative aspiration and the SPGB is confirmed as before. Data on standard radiofrequency or pulsed radiofrequency lesioning, or phenol injection of the sphenopalatine ganglion via this approach appear to be absent in the literature.

Technique Three: Lateral Approach

The patient lies supine with the head inside the C-arm (Figure 7-4A). The anterior position view is then taken (Figure 7-4B).

Site of Needle Entry

The needle is inserted under the zygoma in the coronoid notch. A lateral view of the upper cervical spine and the mandible is obtained, and the head is rotated until the rami of the mandible are superimposed one on the other

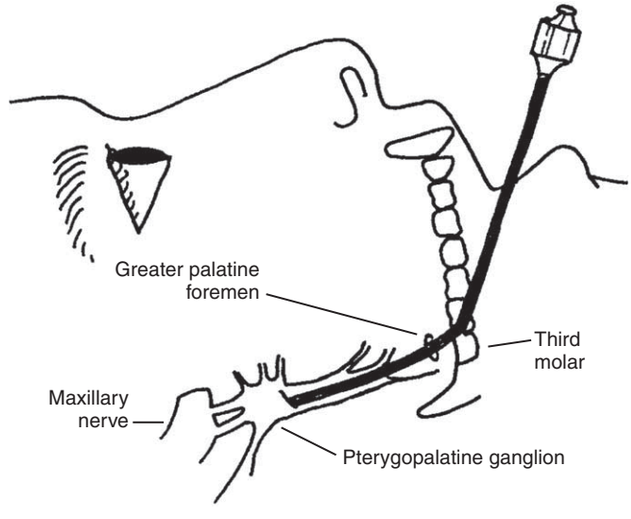


FIGURE 7–3

Greater palatine foramen approach. (From Raj PP, editor: *Textbook of Regional Anesthesia*. St. Louis, Mosb y, 2000, figure 40-3, p. 797, with permission.)

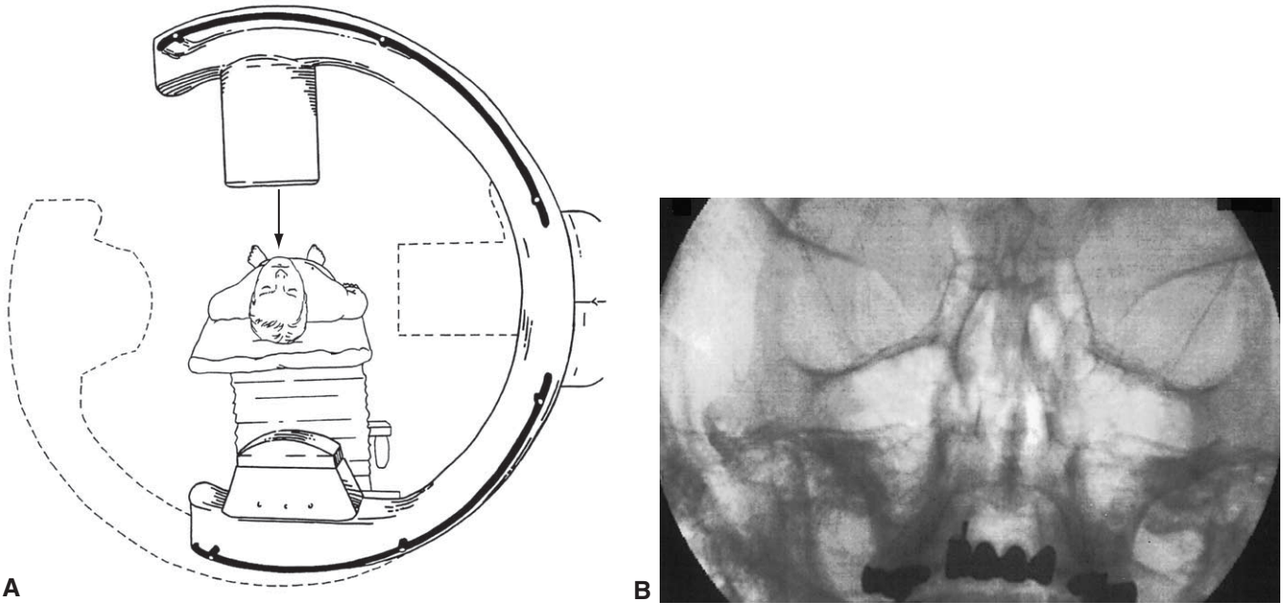


FIGURE 7–4

(A) The patient lies supine. If the fluoroscope is used, the C-arm should visualize the C6-C7 vertebral region in the anteroposterior and lateral views. (B) This radiographic image shows the posteroanterior view of the front of the face, identifying the orbit and maxillary sinus.

(Figure 7-5A). The C-arm is moved slightly cephalad until the pterygopalatine fossa is visualized. It should resemble a vase when the two pterygopalatine plates are superimposed on one another and are located just posterior to the posterior aspect of the maxillary sinus (Figure 7-5b).

Technique of Needle Entry

When a blunt needle is used, a 1-1/4-inch angiocatheter four sizes larger than the blunt needle must be inserted first. The needle is directed medial, cephalad, and slightly posterior toward the pterygopalatine fossa. An anteroposterior view confirms the proper direction and positioning of the needle (Figure 7-6). The tip of the needle should be advanced until it is adjacent to the lateral nasal mucosa. If resistance is felt at any time, the needle must be slightly withdrawn and redirected. The operator takes care to avoid advancing the needle through the lateral nasal wall.

In the lateral view, note that the needle is residing in the inverted vase. Figures 7-7 and 7-8 show the landmarks needed to confirm correct placement of the needle.

Injection of Local Anesthetic

Once it is properly positioned, 1 to 2 ml of local anesthetic is injected, with or without steroid. As much as 5 ml of local anesthetic can be injected for a diagnostic block.

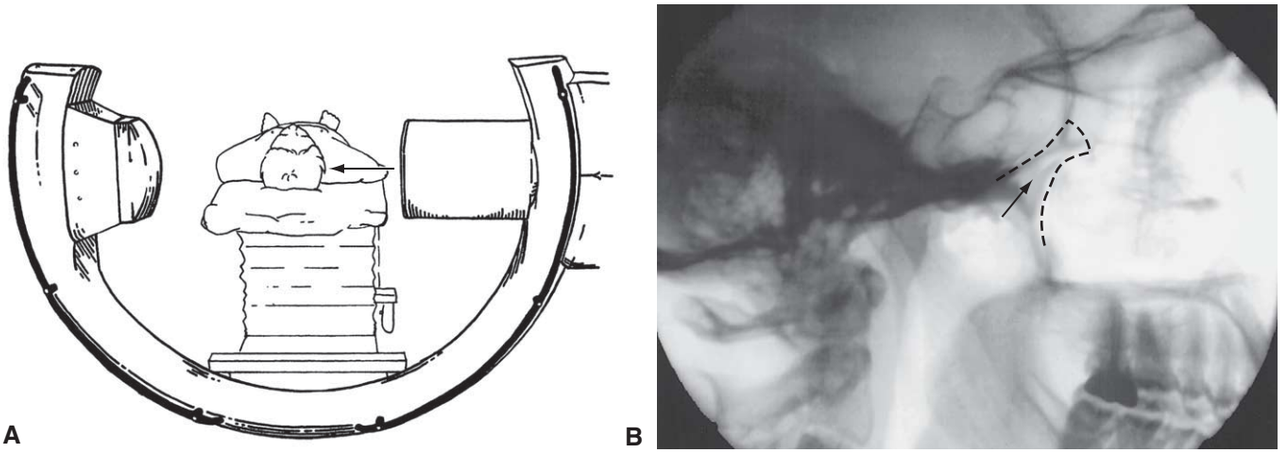
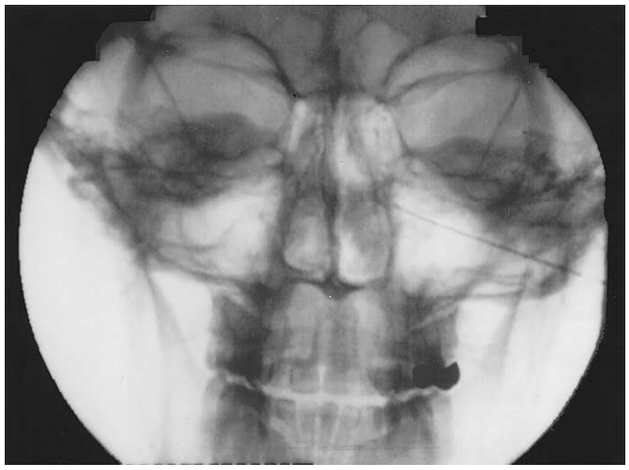
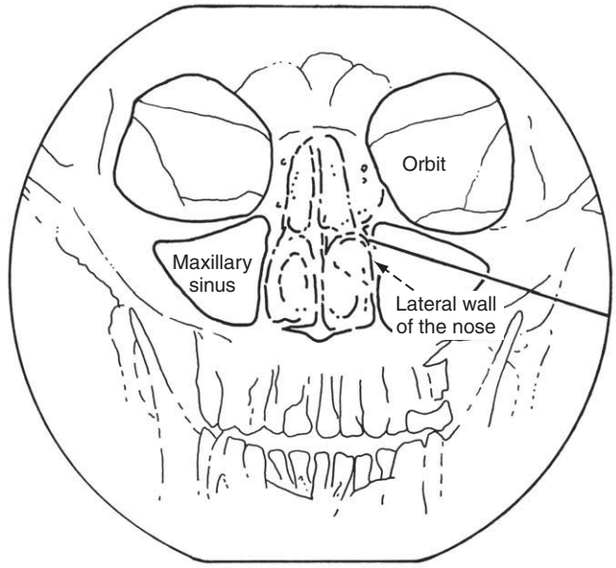


FIGURE 7–5

(A) The C-arm position to obtain the lateral view of pterygopalatine fossa. The beam of the C-arm should be directed toward the root of the nose (arrow). (B) This radiographic view identifies the "inverted flower vase" image of the pterygopalatine fossa (see dotted lines).





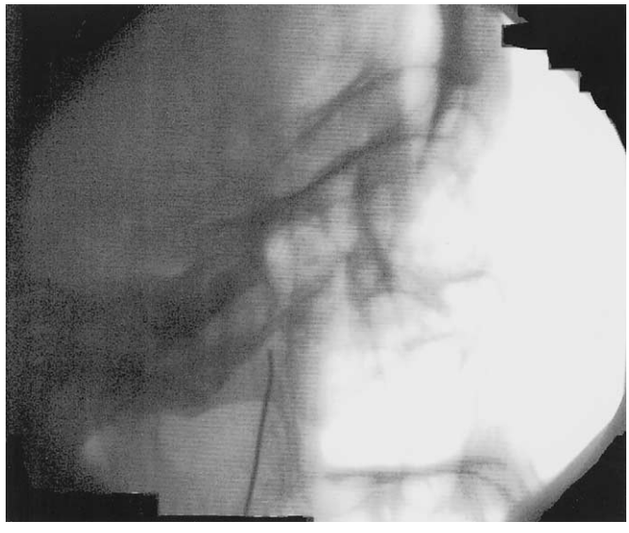
A B

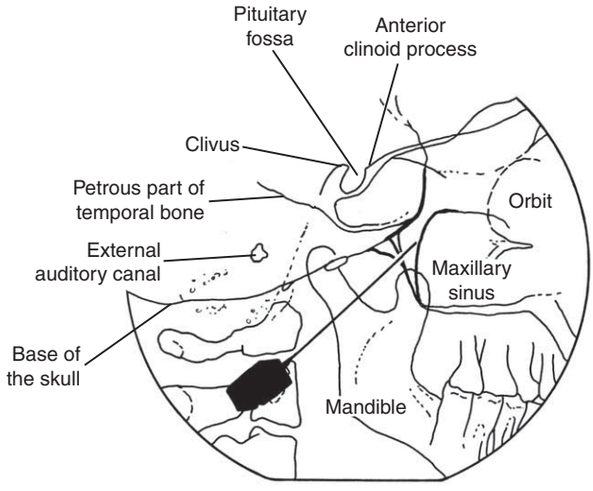
FIGURE 7–6

(A) The radiographic anteroposterior view of the face shows the needle tip at the lateral wall of nose at the superomedial angle of the maxillary sinus. (B) The drawing of the posteroanterior view of the radiograph shows the needle tip at the lateral wall of the nose.

Technique of Neurolysis

Radiofrequency thermocoagulation lesioning. Lesioning of the SPG can be performed with either RFTC or pulsed radiofrequency. With radiofrequency (RF), RFTC sensory testing is done after the needle is correctly placed radiographically. Paresthesia should be felt at the root of the nose 0.5 to 0.7 V at 50 Hz when the needle is correctly situated on the ganglion. If the paresthesia is felt in the upper teeth, the maxillary branch of the trigeminal nerve is being stimulated and the needle must be redirected more caudally. Stimulation of the greater and lesser palatine nerves results in paresthesias of the hard palate. In this case, the needle is anterior and lateral and should be redirected in a more posterior and medial direction. An insulated 20- or 22-gauge, 10-cm, curved, blunt-tipped RFK (Racz-Finch Kit [Radionics, Inc., Burlington, Vermont]) needle with a





A B

FIGURE 7–7

(A) The radiographic lateral view of face shows the radiofrequency needle in the "inverted vase" of the pterygopalatine fossa. (B) Drawing of the lateral view of the face identifies correct needle placement on the sphenopalatine ganglion.

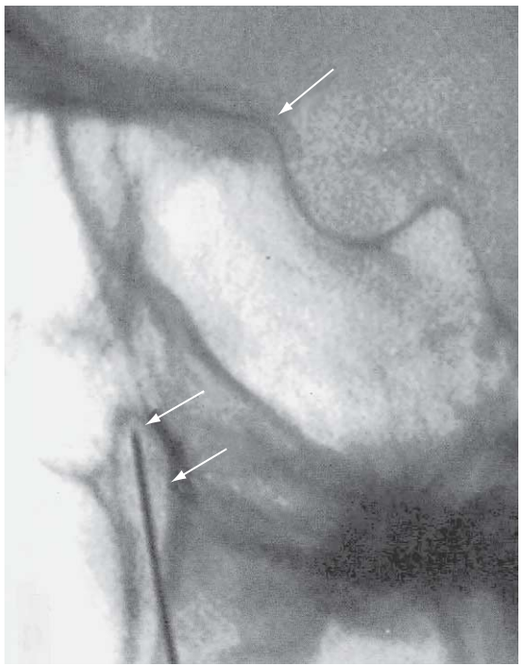


FIGURE 7–8

Close-up radiograph of the lateral view of the pterygopalatine fossa. The following is a key to the letters shown on the figure: (A) anterior clinoid process; (B) pterygopalatine fossa; and (C) needle tip in the pterygopalatine fossa. Correct placement of the needle is seen in the lateral oblique view.

5- to 10-mm active tip is used. After proper placement and stimulation as described in the "Technique" section, RF lesioning is performed for 70 to 90 seconds at 80°C. Two lesions are usually made. Before lesioning, 1 to 2 ml of local anesthetic is injected. Pulsed RF lesioning is performed at 42°C for 120 seconds. Two or three lesions (120 seconds) can be made without local anesthetic, since the temperature of the lesioning is barely above the normal body temperature. Expected effect after local anesthetic block is numbness of the root of the nose and palate. There may be lacrimation from the ipsilateral side of the eye.

COMPLICATIONS

A reflex bradycardia can occur in some patients during RF and pulsed RF lesioning of the SPG.[19]

When the lesioning is halted, the bradycardia is resolved. In some patients, atropine may be needed to complete the lesioning. A reflex resembling the oculocardiac reflex may be the cause. The afferent information may travel back through the vidian nerve, geniculate ganglion, and nervus intermedius to reach the solitary tract nucleus, which has interconnections to the dorsal vagal nucleus.[19]

Infection can occur if proper aseptic technique is breached. Epistaxis can occur if too much pressure is applied to the needle and it is pushed through the lateral nasal wall. Hematoma formation is possible if the large venous plexus overlying the pterygopalatine fossa or the maxillary artery is punctured.

RF lesioning of the SPG can result in hypesthesia or numbness of the palate, maxilla, or the posterior pharynx due to the direct injury or lesioning of the maxillary and mandibulary nerves, but is usually transient.[20]

Destruction of the secretomotor function of the SPG may potentially impair ipsilateral lacrimation or nasal/palatal mucus production.

Mechanical injury to structures superficial to the pterygopalatine fossa must also be considered, such as the parotid gland and branches of the facial nerve.[20]

EFFICACY

To date, no comprehensive prospective randomized clinical trials involving SPG lesioning for atypical facial pain are available. Clinical reports of SPG lesioning for facial pain due to head and neck cancers suggest that early relief may occur in up to 77% of patients,[21] but that recurrence of pain is common.[22] SPG lesioning for intermittent cluster-type headaches has been reported to be quite effective,[23] but the long-term efficacy in chronic cluster headaches is less encouraging.[23]

Recently, a study by Bayer et al.[24] evaluated the efficacy of sphenopalatine ganglion pulsed radiofrequency (SPG-PRF) treatment in patients suffering from chronic head and face pain. Thirty patients were observed from 4 to 52 months after PRF treatment. Fourteen percent of respondents reported no pain relief, 21% had complete pain relief, and 65% of the patients reported mild to moderate pain relief from SPG-PRF treatment. Sixty-five percent of the respondents reported mild to moderate reduction in oral opioids.

One study by Sanders and Zuurmond examined the efficacy of SPGB in 66 patients suffering from episodic and chronic cluster headaches[9]. All had previously been treated with various pharmacologic and/or surgical therapies, without significant pain relief. The patients were divided into two groups—those with episodic pain and those with chronic pain—with sample sizes of 56 and 10 patients, respectively. All received three RF lesions at 70°C for 60 seconds. Thirty-four (60.7%) of 56 patients with episodic cluster headaches and 3 (30%) of the 10 with the chronic type received complete pain relief during a mean follow-up period of 29 months.

Salar and associates reported using percutaneous RFTC of the SPG for sphenopalatine neuralgia in seven patients[2]. Each received two lesions at 60°C and 65°C, respectively, for 60 seconds. One patient required repeat lesioning, and two underwent repeat lesioning, and another two underwent two additional RF procedures. All the patients were pain free over a follow-up period ranging from 6 to 34 months.[1]

Prasanna and Murthy[13] reported complete pain relief for at least 12 months in a patient suffering from herpes zoster ophthalmicus who was treated with SPGB for residual ear pain that had not been alleviated with previous stellate ganglion blocks. The same authors also reported immediate short-term pain relief with intranasal blockade of the SPG in 10 patients suffering intractable pain from

cancer of the tongue and the floor of the mouth.[14] Prospective, randomized, controlled studies to confirm efficacy and safety of sphenopalatine ganglion block for the treatment of chronic pain are still lacking.

STELLATE GANGLION BLOCK

HISTORY

Selective block of the sympathetic trunk of the stellate ganglion was first reported by Sellheim and, shortly thereafter, by Kappis in 1923[25] and Brumm and Mandl in 1924.[26] After 1930, the technique and the indications were established by White and Sweet[27] in the United States and Leriche and Fontaine[28] in Europe.

ANATOMY

The stellate ganglion is named because of its star-shaped appearance resulting from the union of the inferior cervical ganglion with the first thoracic ganglion (Figure 7-9).

Cell bodies for preganglionic nerves originate in the anterolateral horn of the spinal cord; fibers destined for the head and neck originate in the first and second thoracic spinal cord segments, whereas preganglionic nerves to the upper extremity originate at segments T2-T8, and occasionally T9. Preganglionic axons to the head and neck exit with the ventral roots of T1 and T2 and then travel as white communicating rami before joining the sympathetic chain and passing cephalad to synapse at either the inferior (stellate), middle, or superior cervical ganglion. Postganglionic nerves either follow the carotid arteries (external and internal) to the head or integrate as the gray communicating rami before joining the cervical plexus or upper cervical nerves to innervate structures of the neck.

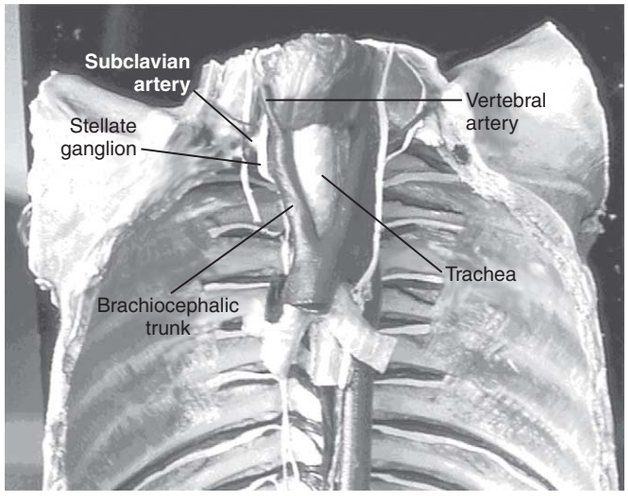


FIGURE 7–9

The anatomy of the head and neck in a cadaver showing the course of the stellate ganglion and its sympathetic chain and relationship to other structures. (With permission from U. Pai, MD.)

To achieve successful sympathetic denervation of the head and neck, the stellate ganglion should be blocked because all preganglionic nerves either synapse here or pass through on their way to more cephalad ganglia. Blockade of the middle or superior ganglion would miss the contribution of sympathetic fibers traveling from the stellate ganglion to the vertebral plexus and, ultimately, to the corresponding areas of the cranial vault supplied by the vertebral artery.[29] Sympathetic nerves to the upper extremity exit T2-T8 through ventral spinal routes, travel as white communicating rami to the sympathetic chain, then pass cephalad to synapse at the second thoracic ganglion, first thoracic or inferior cervical (stellate) ganglion, and, occasionally, the middle cervical ganglion. Most postganglionic nerves leave the chain as gray communicating rami to join the anterior divisions at C5-T1, nerves that form the brachial plexus. Some postganglionic nerves pass directly from the chain to form the subclavian perivascular plexus and innervate the subclavian, axillary, and upper part of the brachial arteries.[30]

In most humans, the inferior cervical ganglion is fused to the first thoracic ganglion, forming the stellate ganglion. Although the ganglion itself is inconstant, it commonly measures 2.5 cm long, 1.0 cm wide, and 0.5 cm thick. It usually lies in front of the neck of the first rib and extends to the interspace between C7 and T1. When elongated, it may lie over the anterior tubercle of C7; in persons with unfused ganglia, the inferior cervical ganglion rests over C7, and the first thoracic ganglion over the neck of the first rib. From a three-dimensional perspective, the stellate ganglion is limited medially by the longus colli muscle, laterally by the scalene muscles, anteriorly by the subclavian artery, posteriorly by the transverse processes and prevertebral fascia, and inferiorly by the posterior aspect of the pleura. At the level of the stellate ganglion, the vertebral artery lies anterior, having originated from the subclavian artery. After passing over the ganglion, the artery enters the vertebral foramen and is located posterior to the anterior tubercle of C6 (Figure 7-10).

Because the classic approach to blockade of the stellate ganglion is at the level of C6 (Chassaignac's tubercle), the needle is positioned anterior to the artery. Other structures posterior to the stellate ganglion are the anterior divisions of the C8 and T1 nerves (inferior aspects of the brachial plexus). The stellate ganglion supplies sympathetic innervation to the upper extremity through gray communicating rami of C7, C8, T1, and, occasionally, C5 and C6. Other inconstant contributions to the upper extremity are from the T2 and T3 gray communicating rami, which do not pass through the stellate ganglion but join the brachial plexus and ultimately innervate distal structures of the upper extremity. These fibers have sometimes been implicated when relief of sympathetically mediated pain is inadequate despite evidence of a satisfactory stellate block.[31]

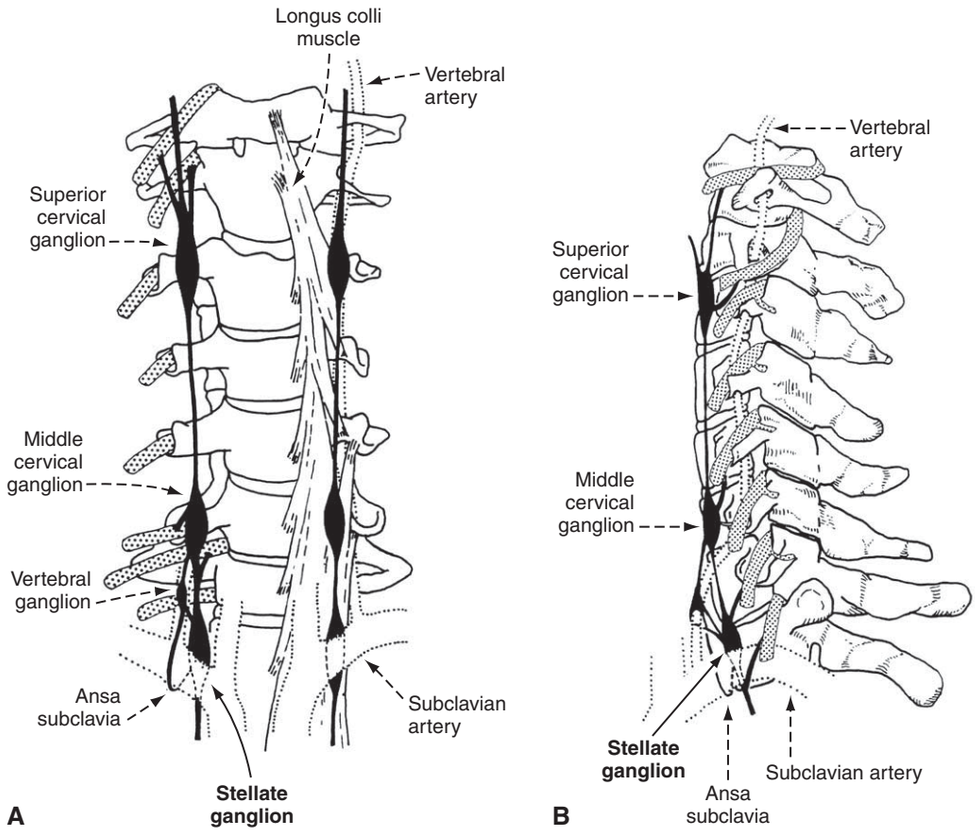


FIGURE 7–10

(A) The anterior view of the anatomy and relations of the stellate ganglion. Note the connections of the stellate ganglion superiorly and its close relation to longus colli muscle. (B) The lateral view of the anatomy of the stellate ganglion. Note the vertebral artery is anterior to the stellate ganglion at C7 and becomes posterior at C6.

INDICATIONS

Stellate ganglion block is useful in the treatment of a variety of painful conditions of the head, neck, upper extremities, and upper thoracic dermatomes.

Pain is due to acute herpes zoster, as well as postherpetic neuralgia; CRPS type I and II; cancer pain of the head, neck, and upper extremities; atypical facial pain; and painful syndromes related with the vascular system such as vascular insufficiency, vasospasm, arterial embolism, and vasculopathy related with Meniere syndrome.[32]

The post-traumatic syndrome, which is often accompanied by swelling, cold sweat, and cyanosis, is an ideal indication for stellate ganglion block.

For patients requiring vascular surgery on the upper extremities, stellate ganglion block has diagnostic; prognostic; and, in some cases, prophylactic value.

Chest pain from angina pectoris may also be an indication. Stellate ganglion block may also be used in the treatment of hyperhydrosis of the upper extremity together with thoracic sympathetic block.

Simultaneous bilateral blocks are not advisable. Nevertheless, in cases of pulmonary embolism, bilateral stellate ganglion block is absolutely indicated as immediate therapy.

Although there is a vast indication for the use of stellate ganglion block in all these painful syndromes, there is very little prospective clinical data assessing the efficacy of the block in the treatment of these conditions. The information related with the stellate ganglion block is based on case series and case reports. Further controlled clinical trials need to be held.

CONTRAINDICATIONS

Absolute contraindications of stellate ganglion block are as follows:

- Anticoagulant therapy, because of the possibility of bleeding if there is vascular damage during insertion of the needle
- Pneumothorax and pneumonectomy on the contralateral side, because of the danger of additional pneumothorax on the ipsilateral side
- Recent cardiac infarction, because stellate ganglion block cuts off the cardiac sympathetic fibers (accelerator nerves), with possible deleterious effects in this condition

Glaucoma can be considered a relative contraindication to stellate ganglion block because provocation of glaucoma by repeated stellate ganglion blocks has been reported.[33] Marked impairment of cardiac stimulus conduction (e.g., atrioventricular block) is also to be regarded as a relative contraindication because blockade of the upper thoracic sympathetic ganglia aggravates bradycardia.

EQUIPMENT

- 25-gauge local infiltration needle
- 22-gauge, 1 inch of 1-1/2-inch block needle
- 5- or 10-cm (2- or 5-mm tip) sharp Sluijter-Mehta (SM®) or Racz-Finch Kit (RTK®) needle for RF
- RF machine

DRUGS

Radiofrequency Equipment

- 0.2–0.5% bupivacaine (0.5-1%) or ropivacaine
- 1–2% lidocaine
- Steroids (optional)
- Phenol (3% phenol in iohexol [Omnipaque 240])
- 0.9% normal saline

Therapeutic Block

1. Local anesthetics and steroids similar to Diagnostic Block are the same as for block with steroids.

PREPARATION OF PATIENT

Physical Examination

- Check neck extension mobility.
- Check for prior radical neck surgery.
- Examine for infection at injection site.
- Examine for thyroid surgery.
- Check for anatomic variations related to surgery.

Preoperative Medication

For preoperative medication, use the standard ASA recommendations for conscious sedation.

PROCEDURE

Patient Preparation

Ideally, proper patient preparation for the stellate ganglion block begins at the visit before the procedure. The patient is much more likely to remember discharge instructions and expected side effects if they are explained during a visit when the patient is not apprehensive about the imminent procedure, what side effects may be expected, and potential complications. Discussions of the realistic expectations of sympathetic blockade should be held before any procedure. The goals of blockade and the number of blocks in a given series differ with each pain syndrome, and these variables should be discussed, when possible, at visits before the actual blockade. Patients are much less likely to experience frustration or despair if they understand beforehand what can be expected. If the cause of pain is unclear and the intended block is considered diagnostic, a complete explanation allows the patient to record valuable information on the effectiveness of the procedure.

Informed consent must be obtained. Potential risks, complications, and possible side effects should be explained in detail. The patient should share responsibility for decision making and must understand the risks and the fact that complications do occur.

Placement of an intravenous (IV) line before the block is not mandatory at all pain clinics, but it facilitates use of IV sedation, when indicated, and provides access for administration of resuscitative drugs should a complication occur. In skilled hands, a stellate ganglion block can be performed quickly and relatively painlessly, so IV administration may not be necessary. All standard resuscitative drugs, suction apparatus, oxygen delivery system, cardiac defibrillators, and equipment for endotracheal intubation, however, need to be readily accessible. For anxious patients and in teaching institutions when the operator is inexperienced or when "hands-on" teaching is expected, preblock sedation through an IV line is beneficial.

There are several approaches for the stellate ganglion block: (1) paratracheal approach, (2) anterior approach, (3) posterior approach, and (4) oblique approach. In the past, stellate ganglion block was performed by blind technique. However, several complications such as pneumothorax were due to this blind approach. Today all approaches should be performed under fluoroscopy.

Paratracheal Approach (Blind Technique)

The patient is made to lie supine with the head resting flat on the table without a pillow. A folded sheet or thin pillow should be placed under the shoulders of most patients to facilitate extension of the neck and accentuate landmarks. The head should be kept straight with the mouth slightly open to relax the tension on the anterior cervical musculature. Hyperextension of the neck also causes the esophagus to move midline, away from the transverse processes on the left.

To ensure proper needle positioning, the operator must correctly identify the C6 tubercle. Identification is most easily performed using firm pressure with the index finger (Figure 7-11). In a left-handed or right-handed stellate ganglion block, the operator's nondominant hand should be used for palpating landmarks. Patients do not tolerate jabbing; rather, gentle but firm probing can easily

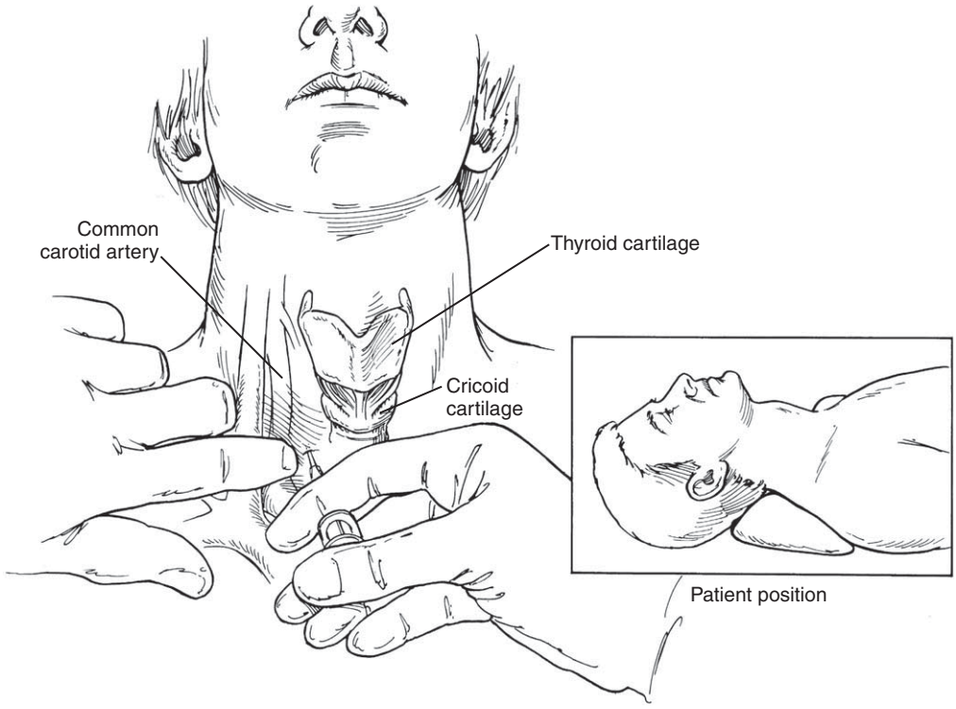


FIGURE 7–11

Stellate ganglion block. C6 anterior tubercle is directly beneath the operator's index finger. The carotid artery is retracted laterally when necessary. The needle is perpendicular to all skin planes and is inserted directly posterior from the point of entry. (Inset) The patient is positioned for stellate ganglion block. A pillow or roll should be placed between the shoulders to extend the neck, bring the esophagus to the midline, and facilitate palpation of Chassaignac's tubercle.

define the borders of the tubercle. A single finger, the index finger, relays the most specific tactile information.

The skin is antiseptically prepared, and the needle is inserted posteriorly, penetrating the skin at the tip of the operator's index finger. Making a skin wheal with local anesthetic is rarely necessary, except in some teaching situations or in patients with obese necks. In both situations, a 5-cm needle (or a 22-gauge B-bevel needle) is used and should puncture the skin directly downward (posterior), perpendicular to the table in all planes. Although a smaller (e.g., 25-gauge) needle can be used, the added flexibility and smaller caliber make it more difficult to reliably ascertain when bone is encountered and then maintain the proper location for injection.

The needle passes through the underlying tissue until it contacts either the C6 tubercle or the junction between the C6 vertebral body and the tubercle. The depths of these structures differ, the tubercle itself being more anterior than the junction between body and tubercle. Regardless of the specific location encountered at C6, if the skin is being properly displaced posteriorly and laterally by the nondominant index finger, the depth is rarely more than 2.0 to 2.5 cm. The important difference between medial and lateral location of bone at C6 relates to the presence of the longus colli muscle, which is located over the lateral aspect of the vertebral body and the medial aspect of the transverse process. It does not cover the C6 tubercle; only the prevertebral fascia that invests the longus colli muscle also covers the C6 tubercle. Therefore, if the needle contacts the medial aspect of the transverse process at a depth somewhat greater than expected, the operator should be prepared to withdraw the needle 0.5 cm to avoid injecting into the longus colli muscle. Injection into the muscle belly can prevent caudad diffusion of local anesthetic to the stellate ganglion. Location of the needle on the superficial tip of the C6 anterior tubercle requires withdrawal of the needle from periosteum before injection.

The procedure is most easily performed if the syringe is attached before the needle is positioned. This prevents accidental dislodgment of the needle from the bone during syringe attachment after the needle is placed. Once bone is encountered, the palpating finger maintains its pressure, the needle is withdrawn 2 to 5 mm, and the medication is injected. Alternatively, once bone is met, the operator's palpating hand can release and fix the needle by grasping its hub, leaving the dominant hand free to aspirate and inject. Even though this technique can be performed blindly, more often fluoroscopy is used to confirm contrast spread (Figure 7-12). With fluoroscopy, correct placement of the needle should be demonstrated by anteroposterior and lateral views with spread of the contrast solution (Figure 7-13A–D).

Final injection. Once proper needle placement is confirmed, injection of medication must be performed in a

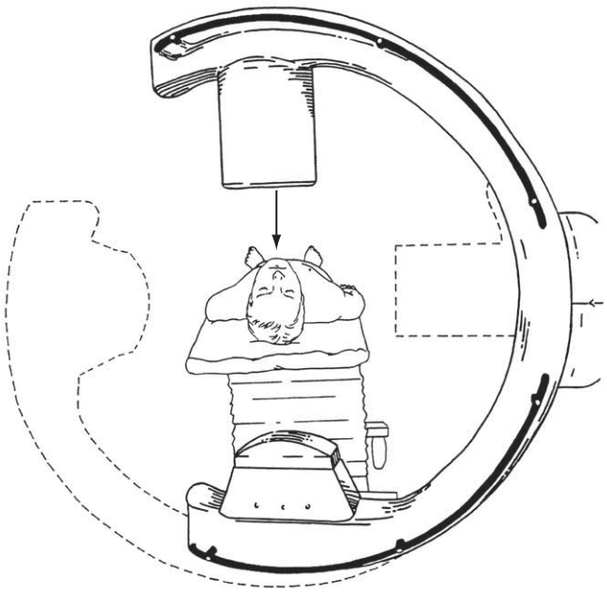


FIGURE 7–12

The patient lies supine. If the fluoroscope is used, the C-arm should visualize the C6-C7 vertebral region in the anteroposterior and lateral views.

routine and systematic fashion. A 50:50 mixture of 2% lidocaine with 0.5% ropivacaine or equipotent ropivacaine and 1 ml of 40 mg/ml of triamcinolone (optional) may be used. An initial test dose must be injected in all cases. Less than 1 ml of solution injected IV has produced loss of consciousness and seizure activity (Raj, personal communication). Before any injection, careful aspiration for blood and cerebrospinal fluid (CSF) must be performed. If the aspiration is negative, 0.5 to 1.0 ml of solution is administered, and the patient is asked to raise the thumb to indicate the absence of adverse symptoms. The patient should be informed beforehand and reminded during the blockade procedure that talking might cause movement of the neck musculature that could dislodge the needle from its proper location. To communicate during the block, the patient can be asked to point a thumb or finger upward in response to questions. After the initial test dose, the operator can inject the remainder of the solution, carefully aspirating after each 3 to 4 ml at a time.

During injection or needle placement, paresthesia of the arm or hand may be elicited. It should always be interpreted to mean that the needle has been placed deeper to the anterior tubercle, adjacent to the C6 or C7 nerve root. Repositioning of the needle is necessary. Aspiration of blood or CSF also demands repositioning of the needle. Even though the needle may be in the correct position, sometimes it is necessary to confirm that the injected solution is not flowing where it is not desired. The correct total volume of solution depends on the type of block is desired.[31] Properly placed, 5 ml of solution blocks the stellate ganglion (Figure 7-14).

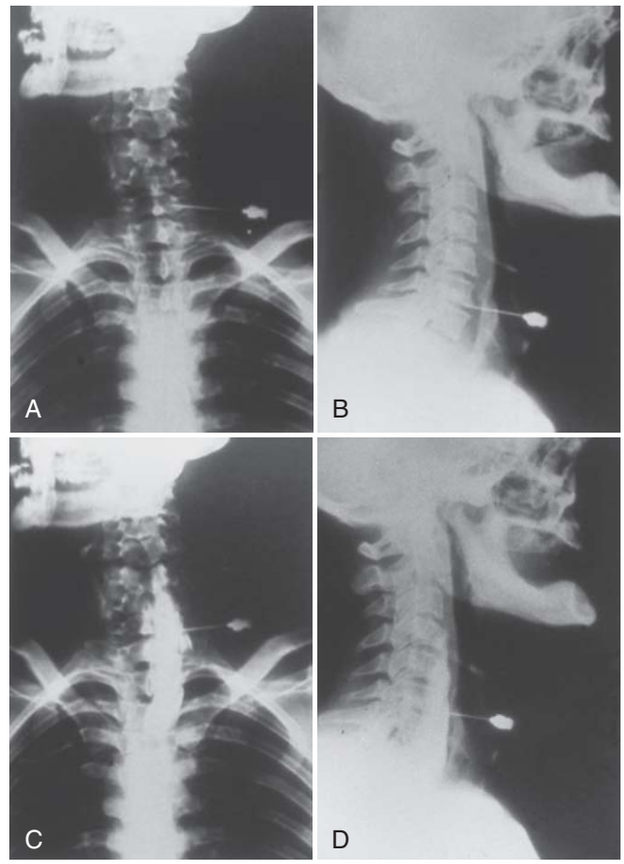


FIGURE 7-13

Anteroposterior and lateral views of correct placement of the needle and the contrast medium spread after injection for stellate ganglion block. Anteroposterior (A) and lateral (B) views of needle placement, and anteroposterior (C) and lateral (D) views of contrast medium spread.

C7 Anterior Approach

The anterior approach to the stellate ganglion at C7 is similar to the approach described at C6. Unlike C6, C7 has only a vestigial tubercle; hence it is necessary to find Chassaignac's tubercle (C6). Then the palpating finger moves one finger-breadth caudad from the inferior tip.

The advantage of blockade at C7 is manifested by the lower volume of local anesthetic needed to provide complete interruption of the upper extremity sympathetic innervation. Only 6 to 9 ml of solution suffices. The bothersome side effect of recurrent laryngeal nerve block is less common with this approach. The technique has two drawbacks: (1) the less pronounced landmarks make needle positioning less reliable, and (2) the risk of pneumothorax increases because the dome of the lung is close to the site of entry. The use of radiographic imaging during the approach helps avoid the complications possible with the blind technique.

Posterior Approach

The posterior approach[32] to the stellate ganglion is used in two clinical situations: (1) where infection, trauma, or tumor

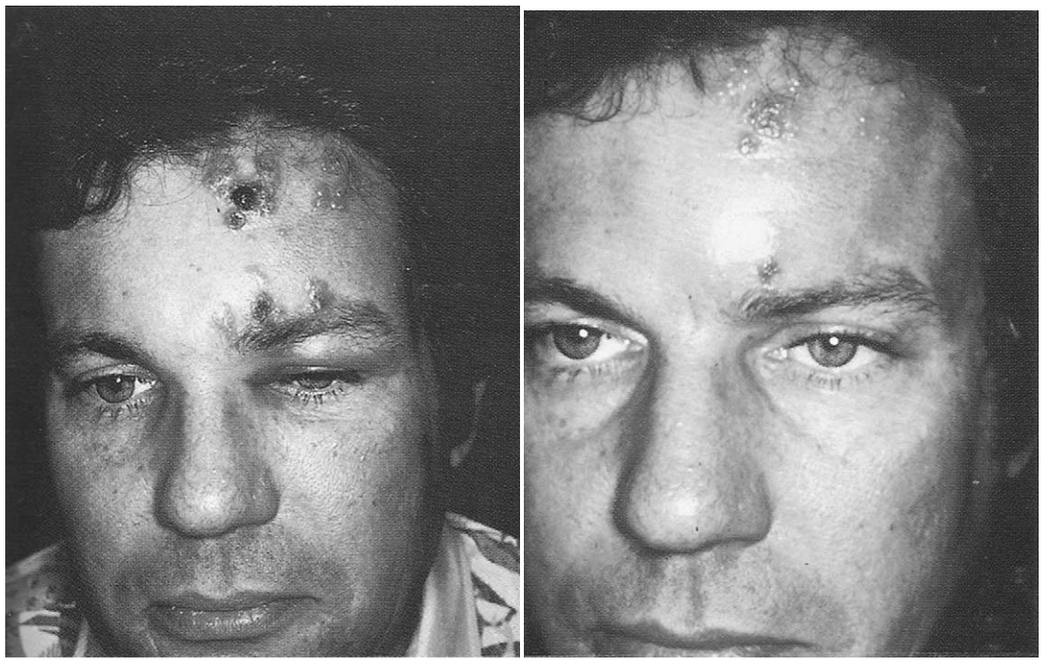


FIGURE 7–14

Acute herpes zoster of the left eye and forehead prior to treatment with intralesional injection and left stellate ganglion block. (B) Appearance 3 days after left stellate ganglion block. Note the clearing of the eye and the lesions of the forehead. (From Raj PP: *Practical Management of Pain*. Chicago, Mosby-Year Book, 1986, figure 22-13, plate 7, with permission.)

precludes use of the traditional anterior approach to stellate ganglion block; or (2) when neurolysis of the sympathetic innervation of the upper extremity is desired. The posterior approach to stellate ganglion block is preferred when neurolytic solutions are being used, because this approach allows the needle to be placed at the more inferior T1 or T2 level, thus avoiding the possibility of superior spread of neurolytic solution with resultant permanent Horner's syndrome. Neurolysis of the sympathetic chain can also be accomplished via the anterior vertebral approach using radiofrequency lesioning.

Technique. The patient is placed in the prone position with the cervical spine in neutral position. Five to 7 ml of local anesthetic without preservative is drawn into a 12-ml sterile syringe. For disease processes that have an inflammation component, such as acute herpes zoster, or disease processes with associated edema, such as CRPS I or II, 80 mg of methylprednisolone is added for the first block and 40 mg of methylprednisolone is added for subsequent blocks.

A point 4 cm lateral to the spinous process of T1-T2 is identified. The skin at this area is then prepared with antiseptic solution, and the skin and subcutaneous tissues are anesthetized with local anesthetic. A 22-gauge, 10-cm needle is advanced until contact is made with the lamina of the target vertebra (Figure 7-15). If bony contact is not made with needle insertion to a depth of 1-1/2 inches, the needle is probably either between the transverse processes of adjacent vertebrae or too lateral. If this occurs, the

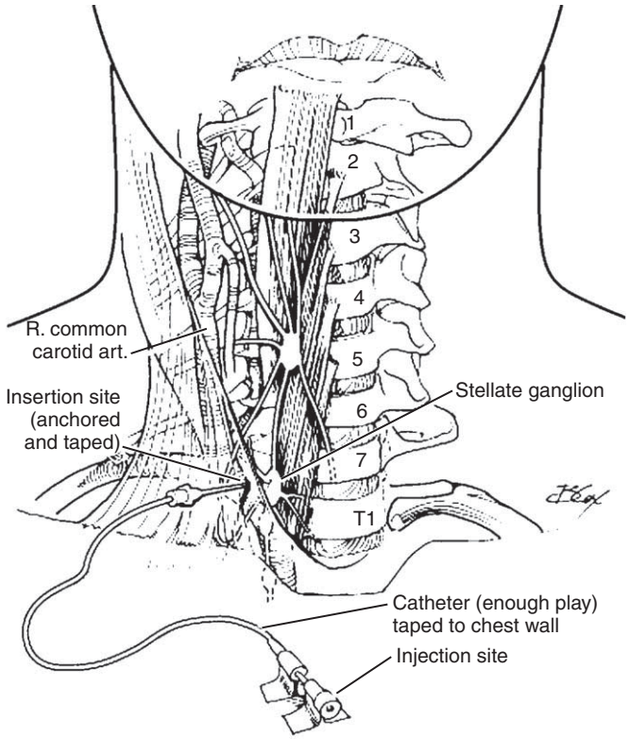


FIGURE 7–15

Posterior approach to stellate ganglion block. (From Abdi S, Zhou Y, Doshi R, Patel N: Stellate ganglion block: emphasis on the new oblique fluoroscopic approach. *Techniques in Regional Anesthesia and Pain Management*, 9:73–80, 2005, figure 6, with permission.)

needle should be withdrawn and reinserted with a more caudad and medial trajectory. After bony contact is made, the needle is then withdrawn and redirected slightly laterally and inferiorly. This allows the needle to slide beneath the transverse process and rib. Ultimately the needle tip should rest just adjacent to the anterolateral border of the vertebral body in a manner analogous to the final needle position when performing lumbar sympathetic block. Careful aspiration is carried out, and 5 to 7 ml of solution is then injected. If neurolytic block is performed, a small incremental dose of 6% aqueous phenol or mixed with Omnipaque or absolute alcohol should be injected while observing the patient's clinical response.

Side effects and complications. The main complication of the posterior approach to stellate ganglion block is pneumothorax. The use of CT guidance should help decrease this complication. Proximity to the aorta also represents a potential risk that can be decreased with careful attention to technique and the use of CT guidance.

Because of the proximity to the spinal column, it is also possible to inadvertently inject the local anesthetic solution into the epidural, subdural, or subarachnoid space. At this level, even small amounts of local anesthetic placed into the subarachnoid space may result in a total spinal anesthetic. Trauma to exiting spinal roots is also a distinct possibility, especially if bony contact with the lamina of the target vertebra does not occur and the needle continues to be advanced.

Inadvertent block of the recurrent laryngeal nerve with associated hoarseness and dysphagia can occur if the injectate comes in contact with this nerve. Should neurolytic solution be inadvertently injected onto this nerve, these side effects could be permanent, with devastating results for the patient. Likewise, superior spread of neurolytic solution can result in a permanent Horner's syndrome. The patient should be forewarned of the possibility of these complications before neurolytic stellate ganglion block using the posterior approach.

The use of CT guidance will dramatically decrease the incidence of complications associated with this technique. Raj[34] reports a 4% pneumothorax rate, which suggests that this procedure should be performed only in a setting where chest tube placement is practical. Given the morbidity of surgical sympathectomy at this level, this technique still has a favorable risk-to-benefit ratio despite the potential for serious complications.

Oblique Fluoroscopic Approach

This technique has been described by Abdi and coworkers.[35] Briefly, the patient is monitored and placed in a supine position as described above with the fluoroscope being directed in the anterior to posterior direction. The C-arm is rotated to the side where the injection is desired until the neural foramina are clearly visualized. The C6 to C7 disc (the disc between the sixth and the seventh cervical vertebra) is flattened with the C-arm directed caudal. This usually requires 30- to 60-degree caudal angulation of the C-arm. In this view, the disc, the foramina, and the uncinate process are clearly distinguishable (Figure 7-16A–D).

A 26-gauge spinal needle is directed onto the vertebral body at the base of the uncinate process and just anterior to the foramina. A total of 1 to 2 ml of radio opaque dye is injected with real time fluoroscopic imaging. This ensures that the injection is along the longus colli muscle and not intravascular or intrathecal. A total of 3 ml of a long-acting anesthetic such as 0.25% bupivacaine is then injected. This typically spreads to at least the first thoracic segment.

There are several advantages of this technique according to the author, such as no need of pressing or pushing the vascular system out of the way or of pressing on Chassaignac's tubercle, which can be uncomfortable and even painful. The chance of intravascular injection or perforation of the esophagus is minimal. The chance of recurrent laryngeal nerve paralysis is minimal. Reduction in the volume of local anesthetics is needed to cover lower cervical through upper thoracic segments.

However, the technique is not without risk. For example, in the individual with emphysematous bulbous pleura, an oblique C7 insertion to the base of the uncinate process could lead to a pneumothorax.

CHEMICAL NEUROLYSIS OF STELLATE GANGLION

The approach for chemical neurolysis is similar to that for stellate ganglion block performed at C7. The patient must be positioned with the neck and head in a neutral position (see Figure 7-18). Under direct anteroposterior fluoroscopy, the C7 vertebral body is identified. A skin wheal is raised over the ventrolateral aspect of the body of C7 with 1 ml of local anesthetic and a 25-gauge needle. A 22-gauge, B-bevel needle is inserted through the skin wheal to contact the body of C7 in the ventrolateral aspect. This is at the junction of the transverse process with the vertebral body. Depth and direction should be confirmed with both anteroposterior and lateral views. The needle tip is positioned deep to the anterior longitudinal ligament. The longus colli lies lateral to the needle tip. The needle should be stabilized with a long-handled Kelly clamp or hemostat. An IV extension should be attached to the needle and used for injection. Approximately 5 ml of water-soluble, nonirritating, nonionic, preservative-free, hypoallergenic contrast medium is injected after negative aspiration. Dye should spread around the vertebra, avoiding IV, epidural, intrathecal, thyroidal, or myoneural (longus colli) uptake. If good spread of the contrast medium is visualized, a mixture of local anesthetic, phenol, and steroid is injected. The total volume of 5 ml should consist of 2.5 ml of 6% phenol in saline, 1 ml of 40-mg triamcinolone, and 1.5 ml of 0.5% ropivacaine. (The total 5-ml dose contains a final mixture of 3% phenol.) The previously injected contrast material serves as a marker for

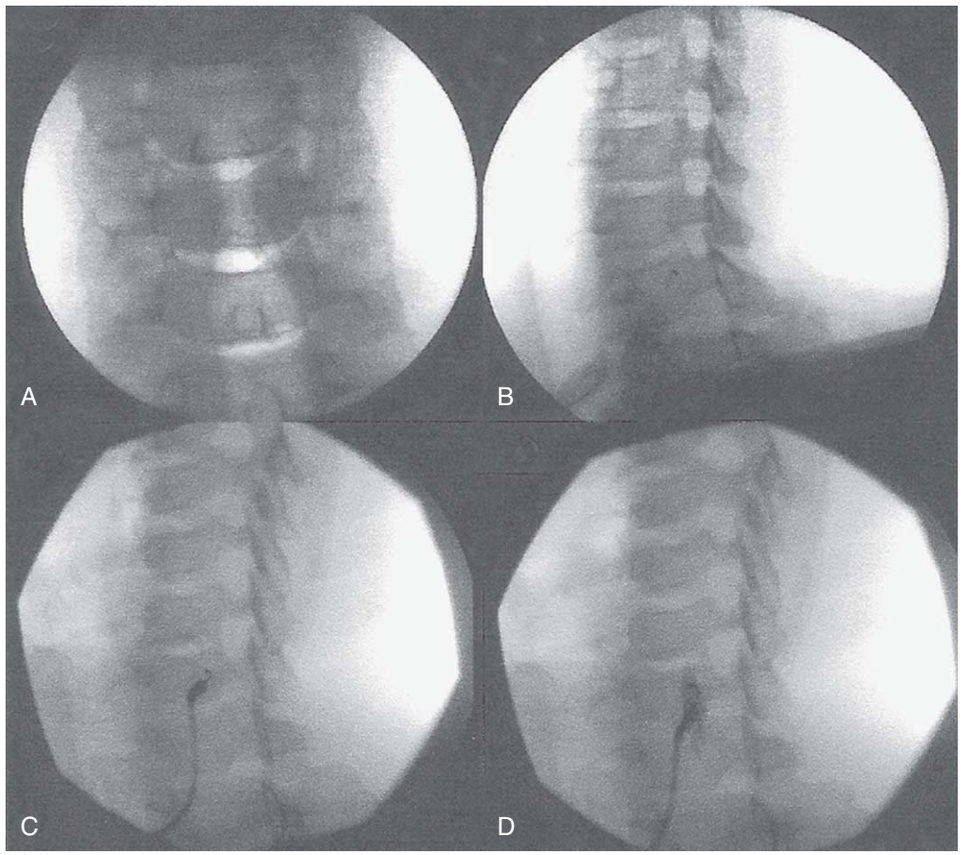


FIGURE 7–16
Oblique approach to stellate ganglion block.

the spread of the phenol. In the anteroposterior view, the contrast should spread caudad to the first thoracic sympathetic ganglion and the inferior cervical ganglion, and cephalad to the superior cervical ganglion. In the lateral view, spread should be observed in the retropharyngeal space anterior to the vertebral body and in front of the longus colli and anterior scalene muscles. After injection, the patient remains supine with the head elevated slightly for approximately 30 minutes to prevent spread of the phenol to other structures.[36]

RADIOFREQUENCY OF STELLATE GANGLION

Radiofrequency of the stellate ganglion may be accomplished under fluoroscopic guidance. After the target area is identified as for chemical neurolysis, a 16-gauge angiocatheter is inserted through the skin wheal instead of the B-bevel needle. A 20-gauge, curved, blunt-tipped cannula with a 5-mm active tip is guided through the angiocatheter at the superolateral aspect. The tip should rest at the junction of the transverse process and the vertebral body. The depth and direction should be confirmed with anteroposterior and lateral views. Correct placement may be confirmed conclusively with the injection of contrast medium (Figures 7-17 to 7-20). A sensory (50 Hz, 0.9 V) and a motor (2 Hz, 2 V) stimulation trial must be performed owing to the location of

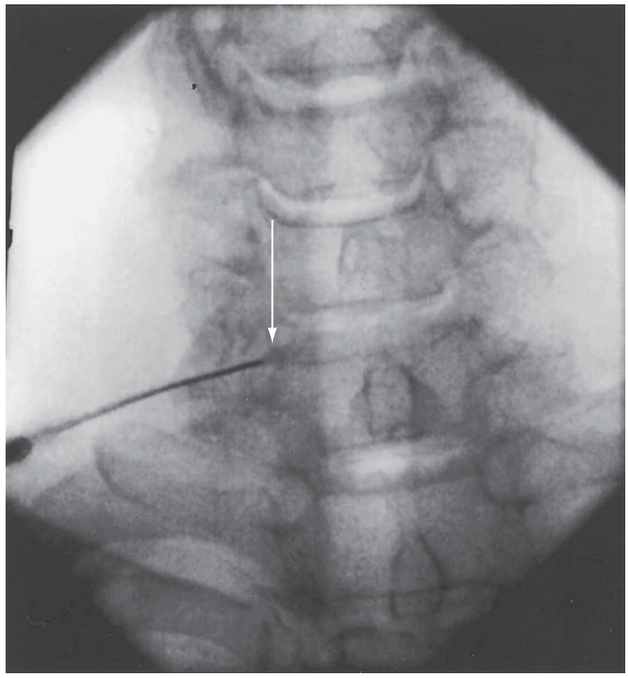


FIGURE 7–17
Posteroanterior radiograph of the cervical spine. Note that at the C7 level, the radiofrequency cannula rests at the junction of the lateral aspect of the vertebral body and the medial aspect of the transverse process (arrow). This represents the correct cannula position for lesioning of the C7 sympathetic fibers.

the phrenic nerve (lateral) and the recurrent laryngeal nerve (anterior and medial) relative to the proposed lesion. While motor stimulation is performed, the patient should say "ee" to ensure preservation of vocal cord function. A small volume of local anesthetic (0.5 ml) should be injected before lesioning. After waiting 10 minutes, the thermal RF is applied for 60 seconds at 80°C. The cannula is then redirected to the most medial aspect of the transverse process in the same plane. Placement is in the ventral aspect of the transverse process in the same plane. Placement in the ventral aspect must be confirmed with a lateral view. Before lesioning, the patient must be retested for sensory and motor stimulation. A repeat dose of the local anesthetic should also be given through the cannula. A third (and final) lesion should be directed at the upper portion of the junction of the transverse process and the body of C7. Potential complications include injury to the phrenic or the recurrent laryngeal nerve, neuritis, and vertebral artery injury.[37,38]

Side effects of a stellate ganglion block should be distinguished from complications. Most unpleasant side effects—ptosis, miosis, and nasal congestion—result from Horner's syndrome.

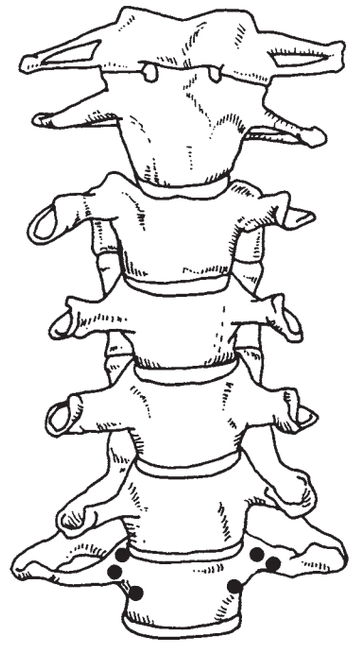


FIGURE 7–18

Drawing of a posteroanterior view of the cervical spine. Dots mark the target points for radiofrequency lesioning of the cervical sympathetic nerves. Note that these are at the junction of the medial aspect of the transverse process with the lateral aspect of its respective vertebral body.

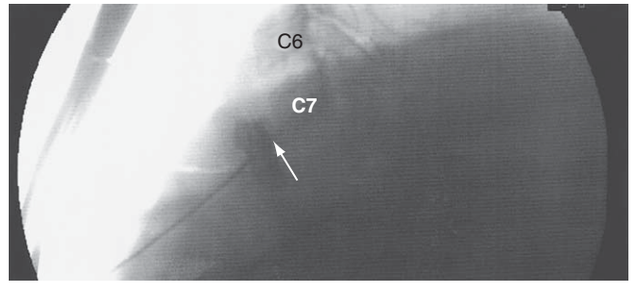


FIGURE 7–19

Lateral view of correct placement of the needle (arrow) and the contrast agent spread after injection for stellate ganglion block.

COMPLICATIONS

The two significant complications of stellate ganglion block are pneumothorax and intraspinal injection. A third significant risk when neurolysis is performed is the possibility of persistent Horner's syndrome. Pneumothorax can be avoided with careful placement of the needle, and, if care is taken that the needle angulation is never lateral and that the needle is advanced through the costotransverse ligaments (posterior and anterior) slowly and cautiously using the loss-of-resistance technique. Intraspinal injection most often occurs by diffusion through the intervertebral foramen and can be avoided by first injecting a contrast solution and checking the needle position radiographically. The optimal method for checking needle position and solution spread is computed tomographic scan.

To check for possible subsequent Horner's syndrome, the clinician can first inject local anesthetic into the region and inspect the patient after 15 to 30 minutes. This practice does not always obviate Horner's syndrome with neurolytic injection, however, and prior local anesthetic injection may not be considered optimal in all situations.

Common complications of a stellate ganglion block result from diffusion of local anesthetic onto nearby nerve structures. These include the recurrent laryngeal nerve with complaints of hoarseness, feeling of a lump in the throat, and sometimes a subjective shortness of breath. Bilateral stellate blocks are rarely advised, because bilateral blocking of the recurrent laryngeal nerve can result in respiratory compromise and loss of laryngeal reflexes. Block of the phrenic nerve causes temporary paralysis of the ipsilateral diaphragm and can lead to respiratory embarrassment in patients whose respiratory reserve is already severely compromised. Partial brachial plexus block can also result secondary to spread along the prevertebral fascia[35] or positioning the needle too far posteriorly. When this complication occurs, the patient should be discharged with the arm in a sling and given careful instructions on how to care for a partially blocked arm.

The two most feared complications of stellate ganglion block are intraspinal injection and seizures induced by intravascular injection. Respiratory embarrassment and the need for mechanical ventilation can result from injection into either the epidural space (if high concentrations of local anesthetic are used) or the intrathecal space. Should either occur, patients need continual reassurance that everything is being appropriately managed and that they will recover without sequelae. No drugs are necessary

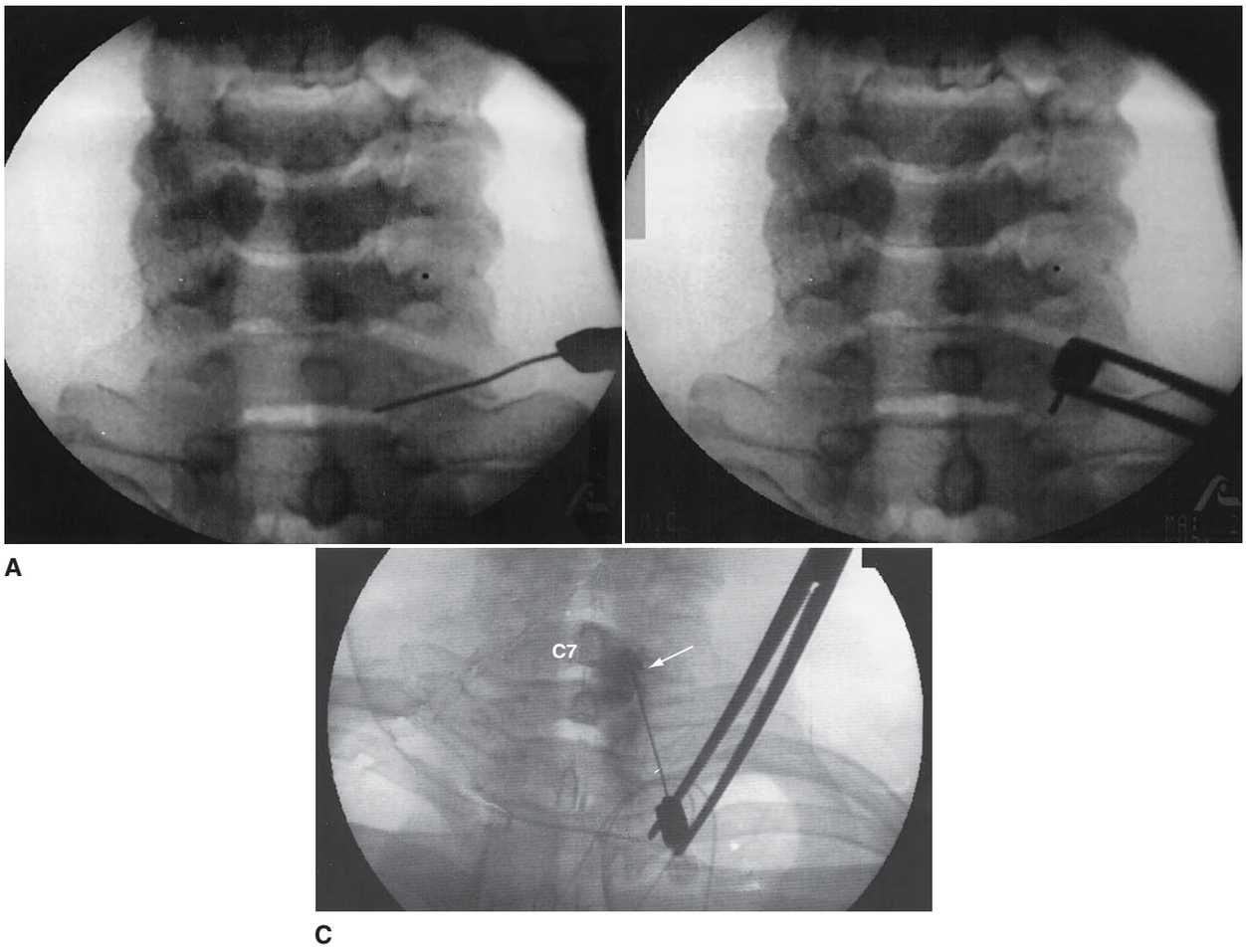


FIGURE 7–20

(A) The needle is in position for radiofrequency at C7 superior (AP view). (B) Inferior needle location at C_7 vertebrae for RF lesioning The arrow (C) shows the tip at the lateral border of vertebral body C7 with contrast.

for endotracheal cannulation because profound anesthesia of the larynx can be expected.

Intravascular injection most often involves the vertebral artery. Small amounts of local anesthetic cause unconsciousness, respiratory paralysis, seizures, and sometimes severe arterial hypotension. Increased IV fluids, vasopressors if indicated, oxygen, and endotracheal intubation may be necessary. If the amount of drug injected into the artery is less than 2 ml, the sequelae just listed are short-lived and self-limiting, with oxygen and increased fluid administration often being the only therapy needed. Care must be taken during a stellate ganglion block to ensure that no air is injected from the syringe. Cerebral air embolisms have been reported from this procedure, and they are preventable.[30,39,40]

The risk of pneumothorax also attends the anterior approach. If the C7 tubercle is used and the needle is inserted caudally, the dome of the lung can be penetrated. Unfortunately, some 10% to 15% of patients suffer postprocedure neuritis, which can last 3 to 6 weeks.[36,41] Persistent cough after stellate ganglion block has been reported.[42]

Severe airway obstruction secondary to acute and delayed retropharyngeal or cervicomediastinal hematoma following stellate ganglion block can occur.[43,44]

Pneumochylothorax is another rare complication of stellate ganglion block, especially when the needle tip is at C7 level.[45] Sudden death following stellate ganglion block has been reported.[46] A 29-year-old woman died 3.5 hours after SGB. Autopsy revealed subcutaneous emphysema of the body, bilateral pneumothorces, and a huge post-tracheal hematoma. The lower half of the trachea was markedly flattened by pressure from this hematoma.

The infection rate is minimal after stellate ganglion. A 56-year-old woman developed pyogenic osteomyelitis of the cervical spine following multiple stellate ganglion

blocks for pigmentation degeneration retinopathy.[47] She had no history of diabetes mellitus and immunodeficiency.

Central nervous system complications after stellate ganglion block could be most devastating. Seizures, hemiparesis, aphasia, locked-in syndrome, brainstem anesthesia, total spinal block, paralysis due to cervical spinal cord lesion and even death have been reported after stellate ganglion block.[48–57]

Continuous fluoroscopy monitoring during injection of contrast may also help to prevent intravascular or intrathecal injection of local anesthetics.

CLINICAL PEARLS

Small amounts of local anesthetics (3 to 5 ml) do not reliably block all fibers to the upper extremities because contributions from T2 and T3 may not be blocked. Injection of 10 ml of solution more reliably blocks all sympathetic innervation to the upper extremity, even in patients with the anomalous Kuntz's nerves. If blockade is being performed for sympathetic-mediated pain of the thoracic viscera, including the heart, 15 to 20 ml of solution should be administered.

Anomalous pathways, termed Kuntz's nerves, can be reliably blocked only by a posterior approach,[31] although the posterior approach is technically more difficult than the anterior approach.

EFFICACY

Sympathetic interruption to the head, supplied by the stellate ganglion, can easily be documented by evidence of Horner's syndrome: miosis (pinpoint pupil), ptosis (drooping of the upper eyelid), and enophthalmos (sinking of the eyeball). Associated findings include conjunctival injection, nasal congestion, and facial anhidrosis. These signs can be present without complete interruption of the sympathetic nerves to the upper extremity.

Evidence of sympathetic blockade to the upper extremity includes visible engorgement of the veins on the back of the hand and forearm, diminution of psychogalvanic reflex, and plethysmographic and thermographic changes. Skin temperature rises also, provided that the preblock temperature did not exceed 33°C to 34°C.

ACKNOWLEDGMENT

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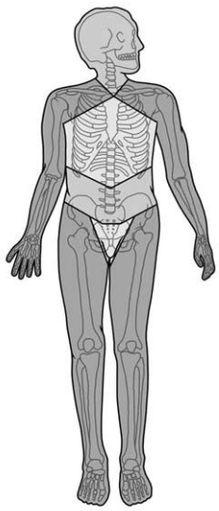
C H A P T E R



8

Spinal Neuroaxial Procedures of the Head and Neck

MICHAEL HAMMER, CARL E. NOE, GABOR B. RACZ, RICARDO RUIZ-LOPEZ, AND CARMEN PICHOT

CERVICAL EPIDURAL STEROID INJECTIONS

HISTORY

Although Pages' description[1] of the paramedian approach to the lumbar epidural space in 1921 is considered the first clinically relevant report of the technique of lumbar epidural nerve block, it appears that Dogliotti[2] was the first to describe the technique of epidural block in the cervical region.[3]

Epidural steroids have been introduced for the treatment of acute radiculopathy. The initial numbers of procedures carried out by physicians were relatively small, and the complexity of the procedure failed to reveal the serious hazards associated with the technique. With better understanding of the anatomy, the consensus seems to be heading in the direction of injecting into the site-specific area.

The most relevant consideration is the proximity of the epidural space to the cervical spinal cord. Care must be taken to avoid subdural, intrathecal, and intravascular injections. The ligamentum flavum is inconsistent in the cervical region and may not be fused in the midline. During cervical epidural needle placement, if the needle tip advances through this open space and loss of resistance is used for establishing the epidural space, the first resistance may be the dura mater and the first loss of resistance may be in the subdural rather than epidural space.[4–6]

Techniques to identify the epidural space have included the hanging drop, loss of resistance, and test dose of local anesthetic techniques. Fluoroscopic guidance with anterior-posterior (AP) and lateral views plus radiopaque contrast use are now commonly used to confirm placement in conjunction with the loss of resistance technique. Bartynski[7] reported a 25.7% incidence of incorrect needle placement without fluoroscopy for lumbar epidural injections.

Lumbar epidural corticosteroid injections have been used for over 30 years.[8] The primary indication has been for spinal radicular pain and, more controversially, for pain related to disk herniations, spinal stenosis and failed back surgery. A review by Nelson and Landau[9] details the history and erroneous argument against epidural steroid injections. Most prospective randomized trials have been in patients with lumbosacral diagnoses rather than cervical syndromes. Dilke[10] reported efficacy with epidural steroid injections in 1973. In the 1980s, Cuckler and Ridley found no benefit.[11,12] In Australia, concern about Depo-Medrol prompted a statement being issued encouraging interdisciplinary treatment for patients with chronic pain.[13] Later, Carette[14] reported temporary benefit in patients with disk herniations but no reduction in the surgery rate. Carette,[15] in a later review, suggested injections as an option for cervical radiculopathy. Meta-analysis has led to different conclusions regarding the effectiveness of epidural steroid injections.[16] Clinical experience justifies the use of epidural steroid injections more for acute radiculopathy rather than chronic painful conditions.

ANATOMY

The superior boundary of the cervical epidural space is the point at which the periosteal and spinal layers of dura fuse at the foramen magnum.[17] It should be recognized that these structures allow drugs injected into the cervical epidural space to travel beyond the injection site as the epidural space is contiguous from the foramen magnum to the sacrococcygeal membrane[18] (Figure 8-1).

The epidural space is bounded by the dura mater and the tissues that line the spinal canal. The cervical epidural space is bounded anteriorly by the posterior longitudinal ligament and posteriorly by the vertebral laminae and the ligamentum flavum. The ligamentum flavum is relatively thin in the cervical region and becomes thicker farther caudad, closer to the lumbar spine.[17] This fact has direct clinical implications in that the loss of resistance felt during cervical epidural nerve block is more subtle than it is in the lumbar or lower thoracic region. The ligamenta flava

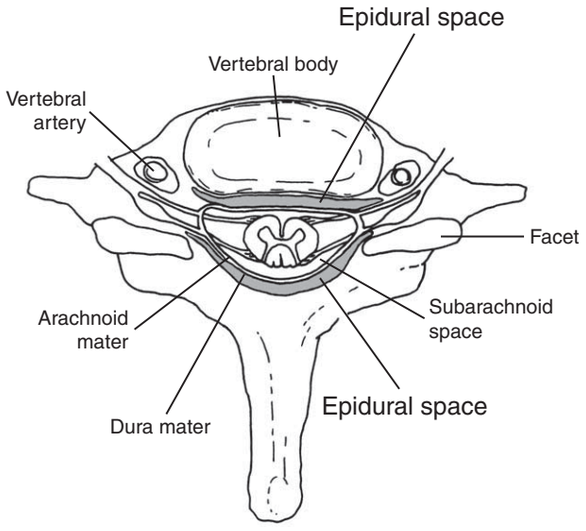


FIGURE 8–1
Drawing shows the relationship of the cervical epidural contents.

connect lamina from one vertebra to the next. Also, the ligamenta flava attach from the facet joint capsule to where the lamina fuses to form spinous processes. The ligamenta flava partially fuse posteriorly with openings allowing venous connections between internal and posterior external vertebral venous plexuses.

Meningovertebral ligaments attach the theca with the tissue surrounding the canal and are most prominent anteriorly and laterally. A midline attachment from the dura to the ligamentum nuchae exists at the first two cervical levels.

The vertebral pedicles and intervertebral foramina form the lateral limits of the epidural space. The degenerative changes and narrowing of the intervertebral foramina associated with aging may be marked in the cervical region. The distance between the ligamentum flavum and dura is greatest at the C2 interspace, measuring 5.0–6.0 mm in adults.[17] Because of the enlargement of the cervical spinal cord, the distance from the ligamentum flavum and dura is only 1.5–2.0 mm at C7.[17] It should be noted that flexion of the neck moves this cervical enlargement more cephalad, resulting in widening of the epidural space to 3.0–4.0 mm at the C7-Tl interspace.[18] This fact has important clinical implications if cervical epidural block is performed with the patient in the lateral or prone position.

Contents of the Epidural Space

The epidural space contains adipose, connective tissue, nerves, arteries, lymphatics, and a venous plexus.

Fat. The epidural space is filled with fatty areolar tissue. The amount of epidural fat varies in direct proportion to the amount of fat stored elsewhere in the body.[17] The epidural fat is relatively vascular and changes to a denser consistency with aging. The epidural fat appears to perform the following two functions: (1) it serves as a shock absorber for the other contents of the epidural space and for the dura and the contents of the dural sac, and (2) it serves as a depot for drugs injected into the cervical epidural space.

Epidural veins. The venous plexus surround the dura in a ringed segmental fashion. The epidural veins are concentrated principally in the anterolateral portion of the epidural space.[17] However, in the presence of obstruction to venous run-off such as epidural scarring, there may be large distended high-pressure veins in the midposterior epidural space. These veins are valveless and so transmit both intrathoracic and intra-abdominal pressures. As pressure in any of these body cavities increases, owing to Valsalva's maneuver or compression of the inferior vena cava by a gravid uterus or a tumor mass, the epidural veins distend and reduce the volume of the epidural space. Because the venous plexus serves the entire spinal column, it becomes a ready conduit for infection.

Epidural arteries. The arteries that supply the bony and ligamentous confines of the cervical epidural space, as well as the cervical spinal cord, enter the cervical epidural space via two routes: through the intervertebral foramina and via direct anastomoses from the intracranial portions of the vertebral arteries.[17,19]

Arteries enter the epidural space via neural foramina anteriorly and posteriorly at multiple levels. The anterior segmental arteries are most commonly at lower cervical, lower thoracic and upper lumbar levels. These anterior segmental arteries supply the anterior spinal artery. Posterior segmental arteries are more numerous and evenly distributed than anterior segmental arteries and supply the posterior spinal arteries.[6]

There are significant anastomoses between the epidural arteries, most of which lie in the lateral portions of the epidural arteries. Trauma to the epidural arteries can result in epidural hematoma formation and compromise the blood supply to the spinal cord itself.

Lymphatics. The lymphatics of the epidural space are concentrated in the region of the dural roots, where they remove foreign material from the subarachnoid and epidural spaces.

INDICATIONS

Indications for cervical epidural corticosteroid injections include cervical radicular pain syndromes, most commonly disk herniation, central or foraminal spinal stenosis, and spondylolisthesis. The entry level of the epidural space should be inferior to the level of stenosis whether a single-shot or catheter technique is used. Many patients have chronic midline pain with exacerbations of radicular pain. While these patients should be classified as having chronic pain, they are sometimes reasonable candidates for subacute, new radicular symptoms.

CONTRAINDICATIONS

Patients with a history of previous cervical spine surgery may have scarring or altered anatomy. Great care is needed to avoid dural punctures or other mechanical complications, such as loculation. Some would advocate a technique such as catheter placement from a thoracic level or transforaminal blunt needle approach as an alternative to a cervical interlaminar approach.

If small-caliber (20–22 gauge) needles are used, it is more likely to penetrate to an unknown depth, especially if a lateral view is not obtained. Unusual contrast cephlocaudal spread may represent intracord injection. Lateral views will confirm the location of the needle tip and injection site. Even small amounts of intracord injection may lead to syrinx formation and/or permanent cord injury.

Procedures for patients who cannot be visualized adequately with fluoroscopy or who have abnormal or questionable contrast distribution should be aborted. Obese patients and patients with posterior hardware are frequently difficult to visualize, and it is best to terminate the procedure and simply explain the safety concern to the patient. Contrast should be visualized in the epidural space, which is distinguishable from subarachnoid collection. Subdural contrast injection is of concern and sometimes requires multiple views to confirm. Intravascular injection may be detected by visualized contrast flow in a vein or artery but also may only be recognized by a lack of contrast collection in the area of the needle. Loculation of contrast may indicate additional risk of cord compression.

Patients with rapidly worsening pain, numbness, weakness, hyperreflexia, changes in bladder function, and other neurological symptoms should prompt a reevaluation and surgical evaluation when indicated. A suspected cord lesion from a hematoma or other compression requires emergent imaging. A contingency plan needs to be in place with the radiology department to short-circuit delays under these circumstances.

Active illness or disease, such as a febrile patient, local infection, or coagulopathy, are contraindications.

An INR (international normalized ratio), prothrombin time, partial thromboplastin time, platelet count, and function studies or bleeding times may be used as indicated.

Anticoagulation with prescription medications is becoming more common for patients with atrial fibrillation, coronary artery disease, cerebrovascular, peripheral vascular disease, and in the postoperative period. Patients need to discuss their periprocedure anticoagulation with the prescribing physician. Patients with mechanical valves, recent deep venous thrombosis, or other conditions prohibiting discontinuing anticoagulation may convert from coumadin to lovenox 5 days prior to the procedure, and lovenox can be held for 12 hours immediately prior to the procedure. An INR coagulation study can be performed if necessary. Plavix should be held for 1 week, and other prescription anticoagulants should be held for the appropriate time. Aspirin is stopped 1 week prior to a procedure and nonsteroidal anti-inflammatory drugs are discontinued 2 days prior to procedures. Patients should be asked about over-the-counter medication including herbal products such as ginko, garlic, ginseng, vitamin E, and so on.

Pregnant patients in the first trimester should be postponed if possible. Patients of reproductive age should be questioned about pregnancy, tested if necessary, and shielded from fluoroscopy.

Uncontrolled diabetes, hypertension, and heart failure may become exacerbated by corticosteroid administration. Patients with substance abuse, pain disorder with predominant psychological factors, or other psychiatric problems should be stabilized or referred for more appropriate care prior to interventional pain procedures. A history of allergy to contrast is not uncommon, and while not an absolute contraindication, it may lead to consideration of other options including catheter techniques.

EQUIPMENT

Patient monitoring equipment for sedation (pulse oximetry) and the procedure is an important consideration. The procedure must be performed under fluoroscopy guidance with appropriate radiation protection equipment including lead aprons, thyroid shields, and leaded gloves. A fluoroscopy unit with a freeze-frame screen and hard-copy printer is preferred.

Special attention to technique is necessary so that the physician's hand is never in the radiation field. Contrast should be water-soluble or nonionized such as Omnipaque 240 or other myelogram-quality contrast.

The field needs to be sterilized, after verifying that the patient is not allergic to iodine-containing solutions such as betadine. Use sterile fenistrated drapes or towels to maintain a sterile field.

It is recognized that the Tuohy needle is most commonly used; however, there is an increasing number of complications from the tip design, where the sharp cutting edge of the needle may cut not only into the epidural space but partially into the subdural space. Potential problems have been recognized for years.[20] If the dye spread is not recognized, the resultant combined subdural and epidural injection can be followed in 15–30 minutes by sudden onset of motor block and respiratory and cardiac arrest, especially if the patient is not observed. Similarly, in the upper cervical area if the needle tip ends up off midline, laceration of segmental arteries can be followed by a rapidly expanding arterial hematoma with pain, numbness, and evidence of cord compression. Midline interlaminar needle placement, especially without lateral fluoroscopic visualization, may result in intracord needle placement and injection.

The needle selection should be a 25–30-gauge needle for local anesthetic infiltration, an 18-gauge, B-bevel needle for opening up a hole through the skin, an 18-gauge, 3-1/2-inch R-X Coudé needle, and in some instances, the

18-gauge reverse R-X Coudé needle. Additional equipment includes a loss-of-resistance, low-friction syringe, preservative-free injectable saline, fentanyl, 0.25% bupivacaine or 0.2% ropivacaine, and 1% lidocaine for skin injection. A 4-inch extension tubing to be able to inject the contrast while the physician's hand is out of the field of radiation is useful.

PREPARATION OF PATIENT

Physical Examination

Examine the area for local infection and the ability to flex the cervical spine. The ability to assume and maintain the position for the procedure is important.

Imaging Studies

Plain films comprise a minimum in order to rule out bone destruction from tumor, spondylolisthesis, or other process. Also, the presence of spondylolisthesis with instability needs to be evaluated. Many patients have had CT or magnetic resonance imaging (MRI) studies, which are frequently helpful in determining the level of likely symptomatology.

Informed Consent

Written informed consent, which includes risks of paralysis, pain, numbness, bowel, bladder or sexual dysfunction, bleeding, and infection, needs to be obtained.

Preoperative Medication

Patients should be advised to avoid ingestion of food and drink, except for their usual medications, prior to procedures requiring sedation or local anesthetic with the potential for spinal block. Patients with diabetes should be placed early on the schedule to prevent losing blood sugar control.

Sedation is essential for some patients and some procedures. However, a responsive patient is a source of a wealth of information during a procedure. Patient history is the cornerstone of diagnosis, even during interventional pain procedures, and it should be surrendered cautiously. Preprocedure education, high-quality bedside manner, and meticulous local anesthetic placement can significantly reduce sedation requirements for many patients.

For preoperative medication, use the standard recommendations for conscious sedation by the American Society of Anesthesiologists.[21] Many patients do well with the combination of fentanyl and midazolam. Some patients desire general anesthesia, and the number of patients being anesthetized with propofol has grown as have the number of complications from the use of general anesthesia for these procedures. It may be safer to avoid general anesthesia as it masks warning symptoms during procedures.

PROCEDURE

Position of Patient and Physician

Proper patient and C-arm positioning is always worth the extra time it takes. The patient may be positioned in the sitting position, but the prone or lateral positions facilitate the use of fluoroscopy. The cervical spine should be flexed as tolerated to open the interlaminar space. The use of pillows or a cervicothoracic positioning device is helpful to avoid positions of cervical extension (Figures 8-2 and 8-3).

STERILE TECHNIQUE

Hand scrub and bactericidal foam are important practices for the physician prior to any procedure. Sterile preparation of the skin with Hibiclens followed by alcohol preparation and sterile drapes and strict technique are essential.

TECHNIQUE

Over the last few years, there has been a trend toward the paramedian approach, with the patient in the left lateral position rather than a prone or sitting position. The technique does call for some experience in making sure that the spinous process is in the midline for the AP visualization of the target. The image intensifier of the C-arm is placed in the cephalad position, and the x-ray tube of the

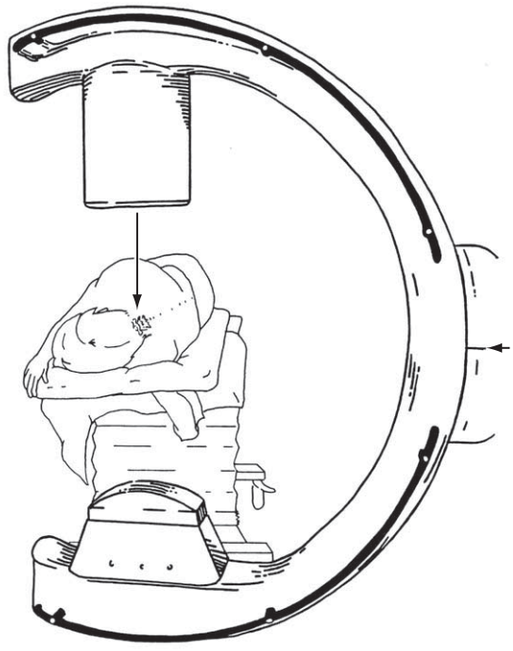


FIGURE 8–2

Patient is seen in a lateral decubitus position. The fluoroscopic C-arm is positioned for obtaining a lateral radiographic image. If it is difficult to keep the patient in a "true" lateral decubitus position, the fluoroscopic C-arm or the fluoroscopic table may be rotated obliquely to create an ideal lateral radiographic image.

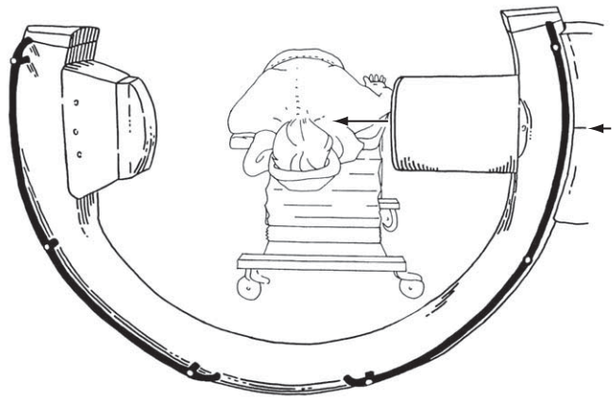


FIGURE 8–3

The typical position of the patient and the fluoroscopic C-arm for the prone position is shown. Note the flexion of the cervical spine for enhanced epidural needle placement.

C-arm is rotated in the caudad direction to visually open up the interspace for improved target selection, usually at C7, T1, or T1-T2 interspaces. The point of skin entry is 1-1/2 segments inferior, approximately 0.25 inch lateral to the midline. The skin is infiltrated with a 25–30-gauge needle. An 18-gauge needle is placed through the skin to open it up.

After local anesthetic infiltration, the needle used is an 18-gauge R-X Coudé needle, which has a wide-open tip (rather than the leading curved cutting end of the Tuohy needle). The 18-gauge R-X Coudé is advanced with a curved tip pointing medially crossing over the lamina (Figures 8-4 and 8-5). The direction that the needle travels can be influenced by rotation of the curved

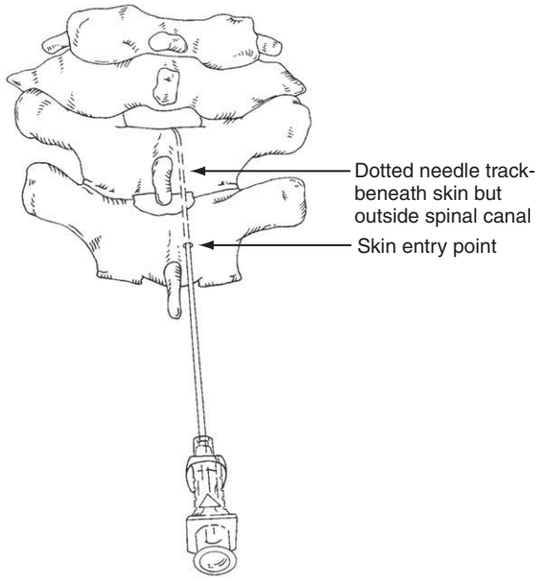


FIGURE 8–4

RX Coudé needle® with tip curving medially (Epimed Int.).

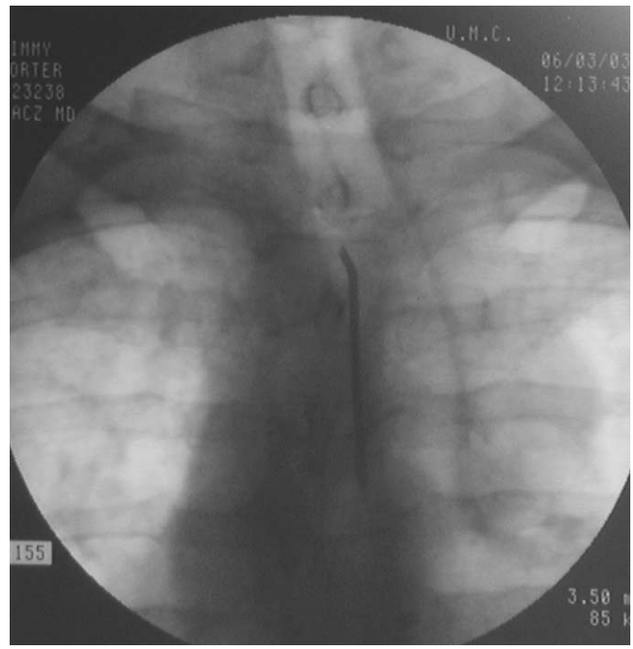


FIGURE 8–5

X-ray of RX Coudé needle® with tip curving medially (Epimed Int.)

tip. When the tip of the needle reaches on top of the T1 lamina, near the midline, the C-arm is rotated into the lateral view. In the lateral view, the shoulder does get in the way, but this problem is solved by obtaining a swimmer's view, which is rotating the image intensifier of the C-arm in the cephalad direction. The angulated lateral view shows a clear path to the base of the spinous process. The base of the spinous process appears as a "straight line," which is actually hockey stick J-shaped. This appearance comes from the four layers of bony cortex on the lamina on the outside, inside, inside, and outside again overlapping. Directly anterior to the straight line is the insertion of the ligamentum flavum. Using this radiographic anatomy, one can precisely determine the location of the ligamentum flavum whether there is any loss of resistance or not from the presence or absence of the gap in the ligamentum flavum. The needle at this point is advanced by rotating the tip of the needle anteriorly and advanced on the lateral fluoroscopic view to the level of the straight line (Figures 8-6 and 8-7). The tip of the R-X Coudé needle is parallel to the ligamentum flavum and slowly advanced until the loss of resistance is obtained (Figure 8-8). If a Tuohy needle was used, the tip of the needle has a sharp cutting end, but the working part of the lumen is farther posterior. The sharp tip penetrates rather easily through structures without necessarily giving the physician a chance to experience the various tissue planes.

Simultaneously, the loss of resistance technique and fluoroscopic guidance are used. Proper use of the loss-of-resistance technique has been lost in translation to

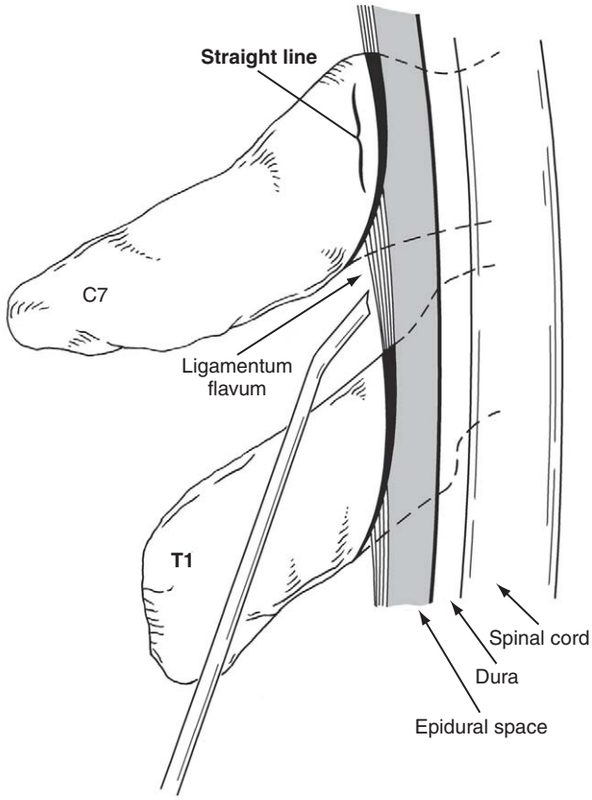


FIGURE 8–6
RX Coudé needle with tip rotated.

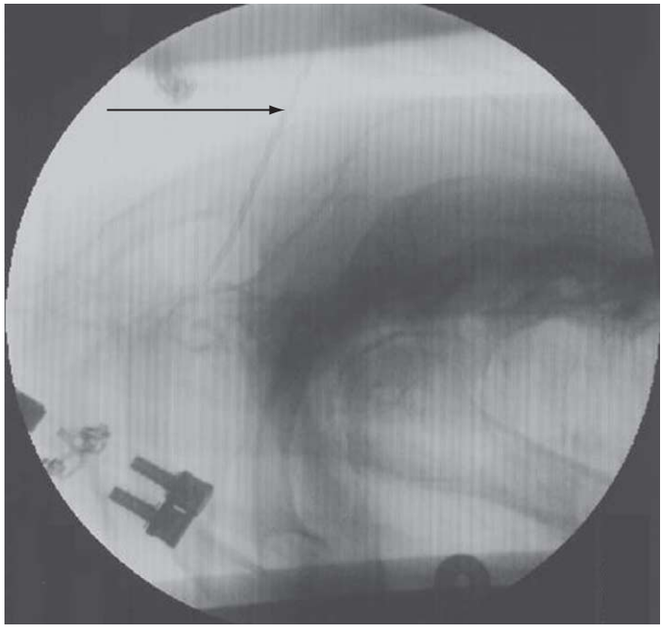


FIGURE 8–7
X-ray of RX Coudé needle with tip rotated. Arrow show the needle tip.

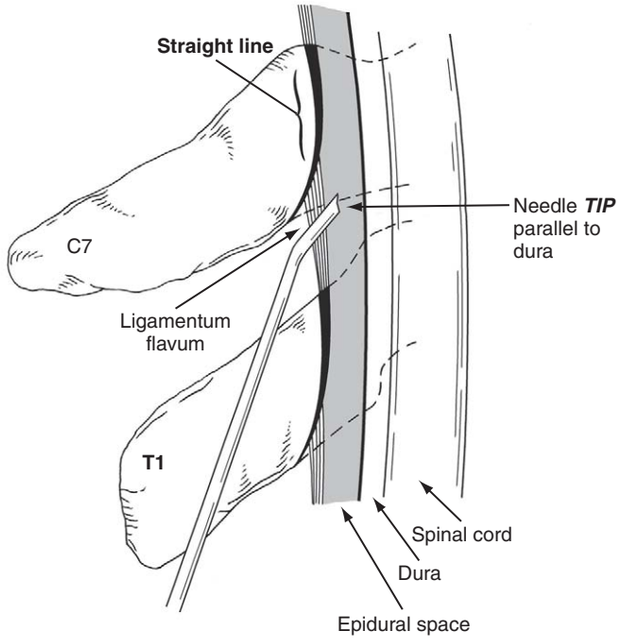


FIGURE 8–8
RX Coudé needle with tip just entering epidural space.

multiple specialties performing epidural procedures. The technique requires one hand on the syringe and, more importantly, the other hand advancing the needle while braced against the spine. This prevents needle advancement by the syringe or patient movement.

The needle is advanced until a loss of resistance or loss of bounce when the needle tip enters the epidural space. Aspiration is performed to test for blood or spinal fluid; however, contrast injection is useful to confirm epidural placement. Contrast injection with Omnipaque or other myelogram-grade contrast is performed to rule out superficial, subdural, subarachnoid, or intravascular injection.

At this point a 0.5 cc of water-soluble contrast is injected and the AP view taken, the contrast injection is observed to spread cephalad and caudad directions. The injected contrast verifies a safe epidural spread. The direction of the injection can be influenced by rotating the tip of the R-X Coudé needle 180 degrees and/or to the left or right, and/or by injecting a small amount of contrast (Figures 8-9 and 8-10). Lateral fluoroscopic visualization with swimmer's view is used to verify the location of injected contrast and the needle tip. Then a test dose injection of the mixture of local anesthetic and steroids can be carried out. Every injection should be proceeded by aspiration as the injected material may be into a vascular structure, whether it is a vein or artery, subarachnoid space, or even the spinal cord. The injected contrast and dye spread will verify the location of the injection. Injected volume should be in small aliquots of 2 cc of local anesthetic, 0.2% ropivacaine, or 0.25% bupivacaine. The total injected volume should be in the 4–6 cc range. The steroid used can be

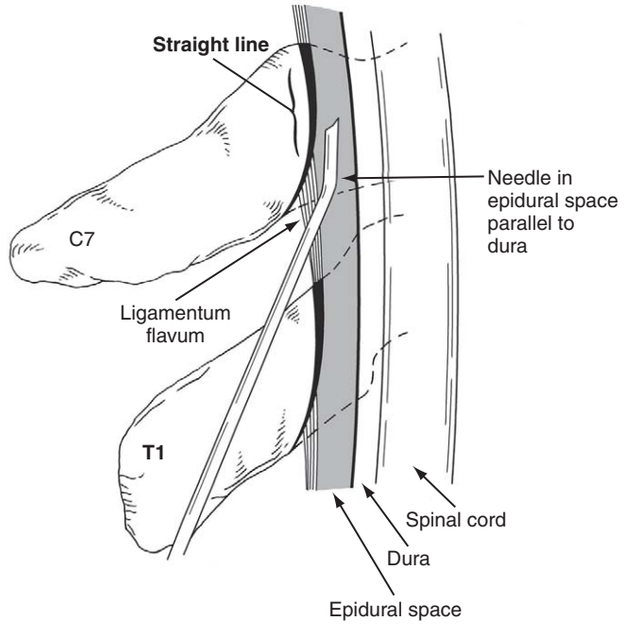


FIGURE 8–9
Needle in epidural space parallel to dura.

4 mg of dexamethasone (decadron), 40 mg of triamcinolone (Aristicort, Kenalog), or 40 mg of Depo-Medrol.

The injection should be made slowly, and if pain or other symptoms are associated with injection the syringe may be disconnected from the needle to allow back flow of injectate to decompress the epidural space. Painful injections may be a sign of compression and an indication to halt injection of further injectate.

The use of alcohol-free, nonparticulate corticosteroid preparations is advocated by some, although a number of positive studies employ depo preparations. These preparations do not have a Food and Drug Administration–approved labeling indication, but epidural steroid injections are a part of multiple treatment guidelines and studies justify their use.

The technique basically incorporates the principle of direction-depth-direction. In the first place, direct the needle toward the target. Second, rotate the C-arms laterally, establish the depth close to ligamentum flavum, and go back to AP view for possible loss of direction during the advancement of the needle to a depth for the third direction; thus, it is a three-dimensional technique. The needle can be rotated for directing the injection to the left or right. Alternative techniques would be the use of a reverse Coudé technique where paramedian approach is done for the first step, but now the needle is rotated cephalad so that the leading cutting edge is sliding underneath the lamina away from the dura. The depth of the injection is verified in the lateral view, and slight angle changes can be achieved to slide in underneath the "straight line" and loss of resistance or loss of bounce to establish the epidural location.

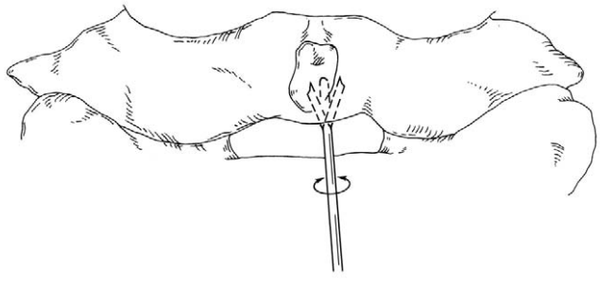


FIGURE 8–10
Coronal view. Needle tip rotated to enhance directional catheter placement or injection.

POSTPROCEDURE MONITORING

Qualified personnel should perform supervised bedside monitoring for 30–45 minutes following the procedure.

Vital signs and neurological checks should be monitored. Some use pulse oximetry and continuous EKG. Discharge instructions should include emergency physician contact telephone numbers and a follow-up appointment. Patients should be instructed to call if they experience worsened pain, numbness, weakness, fever, chills, or other new problems.

COMPLICATIONS

A complication that is difficult to identify involves loculation of hematoma to the lateral aspect of the cervical epidural space. If the interlaminar midline small-gauge Tuohy needle is aimed or accidentally enters the paramedian area, the tip of the needle can cut the artery that follows the nerve root in the posterior aspect of the neural foramen and epidural space to the spinal cord. This loculation in the presence of pathology can remain lateral and is visible but unimpressive on MRI except for compressing the blood supply going to the spinal cord producing a permanent myelopathy. Similarly, large-volume injections in the cervical epidural space may loculate and produce a Brown-Sequard syndrome with ipsilateral weakness and contralateral numbness with pain and temperature reduction.

The presence of degenerative arthritic changes, loss of disc height, bulging discs, and ligamentum flavum can distort and limit the space significantly. Reported complications followed the growing use of epidural steroid injections at the C5 level.

Epidural abscess formation is usually 10–15 days later and may present with fever or worsened neurologic complaints. An emergent MRI is the diagnostic test of choice, followed by cerebrospinal fluid (CSF) analysis, emergent staining for microscopic bacteria, culture, and sensitivity. The usual organism is a *Staphylococcus* sp. Bactericidal antibiotics active against *Staphylococcus* penetrate the CSF poorly, and an infectious disease specialist should be consulted to

manage medical treatment. Surgical consultation is also essential for abscess.

Dural puncture or tear with or without postdural puncture headache is another complication, which is more likely in patients with previous spinal surgery. Hydration and bed rest are first-line treatments. Caffeine and blood patch are two treatments for refractory postdural puncture headache. Rarely, patients with postdural puncture headache develop intracranial subdural hematomas. Other rare complications include air embolus, intra-arterial particulate steroid embolus, and ocular problems.

Risks from cervical epidural steroid injections overlap with other epidural techniques. Possible complications are summarized in Table 8-1.

CLINICAL PEARLS

In the cervical region, after traversing the skin and subcutaneous tissues, the styleted epidural needle impinges on the ligamentum nuchae, which runs vertically between the apices of the cervical spinous processes.[22] The ligamentum nuchae offers some resistance to the advancing needle. This ligament is dense enough to hold a needle in position even when the needle is released.

The interspinous ligament, which runs obliquely between the spinous processes, is encountered next and offers additional resistance to needle advancement. Because the interspinous ligament is contiguous with the ligamentum flavum, the operator may perceive a "false" LOR when the needle tip enters the space between the interspinous ligament and the ligamentum flavum. This phenomenon is more pronounced in the cervical region than in the lumbar region because the ligaments are less well defined.

A significant increase in resistance to needle advancement signals that the needle tip is impinging on the dense ligamentum flavum. Because the ligament is made up almost entirely of elastin fibers, resistance increases as the needle traverses the ligamentum flavum because of the drag of the ligament on the needle. A sudden LOR occurs as the needle tip enters the epidural space. There should be essentially no resistance to injecting the drug into the normal epidural space.

TABLE 8–1 Possible Complications after Cervical Epidural Steroid Injections

Cord injury
Nerve injury
Epidural hematoma
Cardiopulmonary arrest
Bowel or bladder loss of function
Wrong drug/dose
Allergic reaction
Dural puncture
Infection

INJECTION OF DRUGS

When satisfactory needle position is confirmed, a contrast-filled T-piece is carefully connected to the epidural needle. The use of the T-piece allows for aspiration and injection without movement of the needle. The syringe containing the drugs to be injected is attached to the T-piece. Gentle aspiration is carried out to identify CSF or blood.[23] Inadvertent dural puncture can occur in the best of hands, and careful observation for spinal fluid is mandatory.[24] If CSF is aspirated, the epidural block may by attempted at a different interspace. In this situation, drug doses should be adjusted accordingly because subarachnoid migration of drugs through the dural rent can occur. Aspiration of blood can result from either damage to veins during insertion of the needle into the cervical epidural space or, less commonly, IV placement of the needle.[23] If blood is aspirated, the needle should be rotated slightly and the aspiration test repeated. If no blood is present, incremental doses of local anesthetic and other drugs may be administered while the patient is monitored closely for signs of local anesthetic toxicity or untoward reactions to the other drugs. Pain, numbness, or other worrisome symptom during injection may signal neural compression, and it might be best to discontinue the injection.

EFFICACY

Stav et al.[25] compared epidural injections to intramuscular steroid and found significant benefit 1 year after injections. Conflicting results from meta-analyses of lumbar epidural steroid injections may be due to different criteria for study inclusion in each meta-analysis. The applicability to cervical spine syndromes is unknown; however, several recent studies are of interest. Buttermann[26] reported a randomized trial of lumbar epidural steroid injections versus surgical discectomy and found epidural steroid injections to have benefit. Patients had similar pain scores in the long term but a significant number of patients crossed over from the injection group to surgery. Wilson-Macdonald et al.[27] found that lumbar epidural steroid injections provided temporary pain relief but did not change the surgery rate in patients with disk herniations. However, all patients were considered to be surgical candidates prior to enrollment, so perhaps the surgery rate was not reduced for that reason. In another study, aqueous betamethasone had no effect 1 month after translaminar epidural injections in patients with disk herniations or spinal stenosis but Depo-Medrol was helpful.[28] Aqueous corticosteroid may distribute rapidly and have short-term local effect compared to depo preparations. On the 12-month follow-up, following the lysis procedure for spinal stenosis, epidural steroid injection followed by hypertonic saline was significantly effective.[29] It seems that epidural steroid injections help a number of patients tolerate pain from disk herniations long enough for the favorable natural history of disk herniations to result in improvement. Also, some patients will unavoidably choose surgery due to

inadequate pain relief. The use of epidural injections for other diagnoses is common, but fewer data are available regarding outcomes compared to control groups or other treatments. No head-to-head studies comparing nerve root blocks to transforaminal or interlaminar epidural injections have been reported for cervical radicular syndromes.

CERVICAL NEUROPLASTY

HISTORY

Cervical epidural blockade was first reported by Dogliotti in 1933.[30] Cervical neuroplasty is a derivative of caudal neuroplasty. It was conceived by Racz and Holubec in 1989 as a method to address the epidural fibrosis associated with failed neck surgery syndrome, disc bulges, and inflammation not responsive to single-shot epidural steroid injection.[31] Since that time, the technique has been used in the cervical region.[32]

The development of an epidural catheter with less risk of shearing, obstructing and migrating was essential. Because of the narrowness and potential for profound spinal cord injury, fluoroscopy was introduced as a safety tool for the pain physician. Naturally, the use of nonionic, water-soluble radiographic contrast was soon added to monitor vascular runoff. The technique has been modified to include selective nerve root stimulation for localization of the painful nerve root.[33] This modification is called *neural mapping*.

ANATOMY

The cervical spine consists of seven vertebrae and eight nerve roots. Cl and C2 nerve roots exit the central neuraxis posteriorly through the intralaminar space to innervate the posterior upper neck and scalp of the occipital region. Cervical nerve roots C3–C8 exit from neuroforamina. These differences are important for nerve-specific epidural catheter placement and adhesiolysis.

For the placement of the epidural needle, several key points need to be mentioned. The posterior wall of the bony canal is composed of the spinous process and lamina. Knowledge of these landmarks is important for radiographic identification. Just after the bone of the vertebrae is the ligamentum flavum. The interspinous ligament covers the interspinous spaces (Figure 8-1).

Cervical epidural catheters are frequently placed via a thoracic interlaminar space. The interlaminar space is smaller than at other spinal levels, and the spinous processes slope inferiorly. The lamina overlap as well, and these anatomical differences necessitate a skin entry point quite inferior to the level of choice. A paramedian approach to the thoracic epidural space is also frequently used and sometimes required. The ligamenta flava are thicker compared to the cervical levels, but care is needed to avoid the subdural space. Upper thoracic levels are likely to have anterior segmental arteries supplying the anterior spinal artery.

INDICATIONS

- Failed neck surgery syndrome
- Epidural fibrosis
- Cervical radiculopathy
- Cervical disc bulges
- Lateral recess stenosis
- Cervical osteophytes
- Chronic radicular pain syndromes such as radiculopathy and epidural adhesions
- Postradiation radiculopathy

CONTRAINDICATIONS

- Local or other infection
- Coagulopathies
- Unstable cervical spine
- Inability to lie in prone or in lateral decubitus position
- Significant central spinal stenosis
- Syrinx

EQUIPMENT

- 25-gauge, 3/4-inch infiltration needle
- 18-gauge, 1-1/2-inch needle
- 15-gauge Epimed RX Coudé epidural needle
- Epimed Tun-L-XL, 24-cm epidural catheter
- Loss-of-resistance syringe
- 3-ml syringe
- Two 10-ml syringes
- Needle holder
- 3-0 nylon on cutting needle
- Scissors

DRUGS

- 1.5% lidocaine for skin infiltration
- 2% preservative-free lidocaine
- 0.2% preservative-free levobupivacaine or ropivacaine
- 0.9% preservative-free normal saline
- 10% preservative-free hypertonic saline
- 1500 U of hyaluronidase
- Steroid of choice, such as triamcinolone, 40 mg dexamethasone 4 mg/ml, Depo-Medrol 40 ml

PREPARATION OF PATIENT

Written informed consent including risks of paralysis, numbness, weakness, pain, bowel, bladder, sexual dysfunction, bleeding, and infection should be obtained.

PATIENT POSITIONING

The standard used by our team is a left lateral decubitus position (Figure 8-2). In lateral decubitus, the potential of the patient moving into your needle is greatly diminished. By providing an outlet direction for movement, the patient tends to move away from the epidural needle. If the prone or sitting position is used, caution must be used to ensure that the patient does not move. Securing the patient may require extensive strapping and taping of the head and torso.

TECHNIQUE

Because of the potential for dural puncture and spinal cord injury, the upper thoracic approach (T1-T2) is used. After sterile preparation and maintaining sterile technique, the skin entry point is marked one to one and a half vertebral levels lower. Using the spinous process as midline, the site for skin entry is anesthetized with 1.5% lidocaine using a 25-gauge infiltration about 1 cm paramedial on the contralateral side. The entry of the epidural needle is facilitated with a puncture wound from an 18-gauge needle. Using fluoroscopy in the AP view with the C-arm rotated in the cephalad direction to open up the space of Tl-T2, a 15-gauge, 3-1/2-inch Epimed RX Coudé epidural needle is inserted toward the Tl-T2 interspace with the tip of the needle directed to midline (Figures 8-4 and 8-11). At the skin the needle will appear to be progressing in a 70- to 80-degree angle owing to the lordosis of the spine at that level. Once the direction of the needle is considered satisfactory and before the needle crosses the lamina on the AP view and into the interlaminar zone, advancement is stopped and the fluoroscope is changed to a lateral view to visualize the "straight line" of the base of the spinous process (Figures 8-12 and 8-13). In the lateral view, the

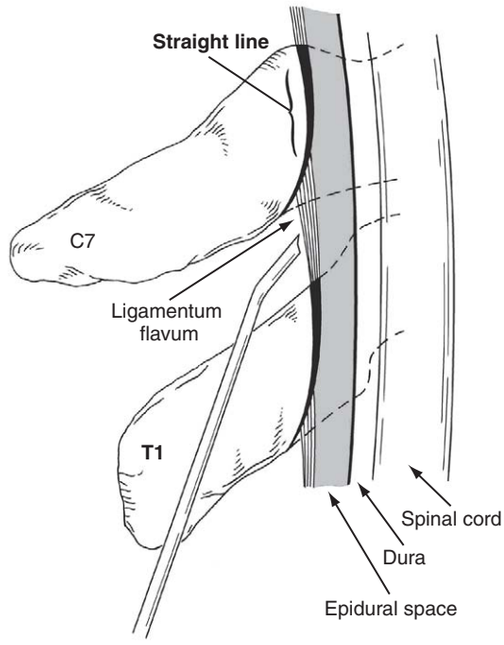


FIGURE 8–12
Lateral view of RX Coudé needle. Note the dark bold line is the "straight line" of the base of the spinous process.

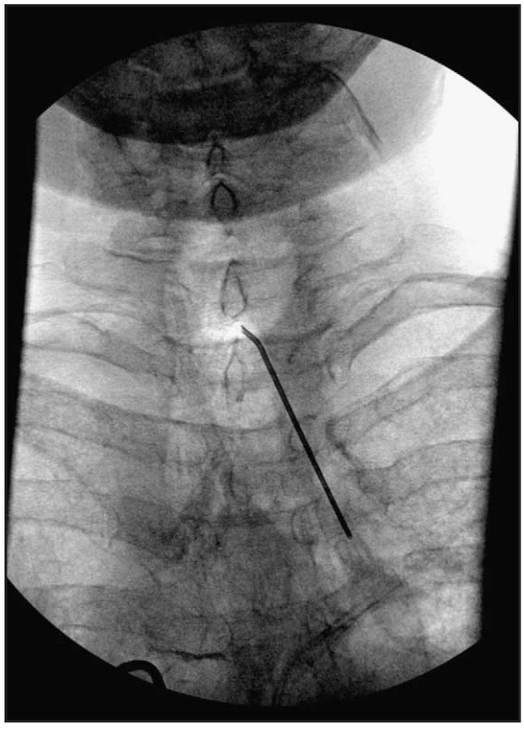


FIGURE 8–11
Posterior-anterior X-ray of RX Coudé needle with tip curved medially and directed at the C_7-T_1 inter laminar space.

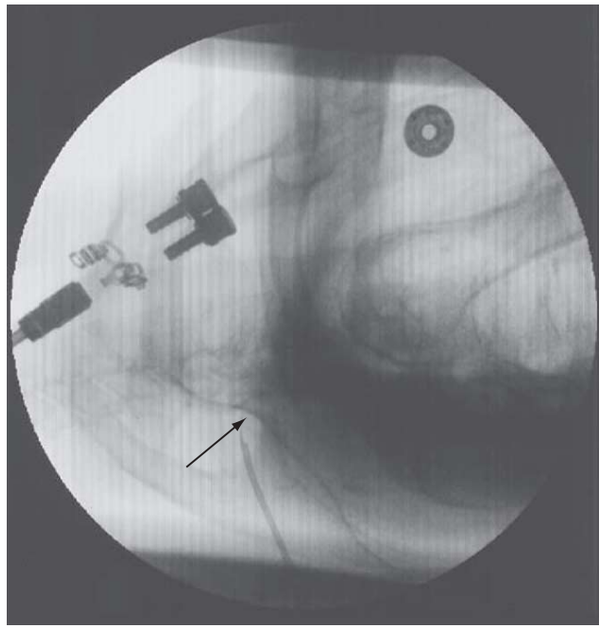


FIGURE 8–13
X-ray of RX Coudé needle. Arrow points to needle tip at the base of the spinous process.

shoulder does get in the way, but this problem is solved by obtaining a swimmer's view, which is rotating the image intensifier of the C-arm in the cephalad direction. The angulated lateral view shows a clear path to the base of the spinous process.

Just before this line is reached, the fluoroscope is returned into the AP view to confirm the proper direction of the needle. The final 2–3 mm of needle advancement is done with the loss-of-resistance technique (Figures 8-14). Our practice is to use 4 ml of preservative-free normal saline and 2 ml of air.

After the needle is in place, 0.5 ml of Omnipaque 240 contrast material is injected to demonstrate communication between the thoracic and cervical epidural spaces. The needle is rotated in a cephalolateral direction toward the target (Figure 8-15). A styleted, Bacitracin solution flushed, Epimed Tun-L-XL 24 catheter is prepared for introduction through the epidural needle. The stylet does not extend to the tip of the catheter but the catheter, tip is nevertheless checked to make sure it is soft. A 10-degree bend is placed at the distal 2 cm of the epidural catheter. The bend facilitates steering of the catheter. By using gentle steering with rotation of the epidural catheter, the tip is guided through the scarred area and specifically placed at that nerve root (Figures 8-16 and 8-17) and catheter placement for nucleus caudalis stimulation for facial pain (Figure 8-18).

If a clearly scarred nerve root cannot be found, the Tun-L-XL 24 catheter lends itself to electrostimulation. The inside of the catheter and tip is metallic, and the uninsulated stylet makes good contact with and can be withdrawn for clamping with alligator clip cables. The negative (black) electrode is placed on the catheter stylet, and the positive (red) alligator clamp is placed on the epidural needle or a 22-gauge needle placed into the skin as a ground. In these situations the catheter tip is placed at the lowest suspected nerve root, and 50-Hz stimulation is carried out to a point of paresthesia. The patient is asked if his or her pain is reproduced, and the response is recorded. The electrode is then moved to the next nerve root. If there is scarring and it is not possible to advance the catheter in the lateral epidural space, 0.5–1 ml of contrast is injected followed by hyaluronidase 1500U/10 ml saline. Then the catheter is injected slowly with hyaluronidase under continuous fluoroscopy. The main concern is loculation of injected contrast and hyaluronidase, and the slow injection continues until the contrast is seen exiting the neural foramen from the perineural space. If the patient complains of arm, neck, and possibly bilateral pain indicating loculation, the patient is asked to rotate the head and neck from left to right. Slow injection of hyaluronidase during rotation thus far has been followed by opening up the epidural space to the outside of the spinal canal in each case. If the contrast is not visible, an additional 0.5–1 ml of contrast is injected. If the catheter cannot be threaded to the lateral epidural space, it is possibly in the subarachnoid or subdural space. Subarachnoid placement will show clear fluid on aspiration. Subdural placement will show a railroad-like picture and extensive spread with a small volume. The danger of subdural catheter placement and local anesthetic injection is

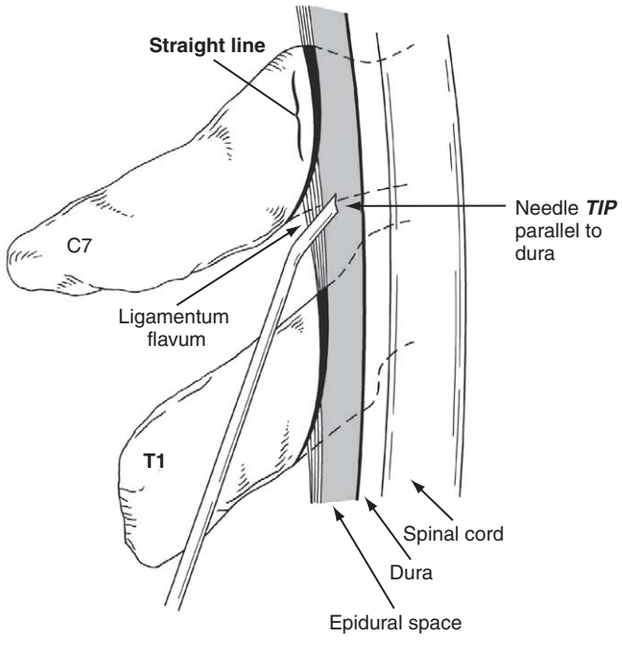


FIGURE 8–14
RX Coudé needle tip in epidural space.

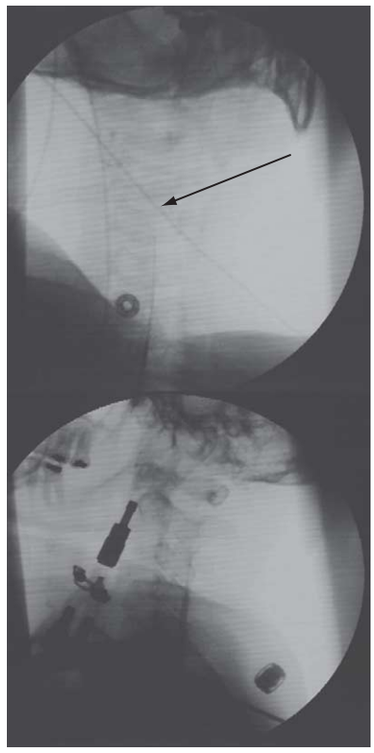


FIGURE 8–15
Epidural catheter placement for stimulation and neuroplasty.

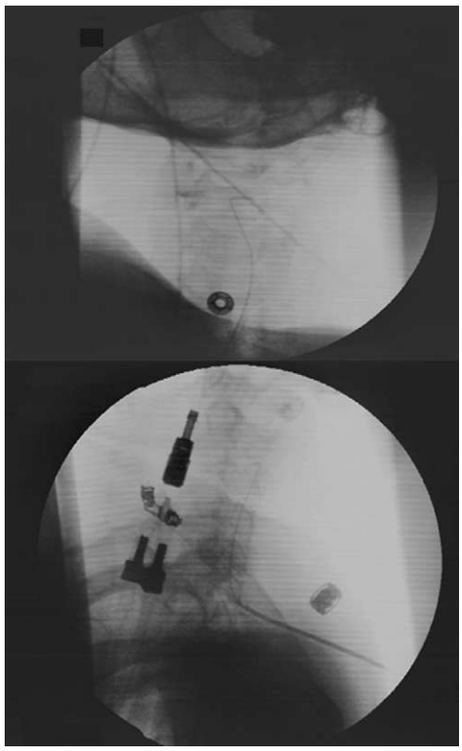


FIGURE 8–16
Epidural catheter placement for stimulation and neuroplasty.

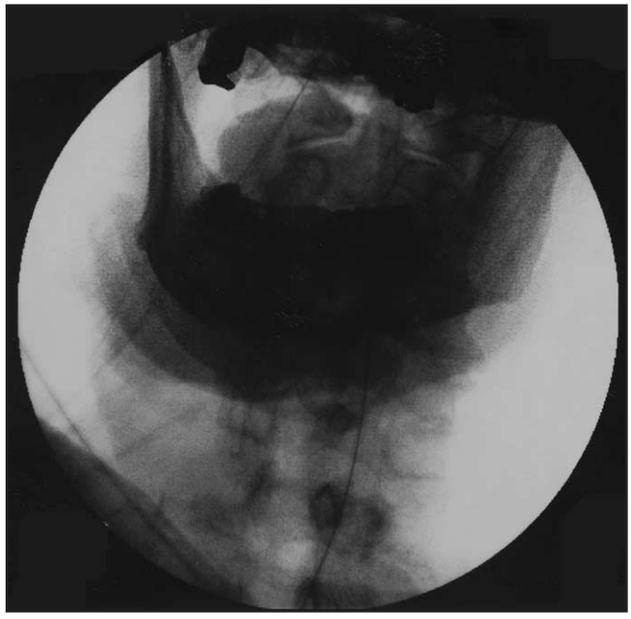


FIGURE 8–17
Epidural catheter placement for stimulation and neuroplasty.

a delayed onset of respiratory arrest in approximately 20 minutes from the onset of motor block.

If the catheter contrast injection shows venous run-off, the catheter is withdrawn and reinjected until contrast remains within the epidural space. The lateral spread from the epidural space also opens up venous run-off converting

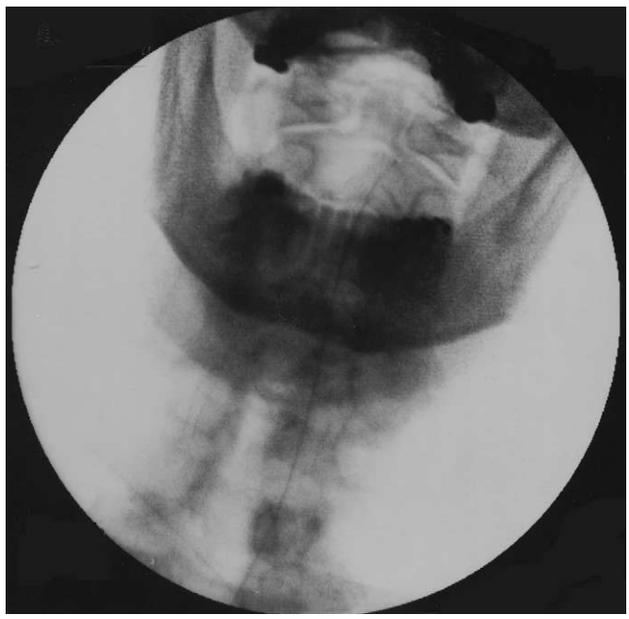


FIGURE 8–18
Catheter stimulation mapping for nucleus caudalis in patient with atypical facial pain C2-C3.

high-pressure veins to low-pressure veins. In over 20 years of using this technique, we have not seen a hematoma. All of the subdural catheter placements that we have experienced and have seen of others have been with the sharp-tipped epidural needles, such as the Tuohy or the spinal cord stimulator placement needles from each company. Recognition that the Tuohy needle's sharp tip can cut through the dura and a small part of the lumen is in the subdural space and some in the epidural space, the catheter will find the flap and opening and end up in the subdural space. Unless this is recognized by local anesthetic boluses, recognizing the problems can be delayed. The same problem can, of course, occur during single-shot epidural injections up and down the spinal canal.

Once the symptomatic nerve root is identified and none of the previously-described events occur, the catheter is left in the lateral epidural space. The catheter is aspirated, and 2-ml increments of 0.2% ropivacaine with 40 mg of triamcinolone or 4 mg of dexamethasone are injected with a period of observation between injections, to a total volume of 6 ml. Prior to local/steroid injection, if hyaluronidase was used, the total volume of injection is limited to the 4–6 ml range.

At this point the needle is removed, and the catheter is sutured in place and connected to sting-ray connector and 0.2-micron filter. Triple-antibiotic ointment is placed at the wound site; a slotted 2 × 2 gauze is placed over the catheter exit site. A small loop of the catheter is then held in place and covered, along with the gauze dressing, by a transparent surgical dressing. After the patient leaves the operating room, the bacterial filter is never removed; if there is accidental disconnect, the

catheter is removed from the patient and no further injections will be carried out.

After 30 minutes of observation for any evidence of motor block, the patient is placed in the lateral position with the painful side down and infusion of the 5 ml of preservative-free hypertonic (10%) saline is delivered over 20 minutes in the recovery room. Should the patient complain of pain, burning, or other noxious stimuli during the infusion of hypertonic saline, the infusion must be stopped and re-evaluated. Occasionally, 1–3 ml of additional 0.2% ropivacaine needs to be injected to anesthetize the newly exposed neural tissue. After 5 minutes, the hypertonic saline infusion can be restarted without further complaints. When the hypertonic saline infusion is complete, the epidural catheter is flushed with 1–2 ml of preservative-free normal saline. The epidural catheter is then recapped.

For neuroplasty stages 2 and 3, the epidural catheter is reaccessed. Aspiration of the catheter should be negative. Once again, 6 ml of local anesthetic (0.2% ropivacaine) is injected in divided doses, with attention given to possible intrathecal, subdural, or intravascular injection. The patient is then asked to lie in the lateral decubitus position with the painful side down to allow for gravitational spread to the affected nerve root. Similar to the previous hypertonic saline infusion, 5 ml is delivered over 20 minutes. Once the infusions are completed, 1–2 ml of preservative-free normal saline is used to flush the catheter. After the third infusion, the suture is cut and the intact epidural catheter is carefully removed. Another application of triple-antibiotic ointment is applied over the wound and covered by a bandage.

POSTPROCEDURE MONITORING

Patients should be monitored for at least 30 minutes following the procedure to observe any signs of subdural block.

COMPLICATIONS

The two major concerns are hematoma and loculation. Subdural block and intracord catheter placement are rare but of major concern. Infection is rare, but if it occurs it is usually during the first 2–4 weeks following the procedure.

EFFICACY

Most studies report benefit for lumbosacral pain syndromes rather than cervical pain syndromes. Gerdesmeyer et al.[34] reported dramatic improvement in Oswestry scores from 64 to 22. Igarashi et al.[35] reported better results in patients with single-level abnormalities compared to multilevel abnormalities. Heavner et al.[36] reported benefit in patients with chronic radicular pain syndromes.

The effect of nonspecific physical therapy, other than to the cervical region, was evaluated retrospectively following 227 cervical catheter patients with three daily injections of local anesthetic steroid and hypertonic saline.[32] The 96 patients who met the inclusion criteria for the study showed no statistically significant change with regards to the inclusion in the physical therapy group.

The catheter technique was found to be more effective in patients with radiculopathies than with facet joint–related pain, evidenced by shoulder pain rather than arm pain. The facet problem needed to be addressed later with nerve blocks of the medial branches at appropriate levels and possibly followed by radiofrequency (RF) lesioning of the nerves. These neuroflossing exercises clearly need to be evaluated in a prospective randomized manner. Therefore, more specific neuroflossing exercises were developed for patients with cervical radiculopathy (Figure 8-19).

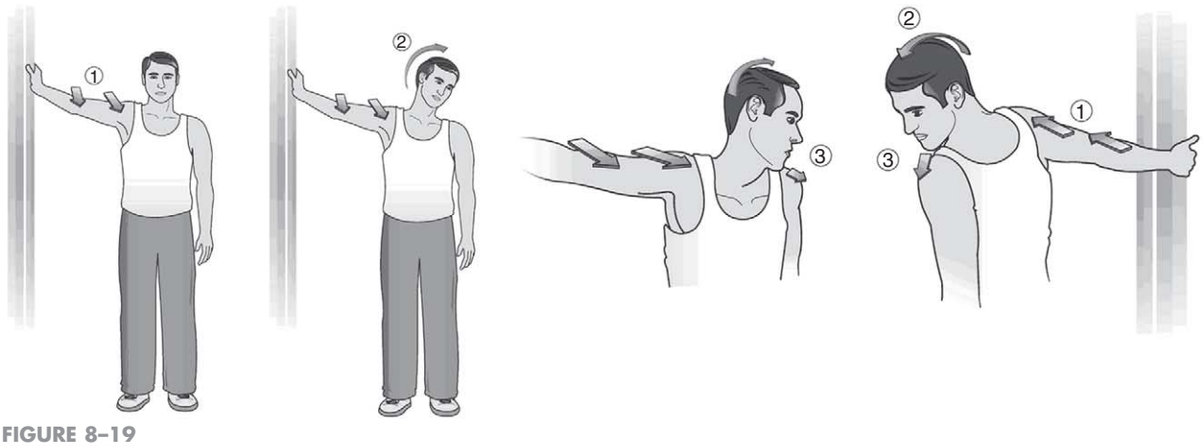


FIGURE 8–19

Step 1: Arm extended and elbow and shoulder pushed forward. Step 2: Head rotated to the opposite side. Step 3: Chin moves to the opposite shoulder stretching the scalene muscles and the strap muscles of the neck. The above triple-stretch rotation is held for at least 20 seconds minimally a twice a day. The above movement facilitates the brachial plexus nerve roots to regain mobility.

CERVICAL CORDOTOMY

HISTORY

Early in 1912, Spiller and Martín[37] concluded that based on the clinical model of Brown-Sequard studies of cord hemisection, despite the fact that pain physiology at that time was poorly understood at the spinal cord level, they performed a cordotomy through a laminectomy, with incision of the antelateral cord in a patient with pain caused by a tuberculoma of the cord. Since then, the operation of open cordotomy has become a standard surgical procedure for the relief of certain types of pain. In later studies, various authors used different-sized lesions in the anterolateral quadrant in the attempt to alleviate contralateral pain. At first, open cordotomy was performed in the thoracic area, but by 1932 open cordotomy had been carried out in the high cervical area as well. Although open cordotomy was effective in many patients,[38–40] the advent of percutaneous cordotomy enabled less-invasive treatment.

Many modifications and technical refinements appeared, like percutaneously applied strontium-90 in the C1-C2 space by Mullan et al.,[41] electrolytic lesions,[42] or impedance monitoring to detect cord penetration.[43] Onofrio[44] added imaging of electrode position by the use of myelography for guidance. Rosomoff and colleagues[45] introduced RF lesion-making cervical cordotomy. More recently, computed tomography-guided cordotomy has been described by Kanpolat et al.,[46] for a more accurate localization of the electrode system in a specific part of the spinothalamic tract.

ANATOMY

The interruption of pain fibers in the spinothalamic tract, the major ascending pathway for information about pain and temperature, leads to sensory deficits to skinfold pinch and temperature sensitivity (Figure 8-20).

Afferent information from Rexed's lamina II is transmitted to second-order projection neurons in laminae IV, V, and VI, the neurons of which also receive some direct innervation from the terminals of the first-order neurons. The axons of these second-order neurons in laminae brain stem IV-VI cross the midline and ascend all the way to the brainstem and thalamus in the anterolateral quadrant of the contralateral half of the spinal cord. These fibers, together with axons from second-order lamina I neurons, form the spinothalamic tract or anterolateral system (Figure 8-21). The mechanosensory pathway is located in the dorsal aspect of the cord and is referred to as the dorsal column-medial lemniscus system.

The location of the spinothalamic tract is particularly important clinically because of the characteristic sensory deficits that follow certain spinal cord injuries and is essential for the cordotomy procedure. Since the pathways for pain and temperature cross the midline to ascend on the opposite side of the cord, diminished sensation of pain below the lesion will be observed on the side opposite the lesion.

According to topographic representation, sacral segments are mostly located posterolaterally and cervical segments more medially and anteriorly within the lateral spinothalamic tract. In addition to the basic topographical organization, there is also a large amount of scattering of the fibers from different segments through the area of the spinothalamic complex.

The fibers responsible for pain sensation at C2 lie between the line of the dentate ligament dorsally and a line drawn perpendicularly from the medial angle of the ventral horn. In close proximity to the spinothalamic tract lie many other ascending and descending tracts, damage to which results in many complications associated with cordotomy. The corticospinal tract is located posteriorly, and injury to it may produce ataxia of the ipsilateral arm. Fibers mediating respiration lie adjacent to the anterior horn in close proximity to the cervical spinothalamic fibers, and its lesion can cause respiratory dysfunction.

The *spinal dura mater* forms a loose sheath around the spinal cord. It is separated from the wall of the vertebral canal by the epidural space, which contains a quantity of loose areolar tissue and a plexus of veins. On each side are the double openings that transmit the two roots of the corresponding spinal nerve, the dura mater being continued in the form of tubular prolongations on them as they pass through the intervertebral foramina.

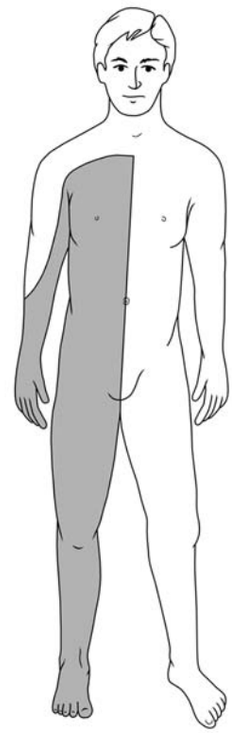


FIGURE 8–20
Analgesic effect of C1-C2 cordotomy on the contralateral hemibody.

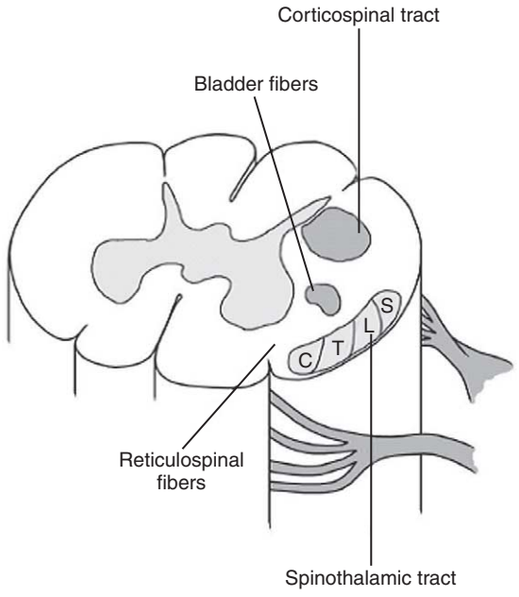


FIGURE 8–21
C1-C2 spinal cord section and topographical distribution of tracts.

The *ligamentum denticulatum* (dentate ligament) is a narrow fibrous band situated on either side of the spinal cord throughout its entire length that separates the anterior from the posterior nerve roots. Its medial border is continuous with the pia mater at the side of the cord. Its lateral border presents a series of triangular tooth-like processes, the points of which are fixed at intervals to the dura mater. There are 21 of these processes, on each side, the first being attached to the dura mater, opposite the margin of the foramen magnum, between the vertebral artery and the hypoglossal nerve; and the last near the lower end of the medulla spinalis.

INDICATIONS

The procedure is restricted to severe unilateral pain due to cancer and refractory to opioids and coanalgesics. According to various authors, duration of effects rarely lasts more than 2 years.

The indications for this procedure have narrowed over the past decade because of the potential to produce additional injuries to the nervous system and painful states more severe than the original problem. Percutaneous cervical cordotomy (PCC) is indicated only in patients in which noninvasive methods of pain control have been attempted without success.

PCC deserves a valuable place for the treatment of patients suffering from cancer in advanced stages with severe unilateral neuropathic or incidental pain. Indications include pleural, chest wall, and intercostal nerve involvement of malignant mesotheliomas; lung cancer with pain radiating to the neck, chest, and arm; carcinoma of the breast; and lumbosacral plexopathy due to cancerous invasion, among others.

CONTRAINDICATIONS

- Medical contraindications to surgery (e.g., bleeding diatheses, infection)
- Midline trunk pain
- Perineal pain
- Respiratory impairment
- Patient's pain should be below the C4-C5 dermatomes for high cervical cordotomy
- Pain should predominate on one side of the body

EQUIPMENT

- A special cordotomy needle electrode system with temperature monitoring (Diros Technology, the Owl Cordotomy system, or Valleylab KCTE Kit, 20–22-gauge thin-wall needle with Teflon hub with an electronically sharpened 2-mm tip projecting beyond the tubing, matched with an 18-gauge lumbar puncture needle; 2 mm is the depth where the spinothalamic tract ascends)
- Compatible RF generator (measuring electrical impedance, temperature, voltage, seconds, and delivering of electrical stimulation from 0–10 V over a range of 2–100 Hz)
- Special head holder
- X-ray guidance
- Surgical theater and resuscitation equipment

POSITIONING OF PATIENT

The patient is positioned supine with a special head holder to optimize positioning (Figure 8-22 A&B). The head is placed in a strictly AP plane with the cervical spine horizontal in order to ease myelogram dye to trap for as long as possible. The procedure can be performed with the patient lying flat on a standard operating table. X-rays are projected to obtain AP and lateral views.

Position is optimal when the C1-C2 interspace is arranged in a strictly lateral image in order to allow tunnel vision of the cordotomy electrode when approaching the spinal canal horizontally.

ANESTHESIA

- IV sedation in the operating room.
- The patient must be admitted to the operating room without prior sedation to avoid impaired cooperation.
- Medications for pain control should be administered before and during the procedure.
- Prophylactic antibiotherapy IV is mandatory.

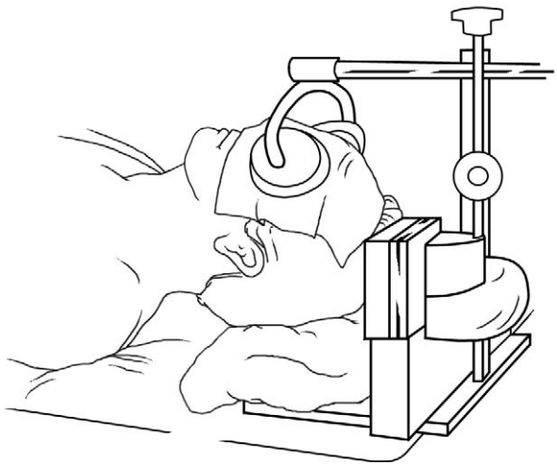


FIGURE 8–22
Patient position for percutaneous cervical cordotomy C2 with Rosomoff's head holder.

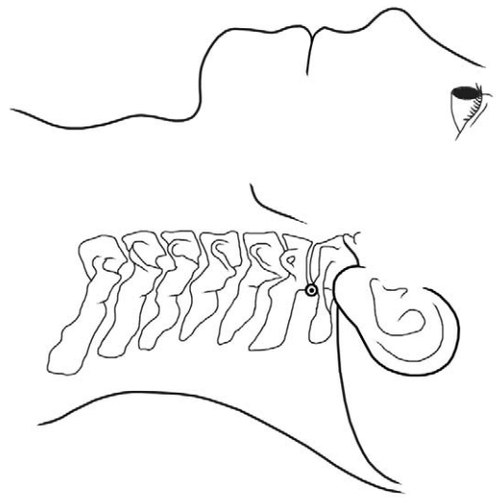


FIGURE 8–23
Line drawing of lateral X-ray position and entry point of cannula at left C1-C2 level.

- Local anesthesia of skin and deep muscle structures.
- General anesthesia should be administered in selected populations (children, elderly, or confused patients). In these instances, CT-guided cordotomy should be performed.

PROCEDURE

Only the unilateral x-ray–guided percutaneous cervical technique will be described. CT-guided technique may also be performed.[47,48] Bilateral procedures can be used in selected cases of bilateral pain, with increased risk.[49]

Radiologic Localization

After suitable preparation of the skin and, if necessary, wetting and retraction of the hair, the tip of the puncture needle is positioned horizontally, and with x-ray guidance, a true image of the C1-C2 interspace is obtained (Figure 8-23). The C1-C2 interspace should be placed in the center of the radiographic image in order to avoid double contours. The spinothalamic tract is located anterior to the dentate ligament at approximately the midpoint of the AP extent of the spinal canal. The anteromedial part of the lateral spinothalamic tract must be targeted (Figure 8-24).

After local anesthesia (lidocaine 2%), an RF needle with a 1-mm active tip is advanced horizontally, and ligamentum flavum and dura are penetrated. At this point, the stylette of the lumbar puncture needle is periodically withdrawn to check for flow of CSF (Figure 8-25), which is normally encountered at a mean depth of 5.7 (±0.3) cm from the skin in men and 5.2 (±0.2) cm in women.[47] Loss of CSF should be avoided by locking the electrode inside the needle or lumbar cannula.

After obtaining CSF, a nonionic (iohexol) water-soluble contrast medium is injected[50] (oil-based dye for myelography is increasingly difficult to obtain). The surgeon can use a length of intravenous extension tubing long enough to keep his hands out of the radiation field while injecting under fluoroscopy. When a sufficient amount of contrast is present in the subarachnoid space, the radiographic image can be "trapped" on the screen for review (Figure 8-26). Well-defined linear images such as the ventral and dorsal root lines may be confused with the dentate ligament, and contralateral structures may be visualized as well, adding to the confusion. If the fine line of the dentate ligament is not seen, the cannula must be repositioned and a second injection of dye can be made. The tip of the needle should be immediately anterior to the dentate ligament (Figure 8-27). Failure to visualize the dentate ligament usually means that the needle is too posterior. It should be withdrawn and redirected more anterior. The electrode is connected, followed by stimulation. The electrode is then advanced, and when it penetrates the cord, the electrical impedance increases dramatically as it leaves the spinal fluid and enters neural tissue.

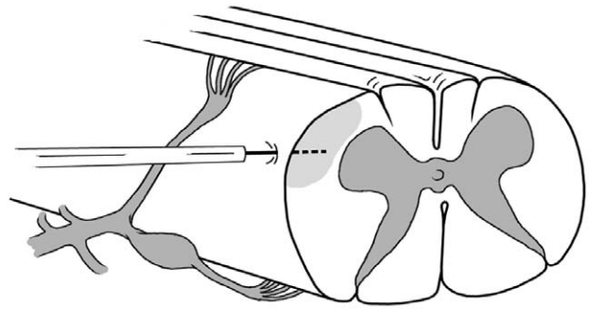


FIGURE 8–24
Cannula position in the spinal cord.

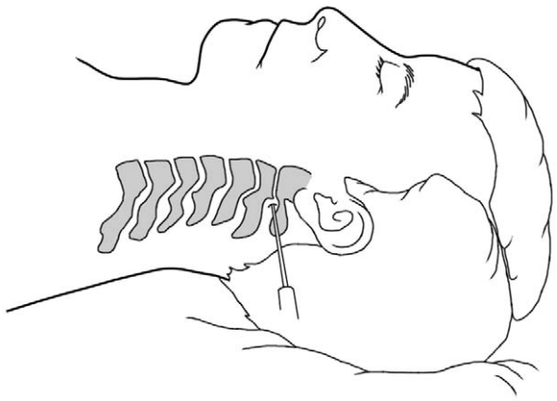
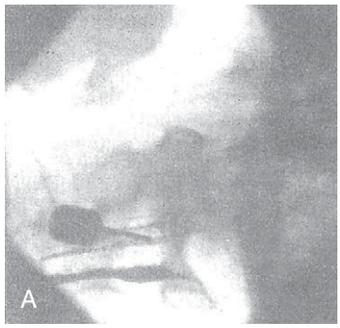


FIGURE 8–25
Cannula entry point at C_1-C_2 level.



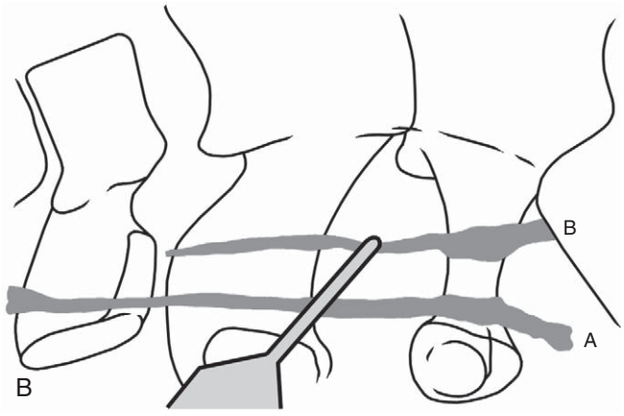


FIGURE 8–26
(A) The radiograph showing a needle directed towards contralateral dentate ligament. (B) A. Ipsalteral L.D. B. Contralateral *ligamentum denticulatum* (dentate)–L.D. After injection od dye (lohexol) in the subarachnoid space.

Impedance Monitoring

Impedance of CSF is approximately 400 Ω (ohms), and of spinal cord 800–1200 Ω or more as the cord is impaled. Physiologic confirmation of electrode positioning is achieved via threshold electrical stimulation at 2 Hz and 100 Hz. The lower-frequency stimulation helps to avoid placement of the electrode too near to the corticospinal tract.

The depth of the electrode in the cord can be measured with an AP x-ray position aimed at the odontoid process through the open mouth. When properly positioned in the spinothalamic tract, the tip should lie at/beyond the midline of the dens, reflecting cord displacement by the thrust of the needle (Figure 8-28).

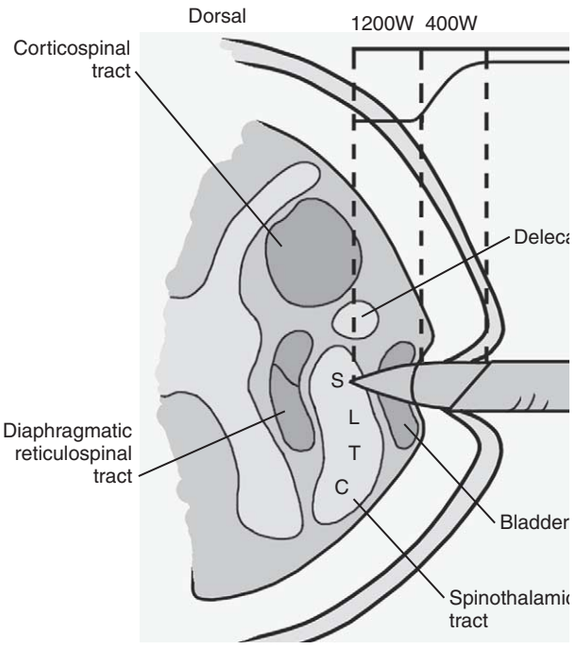


FIGURE 8–27
Entry point of cannula in the anterolateral position of spinal cord.

Contractions in ipsilateral neck or upper limb at low amplitudes indicate a too posteriorly placed electrode. Higher frequency stimulation should produce sensory alterations but no motor tetanization of the electrode if it is in a proper position. At 100 Hz, patients experience contralateral paresthesias, often with a thermal element, followed by a burning pain at higher currents. Occasionally it is possible to accurately select the exact portion of the spinothalamic tract to be lesioned in this manner. More typically, the patient will not be able to precisely localize the stimulation. All that is necessary for a successful cordotomy is to show that the electrode is away from the corticospinal tract and within the spinothalamic tract.

Lesion Making

Since meningeal cauterization elicits pain, sedation must be increased prior to the lesion. The RF lesion is made by raising the temperature of the electrode tip to 60°C for 60 seconds. Based on the depth of analgesia, the lesion can be enlarged by raising the temperature at 70°C or by adjusting the electrode position. Usually two, but only a maximum of three, lesions are made. After completion of the lesion, the level of analgesia to pinprick and deep pain should extend above the level of the patient's pain. Neurologic deficits or ipsilateral hypoventilation should not be present and are an indication to stop the procedure.

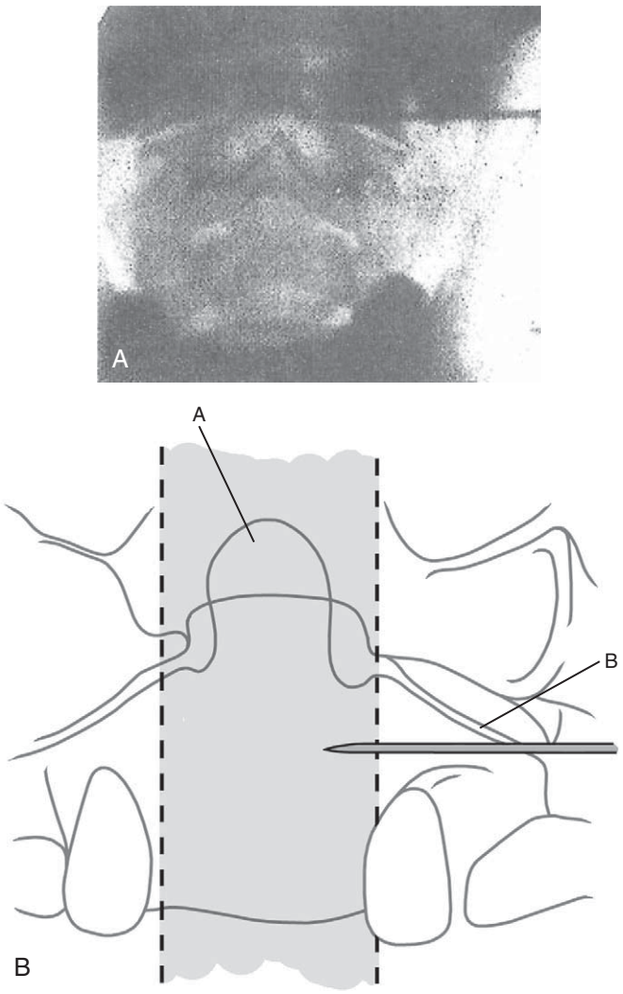


FIGURE 8–28

(A) This AP radiograph at the C_1-C_2 level shows the correct position of the needle for cervical cordotomy. (B) Anterior posterior X-ray view at C_1-C_2 level and cannula tip. (A) Odon toid process. Atlas-axis joint.

After unilateral cordotomy, patients should be monitored for spinal headache, bladder function, respiratory function, and presence of hemiparesis. Many authors state that cordotomy may be repeated with low risk in case of pain recurrence.[48]

COMPLICATIONS

- Complications are much less frequent in unilateral procedures.
- Horner's syndrome (usually transient).
- Mortality rates 0–9% (due in most cases to irreversible postoperative respiratory dysfunction).[51]
- Significant paresis 2–11%.
- Worsening of a pre-existing motor deficit can also occur. This complication is attributed to damage of the corticospinal tract in the lateral white-matter funiculus of the spinal cord. This is presumably due to edema and therefore reversible.
- Bladder dysfunction (in bilateral procedures, 22%) requiring catheterization. It is often transient.
- Respiratory failure (2.6% in unilateral cordotomy).
- Unpleasant abnormal sensations on cutaneous stimulation within the analgesic area are common (hyperpathia, dysesthesia). The likelihood increases with long survival periods (>9 months), and intensely painful dysesthesia may appear.
- New pain in the painless side, or intensification of previously existing pain on the side ipsilateral to the cordotomy, is a less common complication.

EFFICACY

Published reports show a great variation in successful outcome. Tasker's[53] series of 380 cases showed a successful lesion in 95% of cases (lesion within spinothalamic tract, analgesia over area desired), and 88% had total relief of pain. After a 3-month follow-up, only 72% had total relief of pain, but 84% had significant relief. In this series, bilateral cordotomies had 71% of significant relief of pain.

In Sindou and Daher's[54] review of 37 series in the literature comprising 5770 cordotomy cases, 2022 of which were cancer pain patients, early pain relief was achieved in 30–97% of cases. Long-term pain relief was experienced by 75% of patients at 6 months and by 40% after 1 year.

Lahuerta and colleagues[50] obtained 66% complete pain relief in a series of 146 patients and 23% partial relief, with no relief in 11%. The alleviation of pain was more marked in patients with malignant diseases. In general, efficacy drops within 1 year to 40%.[55] Success for percutaneous cervical cordotomy is associated with selection of patients, accuracy of the surgical technique, and criteria for assessing the follow-up persistence of pain.

CERVICAL TRANSFORAMINAL INJECTIONS

HISTORY

The transforaminal approach has the theoretical advantage of more reliable placement of injectate into the anterolateral epidural space compared to single-shot interlaminar epidural steroid injections. Stojanovic et al.[56] reported that only 28% of cervical interlaminar injections result in contrast spreading to the ventral epidural space. A variety of techniques have been described with the final needle tip placement being in a variety of positions in the sagittal plane and in a variety of depths within the foramen.

Transforaminal epidural steroid injections were described in 1992 by Derby and colleagues,[57] and in 1999 by Bardense et al.[58] Rathmell et al.[58] reported their technique in 2004.[59]

Transforaminal injections differ from selective nerve root blocks although some overlap in risks exists. Complications have been reported, and some are of a most significant nature including death from vertebral artery perforation.[60]

Other major neurologic injury has been reported, thought by some to be related to particulate corticosteroid injected into arteries supplying the spinal cord or brain. There may be a trend toward the use of blunt needles for cervical transforaminal injections as they are less likely to penetrate nerves and arteries. All transforaminal techniques employ fluoroscopy or other imaging technique. The most recent relevant information is the presence of arteries in the posterior neural foramen. It had been thought that the posterior foramen was a safe area, but Huntoon[61] reported this new information regarding vascular supply to the cord.

ANATOMY

The most relevant anatomic considerations include the arterial vascularity of the foramen itself and the vertebral artery, which is just anterior to the foramen in the third through sixth cervical levels. In a series of 504 transforaminal injections, Furman and Giovanniello[62] reported that 19.4% had intravascular injections during contrast injection.

The mid and lower cervical neuroforamina are bounded by a superior and inferior vertebral notch in the pedicles above and below. The cervical pedicle extends posterior and lateral from the body, which aligns the foramen in an anterolateral direction. The transverse process extends in the same anterolateral direction and surrounds the foramen transversarium through which the vertebral artery passes.

The deep ascending cervical and vertebral arteries supply anterior segmental medullary arteries that traverse the cervical neuroforamen to supply the anterior spinal artery, as well as posterior segmental medullary arteries.

INDICATIONS

Diagnostic, prognostic, and therapeutic indications exist. Most indications are for nonsurgical cervical radicular pain syndromes such as disk herniations and spinal stenosis. Many prefer transforaminal injections rather than interlaminar injections if a diagnostic question exists with regard to multilevel radiographic pathology.

CONTRAINDI CATIONS

- Severe spinal stenosis, syrinx, spinal instability.
- Infection.
- Pathological or therapeutic disorder of coagulation.
- Patients with severe foraminal stenosis or a history of foraminotomy may be difficult to block with a transforaminal approach. Some clinicians would advocate a catheter technique or nerve root block as alternative procedures.
- Patients who cannot be visualized adequately due to their anatomy or other reasons should receive alternative treatments.
- A history of contrast allergy is a difficult clinical situation, especially with transforaminal injections, since contrast use is so important to the technique. Iodine is an essential nutrient and is too small to be allergenic by itself. Iodine allergies do not correlate well with allergies to contrast, and it is important to explore the history of allergy before the procedure.

PREPARATION OF PATIENT

Written informed consent, including risks of paralysis, numbness, pain, bowel, bladder, sexual dysfunction, bleeding, and infection, should be obtained.

Radiographic imaging of the cervical spine is a most important component of the evaluation of a patient with a cervical radicular syndrome. Correlating the patient's symptomatology and physical examination finding with imaging studies is critical in determining the level for transforaminal procedures.

Patients should be instructed to avoid ingestion of food and drink (NPO) for procedures requiring significant sedation or local anesthetic that could potentially reach the epidural or intrathecal space.

EQUIPMENT

- Lidocaine
- 25-gauge infiltration needle
- Syringes
- T-connecter
- Introducing IV cannula 18-gauge, for 25-gauge, blunt-curve tip needle
- Introducing IV cannula 16-gauge, for 22-gauge, blunt-curve tip needle
- Freeze-frame fluoroscopy
- Omnipaque 240

TECHNIQUE

The patient is positioned in the supine position. The posterior border of the transverse processes are palpated and marked with a skin marker (Figures 8-29 and 8-30). Fluoroscopy is used throughout the procedure. To identify the target site, a 30-degree from horizontal fluoroscopic picture is obtained to visualize the neural foramina. Using a metal pointer in the same view to find the neural foramen, place a cross mark with the skin marker on the previous mark for the transverse process line (Figures 8-32) . This will be the skin entry point for the introducer cannula. It is important to note that there are no vital structures in front of the needle coming in from the lateral side. Extra caution needs to be exercised at the C6 and C7 nerve root as the dome of the lung may get in the way, and a superior to inferior direction needs to be adopted from the C6 level inferiorly.

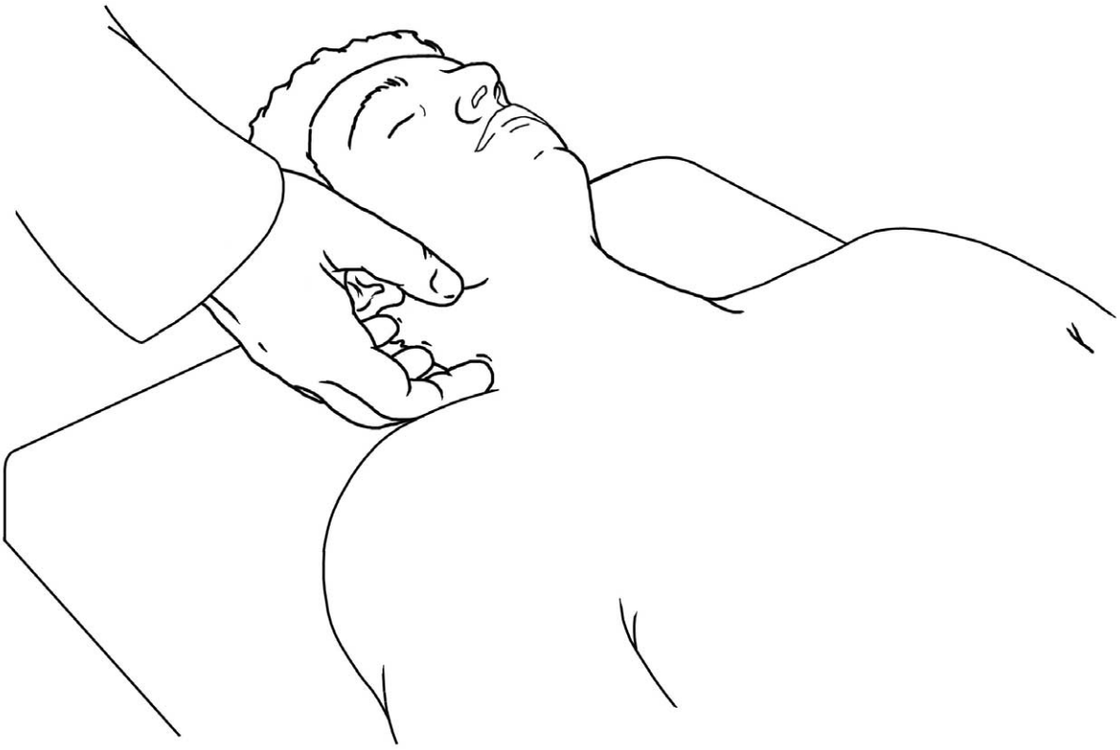


FIGURE 8–29
Mark posterior border of transverse process.

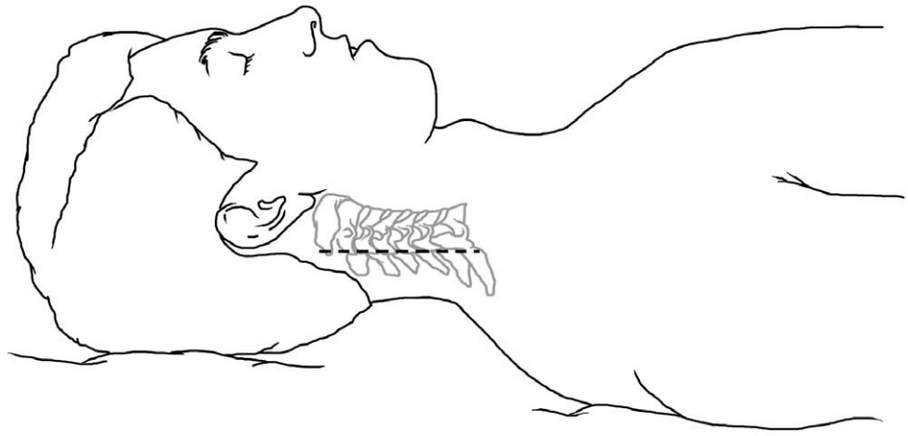


FIGURE 8–30
Find target neural foramen using 30-degree oblique fluoroscopy view and mark skin.

The skin puncture site is infiltrated with local anesthetic. The introducer IV cannula is placed horizontally toward the target, which is the lateral transverse process (Figures 8-33 and 8-34). AP fluoroscopic visualization is used so that the needle does not go beyond the lateral border.

Caution must be exercised because if the needle appears to be medial to the lateral border, the needle could have entered the spinal canal via the neural foramen or posteriorally through the interlaminar space. Lateral fluoroscopic visualization will confirm the direction of the

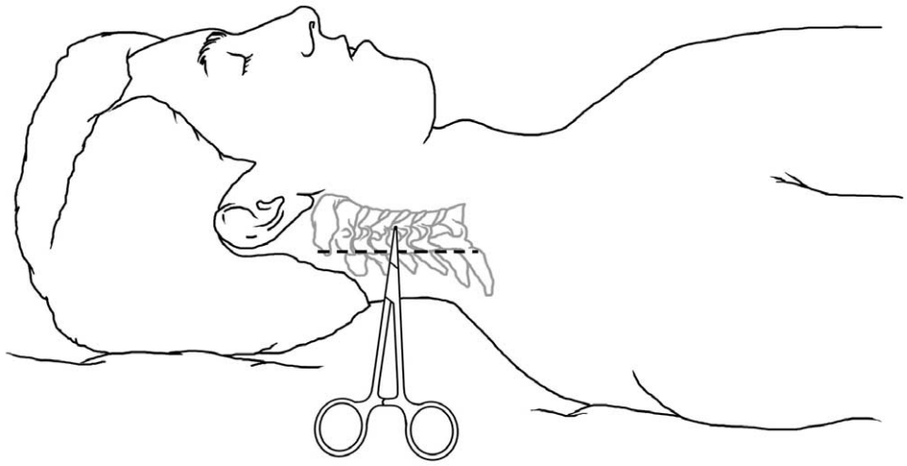


FIGURE 8–31
Anteroposterior view with marker selecting level.

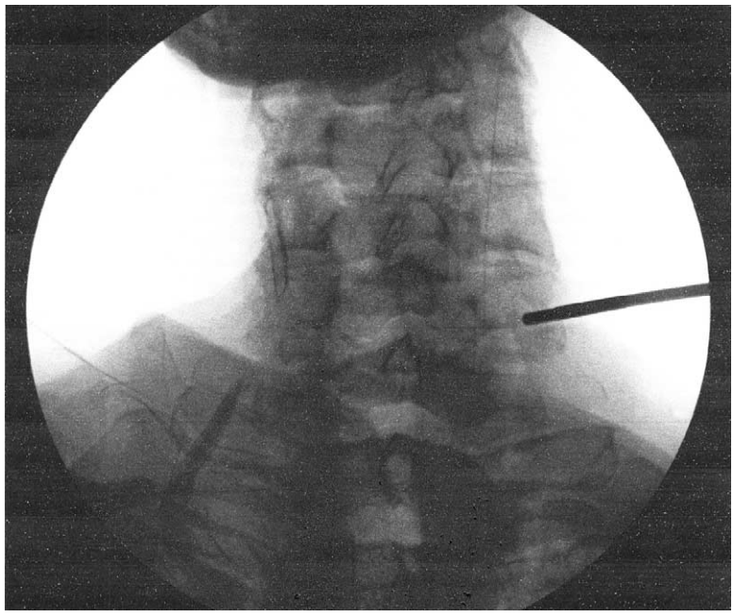


FIGURE 8–32
Introducer cannula.

needle toward the target site but remaining posterior to the neural foramen. The metal needle is removed from the introducer, and the blunt Coudé needle is advanced to touch the transverse process (Figures 8-35 and 8-36). The C-arm is rotated from 30 degrees oblique to horizontal to visualize the neural foramen, and the tip of the blunt Coudé tip is rotated anteriorly (Figures 8-37 to 8-38). The needle is advanced slowly under fluoroscopic guidance. When the tip of the needle is visible in the neural foraminal view, the needle is rotated in a posterior direction to enter the posterior neural foramen (Figures 8-39 and 8-40). The bony contact can be clearly felt. The C-arm is rotated into an AP direction, and the needle tip is advanced halfway into the facet joint markings (Figure 8-41). The T-piece is connected to the blunt Coudé needle, and aspiration is performed and Omnipaque is injected under continuous fluoroscopy.

The injected contrast is used as a marker, 1–3 ml of hyaluronidase is injected under fluoroscopic visualization, and the opening of the perineural space is observed. The

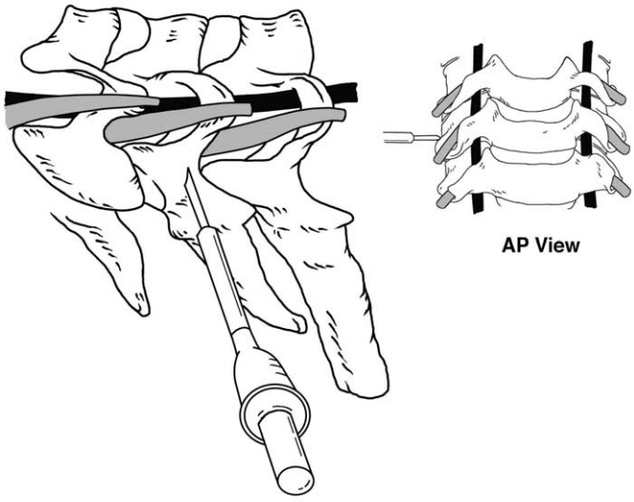


FIGURE 8–33
Oblique view with introducer cannula.

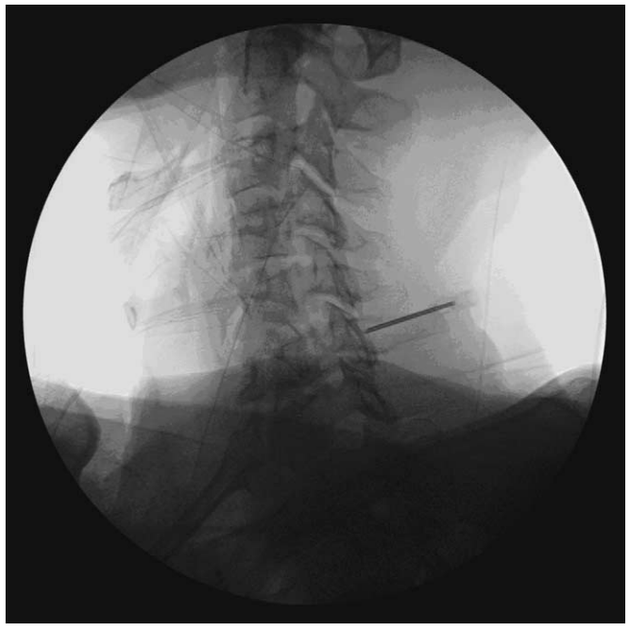


FIGURE 8–34
Oblique view with introducer cannula.

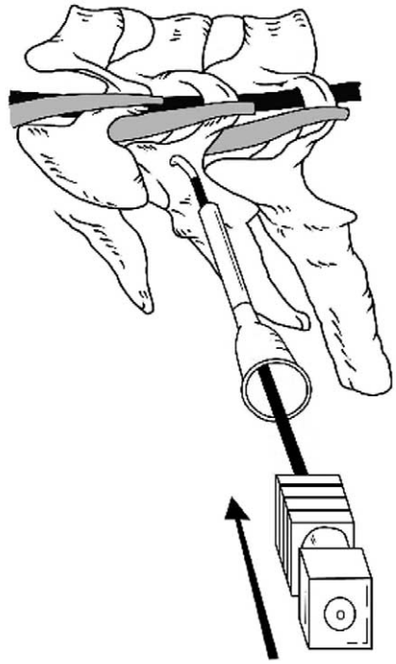


FIGURE 8–35
Curved blunt needle bony contact.

dye spread is often seen not only in the neural foramen but also in the anterolateral epidural space (Figure 8-42). This is followed by injection of local anesthetic and steroids 0.2% ropivacaine and 4 mg dexamethasone or 40 mg of triamcinolone in 5-ml mixture and 1–2-ml volume increments to 3–4 ml total volume of injection.

Paresthesias should not be sought as an endpoint for needle placement, especially with sharp needles. Radiographic endpoints should be used, and the goal is perineural deposition of steroid/local anesthetic.

It is not uncommon to see venous run-off, and this is not viewed as a serious problem. So far, all the reported cases and some nonreported cases of complications occurred during the use of sharp needles. As of this writing, we are not aware of cases of spinal cord compromise following the use of blunt Coudé needles. For this reason, and the convincing animal data that the blunt needles do not enter arteries or nerves, we have completely switched to the use of the 22- or 25-gauge blunt Coudé needles.[63] Introducer cannulae used as described will facilitate the procedure and will not penetrate the skin and some deeper structures easily. The 25-gauge blunt Coudé needle penetrates the fascial planes just posterior to the neural foramen and makes the procedure technically easier.

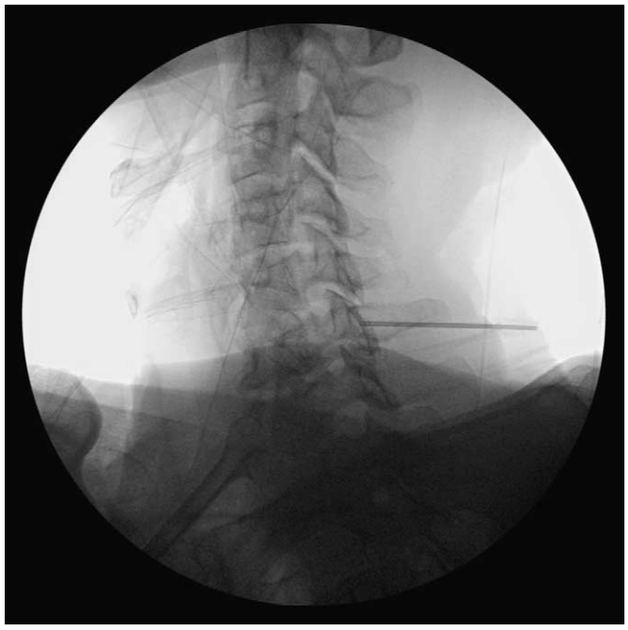


FIGURE 8–36
Curved blunt needle advanced to bone.

The technique described earlier has evolved over a number of years after reports of complications and of many more that have not been reported and cannot be published.

POSTPROCEDURE MONITORING

Patients should be monitored for 30 minutes or more and should be instructed to contact the physician for problems in between the procedure and follow-up visit.

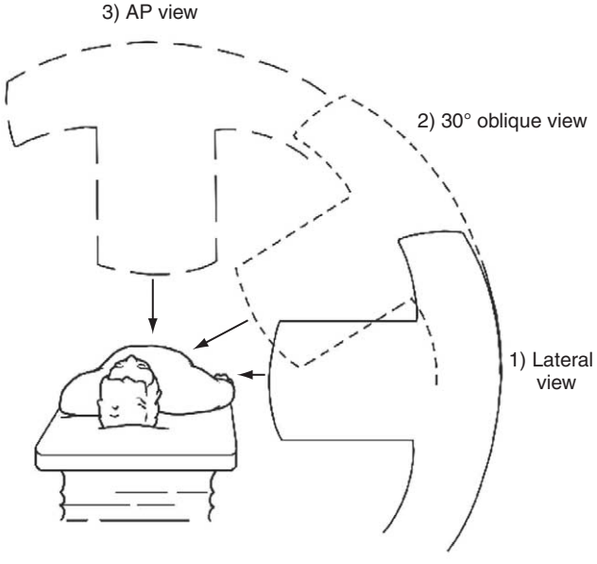


FIGURE 8–37
Patient position and fluoroscopic views for cervical transforaminal injection.

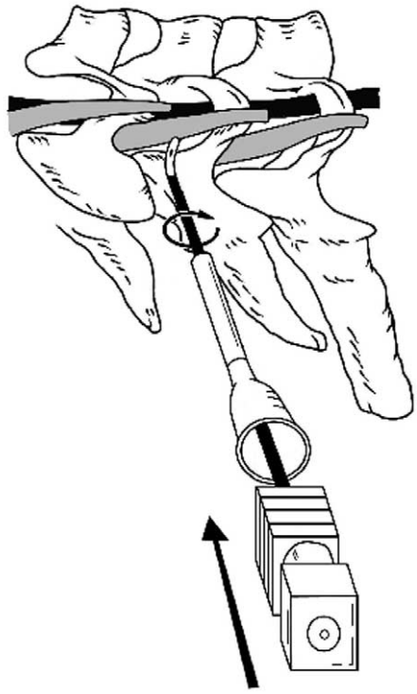


FIGURE 8–38
Rotate needle anterior into oblique fluoroscopy neural foramen view.

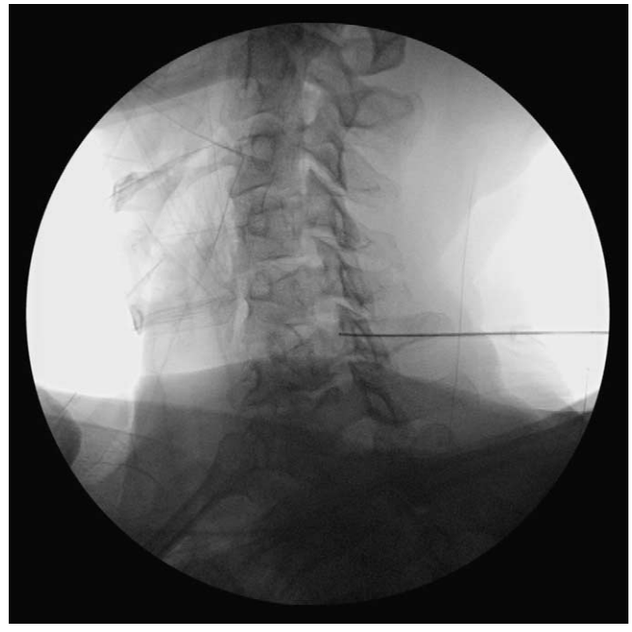


FIGURE 8–39
Curved blunt needle rotated anteriorly.

COMPLICATIONS

The complications reported below are associated with the use of sharp needles and the anterior-oblique approach.

Irreversible complications related to arterial injection or disruptions have been reported with this procedure. Rozin et al.[60] reported death during a transforaminal procedure

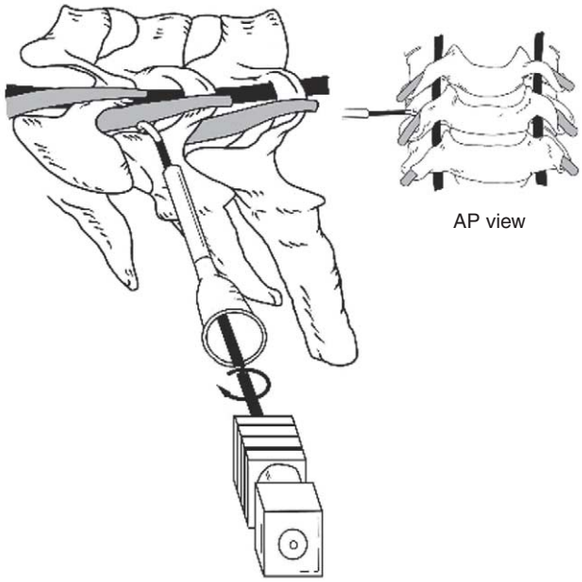


FIGURE 8–40
Needle slides on bone behind nerve root.

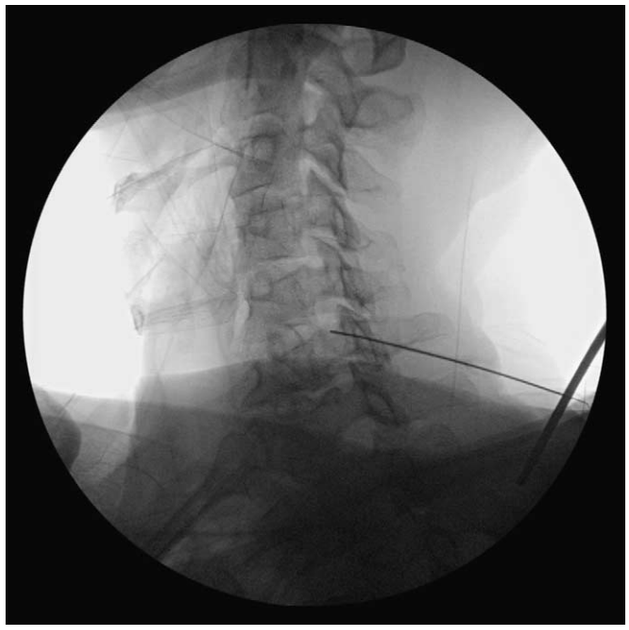


FIGURE 8–41
Curved blunt needle rotated and advanced into foramen.

from a vertebral artery perforation. Tiso and colleagues[64] reported a massive cerebellar infarct after a cervical transforaminal steroid injection. Corticosteroid suspensions and, to a lesser extent, solutions contain particles that the authors propose could embolize. Huntoon and Martin[65] reported a case of paraplegia and spinal cord infarct after a transforaminal injection in a patient with previous spinal surgery. Cortical blindness has been reported.[66]

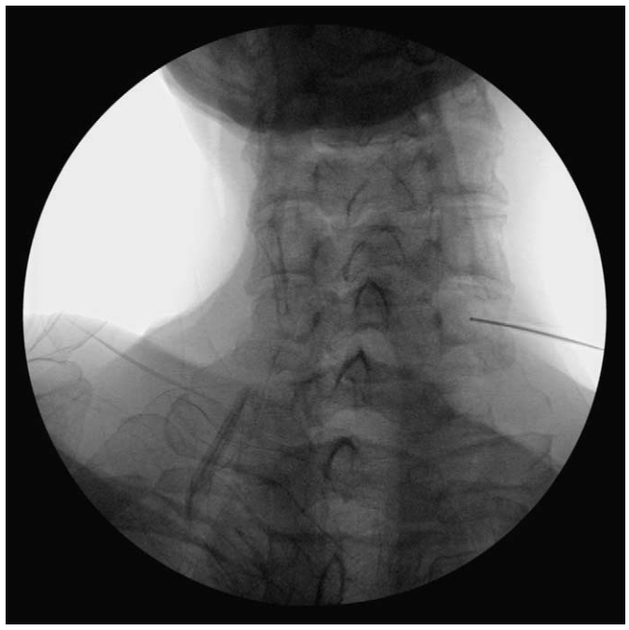


FIGURE 8–41
Final anteroposterior view.

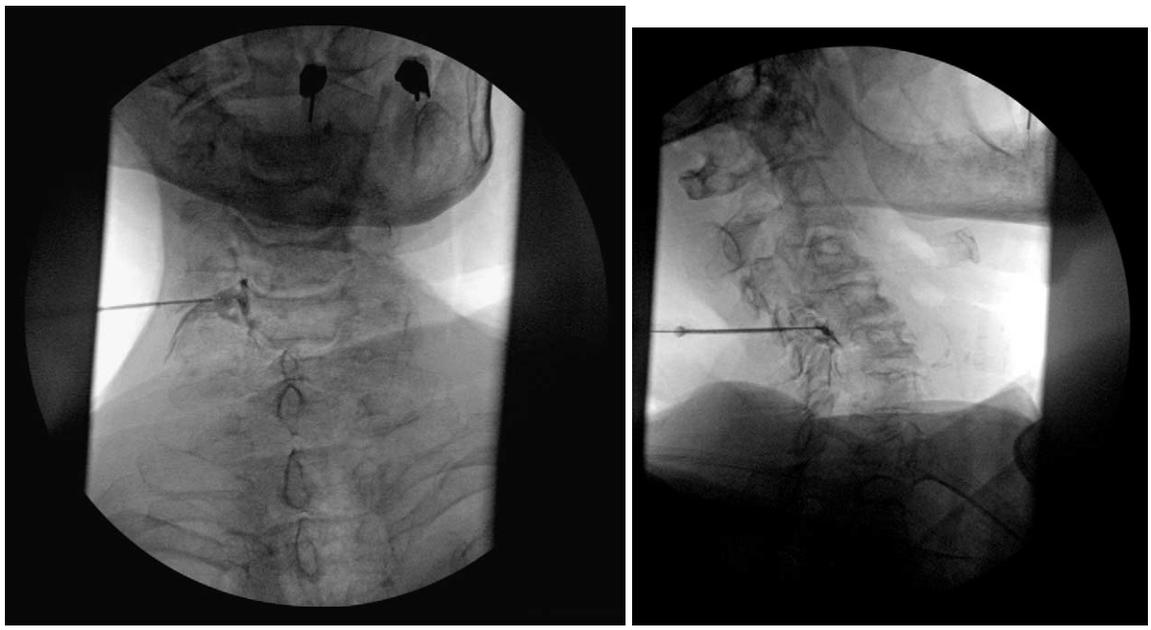
Baker and colleagues[67] reported contrast injection into a radicular artery without ill effects. The procedure was not continued, and no local anesthetic or corticosteroid was injected. However, neurologic complications following contrast injection alone have occurred.

Karasek and Bogduk[68] reported the development of quadriplegia after contrast and local anesthetic injection. Resolution occurred in 20 minutes. This case makes an argument for a test dose of local anesthetic prior to corticosteroid injection.

Some overlapping risks exist among nerve root, transforaminal, and interlaminar blocks. A review of the complication section of the chapters on each block is worthwhile.

The incidence of serious complications with transforaminal injections is unknown. Ma and colleagues[69] reported a series of 844 patients and 1036 blocks. Fourteen patients (1.66%) had minor complications associated with anterior foraminal placement. No correlation was found with needle depth into the foramen.

Based on the review of published cases plus medicolegal (not published) cases, the estimated incidence of permanent cord or other neurologic complication is about 1 in 10,000. Definitive data correlating specific equipment and techniques with complications do not exist, and this type of information may never be convincing due to the low incidence of complications. Nevertheless, complications can be devastating with these procedures and we owe it to our patients and to the future of our field to have open discussions about ideas to prevent complications. Complications related to the vertebral artery are quite serious. The vertebral artery is medial to the lateral border of the midcervical (C3-C6) spine, and an argument can be made to limit the medial positioning of a needle in



A B

FIGURE 8–42

(A) Anteroposterior and (B) lateral view with contrast injection.

the foramen. The artery is also anterior to the nerve roots. An anterior foraminal needle position, as described, would be lateral to the spinal cord and lateral to the vertebral artery. The vertebral artery is not the only artery of concern. Anterior and posterior segmental arteries within the foramen are vulnerable as well. Particulate corticosteroid preparations injected intra-arterially are another concern. Blunt needles may reduce the risk of arterial puncture, and contrast injection after aspiration may be a reliable way to avoid intravascular injections. The use of fluoroscopy is essential to any potential benefit with the use of contrast. Contrast itself is an issue, some preparations are not suitable for use due to potential toxicity.

Giving a test dose of epidural local anesthetic has been a practice in obstetrical anesthesia for years to ensure epidural catheter placement is not subdural. A test dose of contrast followed by a test dose of local anesthetic may be a good idea for transforaminal injections.

With a blunt needle, the urgency to complete the procedure is less than with a sharp needle, and before corticosteroid is injected, local anesthetic effects could be observed before injecting corticosteroid. Minimizing the use of procedures with the greatest risk also makes sense since there are few data showing superiority of one approach over another. Evaluating patients with the bio-psycho-social model of pain in mind will help direct patients toward the most appropriate therapy.

EFFICACY

Most of the literature describes results in patients with lumbosacral radicular pain. The transforaminal injection literature has some overlap with nerve root blocks as some authors describe techniques that could be interpreted as either procedure. No head-to-head studies comparing nerve root blocks to transforaminal or interlaminar epidural injections have been reported for cervical radicular syndromes.

Thomas et al.,[70] in a randomized trial, found transforaminal injections to be helpful compared to interspinous injections. In a review, DePalma and colleagues[71] found evidence in the literature for efficacy with transforaminal injections for radicular pain. Tong et al.[72] reported that patients with disability and jobs with heavy lifting requirements had less response to transforaminal injections compared to other patients in the study.

Rathmell and Benzon[73] posed the question, "Should we continue?" Many may abandon the procedure; however, the use of blunt needles placed lateral to the vertebral artery and the practice of careful interpretation of contrast injection may reduce complications. We have abandoned the use of sharp needles but feel comfortable with blunt needles. All procedures should be performed only after a thoughtful evaluation of the patient.

THIRD OCCIPITAL NERVE BLOCK AND RADIOFREQUENCY NEUROTOMY

HISTORY

The cervical facet joints have been investigated as a source of headache for several decades.[74–80]Sjaastad et al.[81] investigated neck and head pain by performing precision image-enhanced cervical nerve blocks with the use of fluoroscopy. They advocated blocking the cervical dorsal rami near

their origins. Other investigators in Europe adopted a similar approach.[82,83]

Bogduk[84] advocated blocking the respective medial branches of the dorsal rami as they coursed over the bony articular pillars, providing a safer, more selective, and more easily accessible target. This was the first description of the technique of medial branch blocks to block facet joint pain.

At typical cervical levels, that is, C3-C6, proper and correct use of C-arm fluoroscopy enabled precise needle placement onto the target nerve as it crossed centroid of the superior articular process. Hence only a minute amount of local anesthetic, no more than 0.5 ml, was required to anesthetize the target nerve. If injected under controlled conditions, the injectate was confined within the perimeter of the nerve and hence no other structure was likely to be anesthetized, which otherwise would have confounded the block.

By the mid-1980s, Bogduk and Marsland[85,86] had completed two studies showing that 70–80% of patients reported complete relief of their headache by selectively blocking the third occipital nerve (TON), which is the superficial branch of the C3 dorsal ramus and the only nerve that innervates the C2-C3 facet joint. This implicated the C2-C3 facet joint as the primary source of referred pain, perceived as a headache. Because pain is mediated by the TON, the appellation "third occipital headache" was appropriate.

These were the first reports of the diagnostic utility of anesthetizing the medial branch of the C3 dorsal ramus. This block was therefore suggested as a screening procedure for headache mediated by this nerve and advocated as a means of establishing this largely unrecognized diagnosis. Epidemiologic data indicated that cervicogenic headache most often stems from C2-C3 facet joint.[86]

A follow-up study evaluating the effects of cervical medial branch blocking, including the medial branch of the C3 dorsal ramus, demonstrated that 17 out of 24 patients reported relief of head, neck, and shoulder pain. The results of this study led to the first observation of consistent segmental pain patterns and the publishing of a pain-referral map based on relief obtained by medial branch blocks.[87]

These findings prompted other investigators to study pain-referral patterns from cervical facet joints. Using normal volunteers, Dwyer et al.[88] stimulated cervical facet joints by distending their respective capsules with contrast medium. The segmental pain-referral patterns produced coincided with those of Bogduk and Marsland's study.[87] Aprill and colleagues[89] then went on to describe how the segmental location of painful joints could be predicted by these pain patterns (Figure 8-43).

Other investigators have continued to confirm referred segmental pain patterns with the use of intra-articular injections and electrical stimulation of their respective medial branches.[90]

In a recent study to determine the referral patterns of the cervical medial branches and the third occipital nerve by sensory stimulation, Windsor et al.[91] mapped separate and distinct referral patterns from those previously published. Their study was based on the understanding that stimulation of the medial branch above and below the facet joint is required to reproduce referred pain, while anesthetization of the medial branch would be required to block referred pain.[91]

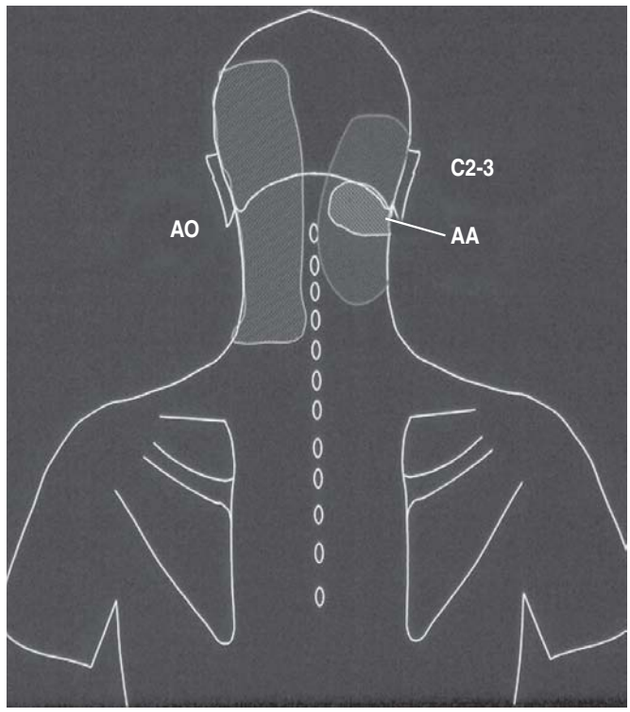


FIGURE 8–43

Pain-referral pattern from C2-C3 facet joint. Distribution of third occipital headache. Note overlapping pain referral from C0-C1, C1-C2 joints. (Adapted from Dwyer A, Aprill C, Bogduk N: Cervical zygapophyseal joint pain patterns. I. A study in normal volunteers. *Spine* 15:453, 1990, with permission.)

A study by Lord et al.[92] in 1994 found that the prevalence of third occipital headache in patients suffering from neck and head pain following whiplash injury was 27%. Moreover, when headache was the dominant complaint, prevalence was 53%. In clinical trials, it has been demonstrated that anesthetic blockade of the third occipital nerve can provide temporary relief from headache.

A follow-up study included neck, as well as head, pain. With a prevalence of 54% facet joint pain, the joints most commonly involved were C2-C3 and C5-C6.[93]

Previous studies had addressed the confirmation of target specificity (face validity)[94] and the ability to discriminate between true positives and false negatives by a paradigm of comparative local anesthetic blocks (construct validity).[95]

The therapeutic utility and predictive validity of cervical medial branch blocks demonstrated that positive control medial branch blocks were predictive of success for percutaneous RF neurotomy at "typical" cervical segments. However, poor results were obtained when using the same criteria with the third occipital nerve. This problem would later be addressed by Govind et al.[96] and published in 2003.

Intra-Articular Facet Joint Injections

There remains controversy as to the therapeutic utility, predictive validity, and face validity of intra-articular cervical facet joint injections. These results stand in contradiction to other published studies that demonstrate possible beneficial aspects of this procedure.[97–100]

In terms of predictive validity, there are no studies to support a positive intra-articular facet injection as a substitute for comparative medial branch blocks. The performance of a C2-C3 facet injection will be described in this chapter for its potential applications. Additionally, physicians who do not have access to an RF generator might find this injection useful.[100]

Barnsley and colleagues[101] demonstrated that there is a high false-positive rate for single zygapophyseal joint injections, and that control or confirmatory blocks must be performed to ensure accurate results. Contradictory or ambiguous results should warrant serious consideration of the need to cease further injections. Conversely, findings of a high percentage of pain relief (80–90% or greater) are confirmatory and RF neurotomy of the third occipital nerve can then be considered, with relief of 90% of pain or above yielding the greatest likelihood of success for RF neurotomy.

Radiofrequency Neurotomy

Several descriptive and long-term reports describing the treatment of cervical and head pain using RF neurotomy appeared in the literature from the late 1970s into the 1980s. Sluijter developed a technique for cervical "facet denervation" by performing thermal RF lesions to the posterior primary ramus. For over a decade, these techniques gained popularity, but they demonstrated inconsistent and only fair long-term results.[83,102–107]

In a 1995 review of those studies, Lord et al.[108] found several variations in study designs that may have led to their lackluster results. These variations included: absence of diagnostic blocks in certain patients, uncontrolled blocks, and use of surgical technique not accurately targeting the cervical medial branches.

The following year, Lord and colleagues[109] reported that chronic neck pain below the C2-C3 facet had been effectively treated by RF neurotomy with complete long-term pain relief. It was further demonstrated by McDonald et al.[110] in 1999 that when pain recurred, similar long-term results could be obtained by repeating a similar RF neurotomy.

Initial attempts to provide similar long-term results in patients suffering from third occipital headache produced disappointing results. Lord et al.[108] concluded that certain technical problems associated with this procedure still needed to be overcome and cautioned against the use of RF neurotomy of the third occipital nerve until further investigation was conducted.

Govind and colleagues,[75] using a revised technique that took many factors into consideration, including the variable pathways of the third occipital nerve, published data that yielded long-term results similar to those demonstrated from Lord and colleagues' earlier work on cervical medial branch RF neurotomy. Additionally, McDonald et al.[110] demonstrated that repeat RF neurotomy of the third occipital nerve could successfully reinstate consistent and profound relief in patients.

A recent study by Govind et al.[75] demonstrated the prevalence of cervicogenic headache emanating from the upper three cervical facet joints, determining their segmental distribution, and proposing an evidence-based diagnostic and therapeutic algorithm. In a double-blind, randomly assisted study (between 2000 and 2004), patients who suffered post-traumatic headaches (unilateral or bilateral) underwent comparative diagnostic blocks. A block was deemed successful when the patient reported total abolition of their pain (VAS=0) on two separate occasions. Of 191 joints investigated, 64% were positive.

The C2-C3 facet joint contributed (revised figures out soon) the C3-C4 joint 13%, and the C1-C2 joint 6%. The remaining results were shared between the lower levels. Segmentally, the respective positive rates at C2-C3 and C1-C2 were 72% and 56%. The upper three joints accounted for 85% of all positive blocks. These results agree with the algorithm for blocking for headache (where a negative C2-C3 "invites consideration" to investigate the C3-C4 joint). However, these new data recommend that C3-C4 should be after the C1-C2 joint has been investigated, which is a technically more demanding procedure.[75]

Another recent study deserves to be mentioned. In a report published in 2004, Stovner et al.[111] attempted to investigate the effects of RF neurotomy in patients with unilateral cervicogenic headache. Based on diagnostic criteria alone, C2-C6 RF medial branch neurotomy was performed along with a "sham" control group. Diagnostic blocks were utilized as investigators wished to test whether the outcomes could be predicted on the basis of percentage of pain relieved by diagnostic blockade. Not a single patient received 100% relief from any block. At 3 months there was no difference between the RF and sham group. They concluded that this procedure is probably not an effective treatment for cervicogenic headache. It was concluded that a near 100% pain relief response should "probably" be among the inclusion criteria in such studies.

This study has been criticized on the basis that the utility of RF neurotomy was based purely on clinical criteria on physical examination, not comparative local anesthetic blocks. When blocks were used, they were only used in such a way as to predict or validate the outcomes based on clinical findings. Not a single patient obtained 100% relief from a control block, which raised questions as to why the authors were attempting to provide grounds that a RF neurotomy would provide complete relief when diagnostic blocks failed to do so. Even though response to blocks was not used as a criterion in their study, blocks were used regardless. Patients without complete relief were enrolled in a controlled trial; therefore, under those conditions the trial was designed to show no efficacy.

It was suggested that the study did reveal that clinical diagnostic criteria have no predictive validity, and therefore the use of a clinical diagnosis should be abandoned. The study predictably demonstrated that patients who did not respond to diagnostic blocks did not respond to medial branch neurotomy. The authors described but did not illustrate their technique, leaving open the question as to whether the procedure was performed correctly. Descriptions of the surgical technique included that the patient was placed in the supine position. In addition, 22-gauge needles with 50-mm electrode lengths were employed upon utilizing stimulation as a criterion for accurate nerve localization. Only three to four parallel lesions against a single plane (sagittal or oblique) were performed at 60 seconds.

In an editorial response, Bogduk[112] states that Stovner's study revealed only that RF neurotomy fails when it is practiced inappropriately and incorrectly, as opposed to what the International Spine Intervention Society recommends in its practice guidelines. He additionally raised concerns that this study could be misused to discredit the already vindicated practice as described in those guidelines.

ANATOMY

The third occipital nerve is a single large nerve of about 1.5 mm in diameter. It projects a transverse course, unlike the remaining medial branches, which are obliquely directed in a posterocaudal direction. The third occipital nerve is the only nerve to cross the facet joint and gives off articular branches to the C2-C3 facet joint from its deeper surface.

The C2-C3 facet joint is unlike the other cervical facet joints–it receives innervation not only from the C3 medial branch, but also from the TON. In experimental studies with normal human volunteers, referred pain from the C2-C3 joint was perceived in the head.[88]

The C3 spinal nerve divides into an anterior or ventral ramus and a posterior or dorsal ramus. The dorsal ramus divides into two medial branches and a lateral branch that innervates the superficial muscles.

The larger, superior branch is the third occipital nerve (superficial median branch), and the inferior branch is the deep median branch. The third occipital nerve curves dorsally and medially around the superior articular process of the C3 vertebra. It crosses the C2-C3 facet joint either just below or across the joint margin. The innervation of the C2-C3 facet joint primarily is from the large, superficial medial branch of the C3 spinal nerve, the third occipital nerve. The deep medial branch of the C3 dorsal ramus is parallel but caudad to the third occipital nerve in the C3 articular pillar.

The innervation of the synovial joints, and disturbance of this innervation, may play a role in the development of degenerative diseases and joint dysfunction. Physiologic, pathomechanical, and pathologic processes lead to articular dysfunction. These include trauma, arthritis, infection, muscle strain or reflex spasm, and nerve injury by trauma or compression.

The receptor fields are large, and one or two nerve endings may be sufficient to monitor the area of each facet capsule. Damage to even a small part of a capsule may denervate that articular structure. This could have important implications for long-term joint function.[113]

Because the C2-C3 joint is innervated mainly by the large third occipital nerve and small communicating branches that originate from the second cervical nerve, third occipital nerve blockade is sufficient to establish the diagnosis of C2-C3 facet pain. The third occipital nerve also provides cutaneous neural supply to the suboccipital area. Blocking the third occipital nerve on the C3 articular process will temporarily block the nerves that innervate this joint and produce cutaneous anesthesia in the distribution of the nerve.

There are several communicating branches between the dorsal branches of C1, C2, and C3, which may also play a role in the anatomic basis for cervicogenic headaches (Figures 8-44 and 8-45).

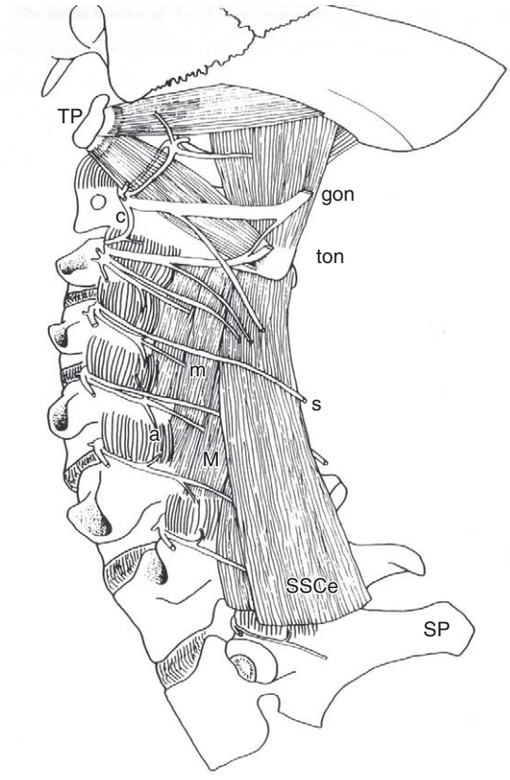


FIGURE 8–44

Dorsolateral view of left cervical dorsal rami in the plane of multifidus (deep to semispinalis capitis). TP, transverse process of C1; GON, greater occipital nerve; TON, third occipital nerve, innervates the C2-C3 Z-joint; variant, communicating branch (c) between C2 and C3 nerves may innervate the C2-C3 joint; below C2-C3, deep medial branches (m) send articular branches (a) to the Z-joints and then to the multifidus (M); SP, spinous process of T1; SSC, sternocleidomastoid. (From Bogduk N: The clinical anatomy of the dorsal cervical rami. *Spine* 7:319–330, 1982, with permission.)

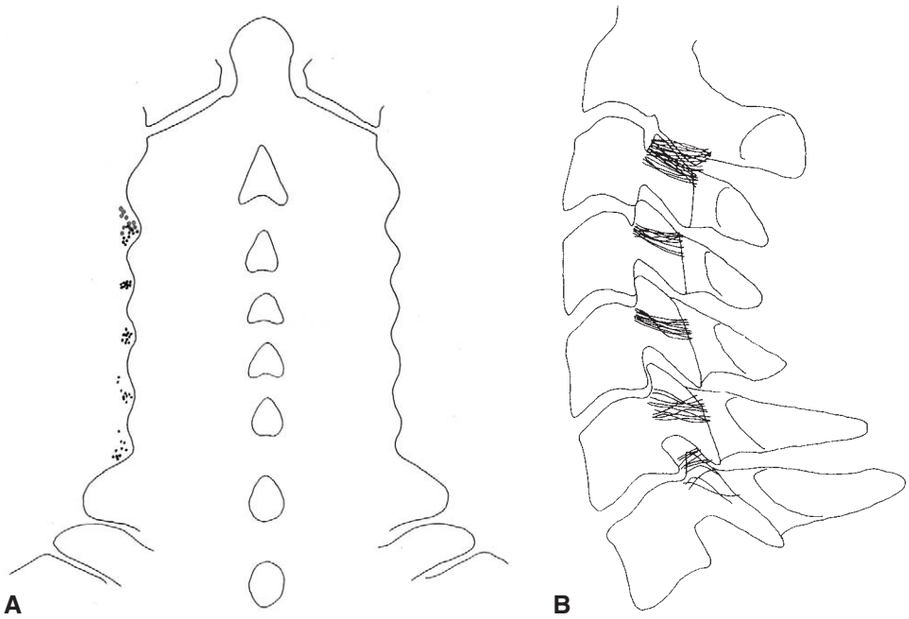


FIGURE 8–45

The coronal illustration (sagittal, pillar view under fluoroscopy) on the left demonstrates the significant variability of the TON. The initial RF lesion is typically the most cephalad. From Lord SM, et al: Percutaneous radiofrequency neurotomy of the cervical medial branches. A validated treatment for cervical zygapohyseal joint pain. *Neurosurg Q* 8:228–308, 1998.

INDICATIONS

The fundamental indication for performance of the third occipital nerve block is to determine if the patient's pain is mediated by that nerve. The only validated treatment for pain mediated by the third occipital nerve is percutaneous RF third occipital nerve neurotomy. If the possibility of using this treatment is being entertained, dual controlled blocks are a prerequisite.

Blocking the third occipital nerve has diagnostic utility. Establishing a proper diagnosis of third occipital headache early will avert therapies and/or surgical interventions based on presumed wrongful diagnosis. Misguided treatment of headaches of unknown origin may lead to disastrous consequences for the patient. Not only will the delay in establishing a correct diagnosis prolong the patient's suffering, but it may also lead to long-term physical, medical, and surgical repercussions (including migraine with long-term unsuccessful medication management trials, emergency room visits, iatrogenic chemical dependence, and spinal fusion).

The performance of a C2-C3 intra-articular block may suggest that the patient's pain is being mediated by that joint. Therefore, it will only be discussed briefly in this chapter as concerns its questionable diagnostic validity. There are no studies to validate the effectiveness of blocking this joint in any algorithmic paradigm. Distention or stressing the joint capsule by joint arthrography may reproduce much of the patient's symptoms, and conversely analgesic injection may reduce or eliminate concordant pain. If a diagnostic/therapeutic intra-articular injection fails to relieve the pain, and seepage from the joint is observed under fluoroscopy, then control blocks would be required to determine if the patient's pain is emanating from that joint.

The C2-C3 intra-articular facet injection as a prognostic procedure prior to RF neurotomy suffers drawbacks similar to any intra-articular injection. These include potential leakage and subsequent spread of injected local anesthetic into the epidural space, thereby rendering it useless in terms of target specificity. Additionally, the size and variability of course of the third occipital nerve requires that anesthetization of the nerve using dual control blocks not only relieve pain stemming from the target joint or joints, but also must produce cutaneous anesthesia of its innervated territory. Furthermore, if RF neurotomy is the only validated long-term treatment, anesthetizing the nerve is essential and false-positive, as well as false-negative responses must be kept to a minimum. The only method of validating that the patient's pain is mediated by the third occipital nerve is by the use of dual control blocks.

CONTRAINDICATIONS

Contraindications for third occipital nerve block follow:

- Coagulopathy (INR >1.5 or platelets <50,000). Anticoagulation medication should be suspended for an appropriate period prior to the conduct of blocks.
- Systemic infection or localized infection at the puncture site.
- Severe allergy to any medications used.
- Pregnancy.
- The patient who is unable or unwilling to consent to the procedure, or who is unable to understand and cooperate with the procedure.
- Any factor that would cause inability to assess the patient's response to the procedure.

- Evidence of another source of neck pain or headaches not related to the facets.
- Motor weakness, absent reflexes, or long tract signs.
- Any anatomical derangements, surgical or congenital, that would preclude safe, successful access.

Contraindications for third occipital nerve RF neurotomy:

- Patients who have had inadequate pain relief or relief for less than 3 months following a previous neurotomy.
- When appropriate, pacemaker, and ICD equipment must be appropriately deactivated prior to RF neurotomy.[114]

PREPARATION OF PATIENT

History

There are no distinguishing features in the history, examination, or imaging that allow a definitive diagnosis of this form of headache. Controlled diagnostic blocks constitute the only means of establishing a diagnosis.[94]

Evaluation of the headache patient poses a significant diagnostic challenge. The physician must first determine if headache is the dominant complaint. If so, all other forms of headache must be ruled out before settling on a diagnosis of third occipital headache, including vasculopathic (i.e., migraine, cluster), supratentorial ("psychogenic" or somatoform, tension headache, etc.), drug-induced, and especially headache secondary to "red flag" conditions such as tumor, metastatic disease, infection, and metabolic process.

Examining the differential diagnosis of the headache patient is beyond the scope of this chapter. Therefore, the reader must be familiar with the characteristics of headaches of other etiologies and evaluation of previous responses to therapeutic modalities (i.e., physical, manual and manipulation therapy, pharmacotherapy, spinal injections, and surgical intervention).

As time passes, it becomes more difficult to determine whether previous interventions have failed because of a wrong diagnosis. Thus, the headache patient who has had a series of medical and surgical failures can easily be mislabeled as malingering, suffering from chemical dependence or symptom magnification, especially when chronic pain behavior denies the patient timely and appropriate care. Therefore, it is imperative that early diagnosis is established because the only validated treatment for third occipital headache is percutaneous RF neurotomy.

A long, protracted course of ineffective headache treatments should not preclude the practitioner from exploring the possibility that an erroneous diagnosis has been made. According to a study by Sjaastad,[115] 13.8% of all headache patients suffer from cervicogenic headache. Disorders of the C2-C3 facet joint have the highest likelihood of producing cervicogenic headache.[84]

Conducting a meticulous review of the patient's history is of paramount importance. By doing so, the practitioner may find that an overlooked diagnosis or other error in judgment led to protracted ineffective treatments.

The ideal candidate should demonstrate characteristics of cervicogenic headache. Patients displaying psychiatric disturbances (severe), symptom magnification, history of substance and alcohol abuse, multiple emergency room visits, and so on may require detoxification and participation in a cognitive/behavioral program. Willingness by the patient to participate in his or her own health and well being are required. Diagnostic criteria supplied by the Cervicogenic Headache International Study Group provide a detailed description of the condition.[81]

Physical Examination

Common features include unilateral headache, ipsilateral shoulder or arm pain, reduced range of motion of the neck, and presence of mechanical precipitation mechanisms.

Focal tenderness over the C2-C3 facet joint is associated with headache in the distribution of the third occipital nerve. Essentially, this is unilateral (very rarely bilateral) pain that can be exacerbated by palpation over the C2-C3 facet joint. Axial loading, including rotation and bending toward the ipsilateral side, may further intensify symptoms.

Patients may have limited head rotation, trigger points confined to the occipital area, palpable cervical crepitus, and abnormal head position. Pain from the C2-C3 joint is located in the upper cervical region and extends at least to the occiput and sometimes into the head, toward the ear, vertex, forehead, and in rare cases to the ipsilateral eye.

Although the physical examination may be quite useful in leading toward a specific diagnosis, there is frequently overlap of pain referral patterns. Aprill et al.[116] demonstrated that patients with C1-C2 joint pain did not differ significantly on clinical examination from patients with pain at other levels. Because of this overlap of pain referral patterns, nerve infiltration with local anesthetic is mandatory in establishing a diagnosis.

The reader is encouraged to become familiar with algorithms for cervical synovial joint blocks, as well as the headache algorithm recently set forth by the International Spine Intervention Society.[114]

EQUIPMENT

- *C2-C3 intra-articular facet injection/third occipital nerve block*
- 60–100-mm, 25-gauge needle
- Short-extension T-piece microbore tubing
- Nonionic water soluble contrast
- 2.0–3.0-cc syringe
- *Radiofrequency neurotomy*

- Radiofrequency generator capable of displaying impedance, voltage, amperage, and temperature
- 16–20-gauge, 100-mm RF needle with 10-mm active tip
- 100-mm compatible thermocouple
- Reference lead attached to a dispersive/ground plate
- Two 3-1/2-inch, 25-gauge needles
- 3.0-cc syringe with short extension "T-piece" tubing

DRUGS

- *C2-C3 intra-articular facet injection/third occipital nerve block*
- Local anesthetics for dual control comparative blocks
- 2% lidocaine
- 0.5–0.75% bupivacaine
- Nonionic water soluble contrast medium
- *Third occipital RF neurotomy*
- Local anesthetic
- 2% lidocaine for skin and subcutaneous anesthesia
- 2–0.5% bupivacaine prior to RF lesion

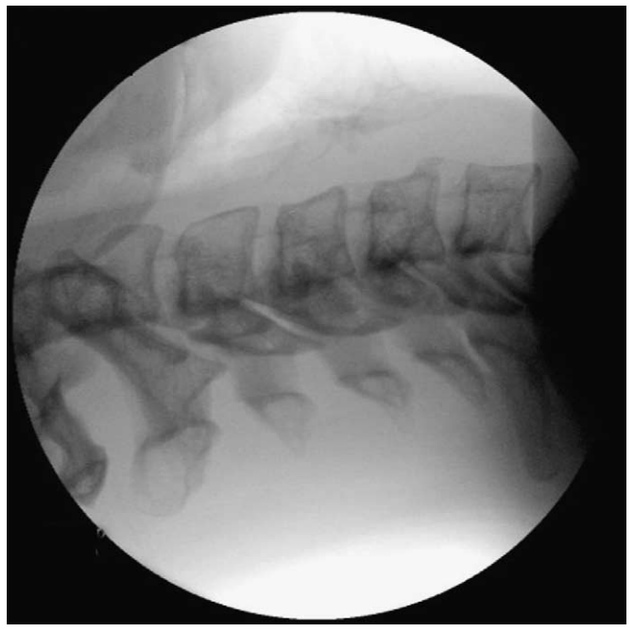


FIGURE 8–46

True lateral view of the C2-C3 facet joint. (Courtesy of Milton Landers, MD. Reproduced with permission of The International Spine Intervention Society.)

PATIENT CONSENT

Candidates for third occipital nerve block must be fully informed of their risks, and a written consent form completed. The consent should be properly witnessed, and all questions from the patient answered in full prior to proceeding. Any evidence of lack of patient understanding or external psychological coercion should result in the immediate termination of the procedure and rescheduling after these issues are resolved. Patients must also understand their responsibility to keep an accurate postprocedure pain diary.

ORIENTATION OF C-ARM

C2-C3 Intra-Articular Facet Injection/Third Occipital Nerve Block

With the patient in the prone, supine, or lateral position, the C-arm must be oriented to ensure a true lateral image. Silhouettes of articular pillars of both sides must be superimposed. Parallax errors are avoided by placing the target point on center-screen by aligning the fluoroscope properly (Figure 8-46).

The C2-C3 joint must be clearly visualized with the silhouettes of the articular pillars superimposed providing a sharp, crisp image. Errors in anatomic visualization, target identification, needle placement, and performance of procedure should be eliminated.

The lateral flange of the inferior articular process of C2 usually overlaps the C2-C3 joint, so a direct lateral approach is usually not possible. A posterolateral approach is required to enter the joint. The C-arm can be adjusted to open the joint using a slight craniocaudal angulation.

SEDATION

C2-C3 Intra-Articular Facet Injection/Third Occipital Nerve Block:

Sedation is not required for either the C2-C3 intra-articular facet injection or the third occipital nerve block. The higher the patient's pain rating at the time of the block, the greater will be the ability of practitioner and patient to rate the percentage of pain. Minimal sedatives may be used if required to combat excessive anxiety. Administration of narcotics will confound interpretation of the percentage of pain relief, and therefore are contraindicated.

PROCEDURE: C2-C3 INTRA-ARTICULAR FACET INJECTION

Patient Positioning

The prone position allows greater stability for the cervical spine, with a foam wedge placed under the patient's chest and head flexed with nasal cannulae or a jelly doughnut. Arms can be secured by the sides with Velcro wrap. The injection can also be performed in the lateral position, but patient cooperation is critical for consistent radiographic imaging without moving the C-arm via patient movements.

Target Identification

The C2-C3 joint requires a more posterior approach due to lateral flange of C2 of inferior articular process over superior process of C3. Demonstrated here (Figures 8-47 and 8-48) are C2-C3 facet joint arthrograms. Typically, only 1/16 ml of contrast is required to confirm intracapsular placement.

PROCEDURE: THIRD OCCIPITAL NERVE BLOCK

Patient Positioning

Supine or prone positions are equally acceptable for the performance of the cervical medial branch block. Some practitioners prefer the patient in the lateral decubitus position. The key element to success in performing this block is meticulous attention to detail in that a near-perfect, true lateral view of the cervical spine is obtained.

Target Identification

Due to the thickness of the third occipital nerve, which is embedded in the pericapsular fascia of the C2-C3 joint, adequate infiltration of the nerve must be ensured by using three target points. This has been termed the "tolerable error zone." This will ensure that the needle will not enter the C2-C3 foramen nor stray from the target zone (Figure 8-49).

First, a longitudinal bisector is made over the C2-C3 joint with three targets:

- High: Opposite apex of C3 superior articular process (sap)
- Low: Opposite base of C2-C3 foramen
- Mid: Midway between

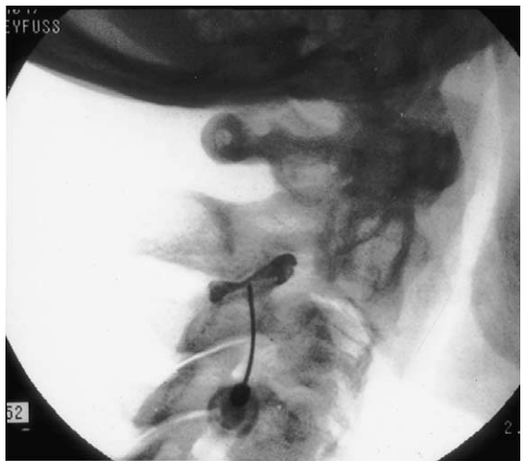


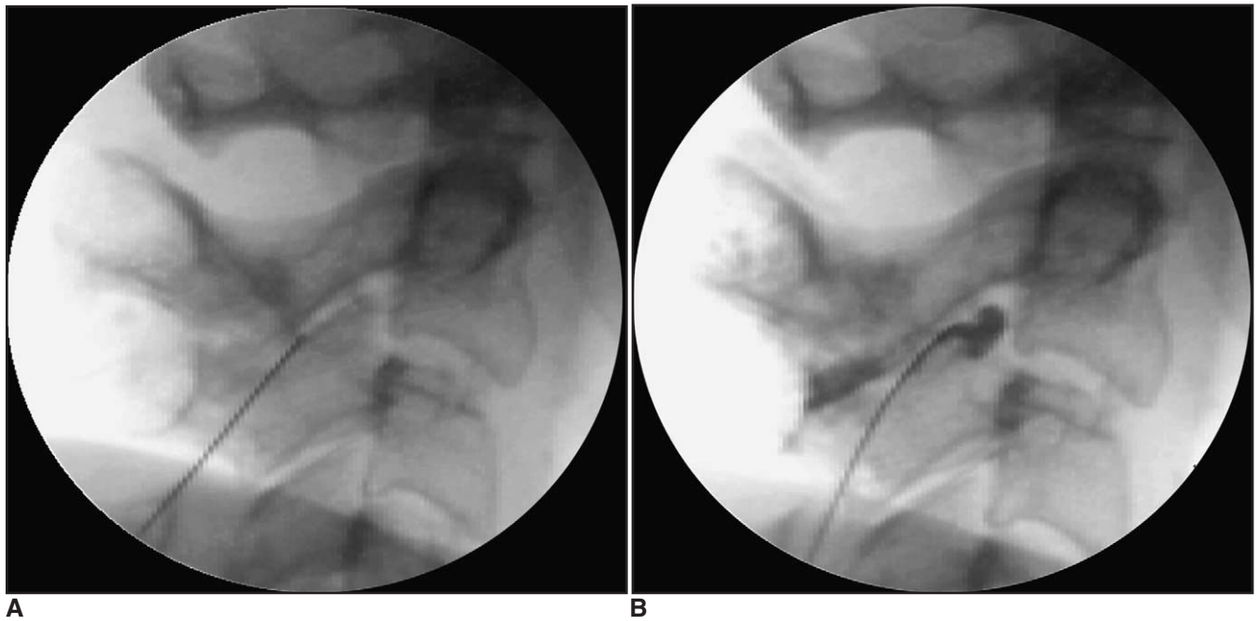
FIGURE 8–48

Posterolateral approach, C2-C3 facet joint injection. (Courtesy of Paul Dreyfuss, MD, and The International Spine Intervention Society.)

Needle Insertion

Ensure a true lateral image. Silhouettes of articular pillars of both sides must be superimposed. Parallax errors are avoided by placing the target point on center-screen by aligning the fluoroscope properly.

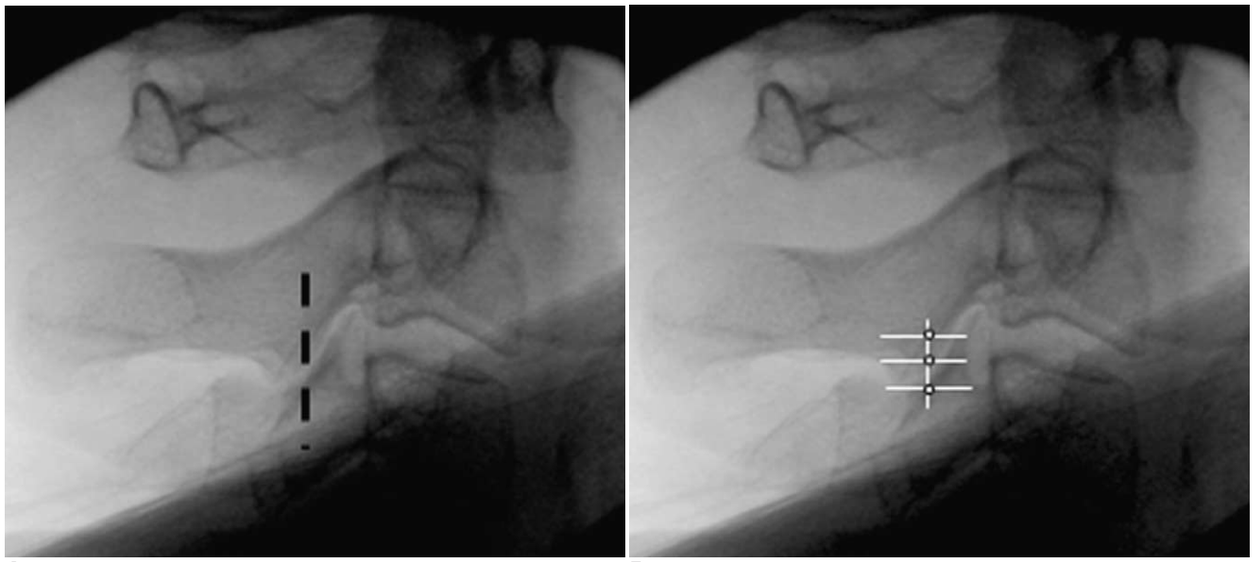
After a puncture point is located on the skin directly over the target zone, the needle is advanced in small increments toward the center of the target points using needle and bevel rotation for directional control. Periodic screening is used to confirm the needle's course. Any deflection away from the target requires withdrawal of the needle with appropriate adjustments to ensure precise target acquisition. The endpoint is contact with bone or pericapsular fascia (Figure 8-50).



A B

FIGURE 8–47

Posterolateral approach. (Courtesy of Charles Aprill, MD, and The International Spine Intervention Society.)



A B

FIGURE 8–49

Target points for third occipital nerve block along longitudinal bisector. (From Bogduk N, editor: *International Spine Intervention Society Practice Guidelines/Spinal Diagnostics and Treatment Procedures*. San Francisco, International Spine Intervention Society, 2004, p. 125, with permission.)

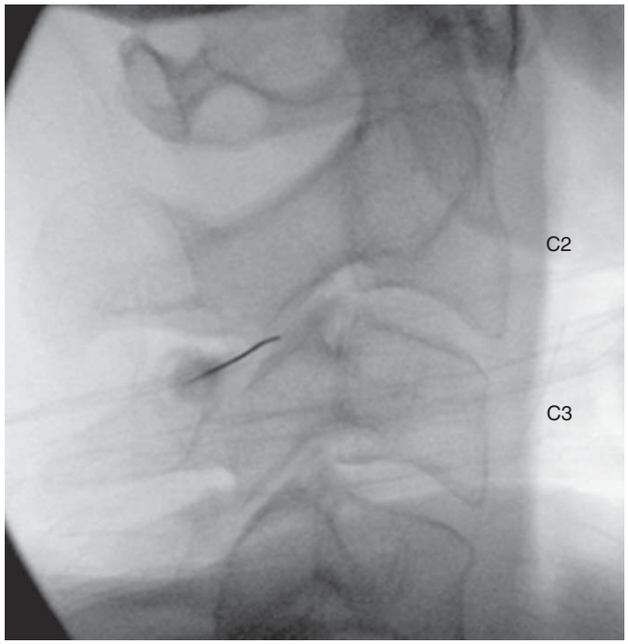


FIGURE 8–50

Needle placement at mid-target point for third occipital nerve, high, and low points marked by dots. (From Bogduk N, editor: *International Spine Intervention Society Practice Guidelines/Spinal Diagnostics and Treatment Procedures*. San Francisco, International Spine Intervention Society, 2004, p. 127, with permission.)

Injection

The needle tip must lie on the extracapsular aspect of the joint, which is approximately 3 mm from bony contact. If the needle is not withdrawn this amount, an intra-articular injection might occur. A minute quantity of contrast medium is injected, just enough to confirm pericapsular spread over the C2-C3 joint. By readjustment of the needle, upper and lower target points are injected with 0.3 ml of local anesthetic followed by the remaining target point. The total volume of local anesthetic injected to block the third occipital nerve must not exceed 1.0 ml (Figures 8-51 and 8-52).

Injection of contrast medium must be performed under real-time imaging to ensure total coverage of the course of the nerve (Figure 8-52) and to rule out inadvertent intracapsular injection or vascular uptake. If venous uptake is observed, the needle must be repositioned and a second injection performed. The volume of contrast medium carried away by vascular uptake, if it goes unnoticed, and is followed by infiltration with local anesthetic, will eliminate a similar volume of local anesthetic and could result in a false-negative block.

POSTPROCEDURE MONITORING

The patient must be watched carefully for untoward side effects from the block. Ataxia and vaso-vagal episodes usually occur early. Vaso-vagal symptoms are treated appropriately by monitoring vital signs and supine bed rest. Upper cervical proprioceptors critical for tonic neck reflexes are anesthesized by third occipital nerve block. This creates a sense of ataxia or unsteadiness (see "Complications" section). Patients should be instructed to keep a postprocedure pain diary (Figure 8-53) to meticulously document their progress after injection. In the diary the patient must note any immediate change in symptoms; he or she must be instructed to keep track of any change in pain in the first 24 hours postprocedure, as well as during the following weeks.

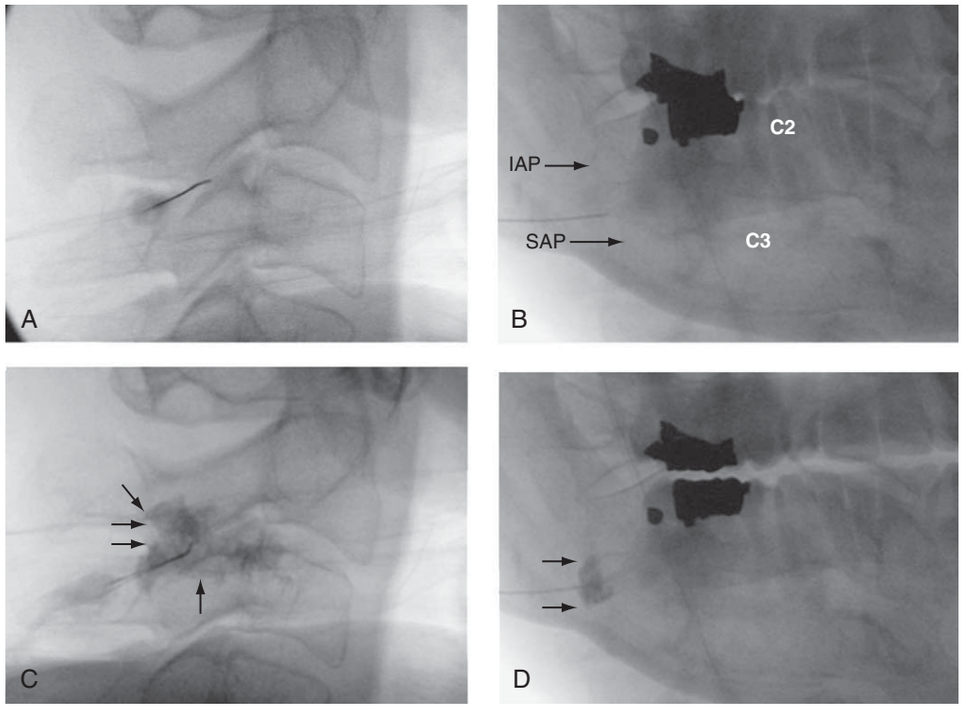


FIGURE 8–51

Radiographs of contrast medium injected onto the third occipital nerve. (A) Lateral view, showing a needle in position prior to injection. (B) Anteroposterior (AP) view, showing a needle in position prior to injection. (C) Lateral view after injection. The contrast medium (arrows) spreads across the lateral surface of the C2-C3 zygapophyseal joint. (D) AP view after injection. The contrast medium (arrows) remains located over the surface of the C2-3 zygapophyseal joint. (From Bogduk N, editor: *International Spine Intervention Society Practice Guidelines/Spinal Diagnostics and Treatment Procedures*. San Francisco, International Spine Intervention Society, 2004, p. 128, with permission.)

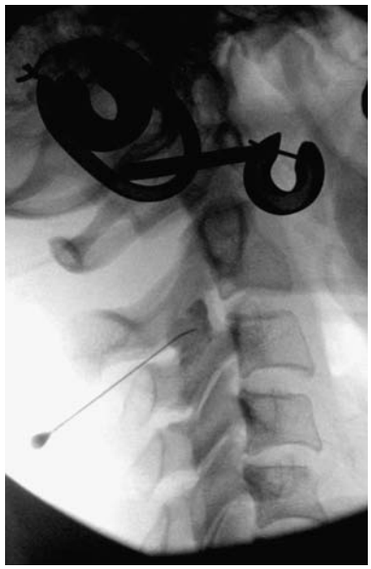


FIGURE 8–52

Posterolateral approach to block the third occipital nerve. Note contrast medium injection covers all possible area where the nerve might course. (Courtesy of Paul Dreyfuss, MD.)

PROCEDURE: THIRD OCCIPITAL RADIOFREQUENCY NEUROTOMY

Facilities Required

The procedure is performed in a room suitable for aseptic procedures. It is advisable that the room be equipped with proper resuscitation facilities. The operator thus will be equipped to handle complications arising from allergic reactions or inadvertent intrathecal or intravascular injection of local anesthetic. Monitoring equipment should include EKG, pulse oximeter, blood pressure cuff, oxygen, and suction devices.

The procedure table must be radiolucent to ensure clear, unobstructed views by the image intensifier. Free rotation of the C-arm is essential in all planes including AP (including pillar view, open mouth view), lateral, and oblique. A sterile transparent bag must encompass the x-ray tube. As with any interventional procedure, hardcopy films or a hardcopy paper printer must be available and in good working order to document needle placement and locations of the RF lesions produced. These "spot films" are mandatory for adequate documentation of the procedure.

Informed Consent

Candidates for RF neurotomy must be fully consented and informed of what RF of the TON entails and what to expect in terms of intraoperative cooperation and postoperative side effects and possible complications. Outcomes based on personal experience, as well as quoting the scientific literature, should help the patient understand what to expect including its limited duration as well as reperformance with similar results. All possible side effects must be clearly understood and accepted. The consent should be explained, witnessed, and performed in a private or semiprivate "nonthreatening" environment and prior to any sedation being administered. Any evidence of lack of patient understanding or external psychological coercion should result in the immediate termination of the procedure and rescheduling after these issues are resolved.

Cervical Medial Branch Blocks

Name of practice or institution

Diagnostic block evaluation sheet: Cervical blocks

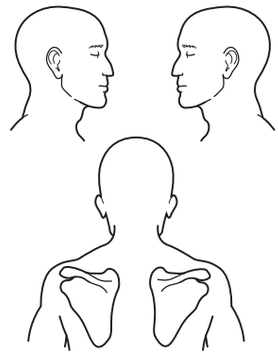
Patient's name: _____ DOB: ___/___/___
Procedure: _____ Date: ___/___/___
Doctor: _____ Assessor: _____

Descriptions

Index pain: _____
Concurrent pain: _____

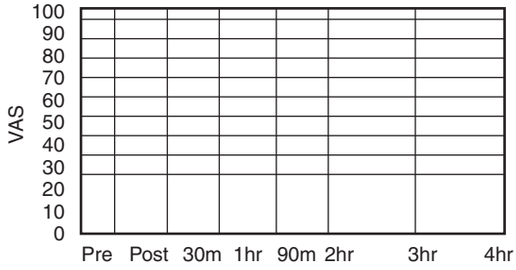
Four activities limited by index pain:
1. _____
2. _____
3. _____
4. _____

Pain map



VAS: Worst pain ever experienced: /10
Worst ever index pain: /10
Index pain today: /10

Response: ADLs restored: 1 2 3 4



Patient's remarks: _____

Assessor's remarks: _____

Corrections/comments: _____

Interpretation of response: _____

Plan of action: _____

Signed: Doctor _____ Assessor: _____
Date: ___/___/___ Date: ___/___/___

FIGURE 8–53

Postprocedure pain diary. (From Bogduk N, editor: *International Spine Intervention Society Practice Guidelines/ Spinal Diagnostics and Treatment Procedures*. San Francisco, International Spine Intervention Society, 2004, p. 12, with permission.)

Patient Positioning

Patients undergoing RF medial branch neurotomy may be placed in either the prone or lateral position with the target side facing upwards. The prone position is most frequently employed for patient stability and for the ease of C-arm rotation to obtain all views. A pillow or foam wedge is placed under the patient's chest, and a small support is placed under the forehead. This allows for greater flexion of the cervical spine, which allows easier identification of facet joints under a pillar view while allowing patient comfort and adequate ventilation.

Have the patient remove any partial plates or dentures that may obstruct clear visualization of vital structures during the performance of the procedure. The arms are

placed by the patient's side, and a Velcro wrap is placed around the patient for stability, pulling the shoulders caudad so as to not obstruct fluoroscopic visualization. Advantages of the lateral position are that it allows "face-to-face" patient–operator contact, facilitates precise placement, and minimizes the chance for excessive anterior placement near the ventral ramus.

Once the patient is satisfactorily positioned, a sterile preparation of the skin covering an adequate region over the posterior needle entry point is made. Solution for skin preparation may include an iodine-based solution (Povidine/iodine, chlorhexadine, or an alcohol based antiseptic). All personnel must wear appropriate lead aprons, personalized x-ray dosimeters, and lead-lined thyroid protection.

A radiology technician should be skilled with specialty training in spinal interventional techniques and familiar with the radiological and surgical anatomy of cervical RF neurotomy.

Sedation

To ensure patient tolerance, judicious amounts of sedative may be administered. But sedation must not impair communication by the patient should any unexpected symptoms or discomfort occur. In the event that an anesthetist is required to provide proper and safe monitoring, the level of patient consciousness, cooperation, and awareness must be communicated prior to the procedure. The effects of sedatives or anxiolytics administered throughout the procedure must be carefully monitored so that the patient remains conversant and may immediately report untoward symptoms or discomfort. Unexpected symptoms such as sudden or increased pain not relevant to the procedure must be communicated and understood by the patient.

Orientation of C-Arm

Both oblique and lateral views are required to identify the target area, perform needle insertion, and properly execute RF lesions with meticulous precision and accuracy. The goal is to effectively cover with thermocoagulation all osseous structures that may contain the variable TON neural pathways.

In order to accomplish this, the C-arm must be positioned for three views for the oblique needle pass: posteroanterior (PA), lateral, and oblique. For the sagittal pass only PA and lateral views are required. An unobstructed view of all vital structures should be performed prior to instituting the procedure.

As the procedure is performed in multiple stages utilizing multiple views, the steps are outlined in sequential order as to clarify which fluoroscopic views are required and at what stages.

Oblique Needle Pass

Block needle placement followed by the spinal (track) needle pass and electrode insertion with final electrode positioning are required to perform this stage of the procedure.

1. The first view is lateral to place the block needle.
2. This is followed by a 30-degree oblique view to place the spinal track needle. For larger needles (16–18 gauge), a spinal needle is placed first in order to anesthetize the subcutaneous tissue, posterior neck muscles, and the tissue immediately surrounding the target zone. This step is followed by passing the electrode down the spinal needle track.
3. The fourth view required is lateral to determine electrode depth, as well as confirm subsequent parallel lesions as they are walked off the anterolateral aspect of the C2-C3 facet joint superiorly.
4. Final AP is then required to ensure that the electrode has not penetrated too medially, which might indicate penetration into the posterior aspect of the intervertebral foramina (IVF), nor strayed laterally, which would then place the electrode away from the vertebral column and the target nerve.

Sagittal Needle Pass

Sagittal target acquisition, spinal needle pass, electrode insertion, and correct position for the RF lesion are required for the second stage.

1. For the second stage of the TON neurotomy, a sagittal insertion is first required. A pillar view is not required, as the TON runs transversely across the lateral aspect of the C2-C3 facet joint. A direct PA approach is therefore utilized.
2. The spinal needle is passed transversely over the lateral flange of the C2-C3 facet joint, and anesthetic delivered to the subcutaneous tissues, posterior neck muscles, and the vicinity surrounding the target zone.
3. The electrode, passed along the track needle path, is then "walked off" the posterior aspect of the lateral convexity of the C2-C3 joint. Lateral views are required to adjust the RF electrode properly.

The only validated treatment for third occipital headache has been described by Govind et al.[96] The median duration of relief was 297 days at time of publication and several other patients still were experiencing ongoing relief. Those requiring repeat neurotomy upon return of symptoms obtained 86% pain relief following repeat RF neurotomy.

To date, no other studies or attempts have been made to replicate these outcomes. These outcomes were performed only when patients were rigorously selected by dual-controlled diagnostic blocks applied using meticulous technique. As of this writing, 16-gauge needles are not commercially available; therefore the use of cannulated larger-gauge needles (18–20 gauge) is recommended.

TON RF neurotomy is performed according to the same principles and following the same precautions as applied to RF neurotomy at typical cervical levels.

Smaller-gauge electrodes reduce the number of lesions required. Large-gauge needles can be flimsy, and thus subject to bending, so that the active tip may be elevated off the periosteum. This misplacement might be imperceptible on AP projections. With the 20-gauge needle, a minimum of four parallel RF lesions would be necessary to approximate the area covered by the original study. It is recommended that the practitioner utilize the largest-gauge, cannulated electrode for the performance of TON RFN (Figure 8-54).

Stimulation: Sensory Testing

There are no valid data that would attest to the accuracy of sensory stimulation. Tissues close to the nerve may respond in a similar manner. Given that the average diameter of each medial branch is about one millimeter (the size of dental floss), sensory stimulation would not guarantee accuracy of electrode placement. A study of lumbar medial branch neurotomy has demonstrated that stimulation thresholds do not correlate with whether or not the nerve is adequately coagulated.[88]

Furthermore, given that local anesthetic is infiltrated to the anterolateral aspect of the superior articular process, any further sensory testing or stimulation is not described here. Therefore, it is not an integral step in the performance of the TON RF neurotomy.

The close proximity of the C3 deep medial branch to the TON might raise concern that an inadvertent C3 RF neurotomy may occur. The "take-off" (i.e., both medial branches may come off the parent ramus close by) makes the deep branch difficult to avoid during RF of the TON. It is invariably sacrificed, for it does not matter because the C3-C4 joint has a dual innervation, as with all the zygapophyseal joints.[117]

On the contrary, during the performance of C3 RF neurotomy, sensory stimulation might be justified. In such a case, paresthesia to the referral zone of the TON would then justify replacement of the electrode. Inadvertent RF of the TON during C3 RF neurotomy has never been reported or described in the literature, despite theoretical concerns.

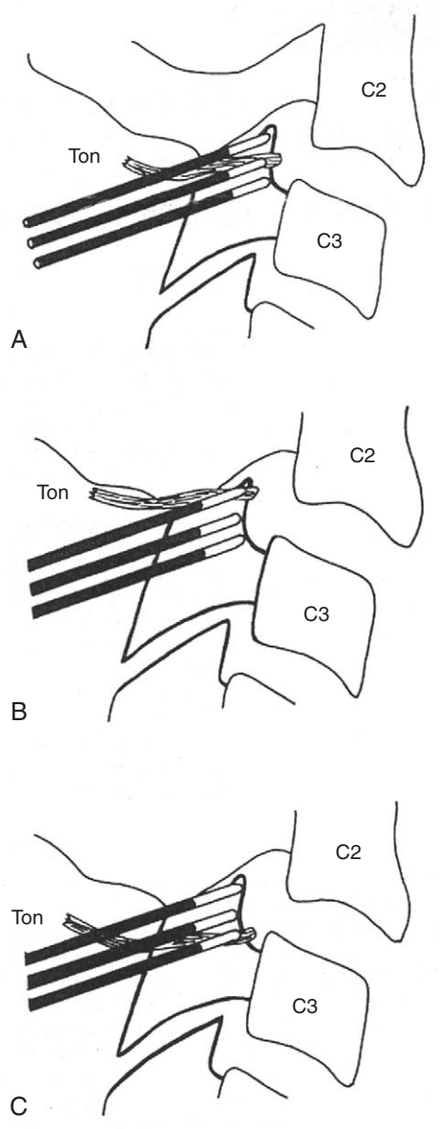


FIGURE 8–54

TON RF neurotomy requires three oblique and three sagittal parallel lesions using a 16-gauge needle at three distinct heights along the superior articular process to ensure that the TON is coagulated. Note the variable locations of the TON. (From Govind J, King W, Bailey B et al: Radiofrequency neurotomy for the treatment of third occipital headache. *J Neurol Neurosurg Psychiatry* 74:88–93, 2003, with permission.)

Motor Testing

On the grounds that an inadvertent thermal lesion may occur to the C3 DRG or ventral ramus, it is justifiable to perform motor stimulation prior to performance of the RF lesion.

This is considered by some to be a superfluous step, as the ventral ramus is significantly distant from the target zone and is therefore not a concern. Many practitioners, despite their level of experience, perform motor testing for documentation, while adding an additional level of security and comfort. The practitioner's open communication with the patient to report untoward or unexpected sensations combined with occasional motor testing is a responsible and safe approach to this step of the procedure.

Technique

Third occipital RF neurotomy is a two-stage procedure where both oblique and sagittal needle passes are used to ensure that the entire length of the nerve is coagulated. The oblique insertion, which is more technically demanding, will be performed first. This will be followed by the sagittal insertion.

The target area is the entire anterolateral surface of the superior articular process from its apex to its base opposite the bottom of the C2-C3 intervertebral foramen (Figure 8-55). On lateral views, the electrode should cover the anterior third of the superior articular process of C3, and its tip should line up with the anterior margin of the process. This is also the posterior boundary of the C3 intervertebral foramen. The electrode tip must not project beyond this margin (Figure 8-55A). On PA views, the electrode tip should project just medial to the lateral silhouette of the C2-C3 facet joint (Figure 8-55B). Line drawings are provided here to demonstrate the consecutive positions of the needles and electrodes for TON (Figures 8-55 and 8-56).

Oblique Electrode Insertion

The technique described is similar to that shown in Govind and colleagues' work. With the consideration that these procedures will be performed with smaller-gauge cannulated needles, one may choose to withdraw the block needle if it serves no purpose other than to add local anesthetic during the RF lesion stage. It is useful in that it can be served as a marker to delineate various positions (high, medium, low) of generating RF lesions.

Under lateral fluoroscopic view, the block needle is advanced laterally to contact the anterolateral aspect of the C3 superior articular process. This step is aimed at capturing the proximal end of the third occipital nerve (Figure 8-57). The third occipital nerve block is performed as described with the exception of needle placement.

A 30-degree oblique is then obtained to determine that the block needle is securely fixed on the target site. The block needle must be firmly planted into the bony surface, which ensures that the needle is not too medial (i.e., into the IVC) or too lateral that it would miss the nerve (Figure 8-58).

A 30-degree oblique view is then obtained, and a puncture point is selected that overlies the target point, which would be where the block needle is seen to be touching the superior articular process. A second spinal (track) needle is advanced down the x-ray beam toward the block needle (Figure 8-59). A lateral view should demonstrate its contact

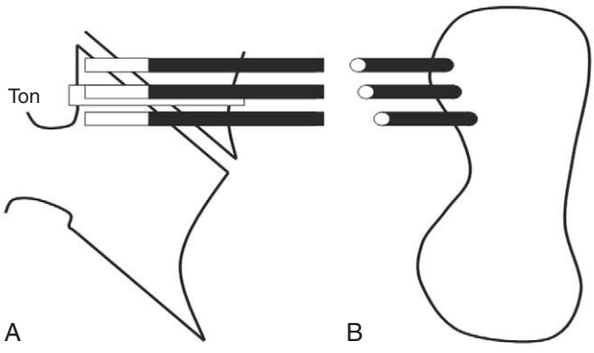


FIGURE 8–55

Sketches of the consecutive appearance of the electrode placements for third occipital neurotomy. (A) Lateral view of the oblique pass. The tips of the electrodes lie over the anterior third of the superior articular process of C3. (B) PA view of the oblique pass. The tips of the electrodes project just medial to the lateral silhouette of the C2 superior articular process. (From Bogduk N, editor: *International Spine Intervention Society Practice Guidelines/Spinal Diagnostics and Treatment Procedures*. San Francisco, International Spine Intervention Society, 2004, p. 277, with permission.)

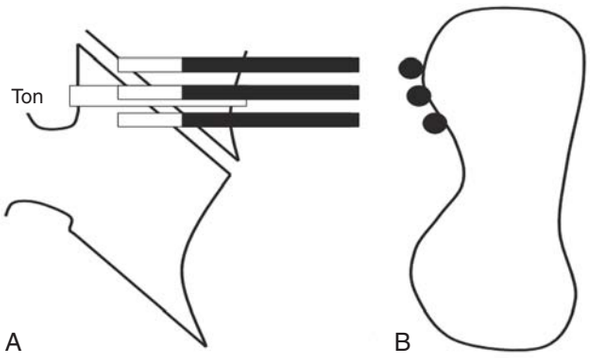


FIGURE 8–56

(A) Lateral view of the sagittal pass. The tips of the electrodes lie over the middle third of the C2-C3 facet joint. (B) PA view of sagittal pass. The electrodes are seen end-on against the lateral surface of the C2-C3 facet joint. (From Bogduk N, editor: *International Spine Intervention Society Practice Guidelines/Spinal Diagnostics and Treatment Procedures*. San Francisco, International Spine Intervention Society, 2004, p. 277, with permission.)

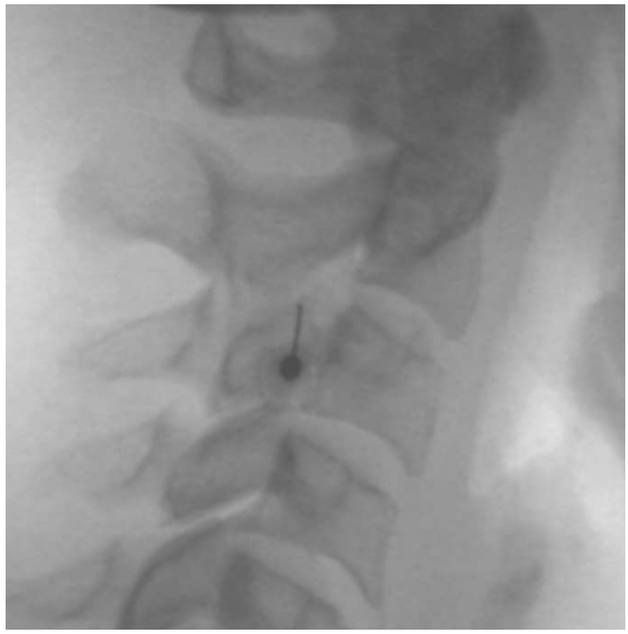


FIGURE 8–57

(A) Lateral view of a needle placed over the anterolateral aspect of the C3 process aimed at blocking the proximal end of the TON. (From Bogduk N, editor: *International Spine Intervention Society Practice Guidelines/Spinal Diagnostics and Treatment Procedures*. San Francisco, International Spine Intervention Society, 2004, p. 278, with permission.)

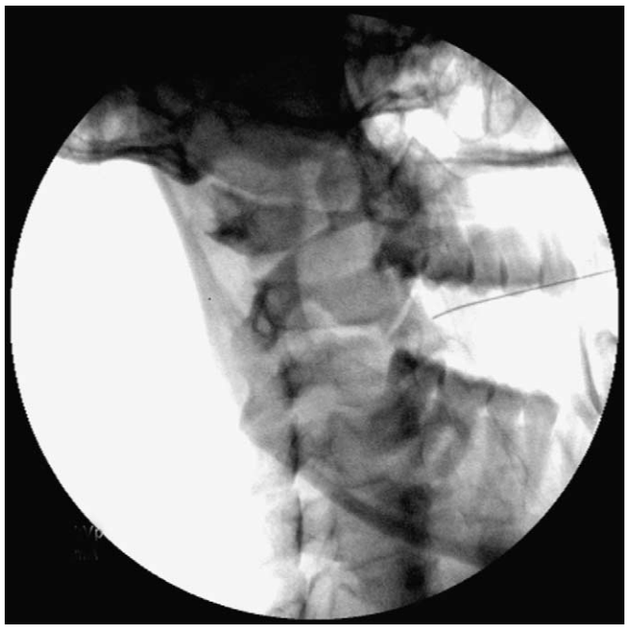


FIGURE 8–58

An oblique open-mouth view confirming placement of the block needle on the periosteum of the C3 superior articular process. (Courtesy of Jay Govind, MD.)

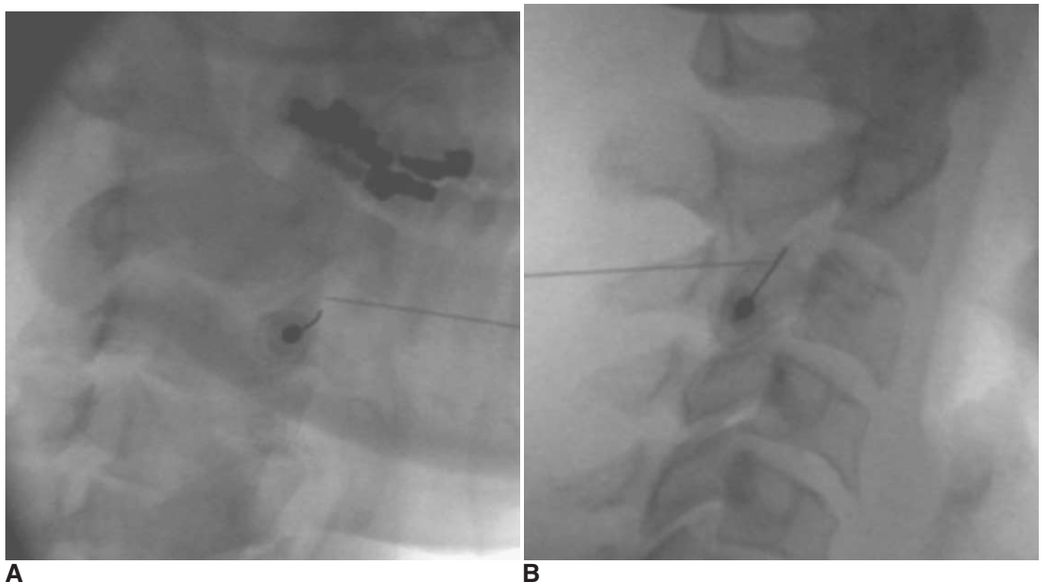
with the block needle. The tissue immediately surrounding the target zone is infiltrated with 0.3–0.5 ml of 0.5% bupivacaine. The track needle is then slowly withdrawn to anesthetize the posterior neck muscles, subcutaneous tissue and skin. Total volume of local anesthetic used should be between 3.0–5.0 ml. The block needle is kept in place.

Oblique Electrode Insertion and TON RF Lesion

The fluoroscope is returned to the previous 30-degree oblique position, and the electrode inserted down the path created by the track needle. In order that the electrode depth is monitored, its first contact during this pass should be the back of the C3 articular process. Once bony contact is made, the needle can be readjusted and advanced for a few millimeters if necessary. A lateral view is then obtained, and the final position of the electrodes adjusted. Three parallel lesions are created with the only difference being the height (i.e., superior, middle, low positions). Finally, a PA open-mouth view is used to confirm that contact has been made between the electrode and the periosteum (Figure 8-60). Sequential images are saved to copy and with a split-screen view box, the location of each lesion can be documented as one walks the needle up or down to the subsequent lesion.

The target area is the entire anterolateral surface of the superior articular process from its apex to its base opposite the bottom of the C2-C3 intervertebral foramen. On lateral views, the electrode should cover the anterior third of the superior articular process of C3, and its tip should line up with the anterior margin of the process (Figure 8-61). This is also the posterior boundary of the C3 intervertebral foramen. The electrode tip must not project beyond this margin.

From an anatomical standpoint, three discrete targets are described. The images shown here are in descending order. The high- and low-lesion PA views are also provided.



A B

FIGURE 8–59

(A, left) Oblique view parallel to the x-ray beam. Needle to block electrode track is inserted parallel to the x-ray beam toward the target zone to which the lateral block needle points. (B, right) Lateral view. The track needle reaches the tip of the lateral block needle. (From Bogduk N, editor: *International Spine Intervention Society Practice Guidelines/Spinal Diagnostics and Treatment Procedures*. San Francisco, International Spine Intervention Society, 2004, p. 278, with permission.)

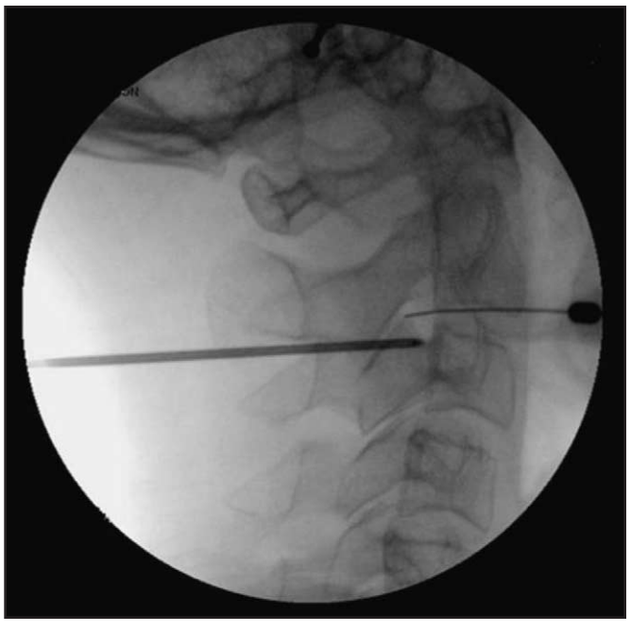


FIGURE 8–60

Lateral cervical spine showing placement of block needle and the electrode. Note that the large RRE electrode has been inserted through the posterior muscles. The tip of the block needle depicts the superior pole of the superior articular process (SAP) and is left in situ during lesioning. This allows for the administration of supplementary local anesthetic as required. In this view, the electrode is placed to RF the "low position" of the TON. Thereafter, lesions are created along the SAP between the trough of the intervertebral foramen and the tip of the block needle, which depicts the uppermost position of the nerve. (Courtesy of Jay Govind, MD.)

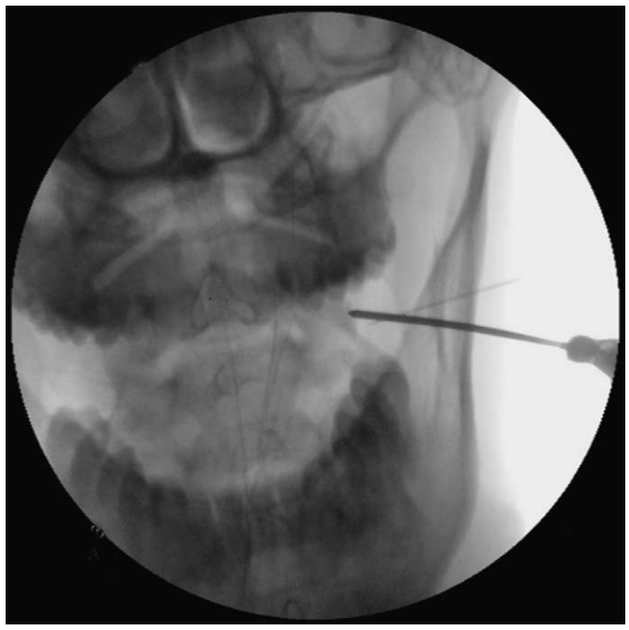


FIGURE 8–61

Anteroposterior (AP) or open-mouth view confirms placement of the electrode against the lateral margin (periosteum) of SAP of C3. There should be no gap between the electrode and the periosteum. Whether the electrode is too lateral and hence away from the nerve or too medial (too far inside the intervertebral foramen) will be revealed in the AP view. Both views must be secured and electrode placement confirmed prior to generating a lesion. (Courtesy of Jay Govind, MD.)

1. High: Opposite the apex of the C3 articular process. Lateral view (Figure 8-62), PA (Figure 8-63).
2. Mid: Between high and low (Figure 8-64).
3. Low: Opposite the bottom of the C2-C3 intervertebral foramen, lateral (Figures 8-65 and 8-66).

It is critical that prior to performing each RF lesion, a PA image is used to check that the electrode tips project medial to the lateral margins or the silhouette of the superior articular process. In addition, these views will hopefully confirm that the electrode has not moved from the surface of the vertebral column.

Once confirmation of correct electrode placement has been secured, a lesion is created at each level in the manner that was performed at other cervical levels. Due to the close proximity of the target zone and the IVF, it is recommended that a slightly "cooler," that is, 80°C lesion for 90 seconds, be performed.

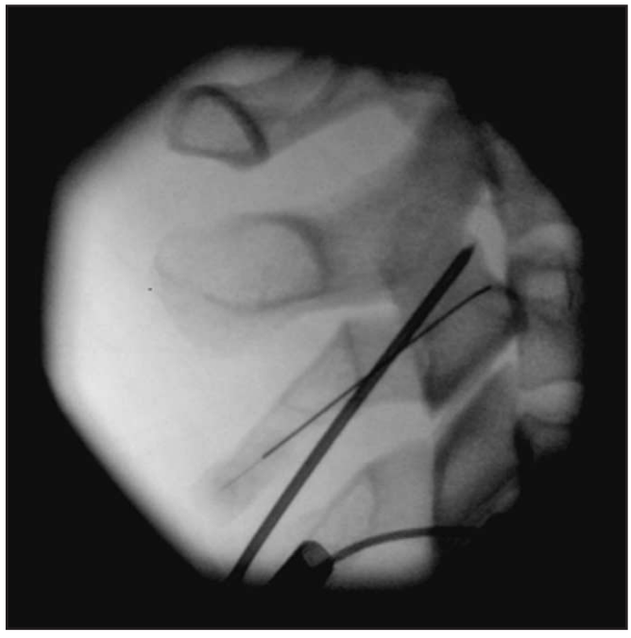


FIGURE 8–62

High position. Parasagittal pass, lateral projection RF TON. (Courtesy of Jay Govind, MD.)

Sagittal Electrode Insertion and TON RF Lesion

The second stage of the TON neurotomy involves creating a series of three sagittal lesions created from a "nonpillar" PA view over the lateral aspect of the C2-C3 joint (Figure 8-67) (note that the block needle, still in place). A similar protocol is utilized as in the oblique, where the electrode is initially advanced to the posterior aspect of the joint and readjusted over the lateral aspect of the C2-C3 joint. Lateral views are obtained to cover the target zone with three parallel lesions (again, all performed with respect to height—high, low, mid). These three targets are described:

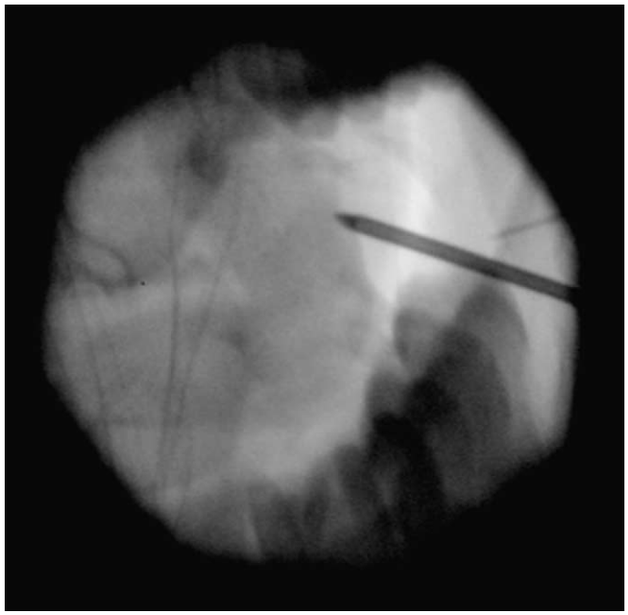


FIGURE 8–63

Posteroanterior view, high position. Confirms contact between electrode and periosteum. Electrode tip seen medial to lateral silhouette. (Courtesy of Jay Govind, MD.)

1. High: The tip of the electrode lies in the apex of the C3 superior articular process.
2. Low: The electrode should lie opposite the bottom of the C2-C3 intervertebral foramen.
3. Mid: The tip of the electrode lies between these two positions.

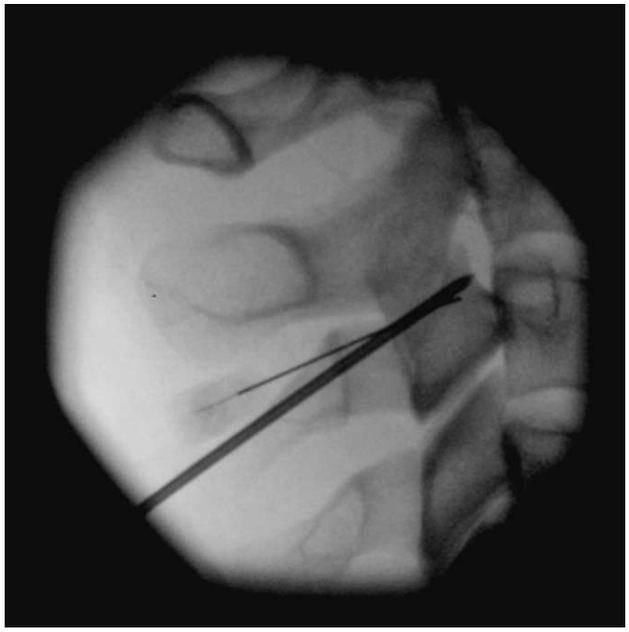


FIGURE 8–64

Mid-position. Parasagittal pass lateral projection. (Courtesy of Jay Govind, MD.)

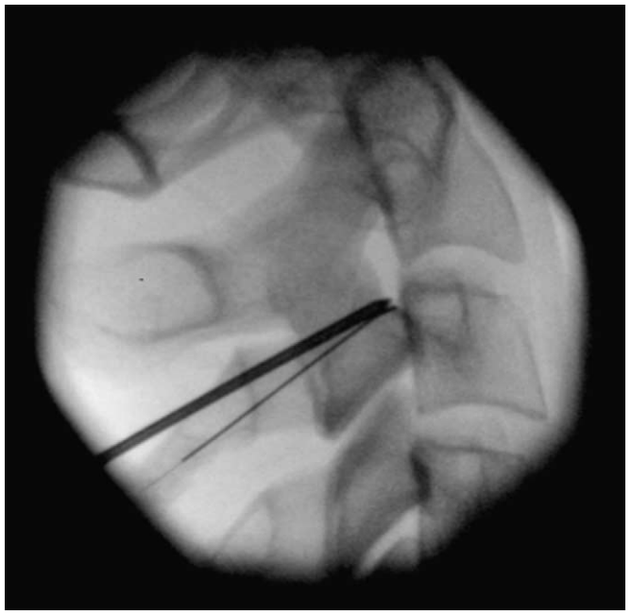


FIGURE 8–65

Low position. Parasagittal pass, lateral position RF TON. (Courtesy of Jay Govind, MD.)

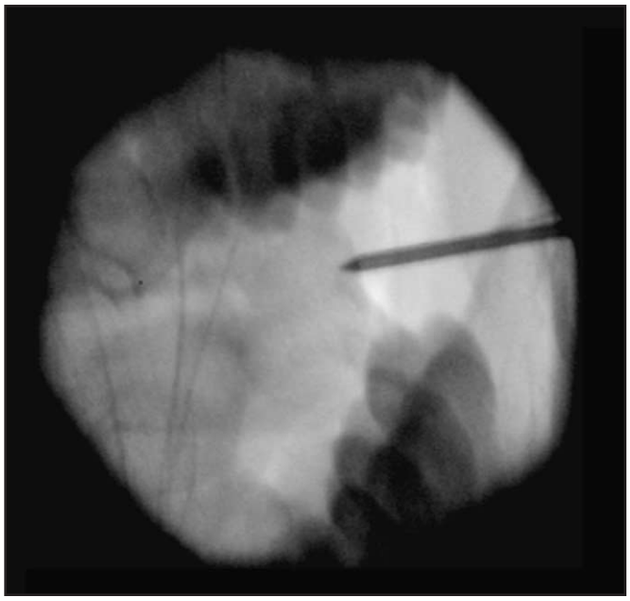


FIGURE 8–66

Posteroanterior view. Low position. Parasagittal. Note electrode tip low on SAP. (Courtesy of Jay Govind, MD.)

Following confirmation of each electrode's placement, PA views are used to check that the electrodes have not strayed laterally of the bone. Note any identifiable "gap" between the tip of the electrode. In the high position, the electrode lies typically opposite the equator of the rounded eminence of the C2-C3 facet joint. In the low position, it lies just above the waist of the C3 articular pillar. In the middle position, it lies in the middle of the lower half of the joint.

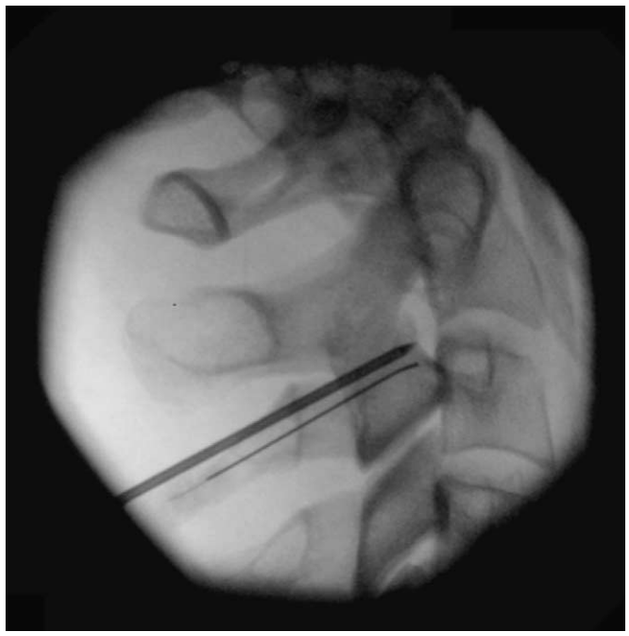


FIGURE 8–67

Parasagittal pass. Lateral view. Note that the tip of the electrode lies flush with or preferably slightly behind the superior border of the superior articular process in order to coagulate the nerve along the lateral aspect of the superior articular process. (Courtesy of Jay Govind, MD.)

Following confirmation of correct electrode placement in each of its positions, the RF lesion is generated using standard protocol. The sagittal lesions can be performed at typical temperatures (i.e., 85–90°C), with no risk to adjacent structures.

Postoperative Care

Before rising from the procedure table, patients should be reminded that they may experience unsteadiness, especially after undergoing third occipital blocks and neurotomy. Patients should be discharged into the care of an accompanying and responsible adult. They should be reminded to engage visual cues to establish horizontal. Application of cold packs for 1 or 2 days is recommended, as well as simple analgesia as required.

COMPLICATIONS

Temporary ataxia and numbness have been reported for third occipital nerve RF neurotomy. Patients must also be willing to accept possible side effects from local anesthetic block and RF neurotomy. In a study of 49 patients, Govind et al.[96] reported the following side effects:

- Numbness (in 97% of patients). Based on the cutaneous distribution of the third occipital nerve, it is expected that complete anesthesia or numbness will occur; this is a sign that the nerve has been completely coagulated. Absence of numbness indicates a technical failure. Prior to third occipital nerve block patients must be asked whether the temporary numbness they will experience will be an acceptable alternative to the pain. The numbness may last weeks, and possibly months.
- Ataxia (95%). A large portion of the semispinalis capitis is supplied by the third occipital nerve. RF denervation of a substantial portion interferes with tonic neck reflexes. The unsteadiness created, especially when looking downward, is not disabling, but the patient must understand and expect this side effect prior to the procedure. Visual cues, such as fixing on the horizon, can minimize this result. As in the prognostic third occipital nerve block, this ataxia must be discussed as an alternative to the pain. This side effect may last weeks.
- Dysesthesia (55%). This represents central disinhibition of adjacent cutaneous nerves that overlap into the third occipital nerve territory. It occurs at the border of the area innervated by the third occipital nerve and is short-lived.
- Hypersensitivity (15%). Duration is in days, rarely weeks.
- Itch (10%). Duration is in days or weeks.

Regeneration of the TON with return of pain is preceded by resolution of the above side effects. Return of normal sensation is a cue that the nerve is regenerating.

Note that these side effects were based on RF lesions created using multiple coagulations of the third occipital nerve with a 16-gauge Ray RRE electrode. Therefore, less-complete destruction of the nerve (i.e., utilizing smaller-diameter electrodes such as 18–20-gauge electrodes with a modified approach) may decrease the incidence of these side effects.

Transiently increased pain is the most common complication following cervical facet blocks and RF, with an incidence of 2%, lasting from several weeks to 8 months maximum.[118]

Chemical meningism has been noted following medial branch blocks.[119,120] A possible cause could be inadvertent dural puncture. The practitioner must confirm needle placement in more than one view (particularly in the lateral view) to establish that the needle tip is placed posterior to the neural foramen.

The practitioner must also be constantly alert for the maintenance of sterile technique. Failure to maintain sterile technique may result in less severe but still troublesome complications. For example, paraspinous tissue infections may result in abscesses.[84,121,122]

CLINICAL PEARLS

Pertinent to the TON, note that it is a single large nerve of about 1.5 mm in diameter and projects a transverse course, unlike the remaining medial branches, which are obliquely directed in a posterocaudal direction. The TON

is the only medial branch to cross a zygapophyseal joint and gives off articular branches to the C2-C3 zygapophyseal joint from its deeper surface. For RF to be successful, a larger electrode should be used in order to maintain parallelism; the electrode must be inserted transversely.

Advantages of the lateral view are that it allows the operator to maintain "face-to-face" patient contact, and that it facilitates precise placement (i.e., not too posterior, and certainly not too anterior). The tip of the electrode should not venture anterior to the anterior margin of the superior articular process (SAP). Lateral projection also allows you to plan the number of lesions permissible, depending on the height of the pillar.

With respect to avoiding inadvertent RF lesioning of the TON while performing medial branch lesions to C3, the take-off of the C3 deep branch is variable only with respect to the TON. More often the TON occupies the upper/lower or the mid-portion of the trough and the TON has a transverse trajectory. The deep medial branch runs obliquely and posterocaudally. If the electrode is kept "parallel" to the nerve and the tip slightly distal or behind the lowermost portion of the trough, RF-ing the TON can be avoided. Sensory stimulation is unnecessary.[75]

Because ataxia is common in patients who undergo procedures to the upper cervical region, and in particular those who undergo third occipital nerve blocks, the practitioner should be prepared to assist the patient in preparing for this possibility. This ataxia occurs because the blocks anesthetize the proprioceptors in the upper cervical region, which are essential to tonic neck reflexes.

In order to compensate for this ataxia, patients should be encouraged to establish visual cues at all times, focusing on horizontal objects such as window frames, doorposts, and the horizon. It is reassuring to instruct patients that this ataxia is temporary, and that most patients adjust to the new sensation within a half-hour or less. However, patients must be instructed that they should not drive a motor vehicle, which may necessitate sudden turning of the head and loss of contact with the horizon, producing giddiness and possible loss of control of the vehicle.

EFFICACY

This technique produced admirable results, reversing the poor results from use of the traditional neurotomy technique on the third occipital nerve. Using this technique, Govind et al.[96] found that 86% of 49 patients obtained complete relief of pain. When the report was published, the median duration of relief among those patients was 297 days, with eight patients still experiencing complete relief. Of the 14 patients who underwent a repeat neurotomy procedure when pain recurred, 12 regained complete relief.

Several studies have demonstrated that long-term pain relief can be produced using cervical medial branch neurotomy, and that patients who experience an eventual return of symptoms can be effectively treated with a repeated use of the same procedure.[97,109,110,123]

CERVICAL NERVE ROOT BLOCK

HISTORY

Pauchet and colleagues[124] are credited for injecting sacral nerve roots to salvage inadequate caudal anesthesia in 1914. Later, paravertebral blocks were used for surgical anesthesia. Nerve root blocks have been shown to be useful for diagnostic, prognostic, and therapeutic purposes. In 1974, Krempen and Smith[125] reported the use of nerve root blocks for evaluating sciatica. Riew and colleagues,[126] in a randomized controlled trial, demonstrated pain relief and a reduction in the surgery rate for patients undergoing nerve root blocks for lumbar radicular pain.

After it was observed that injectate can spread into the epidural space and then to other levels, Furman and O'Brien[127] asked the question, "Is it really possible to do a selective nerve root block?"

ANATOMY

In a study by Slipman et al.,[128] mechanical stimulation of the nerve root produced a distribution of pain that was different than dermatomal patterns. This technique, using mechanical stimulation of cervical nerve roots, produces pain patterns with much overlap, making the interpretation of diagnostic blocks less specific.

A more specific and predictive approach was published by Larkin et al.,[129] where individual cervical nerve roots were electrically stimulated to identify painful nerve roots by the use of a Racz catheter. Subsequent surgeries were successful. This multicenter prospective study leads to the conclusion that identifying painful nerves should lead to better treatment and outcome from nonsurgical and surgical approaches.

The anterior and posterior roots of cervical nerves C2 to C4 emerge from the spinal canal through their respective intervertebral foramina. Eight pairs of cervical nerve roots are numbered with the first root exiting between the occipital bone and atlas (C1). The number of the cervical root corresponds with the number of the cervical vertebral body below it until the eighth cervical root, which is below the seventh cervical vertebrae. Ventral and dorsal rami separate within the foramen and may trifurcate producing the ramus intermedius. The posterior primary ramus has a medial branch that innervates the zygohypophyseal joints. The sinuvertebral (recurrent meningeal) branch comes off the ventral ramus, and a grey ramus communicans intersects. Within the spinal canal, the sinovertebral nerve ascends and descends to innervate intervertebral disks and other structures above and below the level of its origin, including the dura in the posterior cranial fossa.

The first cervical nerve emerges between the occipital bone and the posterior arch of the atlas. The posterior sensory root of this nerve is much smaller than the anterior motor root, or may be much smaller than the anterior motor root it may be entirely absent.[130] Cervical nerve roots increase in size from upper to lower.

After the mixed nerves are formed by the union of the anterior and posterior roots, they divide into anterior and posterior primary divisions. The exception is the first cervical nerve, which seldom has an anterior division. Because the first cervical nerve is composed almost exclusively of motor fibers to the muscles of the suboccipital triangle and only rarely has any significant sensory component, it is usually unnecessary to block this nerve.

Dura mater may extend into the neural foramen and is tethered to the walls of the foramen and transverse process. The subarachnoid space may be present along the nerve root but not as far out as the dura.

The nerve root may exit the foramen above or below the transforaminal ligaments. After exiting the intervertebral foramina, the anterior primary rami of C3-C4 pass in an anterior-caudal lateral direction behind the vertebral artery and vein, in the gutter formed by the anterior and posterior tubercles of the corresponding transverse processes of the cervical vertebrae.[131,132] The tubercles of the transverse processes lie 0.5 inch (1.3 cm) to 1.25 inches (3.2 cm) below the skin, depending on the size of the patient and the cervical level. The lower cervical tubercles are more superficial than the tubercles of the upper cervical transverse processes.[133] The anterior tubercles are located farther cephalad and medial than the posterior tubercles.[130]

The first cervical nerve passes under the vertebral artery in its relationship to the posterior arch of the atlas and is held in place by a fibrous tunnel.[130] The anterior primary rami of C2-C4 are also held firmly on the transverse process by a fibrous tunnel. After leaving the transverse processes, these nerves are enclosed in a perineural space formed by the muscles and tendons attached to the anterior and posterior tubercles of their respective cervical vertebrae. The muscles and tendons of the anterior tubercles are the longus colli, the longus capitis, and the scalenus anterior. Those attached to the posterior tubercles are the scalenus medius, scalenus cervicis, and longissimus cervicis.[130–132]

The ascending branches (lesser occipital and greater auricular nerves) supply the occipitomastoid region of the head, auricle of the ear, and parotid gland; the transverse branch (superficial cervical) innervates the anterior part of the neck between the lower border of the jaw and the sternum; and the descending branches (suprarenal, supraclavicular, and superacromial) supply the shoulder and upper pectoral region.[134–137]

The deep cervical plexus supplies mainly the deep structures of the anterior and lateral neck and sends branches to the phrenic nerve. It also contributes to the hypoglossal loop.[137] One group of nerve branches—the lateral (external) group—proceeds from beneath the sternocleidomastoid muscle in a posterolateral direction toward the posterior triangle. This group provides muscular branches to the scalenus medius, sternocleidomastoid, trapezius, and levator scapulae muscles. The medial (ventral) group runs medially and forward to the anterior triangle. It provides muscular branches to the rectus capitis lateralis and rectus capitis anterior, longus capitis, and longus colli muscles and to the diaphragm via the phrenic nerve. By means of the ansa hypoglossi, it also innervates the thyrohyoid, geniohyoid, omohyoid, sternothyroid, and sternohyoid muscles.[131,134,137]

The cervical plexus also communicates with the vagus, hypoglossal, and accessory cervical nerves.[131] These communications may explain some of the side effects often seen with cervical plexus blockade.

INDICATIONS

Cervicogenic headaches and upper cervical pain may be evaluated and treated with cervical selective nerve root blocks. Single-level, unilateral radicular syndromes are the most appropriate diagnoses for nerve root injections. Diagnostic and therapeutic indications exist. Concerns about bleeding or severe spinal stenosis may be attractive reasons to pursue this approach as an alternative to transforaminal or translaminar techniques.

CONTRAINDICATIONS

- Local infections
- Coagulopathies
- Suboccipital craniotomy with no bone and/or distorted anatomy
- Vertical metastasis
- Contrast allergy is problematic as contrast use is important for the technique
- Cervical spinal instability
- Cervical fracture

EQUIPMENT

Local anesthetic block
25-gauge, 3/4-inch infiltration needle
16-gauge, 1-1/2 blunt Coudé, 1-inch IV cannula-introducing needle
22-gauge, 3-1/2-inch spinal needle
3-ml syringe
5-ml syringe
IV T-piece extension set
Pulsed electrode magnetic fields and RF thermocoagulation
16-gauge, 1-1/4-inch angiocatheter
10-cm with 10-mm blunt active-tip RF thermocoagulation needle
RF thermocoagulation set with cables

For cryoneurolysis
12-gauge, 1-1/2-inch angiocatheter
Cryoneurolysis probe set

DRUGS

- 1.5% lidocaine
- 0.5% bupivacaine or ropivacaine
- Steroids (optional)
- Iohexol (Omnipaque 240) contrast medium

PREPARATION OF PATIENT

Written informed consent including paralysis, numbness, weakness, worsened pain, bowel, bladder, sexual dysfunction, bleeding, and infection should be obtained.

Physical Examination

Active range of motion of the cervical spine is generally performed to provoke the patient's symptoms and to assess limitation of motion. Pain provocation can be isolated to, or emphasized in, the upper cervical spine by means of testing rotation or lateral bending from a position of protraction and retraction. The flexion and extension of the C0 to C2 segment are greatest during retraction and protraction, respectively.[134] Thus, an affliction of these segments is more accurately elicited by protraction or retraction rather than by general extension or flexion of the entire cervical spine.

Preoperative Medication

For preoperative medication, use the standard recommendations for conscious sedation by the American Society of Anesthesiologists.

TECHNIQUE: CERVICAL SELECTIVE NERVE ROOT

See Figures 8-68 through 8-71.

Indications and Precautions

The cervical nerve roots at C1 and C2 exit the canal in a posterior direction. C3 is somewhat unique in that while it courses in an anterolateral and lateral direction, it is the most posterior located cervical root. C4, C5, C6, and C7 are the most commonly injected cervical nerve roots for diagnostic and therapeutic purposes. Unique hazards are associated with C6 and C7. C8 has been described by Sluijter as a forbidden zone because of the proximity to the vertebral artery and lung. C6 and C7 should also be approached cautiously, especially if sharp needles are used because of the proximity of the lung.

C3 has rare but unique problems, especially if a posterior approach and sharp needles are used for the purpose of diagnostic facet innervation block or RF denervation. The posterior border of the C3 neural foramen in the lateral view cannot be clearly defined if there is a slight rotation of the neck. Following verification with injection of contrast, and switching the injectate to local anesthetic and steroid, a sharp needle may partially migrate into the C3 nerve root. The injected fluid can readily tract back into the nerve root and spinal cord and cause permanent myelopathy.[138] The frequency of C3-related procedures has increased since the description of the third occipital syndrome by Bogduk.[139] There are three myelopathy cases

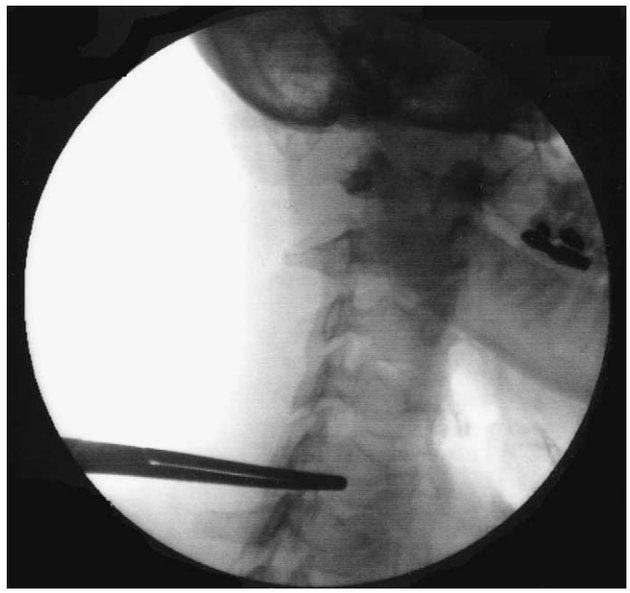


FIGURE 8–68
Thirty-degree oblique view to mark selected C5-C6 neural foramen. Skin is marked with pen at posterior cervical transverse process.

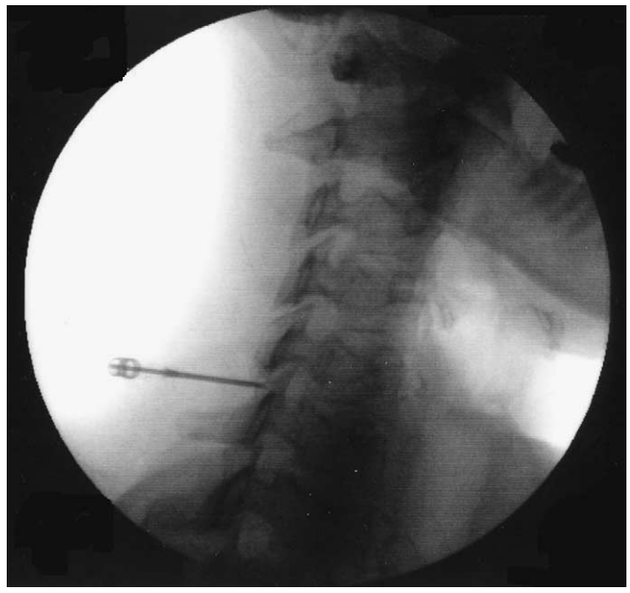


FIGURE 8–69
Sharp needle is removed, and the blunt Coudé needle is advanced through the cannula, to lateral cervical spine.

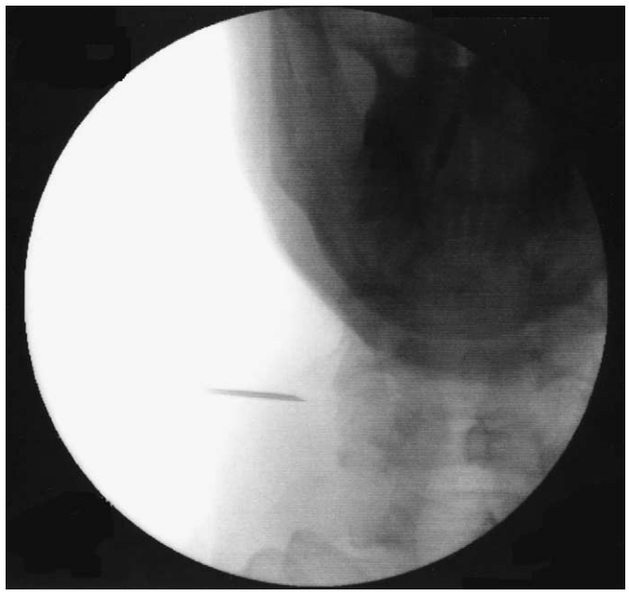


FIGURE 8–70
The 16-gauge 1-1/2" cannula introduced horizontally at skin mark, thirty-degree oblique view.

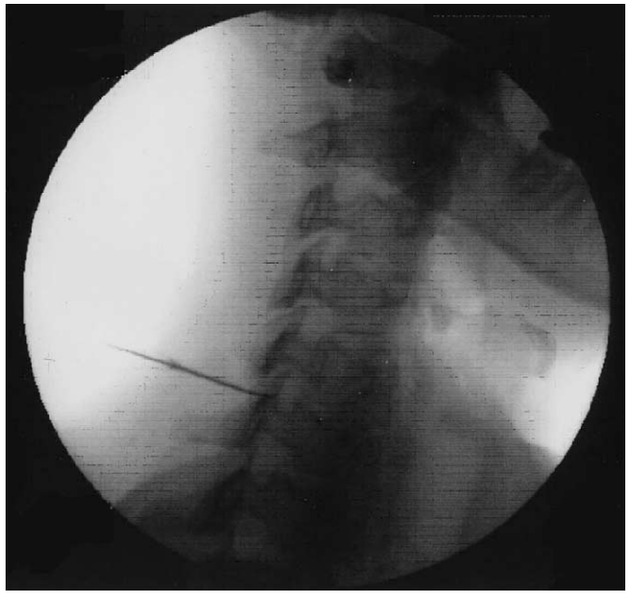


FIGURE 8–71
Thirty-degree oblique view. Blunt Coudé needle advanced until visible in neural foramen.

from the medical-legal area that have not been published. Of these cases, two had local and steroid injected and the third one local anesthetic alone, yet permanent myelopathy followed in all three procedures.

Our approach, especially at C1, C2, C3, and C8, is to use blunt Coudé needles through an introducing cannula.

TECHNIQUE: C1 SELECTIVE NERVE ROOT

See Figures 8-72 through 8-76.

Indication

Movement, particularly nodding motion–related pain with deep lateral occipital pain, is the only indication.

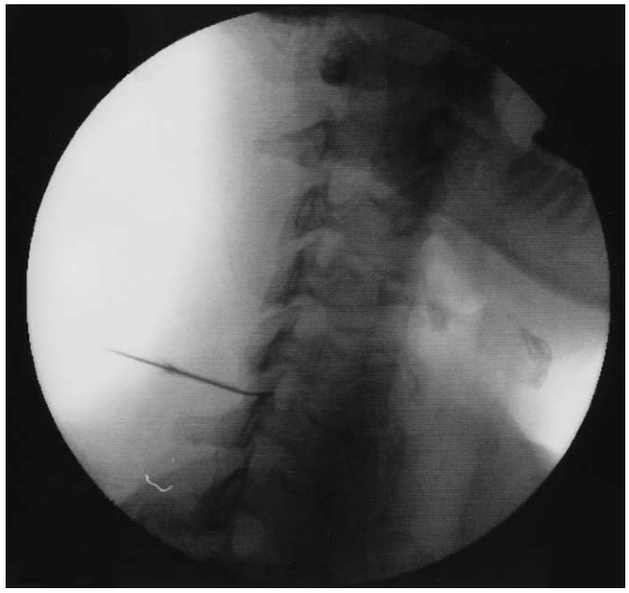


FIGURE 8–72
Blunt needle is rotated posteriorly behind the C6 nerve root and advanced on the anteroposteriorAP view to mid-facet position.

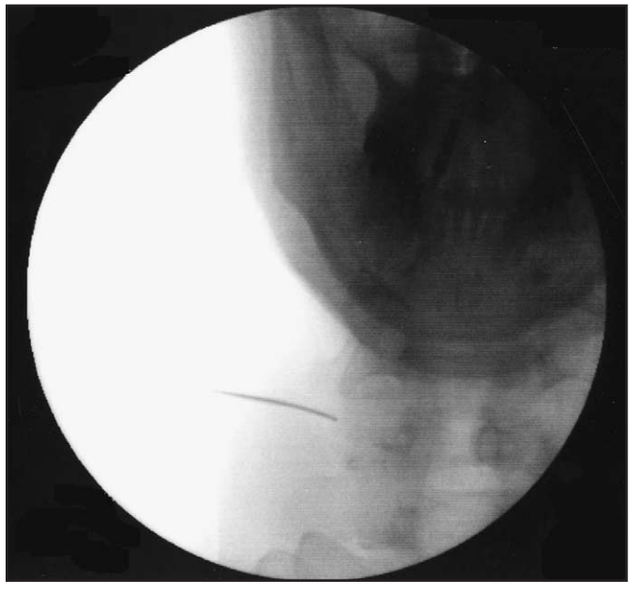


FIGURE 8–73
Anteroposterior view. Sharp needle is still present within the cannula.

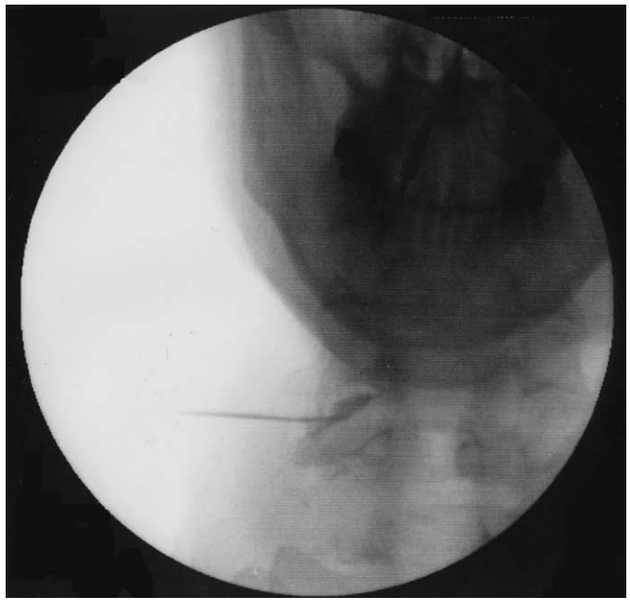


FIGURE 8–74
Anteroposterior view. Advance needle to mid-facet position.

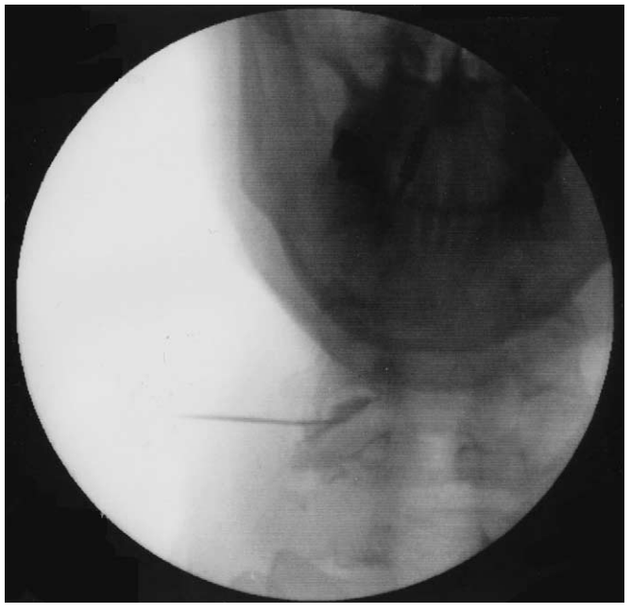


FIGURE 8–75
Small volume, 0.5 ml of Omnipaque injected (nerve root injection).

Technique

With the patient in a prone position, a paramedian approach is used. The entry point is approximately 1/2 inch from the midline. Following local anesthetic infiltration, a 16-gauge introducing cannula is passed on a lateral fluoroscopic view in the direction of the arch of C1. The metal needle of the cannula is removed and a 22-gauge blunt Coudé needle curving in a caudad direction is advanced to touch the arch of C1.

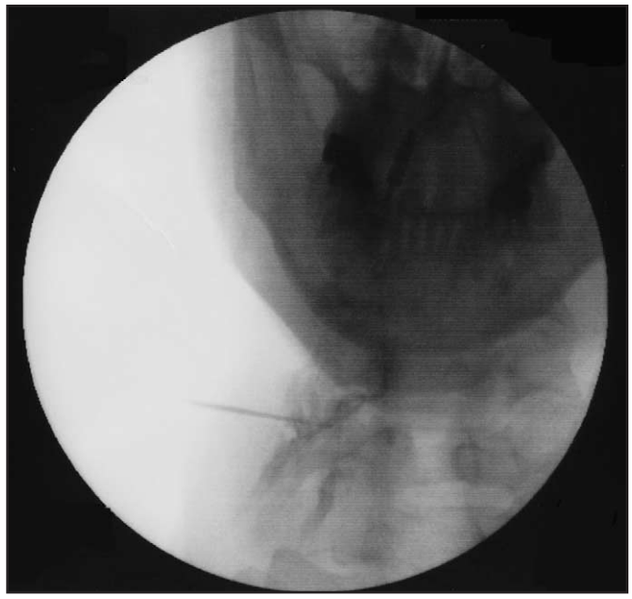


FIGURE 8–76
Larger volume, 2 ml of Omnipaque followed by 2 ml of 0.2% ropivacaine and 40 mg of triamcinolone injected. (Transforaminal injection—more spread to epidural space.)

The needle is rotated cephalad and felt to slide proximal to the arch, and then rotated in a caudad direction just over the superior edge of arch of C1. At this point, aspiration is carried out and the needle may be stimulated to verify proximity to the C1 nerve root.

If aspiration and stimulation confirm said location, 0.5–1 cc of contrast may be injected and 1–2 cc of local anesthetic/steroid solution may be injected. A specific hazard for this injection is related to the use of a sharp needle. The vertebral artery is very close and intraneural, subdural or subarachnoid injection is more of a possibility than when the blunt Coudé needle is used.

C2 INJECTION

The C2 nerve very often is involved in painful conditions especially because of the entrapment that is caused by the inferior oblique muscle. The nerve exits the spinal canal at the base of the C1-C2 joint. The patient is placed in the prone position. The C2 vertebral arch is identified. Local anesthetic is infiltrated, followed by placement in the direction of the superior pars of C2 and the medial edge is the direction where the cannula is placed. The C-arm is rotated to the lateral view because the target is more anterior than the spinous process of C2. The metal needle is removed and the blunt Coudé is advanced to come in contact with arch of C2. The tip of the needle is rotated cephalad, and as it is felt to slide over the bony edge is rotated in a caudad direction. Aspiration is carried out, and 50 Hz stimulation can be used to identify proximity to the C2 nerve root; 0.5–1 cc of contrast can verify the spread of injection; and 1–2 cc of local steroid can be deposited on the C2 nerve root.

C3-C7 INJECTIONS

These injections are carried out from a lateral approach. C6 and C7 have a more superior entry point to avoid perforating for the dome of the lung.

Our concerns regarding the C3 injection and its more posterior location has been described above. The technique for the lateral approach for the nerve root of C3, C4, C5, C6, and C7 is described below.

The patient's cervical spine is palpated, and the surface skin marking is marked for the posterior border. The patient is placed in a supine position. Thirty-degree lateral oblique fluoroscopic visualization is used with a metal skin marker where fluoroscopically the nerve root is visualized to overlay with the metal marker.

Directly underneath the marker the skin is marked with a pen so that the cross marks the skin entry. Local anesthetic infiltration of the skin is followed by the placement of an introducing cannula. On a lateral fluoroscopic visualization, the needle is advanced to the lateral mass or transverse process of the appropriate level.

Anteroposterior fluoroscopic visualization is used to confirm that the needle does not go beyond the lateral border. The metal needle is removed. The blunt Coudé needle is used with the tip curving posteriorally and is brought in contact with the transverse process. The needle tip is rotated 180 degrees in an anterior direction, and the fluoroscopic beam rotated to a 30-degree oblique view. The needle is advanced until the tip of the needle is visible in the upper part of the neural foramen. The needle is then rotated in a posterior direction and is slid into the neural foramen. Bony contact can be felt as the needle is advanced into the foramen halfway to the depth of the facet joint line.

The safety concerns regarding the transforaminal cervical injections clearly include the initial or secondary migration of a sharp needle tip into the segmental arteries that come off from the deep cervical arterial plexus and secondary infarction of the cord.[140] Therefore, over several years of avoiding sharp needle injections, our practice recommends doing cervical transforaminal injections for diagnostic and therapeutic purposes.

C8 selective nerve root is carried out rarely. The specific hazard with C8 is the small space in which the vertebral artery, lung, and the nerve root are together. The small triangular opening is visualized with the patient in the prone position, and the C-arm is rotated cephalad and slightly laterally. Local anesthetic injection is followed by placement of a 16- or 18-gauge introducing cannula in the direction of the opening between the transverse process and the rib. The metal needle is removed, the curved blunt needle is directed toward this opening, and 50-Hz stimulation is carried out as the needle is advanced. The sensory stimulation of C8 will be followed by reproduction of patient C8 radiculopathy, and following aspiration, an injection of contrast 1–2 cc of local/steroid can be injected. In patients who have C8 neuropathic pain following successful diagnostic block, pulsed RF, lesioning has resulted in long-lasting pain relief.

The "forbidden zone" concept came from attempts to inject the C8 nerve root with sharp needles, and the several infarctions secondary to those particulate steroids embolizing into the spinal cord have led to this long-held yet rarely published concern regarding C8. With the approach of the above-described blunt needle technique we have not seen intra-arterial or intraneural injection.[138]

The long-held technique of the anterior-oblique posterior-neural foramen placement of sharp needle injections has, for practical purposes, been abandoned because of unacceptably large incidence of paraplegia, quadriplegia, and death. The most likely explanation has been the secondary pathological changes to unintentional intra-arterial injection of particulate steroids. The recent description of cervical segmental arteries in the posterior neural foramina in the cervical area only added weight to this recognition that this is an area that should be avoided when using sharp needles. Blunt needle approaches have clearly added a safer therapeutic and diagnostic option for our patients.[141]

Patients who have responded favorably to the cervical selective nerve blocks, the cervical sleeve injections, or the cervical lysis of adhesion procedures often need cervical facet injections and RF lesioning at the appropriate levels. During this time patients may have back pain, and it is especially noticed first thing in the morning. Therefore, the patient is advised to wear a soft cervical collar at night to reduce the strain on the facet joints structures while sleeping. Additionally, renewal flossing exercises are recommended and instructions to the patient are given (Figure 8-20). The exercises are to be done twice a day. The main point to stress to the patient is the 20–30 seconds of sustained stretch to retain nerve root mobility.

Postprocedure Monitoring

Monitoring patients for 30 minutes following cervical nerve root injections for numbness and weakness is essential.

COMPLICATIONS

Huntoon[140] reported a case of paraplegia and spinal cord infarct after a transforaminal injection in a patient with previous spinal surgery. Houten and Errico[142] reported three cases of sudden paraplegia after steroid injection during nerve root block procedures.

Some overlapping risks exist among nerve root, transforaminal, and interlaminar blocks. A review of the complication section of the chapters on each block is worthwhile.

The incidence of serious complications is unknown. But based on historical background, it appears that the

incidence of serious complications is in the range of 1 in 10,000. No reported cases when blunt needle and introducers are used. Huston et al.[143] reported a series of 181 patients who had 306 selective nerve root injections with no major complications.

With proper technique, complications within the spinal canal, such as direct cord trauma, hematomas, and infection may be reduced. However, injectate can dissect along the nerve root to the canal and produce complications associated with other techniques, including spinal block and postdural puncture headache.

CLINICAL PEARLS

It must be emphasized that, like C2 cervical dorsal root ganglion lesioning and percutaneous cordotomy, the technique of C2 ramus communicans lesion is technically demanding. A considerable potential for patient morbidity exists if the procedure is performed by inexperienced practitioners unfamiliar with advanced fluoroscopy-guided procedures and upper cervical bony and soft tissue anatomy.

EFFICACY

Diagnostic Blocks

Strobel and colleagues[144] reported that pain relief following nerve root block was correlated with MRI evidence for stenosis and was inversely correlated when no MRI abnormality was present. Perhaps diagnostic blocks are a reasonable indication for selective nerve root blocks when imaging studies fail to confirm a diagnosis. It is unclear if therapeutic blocks are helpful following positive diagnostic blocks when imaging is negative.

Prognostic Blocks

In a study by Anderberg et al.,[145] 18 patients with cervical radicular syndromes and correlating MRI findings underwent blocks followed by provocation with neck motion. All 18 reported pain relief during provocation and postoperatively.

Therapeutic Blocks

In a randomized controlled trial, Riew et al.[126] demonstrated pain relief for lumbosacral radicular pain with nerve root injections. Riew and colleagues may be the only investigators to report a reduced surgery rate as a result of pain relief from injections. Slipman et al.[146,147] reported that selective cervical nerve root blocks were helpful in atraumatic radicular pain syndromes but not in traumatically induced syndromes.

No head-to-head studies comparing nerve root blocks to transforaminal or interlaminar epidural injections have been reported for cervical radicular syndromes.

CERVICAL PROVOCATION DISCOGRAPHY

HISTORY

In the 1940s, Lindblom,[148] a Swedish radiologist, performed the first diagnostic disc puncture and coined the term discography.[148] In 1957, Smith and Nichols[149] emphasized reproduction of the patient's pain as the key diagnostic indicator of the procedure. Cloward,[150,151] a pioneer of cervical discography, described two different types of pain caused by discography—discogenic pain and neutrogenic pain. Hirsch[152] employed the procedure to identify painful discs in patients with lumbago and sciatica. The diagnostic aspect of the procedure was the pain response, thus the name provocative discography. Lindblom[148] continued to modify the technique to use the injection of radiographic contrast to visualize radial disc rupture, and the diagnostic criteria were expanded to include the radiographic appearance of the disc and the patient's response to the injection (i.e., to provocation).

Wise and Weiford[153] were the first in the United States to visualize and study internal disc morphology. Cloward and Busade[154] continued the work and described the technique and injections for discography and the evaluation of normal and abnormal discs.

In his often-cited study of inmates in the 1960s, Holt[155] questioned the role of discography as a reliable test. While that study was flawed and has since been refuted in numerous papers, provocative cervical discography remains controversial.

Schellhas and colleagues[156] compared responses to discography in both symptomatic and asymptomatic volunteers. Patients with neck pain consistently reported higher pain scores—at least 7 on a 10-point numerical scale—opposed to the control group, which never reported pain higher than 6. The recommended operational criterion was that an evoked pain intensity of at least 7 is required, which would guard against implicating a positive disc that was only moderately painful but otherwise asymptomatic. Schellhas et al.[156] concluded in the same study that cervical disc fissures are normal age-related changes and do not indicate symptomatic pathology.

Based on Schellhas et al.[156] and Grubb and Kelley,[157] the patterns for cervical discogenic pain are indistinguishable from those of zygapophyseal joint pain. They concluded that pain patterns or mapping offer clinical utility only when the patient can identify a distinct pattern. However, they reflect the innervation of the source of pain but do not implicate a particular structure as the source of pain.

By mapping concordant pain referral patterns provoked during cervical discography, Slipman and colleagues[158] concluded that the discographer can make accurate assumptions regarding disc-level involvement by pain location and symptom manifestation. Pain patterns previously described by Schellhas et al.[156] were reproduced in this study, which also addressed whether multiple discs could create similar patterns

and whether provoked pain may emanate from multiple levels and be equally referred unilaterally as bilaterally. Unilateral symptoms were provoked as frequently as bilateral symptoms. Slipman et al.[158] concluded that, based on symptom manifestation (i.e., face, occiput, posterior neck, etc.), accurate assumptions could be made as to which cervical discs require investigation and which would not.

Bogduk and Aprill[159] demonstrated that the pain of positive discography could be relieved in certain patients by anesthetizing the facet joints at the same level, thus yielding a false-positive rate of 68% unless the facet joint pain is first excluded. To guard against false-positive responses and maximize the specificity of cervical discography, they recommended that operational criteria include the exclusion of facet joint pain.

Surgical outcomes following positive cervical discography by Slipman et al., in a 4-year follow-up study, reported outcomes on anterior cervical discectomy and fusion as perceived by patients. Patients who had a "clean" or classic discography pattern, defined as a significant concordant reproduction of pain at the affected levels but no pain at the adjacent control levels, were compared with patients who reported nonconcordant pain at adjacent segments rated greater than 4 on a 10-point scale. Good to excellent outcomes were reported more often in the group with clean discographic patterns (91%) than in the nonclassic group with nonconcordant pain at the adjacent segments (68%).[160]

Zheng and colleagues[161] examined the correlation of cervical MRI and discography in cervical spine degenerative disc disease. The objective was to compare the value of cervical MRI versus discography in selecting patients for fusion, as well as in evaluating surgical outcomes. Surgical planning was based on correlating information from cervical MRI and CT/discography. The authors concluded that MRI has a false-positive rate of 51% and a false-negative rate of 27% based on discography results with hypointense (dark) discs on MRI. Despite the fact that MRI could predict most of the painful discs, they were not always symptomatic by discography. Therefore, discography can save certain levels from being unnecessarily fused, and the combination of MRI with discography can improve surgical outcomes.

ANATOMY

A study of the ligaments and annulus fibrosis of human cervical intervertebral discs demonstrates that anatomically they are distinct from lumbar discs.

The cervical disc is composed of anterior and posterior longitudinal ligaments, periosteofascial tissue, intrinsic fibers of the annulus fibrosis, and a deep core of fibrocartilaginous material[162] (Figure 8-77).

The annulus fibrosis forms a concentric mass of thick collagen anteriorly that thins out toward the uncinate process. The anterior crescentic mass is likened to an interosseus ligament rather than a ring of concentric fibers surrounding the nucleus pulposis (Figure 8-78). The primary thickness is anterior and this is the most likely source of pain when subject to strain or tears. Minor injuries to the anterior, annulus include transverse tears at the vertebral rim, which are thus called rim lesions.[163] The annulus tapers laterally toward the uncinate process. Posterolaterally, it is essentially deficient.

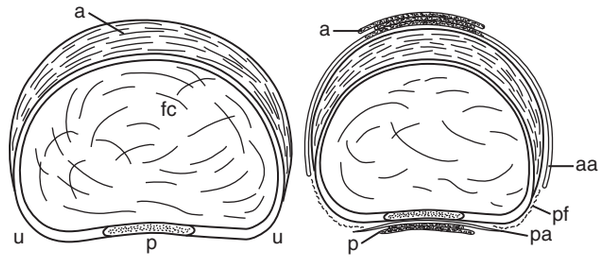


FIGURE 8–77

The cervical intervertebral disc. aa, anterior annulus; pf, periosteofascial tissue; pa, posterior annulus. (From Mercer S: The ligaments and annulus fibrosis of human adult cervical intervertebral discs. *Spine* 24:619, 1999, with permission.)

Posteriorly, the annulus is represented by a thin layer of paramedian vertically oriented fibers, which represent the only barrier between the disc and the posterior longitudinal ligament, which contains longitudinal and alar fibers (Figures 8-79 and 8-80). These fibers are the only barrier to prevent herniation of nuclear material posteriorly. An absence of posterior annular fibers leaves these alar fibers as a probable cause of discogenic pain, especially when stretched by a bulging intervertebral disc (IVD) of cervical discogenic pain.

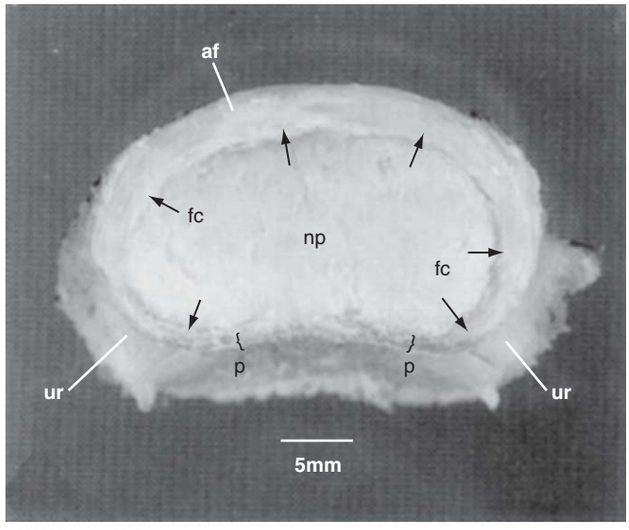


FIGURE 8–78

The three-dimensional architecture of the cervical annulus fibrosis is more like a crescentic anterior interosseous ligament than a ring of fibers surrounding the nucleus pulposis. (From Mercer S: The ligaments and annulus fibrosis of human adult cervical intervertebral discs. *Spine* 24:619, 1999, with permission.)

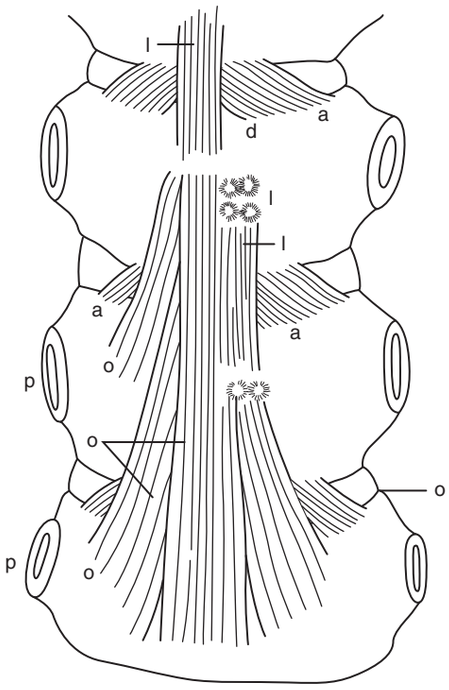


FIGURE 8–79

Anterior longitudinal ligament and fibers. S, superficial fibers; E, lateral extensions; I, intermediate fibers; D, deep fibers and thin alar extensions; T, tubercles. (From Mercer S: The ligaments and annulus fibrosis of human adult cervical intervertebral discs. *Spine* 24:619, 1999, with permission.)

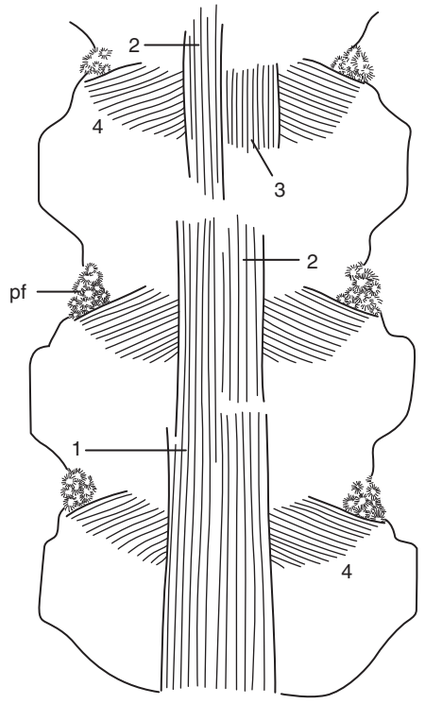


FIGURE 8–80

Posterior longitudinal ligament and fibers. 1, superficial longitudinal fibers; 2, Intermediate longitudinal fibers; 3, deep longitudinal fibers; 4, alar fibers; Pf, periosteofascial tissue. (From Mercer S: The ligaments and annulus fibrosis of human adult cervical intervertebral discs. *Spine* 24:619, 1999, with permission.)

Normal cervical movement, otherwise known as translation, causes a shear effect across the discs, forming lateral uncovertebral clefts at puberty. By the mid-30s, complete transverse fissures form through the posterior half of each disc. These age-related fissures must therefore be distinguished from those secondary to injury.[164]

The anterior portion of the cervical disc space is larger than the posterior portion, which makes it difficult for the nuclear material to move anteriorly unless great force is applied to the disc. The tough outer annulus is also thicker in the anterior portion of the cervical disc, so posterior bulging is more likely.

Radicular symptoms attributable solely to disc herniation are much less common in the cervical region than in the lumbar region. For the cervical disc to impinge on cervical nerve roots, it must herniate posteriorly and laterally. If the posterior cervical disc herniates laterally, it can impinge on the cervical roots as it travels through the intervertebral foramen, producing radicular symptoms. If the cervical disc herniates posteromedially, it can impinge on the spinal cord itself, producing a myelopathy that may cause upper and lower extremity neurologic signs and symptoms along with bowel and bladder dysfunction. Severe compression of the cervical spinal cord may result in quadriparesis or, rarely, quadriplegia.

Innervation of Cervical Disc

Nerve fibers appear to enter the disc in the posterolateral direction and course perpendicular to the fibrocartilaginous bundles in the deep layers of the annulus fibrosis (Figure 8-81). Nerves are more numerous in the middle

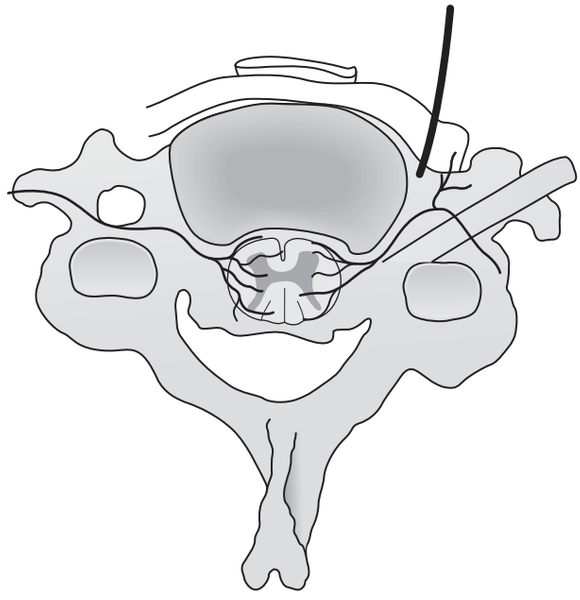


FIGURE 8–81

Innervation of the cervical disc. (Courtesy of Nikolai Bogduk, MD, and The International Spine Intervention Society.)

third of the disc. Both types of receptors were most prevalent in the posterolateral regions of the annulus fibrosis. Only three or four mechanoreceptors were identified per disc. The circumferential arrangement of the nerve bundles about the disc and the superficial to deep location of the mechanoreceptors may enable the IVD to sense peripheral compression or deformation, as well as alignment.[165]

Posteriorly, the annulus receives fibers from the sinuvertebral nerves. Laterally, fibers from the exiting spinal nerve roots provide sensory innervation and the anterior portion of the disc receives fibers from the sympathetic chain.

INDICATIONS

Neck pain is a common complaint. Overall, 35–45% of people suffer from neck and arm pain at some point in their lives. Of these patients, 30% may develop chronic pain symptoms as a result.[166] In every 100,000 people, there are 83.2 cases per year of cervical radiculopathy[167] and 38.4 cases of definite radiculopathy that are proven to be caused by disc prolapse.[168]

The most common cause of cervical degenerative disc disease is due to age-related changes. However, the degenerative process is also affected by lifestyle, genetics, smoking, nutrition, and physical activity, which reveal degenerative disc changes, may reflect simple aging, and do not necessarily indicate a symptomatic process.

Isolating the source of neck and cervical radicular pain can be a difficult challenge. While MRI can identify most painful discs, it still has relatively high false-negative and false-positive rates.[161] And, while there is a high likelihood that hypointense signals and small herniated discs are the source of pain, they are not always symptomatic.

Provocative cervical discography offers an additional diagnostic tool to determine if discogenic pain is the source of the pain and identifies which disc(s) may be causing that pain. Cervical discography is indicated as a diagnostic maneuver for a carefully selected subset of patients suffering from neck and cervical radicular pain suspected to be of discogenic origin.

Provocative cervical discography should never be the initial diagnostic tool for discogenic pain nor evaluation of such pain. It is a test of exclusion when other minimally invasive tests and therapies have failed to provide an answer. The patient should have a full clinical assessment, including neurologic exam, and have undergone MRI or CT scanning of the cervical spine. The ideal patient exhibits idiopathic neck pain consistent with cervical disc disease. Other sources, notably facet pain, are eliminated prior to discography.

Disc stimulation is based on the premise that if a particular disc is painful, then stressing that disc should reproduce the patient's pain. Alternately, if the disc is not the source of pain, stressing it should not be painful or should produce pain unlike the patient's accustomed pain. Therefore, the objective of provocative cervical discography is to reproduce the patient's usual (concordant) pain. Using a low-volume injection, the disc is stimulated and the patient is asked to rate their pain. A patient response concordant with their usual pain and symptoms is considered a positive response at that disc level. Accordingly, the diagnostic criteria of the International Association for the Study of Pain (IASP) are that provocation of the target disc reproduces the patient's pain, while provocation of adjacent discs does not reproduce pain.[169,170]

Knowledge of the symptomatic level of cervical pain can guide appropriate therapy, including surgical interventions. Cervical discography also has therapeutic utility by protecting patients from undergoing unnecessary and unjustified surgical procedures.

Patients with the following conditions/symptoms may benefit from discography:

- Persistent neck and/or cervical radicular pain when traditional diagnostic modalities, such as MRI, CT, and electromyography, have failed to identify the etiology of the pain
- Findings, such as a bulging cervical disc identified on traditional diagnostic modalities, are indeterminate for deciding whether such abnormalities are responsible for the pain
- Scheduled for cervical fusion to identify which segmental levels should or should not be fused
- Previous cervical fusion to help identify whether levels above and below the fusion are causing persistent pain (i.e., posterolateral fusion, pseudoarthrosis, etc.)

CONTRAINDICATIONS

Absolute

The patient who is unable or unwilling to consent to the procedure, or is unable to understand and cooperate with the procedure.

Any factors that would cause inability to assess the patient's response to the procedure.

Evidence of another source of neck pain.

Canal diameter less than 10 mm.

Cervical spinal stenosis, including stenosis secondary to disc herniation (Figure 8-82). Pressurization might further increase herniation, resulting in increased pain, spinal cord compression and myelopathy as a result of disc distention.

Localized infection at puncture site.

Relative

Allergy to injectates.

Extensive anterior osteophytic growth.

Pregnancy.

Anticoagulants–coagulopathy (including INR >1.5 or platelets <50,000). Anticoagulation medication

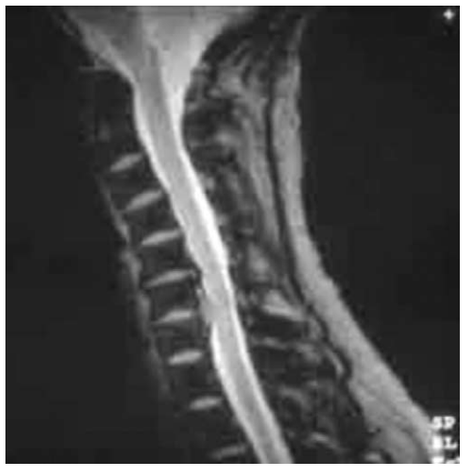


FIGURE 8–82

Cervical disc herniation at C5-C6 resulting in stenosis and a contraindication to cervical discography. (Courtesy of The International Spine Intervention Society.)

should be suspended for an appropriate period prior to executing blocks.
Any anatomic, surgical, or congenital derangement that may prohibit access to the disc or compromise safety of the procedure.
Significant cardiorespiratory compromise.
Immunosuppression.
Systemic infection.

PREPARATION OF PATIENT

History

1. Pain history.
2. Pain onset, location, quality, referral map/patterns, severity (VAS), exacerbating and alleviating factors, and associated symptoms such as numbness or weakness.
3. Prior treatments, with *response* to previous treatments.
4. Past medical history.
5. Rule out other medical conditions.

Differential Diagnosis

Evaluate for "red flag" conditions, such as malignancy and infection, metabolic phenomena, and associated symptoms such as recent onset, fever, weight loss, and night sweats. Spinal tumors or metastasis are best seen on contrast-enhanced MRI.

Physical Examination

Palpation
Location of point of maximal tenderness
Musculoskeletal
Range of motion.
Segmental restriction–useful in pre- and post-procedure assessment
Pain with movement–useful in pre- and post-procedure assessment
Atrophy
Asymmetry
Neurological examination
Sensory, motor, deep tendon reflexes, and compression testing (Spurling's maneuver) are most useful

Radiographic Imaging

The patient's MRI must be read and interpreted appropriately prior to performing discography. CAT scans are excellent at revealing bony abnormalities that might contribute to the patient's pain. The CT/myelogram is considered the "gold standard" by many but should be a secondary test because of its more invasive nature.

CT is useful, but not as accurate in delineating detail of soft tissue morphology. Bony structures are better defined under CT, as well as canal diameter, and bony abnormalities.

CT Technique

- Thin axial sections at 2–3 mm.
- Gantry angled to plane of midlevel.
- Bone detail and soft tissue windows.
- Occiput to C3 reveals only "bare bones."
- C3-C7-T1 soft tissues recognized.
- Sagittal, coronal, axial reformations from craniocervical to cervicothoracic junction.
- Plain x-rays are useful only in the context of trauma, or to rule out spondylotic foraminal narrowing.

Reviewing pain locations and referral patterns can yield valuable information. In evaluating cervical disc pathology, independent or concomitant facet joint pain must be evaluated in hopes of eliminating false-positives and -negatives. By performing bilateral medial branch blocks at the same segment as the suspected disc pathology, patients may be spared unnecessary surgery if the facets are found to be the source of the pain.

The patient must understand the rationale and potential consequences for performance of discography, and that the possibility of a surgical option exists—fusion. Similarly, the most efficacious treatment to date for facet pain is RF medial branch neurotomy. Therefore, minimally invasive percutaneous diagnostic and treatment options could lead to better patient outcomes.

Pain referral patterns from disc and facet are almost indistinguishable (Figures 8-83 and 8-84); therefore, posterior column (facet) pain must be ruled out prior to discography.

Informed Consent

- Candidates for cervical discography must be fully consented and informed of the risks of the procedure.

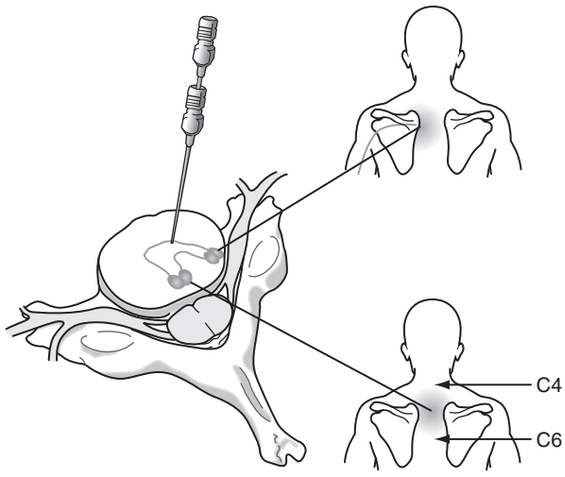


FIGURE 8–83

Reproduction of patient's concordant pain with disc stimulation. Note overlapping pain referral patterns with facet joint stimulation. (From Cloward RB: Cervical discogenic: techniques, indications and use in diagnosis of ruptured cervical disks. *Am J Radiol* 79:563–574, 1958, with permission.)

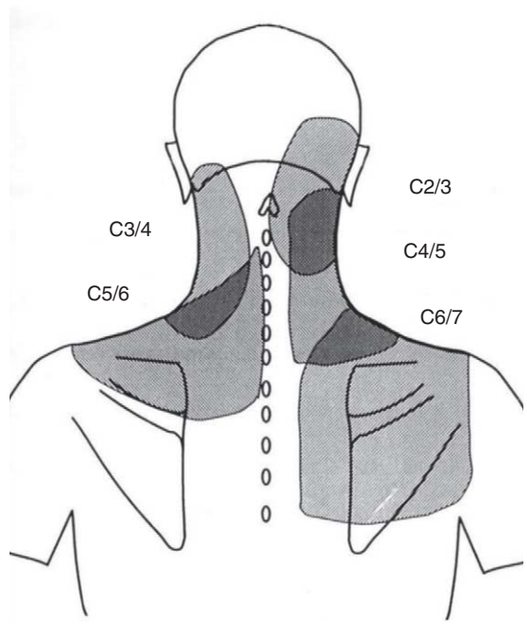


FIGURE 8–84

Typical distribution of pain referred from each cervical facet joint when stimulated in normal volunteers. (Courtesy of The International Spine Intervention Society.)

- The patient must consent to this procedure before any medications are administered parenterally or otherwise.
- The consent should be properly witnessed and all questions from the patient answered in full prior to proceeding.
- Any evidence of lack of patient understanding or external psychological coercion should result in the immediate termination of the procedure and rescheduling after these issues are resolved.

The contrast-enhanced MRI should always be reviewed preprocedurally. Spinal stenosis, herniated nucleus pulposis, and other contraindications mentioned above should be ruled out. Canal diameter must be evaluated at all levels undergoing investigation. A canal diameter of less than 10.0 mm contradicts discography (Figure 8-85).

Radiologists require instruction to measure all canal diameters. If narrative reports do not mention these diameters, the radiologist must be instructed to provide them prior to performance of the procedure. The discographer should always have a rule graded in millimeters in the procedure suite if the report is generated without narrative canal diameters.

The patient should stop all pain medications on the day of the procedure to allow for greater diagnostic accuracy. Beards should be shaved in advance to avoid freshly abraded skin. The patient should be thoroughly counseled in regard to the objectives of discography and the importance of their compliance with the procedure. Patient compliance is critical. The patient should be clearly aware that the goal is to identify the source of pain and in doing so pain will be elicited during the procedure. They should understand the concept of being asked whether that pain is similar (concordant) or dissimilar to their usual pain. Reproduction of the patient's usual pain is the critical component of provocative cervical discography. The patient should also be familiar with a numerical rating scale (1–10), not only in terms of intensity but location as well.

Additionally, the patient must be aware of the possibility of difficulty in swallowing or repetitive swallowing, production of extremity pain, unexpected neurological symptoms (production of pain at a distant site other than expected referred pain), dizziness, nausea, and so on. Inform the patient that constant and accurate communication is of utmost importance in the performance of a safe and accurate procedure.

Preoperative Medication

The patient's response to provocative discography is critical to its validity. The patient must be able to respond appropriately during the provocative phase of this procedure. While it is ideal to use no sedation in the procedure, if it

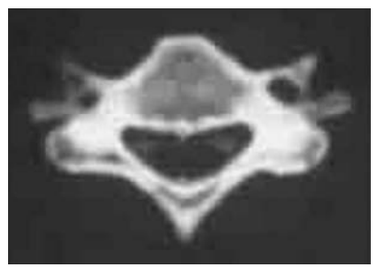


FIGURE 8–85

Mid-body–mid-sagittal diameter is 9.0 mm. (Courtesy of The International Spine Intervention Society.)

is necessary the most minimal sedation possible should be used. Careful titration of a short-acting sedative may be required in patients with unusual levels of anxiety that might prevent a clear interpretation of the procedure. Oversedation or the use of opioids must be avoided as the patient might under-report perceived pain, resulting in a false-negative response. The use of short-acting hypnotics may disorient the patient and must be avoided.

Antibiotics

- Cefazolin 5 mg or clindamycin 4 mg, for intradiscal injections.
- Cefazolin 1 g, clindamycin 900 mg, or ciprofloxacin 400 mg IV are administered within 30 minutes of starting the procedure.

EQUIPMENT

Facilities
Clean procedure room
High-resolution C-arm fluoroscopy
Radiolucent procedure table
Emergency supplies
Supplies
25-gauge, 3/4-inch infiltration needle
22–26 gauge, 3-1/2-inch spinal needle
3-ml Luer lock syringe
5-ml Luer lock syringe
IV T-piece extension

DRUGS

- Local anesthetic for skin if other than a 25-gauge needle used
- Nonionic contrast

PROCEDURE

Cervical Discography Technique

The patient is placed supine with the neck slightly extended and the head turned slightly to the contralateral side. The anterior right neck is usually chosen as the needle entry point, because the esophagus tracks to the left as it descends through the neck (Figure 8-86). The skin overlying the anterior and lateral neck is prepared with antiseptic solution.

Initially, the C-arm is placed in the AP position (Figure 8-87), with subsequent cephalad and caudal angulations to square off the vertebral endplates and optimally visualize the disc space (Figure 8-88). The C-arm is then rotated oblique and ipsilateral to the procedural side (patient's right) approximately 20–30 degrees, which will provide a view in which the trachea and esophagus will be displaced out of the center of the beam.

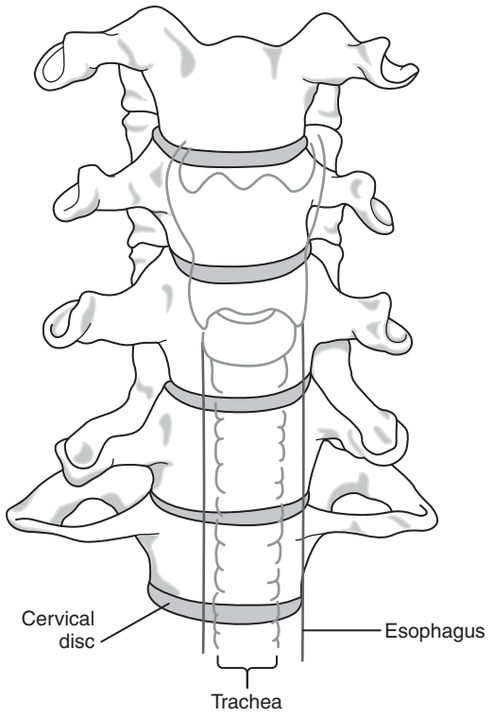


FIGURE 8–86
Drawing shows the relationship of the esophagus to the cervical spine on the left side. Because of this relationship, the needle is usually put on the right side of the disc.

To create a clear path for needle insertion, the index finger of the palpating hand displaces the carotid artery and jugular vein laterally and the ring finger pushes the trachea and esophagus medially. This displacement must be maintained to ensure correct and safe needle placement.

A 22–26-gauge discogram needle is removed from its protective cover just prior to performance of the procedure, and its distal portion is never touched by the discographer's glove to minimize contamination. A bend at the tip may facilitate fine movements as the needle can be rotated to permit easy disc entry, thus minimizing patient discomfort. The needle is then carefully advanced along a parallel beam of the fluoroscope to the anterolateral border of the superior endplate of the vertebral body below the target disc. Once the hardened osseous surface is contacted, confirmation of correct needle placement is performed using AP and lateral fluoroscopic imaging. The needle is cautiously "walked off" the bony surface in a cephalad direction until penetration of the disc annulus is perceived by a change in resistance from a feel of bone to a feel of rubber-like disc. The forward motion of the needle is stopped and the needle is held in a stable position (Figure 8-89).

At this phase of the procedure the patient must be closely observed for any signs of complications, particularly due to irritation or penetration of the trachea, esophagus, neural elements, retro pharynx, or vascular structures. These may include coughing; repeated swallowing;

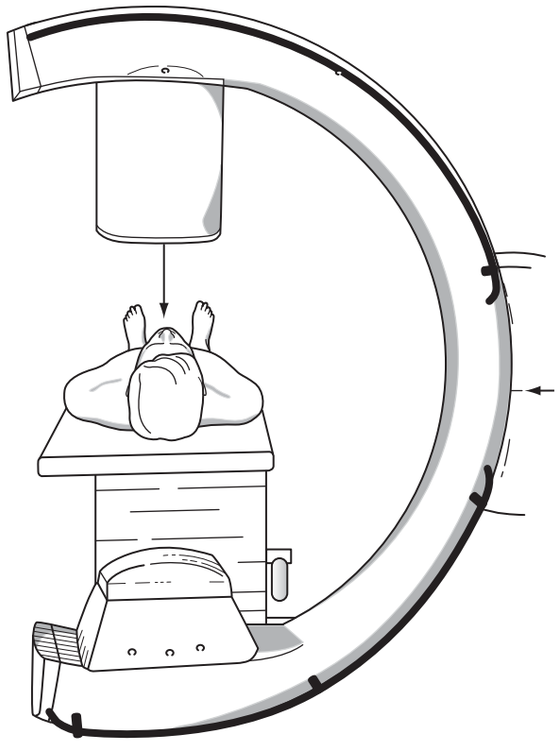


FIGURE 8–87
The patient lies supine with the C-arm in position for a posteroanterior view of the cervical spine.

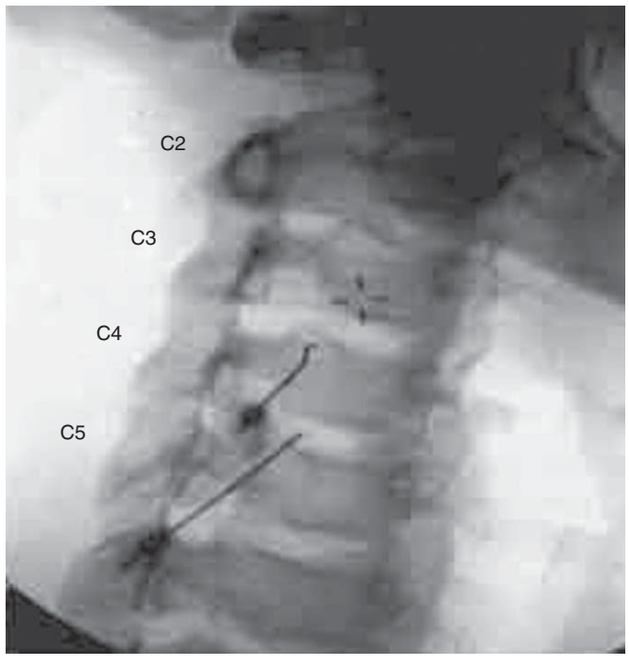


FIGURE 8–89
An oblique radiograph of the cervical spine showing a needle in preliminary position, on the superior edge of C4, en route to the C3-C4 disc. (A needle has already been placed in the C4-C5 disc.) (Courtesy of Michael Kaplan, MD, and The International Spine Intervention Society.)

struggling; respiratory distress; sudden electrical or parasthetic sensation, particularly into the upper or lower extremities; and bleeding or swelling at the injection site. Sudden onset of respiratory distress or cardiovascular instability calls for immediate removal of the needle and instituting proper medical attention.

The position of the needle must be checked under lateral imaging, and the needle advanced to the midposition of the disc (centroid) and no farther. Once confirmed as to its location in the center of the target disc, the C-arm is rotated back to an AP view, which should similarly show the needle in the centroid (Figure 8-90). The needle should never stray beyond the center of the disc in either projection. If the needle was erratically placed, readjustment or repositioning is required. The discographer must always remain vigilant as to the position of the needle tip and its relative location to the spinal canal to avoid overpenetration. Extreme care should be taken to ensure that the needle is not advanced completely through the disc and into the cervical spinal cord.

Well-aligned PA and lateral views should be taken to definitively confirm appropriate needle position in the center of the disc (Figure 8-91). Once the needles have

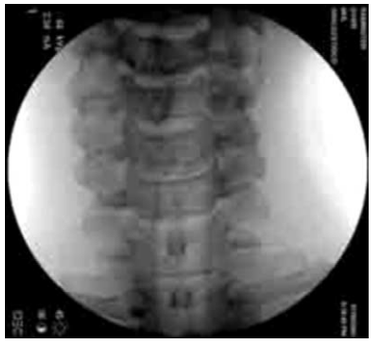


FIGURE 8–88
Cephalad and caudal angulations used to "square off" vertebral endplates. (Courtesy of The International Spine Intervention Society.)

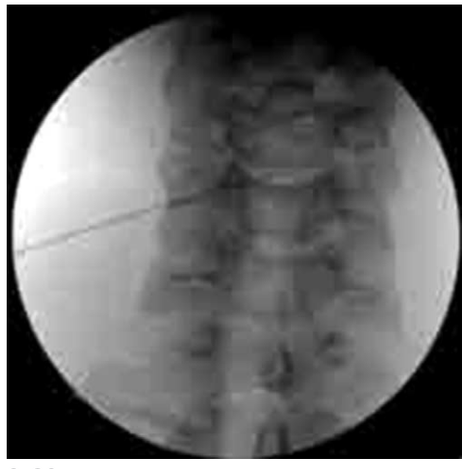


FIGURE 8–90
Posteroanterior image of needle in "centroid" position of IVD. (Courtesy of The International Spine Intervention Society.)

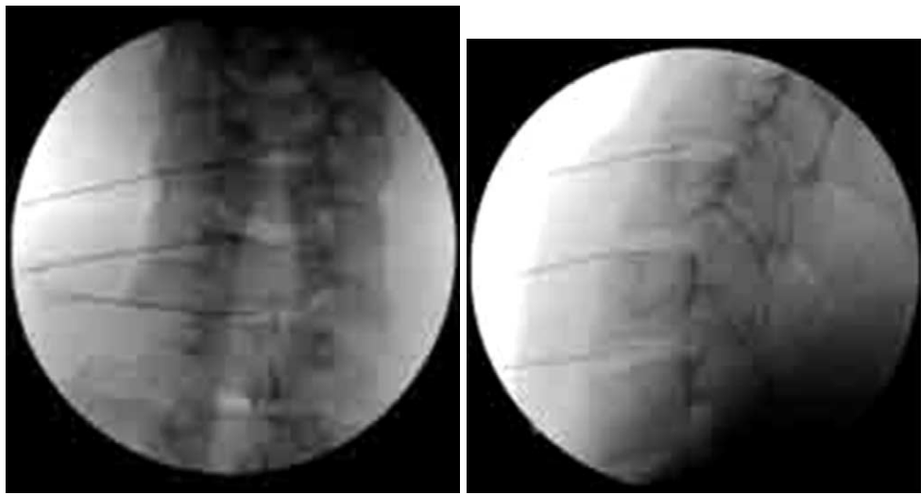


FIGURE 8–91

(A) Anteroposterior view of the appropriate needle placement into the center of the disc. (B) Lateral view of appropriately placed needles at center of target discs on three adjacent cervical discs. (Courtesy of The International Spine Intervention Society.)

been appropriately placed, the stimulation or provocative phase may begin. The discographer must have a high level of confidence that the patient is alert and oriented. The onset, severity, location, and concordancy of pain are evaluated. The quality and similarity to the patient's ongoing pain are particularly important. A verbal analogue scale is useful to help quantify the degree of pain as compared with that from injection of adjacent discs.

Injection of contrast medium into the disc is the critical diagnostic component of this procedure (Figure 8-92). Using a 3-cc syringe with a T-piece extension, contrast medium is carefully injected at 0.1–0.2 cc increments under continuous fluoroscopic visualization. A 3-cc syringe offers optimal resistance and control of injection. The syringe and extension should be free of air, so as to not adversely affect the ability to assess the pressure of the injection. The resistance to injection should be noted since intact discs offer firm resistance.

Typically, a disc will hold no more than 0.5 cc of contrast. If resistance is minimal, take care not to overfill the disc. If neither resistance nor pain is encountered, note on fluoroscopy whether contrast is escaping from the disc.

Injection is stopped under the following conditions:

- Contrast freely escapes from the disc into the epidural space.
- Firm resistance to injection is encountered.
- The patient's pain is greater than 6 of 10.
- The patient experiences nonconcordant pain (negative result).
- There is visible endplate distraction.

Concordant pain upon stimulation of the disc is considered a positive result. Pressurization should be repeated to ensure that the patient has a consistent response to repeat stimulation. For diagnostic purposes, a single-level cervical discogram is inadequate. The diagnosis of single-level discogenic pain is made if concordant pain is produced at an intensity of at least 6 of 10 and no pain was elicited upon stimulation of control discs. It is imperative that control discs, or injections of adjacent nonpainful discs, are stimulated when there is a positive disc to ensure validity and if that surgical intervention is considered. Typically, these should be performed at adjacent levels.

Discogram

Disc morphology, as demonstrated on fluoroscopy, is immaterial to the diagnosis of discogenic pain. This is called the discogram, and the internal (as well as external) architecture must be recorded on hard copy and dictated in the procedure report.

The nucleogram of a normal cervical disc shows a lobulated mass with posterolateral clefts, which develop as part of the normal aging process of the disc. Therefore, evaluation of dispersion patterns of contrast within or outside the disc serves only to confirm that the injection has been made into the nucleus pulposis.

In the normal or damaged disc, contrast material may flow into tears in the inner-annulus, producing a characteristic transverse pattern. If the tears in the annulus extend to the outer layer, a radial pattern is produced.

Contrast material may also flow between the layers of the annulus, in a circumferential pattern. Complete disruption of the annulus allows the contrast to flow into the epidural space or into the cartilaginous endplate of the vertebra itself (Figure 8-93).

Assessment

Patient parameters to be assessed and recorded are presence or absence of pain, severity of pain, and concordance or discordancy of pain.

POSTPROCEDURE MONITORING

After the procedure is complete, the patient should be observed for 30 minutes prior to discharge. The patient should be told to expect minor postprocedure discomfort,

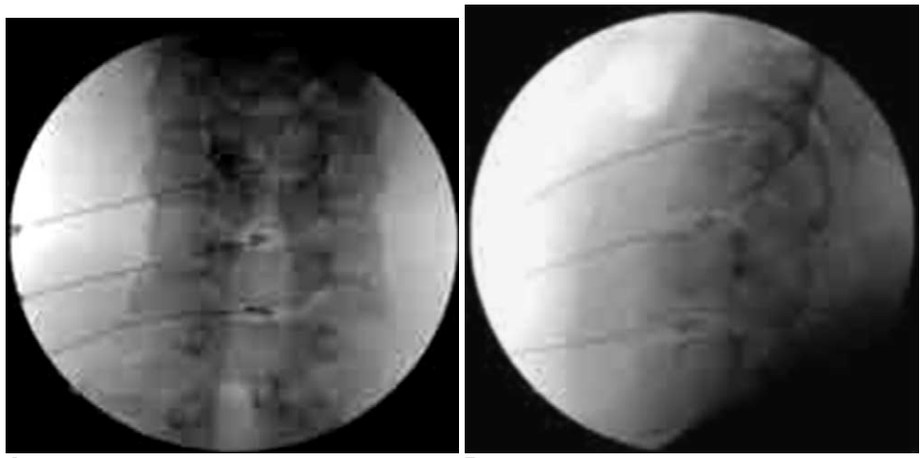


FIGURE 8–92

Top or most cephalad discogram shows lobulated mass with posterolateral clefts. This may or may not be concordant. If concordant, another adjacent control disc must be stimulated. (A) Anteroposterior view. (B) Lateral view. (Courtesy of The International Spine Intervention Society.)

which may include some difficulty swallowing. Ice packs placed on the injection site for 20-minute periods help decrease these untoward effects. The patient should be instructed to call immediately if any fever or other systemic symptoms suggestive of infection develop.

Patients are asked to report any unusual pain or pain not relieved by the prescribed medications. Severe or unusual pain may be a symptom of discitis. The incidence of this complication is extremely low, but the symptoms of discitis can present as late as 6 weeks after the procedure.

COMPLICATIONS

The proximity of the pharynx and esophagus, especially at the upper cervical levels, increases the risk of penetration with resultant contamination of the disc. Potential complications are listed below.

- Discitis. With the use of prophylactic antibiotics, the incidence is 6.4/1000 cases.
- Spinal cord trauma. Prolapse in patients with spinal stenosis or bulging discs may result in impingement.
- Vertebral osteomyelitis.
- Epidural abscess.
- Hematoma or vascular injury.
- Nerve root irritation.
- Drug-related allergic reactions.
- Inadvertent puncture of the thecal sac (which rarely results in arachnoiditis).
- Headache.
- Superior laryngeal nerve damage.
- Laryngeal damage or penetration, particularly at the C2-C3 level.
- Pneumothorax, particularly when operating at the C7-T1 level.
- Vaso-vagal response due to compression of the carotid body when manually displacing the carotid artery. (Atropine should be available.)

EFFICACY

While there has been controversy regarding the role of provocative cervical discography in the assessment of discogenic pain, it has proven to be a good diagnostic tool. Several studies have shown 70–76% of patients with satisfactory results after cervical discectomy and fusion selected by cervical discography.[171,172] Zheng et al.[161] yielded good or excellent results in 76% of patients when discography was used to select the symptomatic levels to be surgically corrected. While discography should never be the initial screening procedure to confirm discogenic pain, it is a valid measure of symptomatic cervical disc pathology. Along with physical examination and MRI, cervical discography is best used as a confirmatory procedure. Appropriately utilized, provocative cervical discography can aid in the diagnosis and management of cervical discogenic pain.

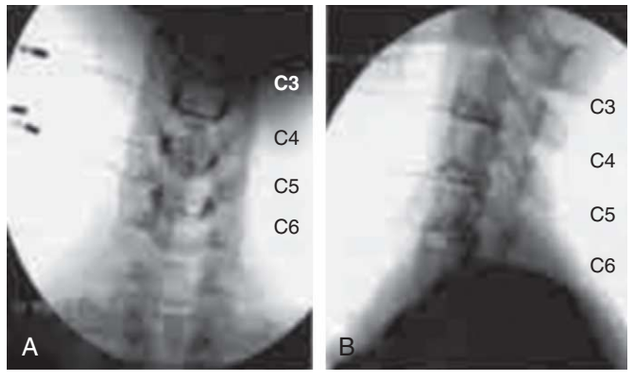


FIGURE 8–93

Spread of contrast medium out of uncovertebral joints with extensive posterolateral fissuring. Note abnormal disc morphology does not implicate the disc as positive or negative. (Courtesy of Michael Kaplan, MD, and The International Spine Intervention Society.)

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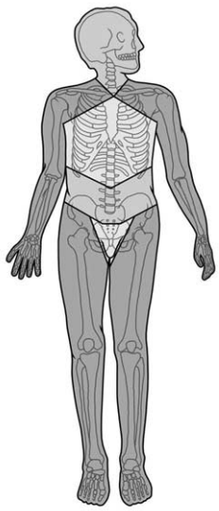
C H A P T E R



9

Joint Blocks of the Head and Neck

MICHAEL HAMMER, CARL E. NOE, AND GABOR B. RACZ

TEMPOROMANDIBULAR JOINT BLOCK

HISTORY

Toller[1] described injecting corticosteroid into the joint in 1970. However, today injections are limited to perhaps one injection with corticosteroid for inflammatory conditions such as rheumatoid arthritis. Diagnostic injections with local anesthetic alone and lavage are more common. Much of the attention focused on temporomandibular joint dysfunction has been directed away from the joint itself toward other mechanisms for the syndrome such as pain in the masticatory muscles, the bio-psycho-social model of pain, and the neuromatrix model. Myofascial trigger point injections are commonly performed for the myofascial component of temporomandibular joint dysfunction.

Alpaslan et al.[2] reported that arthrocentesis and sodium hyaluronate injection might reduce nitrite, nitrate, and thiobarbituric acid–reactive substance that may be involved in the development of temporomandibular syndrome.[2]

Yura and Totsuka[3] reported no correlation between improvement after arthrocentesis and magnetic resonance imaging findings, but that there was more improvement among patients with less opening at baseline.

ANATOMY

The temporomandibular joint is bounded superiorly by the articular (mandibular or glenoid) fossa and inferiorly by the mandibular condyle. An articular disk separates an upper and lower joint space. The axis of the joint is tilted anteriorly to the occlusal plane about 25 degrees. The superior joint space extends anteriorly to the articular eminence, anterior to the articular fossa, and articular tissue extends farther to the preglenoid plane. The inferior joint space is confined to the area of the condyle.

Masseter, temporalis, and lateral pterygoid muscle spasm are common in patients with temporomandibular dysfunction. Nociceptors are present in the joint capsule, lateral ligament, and posterior disk. Nerves are supplied via branches of the auriculotemporal and masseteric nerves and postganglionic sympathetic fibers.

INDICATIONS

Diagnostic injections with only contrast and local anesthetic are most common after other diagnoses have been reasonably excluded by noninvasive means. Joint lavage has been advocated. A single injection with corticosteroid may be useful in patients with inflammatory conditions after the diagnosis of a noninfectious inflammatory disorder of the temporomandibular joint is reasonably certain.

Pain with function (eating, speaking), pain with opening, joint tenderness, and crepitus are the four signs of temporomandibular dysfunction, but these are not specific to temporomandibular joint pain. Inflammatory, degenerative, neoplastic, and post-traumatic processes involving the temporomandibular joint respond to principles of painful joint treatment including analgesia and range of motion exercises regardless of the joint involved.

For neurolytic procedures, failed TMJ surgical procedures are the primary indication.

CONTRAINDICATIONS

Patients with chronic pain and pain disorder are less likely to respond as are patients who have been injected previously. A series of three corticosteroid injections is not consistent with any guideline for treatment of temporomandibular joint dysfunction.

PREPARATION OF PATIENT

Written informed consent including risks of pain, no pain relief, infection, and bleeding should be obtained.

EQUIPMENT

- 25-gauge needle
- 22-gauge B-bevel 1-1/2-inch needle
- Connecting T-piece
- Two 3-ml syringes for contrast agent and local anesthetic/steroid
- One 1-ml syringe for local anesthetic

DRUGS

Local anesthetic block lidocaine
1 ml bupivacaine
Neurolytic block
0.5 ml local anesthetic, 0.5 ml corticosteroid
6% phenol in saline

TECHNIQUE

The patient is positioned in the supine position. Fluoroscopy is used for lateral and anteroposterior (AP) views (Figures 9-1 and 9-2). The zygomatic arch is palpated in front of the external auditory meatus. The patient is asked to open and close the mouth. The moving condyle of the mandible is palpated and marked with a marking pen. After sterile preparation and draping, horizontal fluoroscopy is carried out where the angles of the mandible line up to be seen overlapping. Local anesthetic infiltration is carried out just inferior and posterior to the skin mark. The 22-gauge and 1-1/2-inch needle is connected to the T-piece and 3-ml syringe with contrast Omnipaque 240. The needle is passed beneath the zygomatic arch medially, superiorly, and slightly anteriorly until bony contact is made (Figure 9-3). The needle is held in position, and the patient is asked to slowly open the mouth as contrast is injected. The glenoid process will be seen outlined by contrast in a concave shape (Figure 9-4).

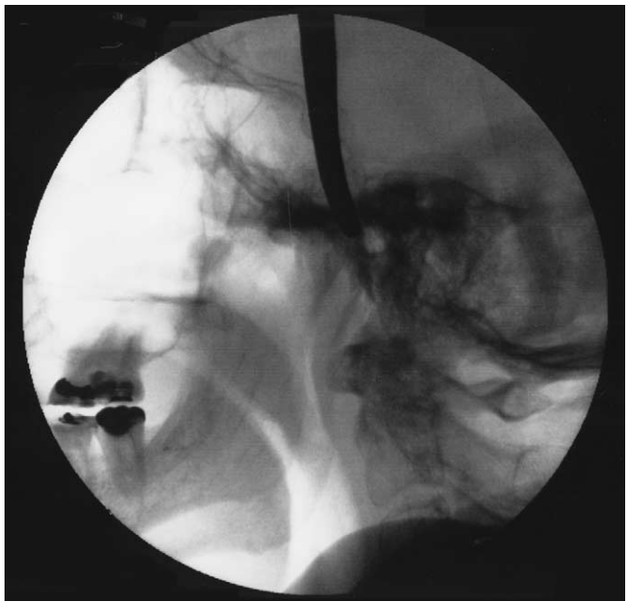


FIGURE 9–1
Lateral view with forceps used as marker.

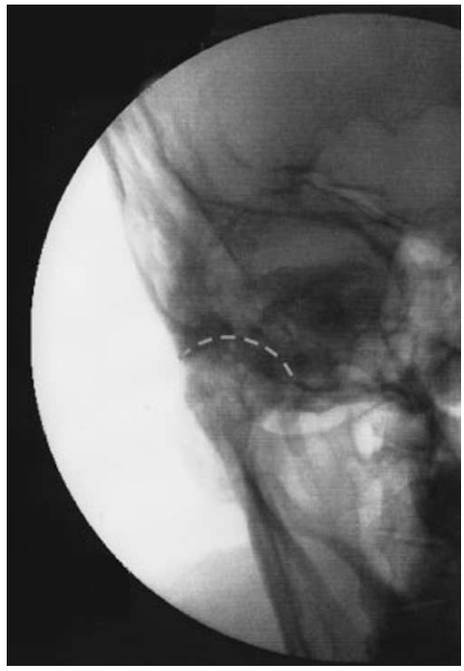


FIGURE 9–2
Anteroposterior view with and without outline of temporomandibular joint.

The C-arm is rotated to an AP position, and the tip of the needle and contrast is verified to be within the medial border of the mandible (Figure 9-5). One to 2 ml of local anesthetic and steroid are injected. The needle is removed, and pressure applied.

CASE EXAMPLES

Case 1

A 32-year-old, x-ray technician slipped and fell hitting the tip of the mandible. Postinjury, he developed posterior mandibular pain that failed to respond to medications and splinting by an orthodontist. On evaluation he was diagnosed with temporomandibular joint pain; after undergoing the above procedure, he experienced 3 months of pain relief. The pain returned to a lesser degree, and the procedure was repeated with no pain at the 4-month follow-up.

Case 2

A 34-year-old computer programmer with a history of seven temporomandibular joint surgeries including prostheses, rejection, and failed medical and psychological therapy was barely able to open his mouth. Following repeated

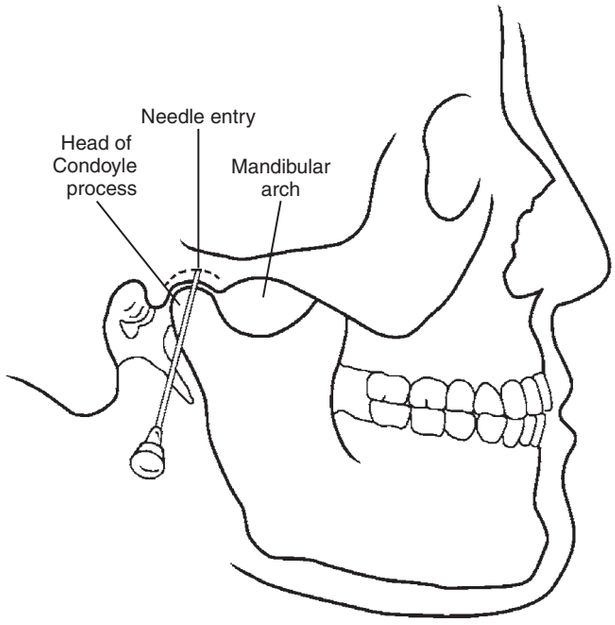


FIGURE 9–3
Lateral approach to temporomandibular joint.

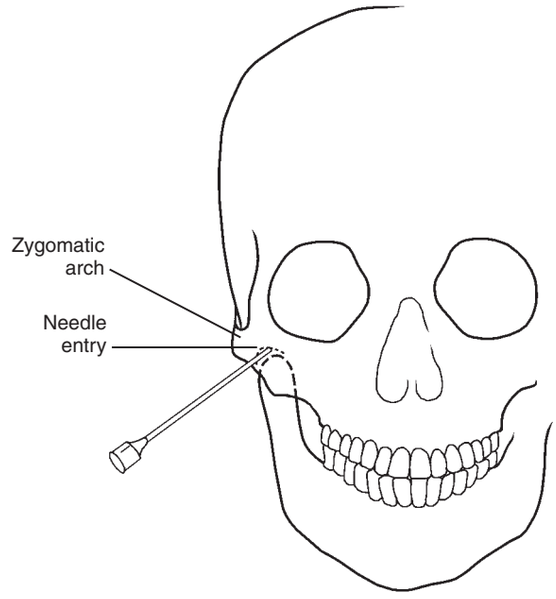


FIGURE 9–5
Anteroposterior drawing of temporomandibular joint injection.

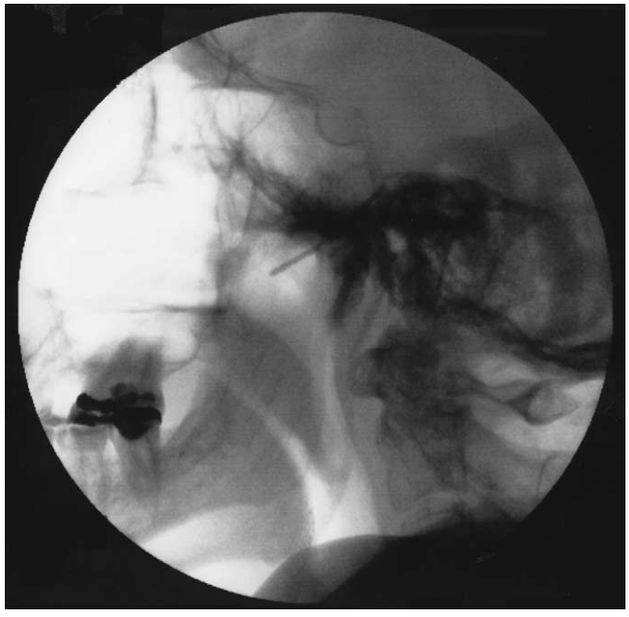


FIGURE 9–4
Contrast filling temporomandibular joint (see arrow).

injections that gave positive but temporary responses, a neurolytic 3% phenol injection was performed with an encouraging response. The phenol injection was repeated 2 months later, allowing the patient to open the mouth but still with pain. The patient was placed on continued psychological treatment and a nonescalating dose of 15 mg of methadone three times a day. His condition remained unchanged during the subsequent 14 years.

Case 3

A 47-year-old patient suffering from TMJ pain unresponsive to medical, psychological, and orthodontic treatment was losing weight due to pain associated with eating. Following TMJ diagnostic block twice with a favorable response, she had a single 3% phenol TMJ injection, allowing her to eat and gain 30 lb and achieve normal body mass index. She remains on low-dose medication.

POSTPROCEDURE MONITORING

Patients should be monitored for effects of sedation and local anesthetic.

COMPLICATIONS

- Infection
- Bleeding
- Pain

EFFICACY

The outcomes literature is scant. Vallon and colleagues[4] did not detect a significant long-term benefit from corticosteroid injections compared to injection of other substances used for analgesia. Bryant et al.[5] reported that morphine injections into the joint did not provide relief.

ATLANTO-OCCIPITAL JOINT BLOCK

HISTORY

Busch and Wilson[6] described injection of the atlanto-occipital and atlantoaxial joints in 1989. Dreyfuss and colleagues[7,8] described a lateral approach and also mapped referred pain patterns from the joints with provocative injections. The atlantoaxial joint refers pain to a more narrow transverse band at the level of the joint compared to the atlanto-occipital joint, which refers pain to a more broad area from the occiput to the base of the neck.

ANATOMY

The atlas (C1) is unique in that it lacks a vertebral body and, instead, functions as a disc, or "relay center," between the occiput and C2. The cranial articular surfaces for the occiput are large and biconcave, complementing the occipital articular surfaces. These joints are more anterior compared to facet joints from C2-C3 and inferior. The innervation to the joint is from the ventral root, so no medial branch block is possible at this level. The posterior arch lies deep under the skin, hence palpation is challenging. The anterior and posterior arches form the triangular spinal foramen that accommodates the brain stem. The transverse processes are long and perforated, accommodating the passage of the vertebral arteries through the transverse foramina. After exiting these transverse foramina, they course through grooves that can be observed posterior to the lateral masses. These grooves, or occasional tunnels, accommodate the vertebral arteries as they loop for a second time in the upper cervical region. Bone changes can occur here that have the potential to compromise vertebral artery function and promote symptoms associated with vertebral basilar insufficiency.[9] The architecture at these grooves is different in men and in women; as a consequence, women may be more susceptible to arterial compromise.[10]

Noteworthy is the location of the accessory nerve nuclei, found in the spinal cord between C1 and C4.[11] Patients who suffer from chronic upper cervical conditions may experience increased tone in their upper trapezius muscles ("tight traps") secondary to sensitization and reorganization of interneurons at those same levels. This reorganization sensitizes the cranial nerve nuclei, increasing the efferent signals to the trapezius. A similar condition can arise from the trigeminal nuclei in the spinal cord between C1 and C4.[11] Chronic afferents from the cervical spine can also sensitize these cranial nerve nuclei, resulting in chronic headache in the cutaneous trigeminal distribution.[12] Note that the vertebral artery at this level lies lateral to the atlantoaxial articulation as it courses through the C1 and C2 foramina.

INDICATIONS

Deep suboccipital pain that is movement related can be one of the most elusive pains to diagnose and treat. Busch and Wilson[6] used intracapsular injection of local anesthetic and steroid for suboccipital pain instead of putting the patient's head in a neurosurgical frame to immobilize it entirely.[13] The atlanto-occipital joint pain is usually associated with "nodding" (flexion-extension) as opposed to atlantoaxial joint pain, which is usually associated with rotation. Theoretically, once the motion ceases, the pain responds favorably to the stabilization of the fusion. Most patients, however, prefer repeat injections in the hope that the anti-inflammatory effect of injections will "settle the disease down" and limit pain from the swollen joint.[14]

Another indication is for headaches and pain caused by isolated injury to the atlanto-occipital and atlantoaxial joints. This is especially true when the pain is occipital or suboccipital and is exacerbated by the neck movements typically associated with these joints.[14]

CONTRAINDICATIONS

- Local infection
- Coagulopathy
- Cervical vertebrae/spine instability

EQUIPMENT

- 25-gauge, 3/4-inch infiltration needle
- 22-gauge, 1-1/2-inch spinal introducer needle
- 25-gauge, 3-inch spinal needle
- 3-ml syringe
- 1-ml syringe
- IV T-piece extension

DRUGS

- 1.5% lidocaine for infiltration
- 2% preservative-free lidocaine
- 0.25% levobupivacaine/0.2% ropivacaine
- Iohexol (Omnipaque 240) radiographic contrast medium
- 1 ml dexamethasone, 4 mg/ml

PREPARATION OF PATIENT

Written informed consent including risks of quadriplegia, ataxia, pain, no pain relief, bleeding, and infection should be obtained.

Physical Examination

When rotation is performed at the end of the range of protraction or retraction, the segment most likely to be painful is C1-C2. On the other hand, when side bending is

performed at the end of the range of protraction or retraction or nodding, the joint most likely affected is C0-C1.

A limitation of motion at the C0-C1 or C1-C2 segments renders the upper cervical spine incapable of compensating for coupling at cervical disc segments. This can manifest itself in a number of "deviated" patterns during active cervical motions.[15]

Nodding the head from a position of end-range cervical axial rotation allows assessment of the range of upper cervical spine flexion. It is primarily the C0-C1 segments that perform this movement; thus, a lesion at this level could cause pain or limitations of motion during this test.

Laboratory Studies

Complete blood count and other appropriate testing to rule out osteomyelitis may be appropriate.

Preoperative Medication and Monitoring

For preoperative medication, use the standard recommendations for conscious sedation and monitoring by the American Society of Anesthesiologists.[16]

PROCEDURE

Position of Patient

The patient is placed in prone position with the neck slightly flexed; the fluoroscope is rotated in a caudal-cephalid direction to open up the atlanto-occipital joint (Figure 9-6). The posterior approach to the atlanto-occipital and atlantoaxial joints is commonly used because of the safety it affords due to the tortuosity of the vertebral artery complicating a lateral approach.[17] Place several blankets under the chest to allow the head to be slightly flexed.

The fluoroscope C-arm approaches the table from the head in an anteroposterior direction. It is then rotated in the sagittal plane so that the beam passes from the anterosuperior aspect to the posteroinferior aspect.[18] This rotation is done under fluoroscopic visualization until the atlanto-occipital joint is visualized.

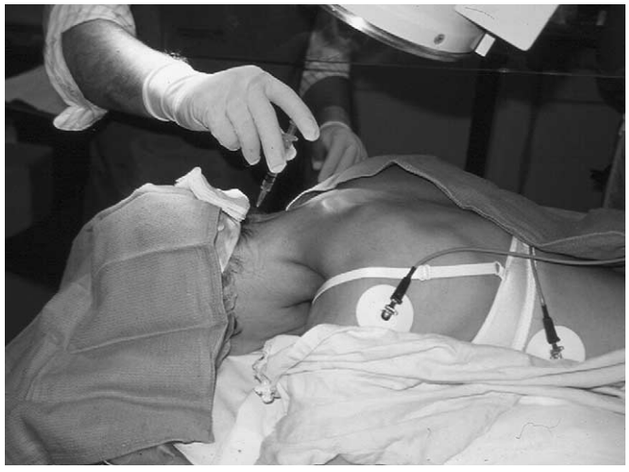


FIGURE 9–6
Patient positioning and initial C-arm orientation for atlanto-occipital block.

Technique of Needle Entry

The skin is prepared and draped in the usual sterile fashion, and a skin wheal is raised with local anesthetic at the insertion site. Use a 22-gauge, 1-1/2-inch spinal introducer needle from slightly medial to lateral "gun-barrel" view. For reasons of safety, an introducer needle is preferred to break the skin and establish the direction toward the target. The 1-1/2-inch needle will not reach the contents of the spinal canal. The C-arm is rotated into the lateral view to verify that the shaft of the introducer needle is pointing at the posterior lip of the atlanto-occipital joint (Figure 9-7). If necessary, readjust the direction to the joint. Rotate the C-arm back to the "gun-barrel" sagittal view, verifying that the shaft of the needle is following the x-ray beam (Figure 9-8).

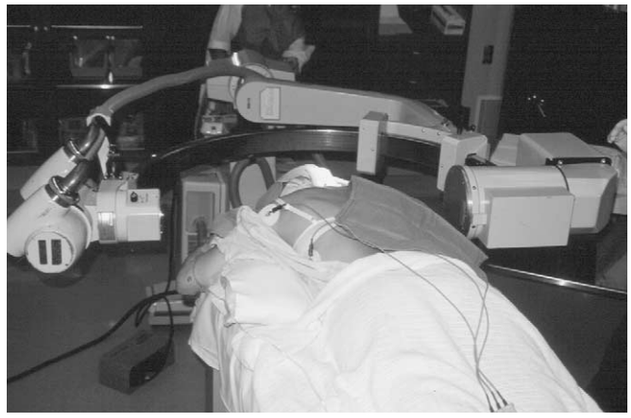


FIGURE 9–7
Lateral C-arm orientation for atlanto-occipital block.

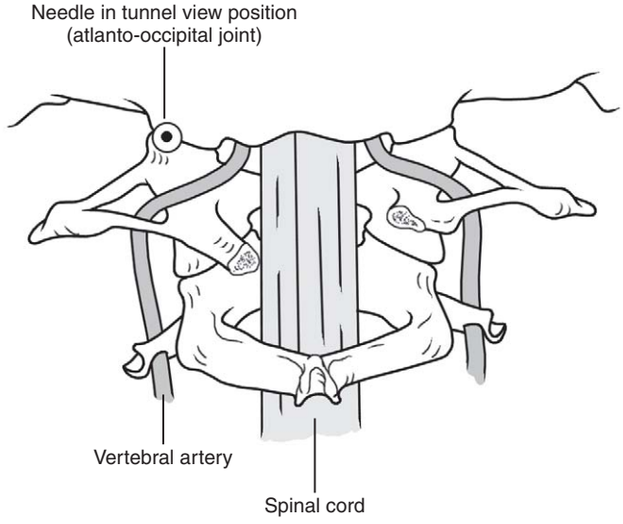


FIGURE 9–8
Needle in tunnel view position.

The introducer needle, appearing as a small point on the screen, is placed and directed toward the posterolateral aspect of the atlanto-occipital joint. The 25-gauge, 3-inch needle with a 1/4-inch, 15-degree bend, and the connecting T-piece are attached to the needle to indicate the direction of the bend. The 25-gauge needle is advanced through the introducer needle in small movements. Each movement is followed by a new fluoroscopic picture to appropriately steer the needle into the atlanto-occipital joint. Before reaching the joint one or two lateral fluoroscopic views are taken to verify that the direction to the atlanto-occipital joint is maintained. When the needle reaches the joint a distinct "pop" is felt. The C-arm can then be rotated to the horizontal plane, and the needle can be seen to have entered the joint. The atlanto-occipital joint is anterior to the posterolateral columns of the spinal cord.[14] The fluoroscopic image then further confirms positioning the needle in the atlanto-occipital joint (Figures 9-9 and 9-10).

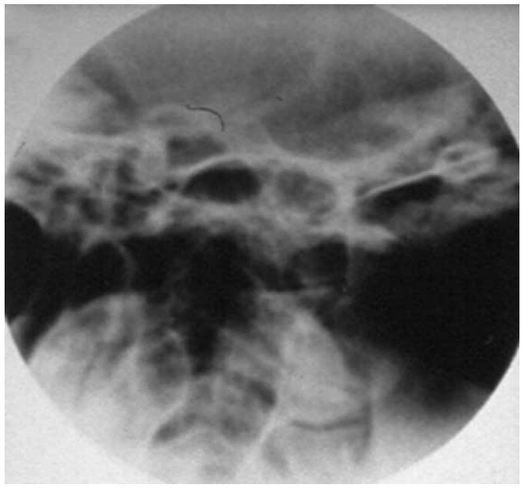
Prior to injection, aspirate; if the results prove negative on the lateral view, 0.2–1.0 ml of Omnipaque 240 contrast is injected to verify appropriate outline of the atlanto-occipital joint under fluoroscopy using a flexible T-piece connected to the spinal needle (Figure 9-11). Attention to the above detail is important because the atlanto-occipital joint is anterior to the posterolateral columns of the spinal cord. Reproducing the patient's pain by the provocation of injection is extremely common.

The vertebral artery lies inferior and medial to the atlanto-occipital joint, having passed from lateral to medial below it.

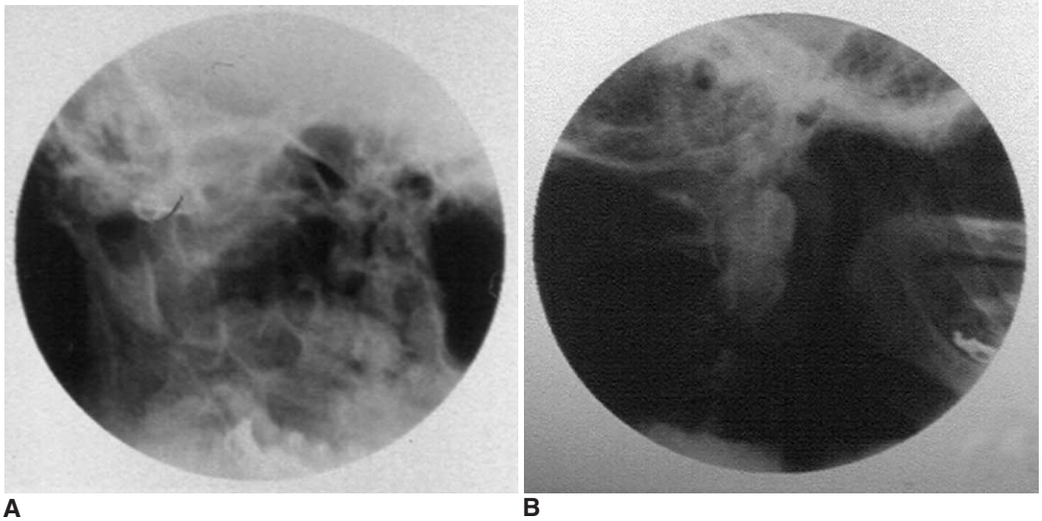
Injection of Contrast Agent and Its Interpretation

With good placement, the classic bilateral concave dye pattern is seen on lateral fluoroscopic view, representing the dye's lining of the joint capsule. It is not uncommon, in the presence of trauma, for the dye to penetrate the torn capsule and enter the cervical epidural space.

Venous runoff of dye almost always heralds placement of the needle outside the joint, which is surrounded by a rich venous plexus. In the presence of venous runoff or spread of dye into the epidural space, the injection is stopped and the block is abandoned. The incidence of venous runoff in our practice over the previous 15 years dropped after the switch to the use of the introducer spinal needle and 25-gauge spinal needle with a 15-degree curve at the distal 1/4 inch. Biplanar views are again checked before injection of local anesthetic to ensure safe and appropriate placement of the needle. If the dye spread remains circumscribed and the joint is outlined as described earlier, 1–1.5 ml of 0.2% ropivacaine with 20 mg of triamcinolone or 4 mg dexamethasone is slowly injected (Figure 9-12).[14]



FIGURE–9–9
Atlanto-occipital block: anteroposterior view.



A **B**

FIGURE 9–10
(A and B) Atlanto-occipital block.

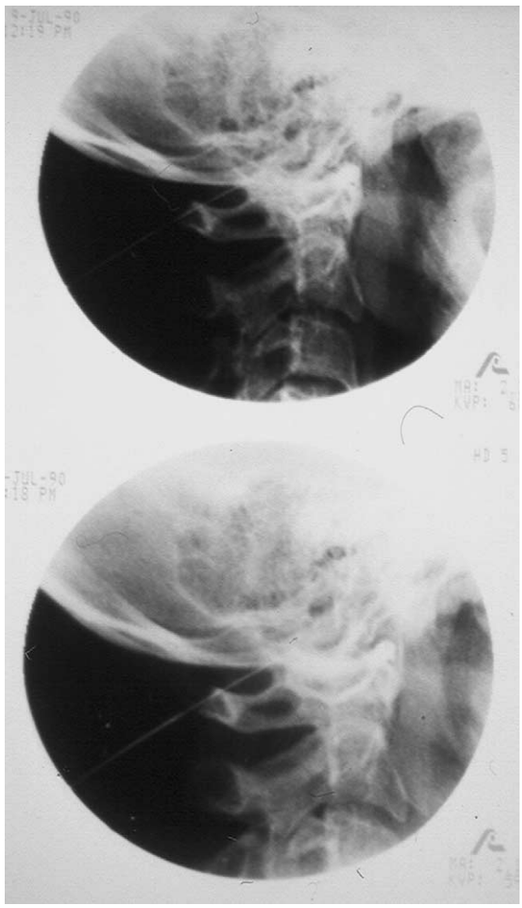


FIGURE 9–11
Atlanto-occipital block. (A) Needle placement. (B) Contrast filling joint.

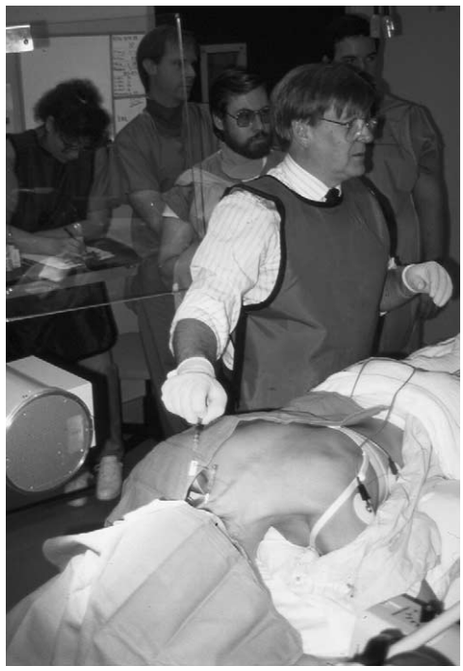


FIGURE 9–12
Injection must be carried out under live continuous fluoroscopic visualization.

Hyalgan Injection

One milliliter of Hyalgan, a lubricant used for knee injection, has also been employed. If helpful, this injection is repeated three to five times.

POSTPROCEDURE MONITORING

Brief periods of ataxia in the immediate postblock period have been noted by the authors and others.[6,14] This ataxia may be secondary to the reproduction of pain by joint distention or by the absorption of local anesthetic into either the vertebral arteries or the valveless venous plexus that is posterior to the facets. Patients are therefore observed for approximately 30 minutes before discharge.[14]

COMPLICATIONS

Complications of atlanto-occipital blocks include epidural and intrathecal injections and intravascular injections into the adjacent venous plexus, vertebral artery, and possibly the carotid artery. The reasons for all the above-described precautionary steps are the proximity of the spinal cord, vertebral artery, and nerve roots. One must always keep in mind that the atlanto-occipital joint is anterior to the upper cervical spinal cord! Although only a small amount of local anesthetic is injected, its proximity to the brain ensures a higher intracranial concentration than would be anticipated, and it is possible for symptoms common to local anesthetic central nervous system toxicity to result.[14] In over 15 years of practice, we have not seen any serious complications in our university-based practice when the above principles have been utilized.

CLINICAL PEARLS

There are known cases of quadriplegia following injection of particulate steroids. The physician implementing the procedure must avoid placing a needle into the brain stem or the vertebral artery and injecting either air or particulate matter such as precipitated steroids.

EFFICACY

Using a posterior approach for atlantoaxial injections, Glemarec et al.[19] reported better results in patients with inflammatory conditions compared to mechanical. The authors used 1 ml of contrast and 1 ml of corticosteroid without local anesthetic, presumably to avoid local anesthetic complications. Clinical experience justifies its use,

especially when the alternative is a major surgical procedure with instrumentation and fusion.

LATERAL ATLANTOAXIAL JOINT BLOCK

HISTORY

The discovery of an effective diagnostic procedure for upper cervical pain and headache proved to be a difficult task for decades. Even though the upper cervical synovial joints have been pinpointed as a likely source of that pain for at least 90 years,[20] standard imaging techniques and other assessment measures were unreliable for diagnosis.

Beginning in the 1980s, practitioners began to use diagnostic blocks to analyze pain caused by the upper cervical joints. Attention focused on headaches and C2-C3 zygapophyseal joint pain.[21,22] Techniques were also developed to diagnose the condition of the atlantoaxial (C1-C2) joint.

One such technique described by Bogduk[23] involved anesthetizing the C2 spinal nerve, which innervates the atlantoaxial joint. Another, described by Ehni and Benner,[24] used peri-articular blocks to determine if arthritic lateral atlantoaxial joints were symptomatic. Intra-articular injection of the atlantoaxial joint was described by McCormick[25] in the late 1980s.[25]

After Dwyer et al.[26] demonstrated that noxious stimulation of the C2-C3 zygapophyseal joint could induce headache in normal volunteers, Dreyfuss and associates[27] conducted similar tests of the lateral atlantoaxial joint. Stimulation of the joint induced occipital and suboccipital pain.

Although Busch and Wilson[28] reported producing headache relief following lateral atlantoaxial joint blocks, there have been few clinical studies of the technique. The only full paper on the subject was published recently by Aprill et al.[29] In a practice audit they reported headache relief using blocks in 60% of the patients whose pain was suspected to stem from the lateral atlantoaxial joint.[29]

ANATOMY

The axis (C2) is best recognized by the dens, or odontoid process, which projects craniad from the front of the bony segment, and in appearance resembles an asparagus spear. The dens and C2 form a pivot around which turn C1 and the head (Figure 9-13).[30]

The anterior atlantoaxial (or atlantodental) joint is located between the dens and the anterior arch of the atlas. Posterior to the dens is the joint between the dens and the transverse ligament of the atlas (TLA), the inferior part of the cruciform ligament, which can also be interpreted as the bursa atlantodentalis.[30]

The lateral atlantoaxial joint, seen from a sagittal view, is biconvex on both the left and the right sides (Figure 9-14).

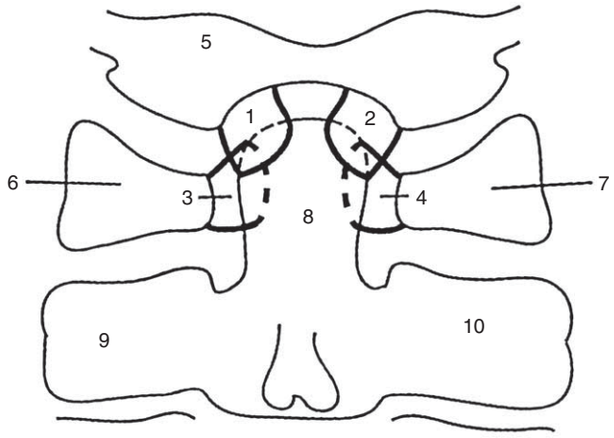


FIGURE 9–13

Posterior view of the atlanto-occipital and atlantoaxial joints, showing the attachments of the alar ligaments. The numbers following correspond to the numbers on the figure: 1, left occipital alar ligament; 2, right occipital alar ligament; 3, left atlanto-alar ligament; 4, right atlanto-alar ligament; 5, occiput; 6, left C1; 7, right C1; 8, dens; 9, left C2; 10, right C2. (Courtesy of International Academy of Orthopedic Medicine, United States.)

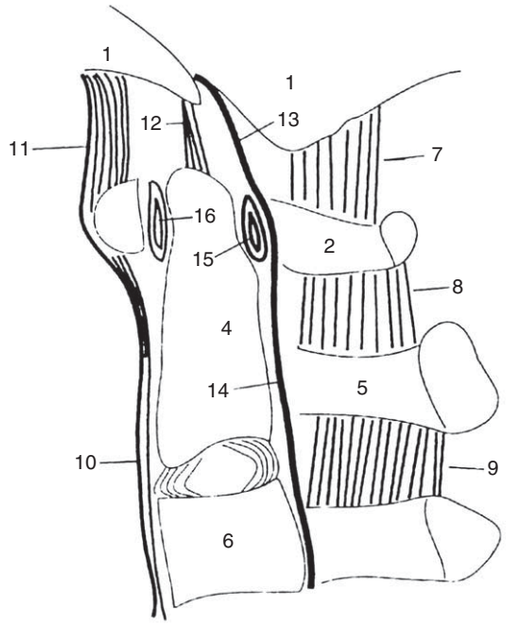


FIGURE 9–14

Sagittal view illustrating the relationship between the atlantoaxial and atlanto-occipital joints. The numbers following correspond to the numbers on the figure: 1, occiput; 2, posterior arch of the atlas; 3, anterior arch of the atlas; 4, dens; 5, posterior arch of C2; 6, vertebral body of C3; 7, posterior atlanto-occipital membrane; 8, ligamentum flavum C1-C2; 9, ligamentum flavum C2-C3; 10, anterior longitudinal ligament; 11, anterior atlanto-occipital membrane; 12, apical ligament of the atlas; 13, tectorial membrane; 14, posterior longitudinal ligament; 15, transverse ligament of the atlas; 16, synovial space between the dens and the anterior arch of the atlas. (Courtesy of International Academy of Orthopedic Medicine, United States.)

These convexities are accentuated by increased thickness in articular cartilage (1.4–3.2 mm). That increased thickness is accommodated by large, intra-articular menisci emerging from flaccid, roomy joint capsules. The menisci are subject to degradation, producing interposition with rotation between C1 and C2 and resulting in sharp, local, catching pain. It is important to note that these four systems allow for flexion, extension, and rotation, but afford very little lateral bending. Because of the strong stabilizing influence of the TLA on a normally configured dens, there is practically no axial separation between C1 and C2. The TLA runs between lateral masses of C2 behind the dens, and this prevents any separation between C1 and C2.

The ligament also prevents posterior movement of the dens into the brain stem and spinal cord during forward flexion of the head, which would cause the patient to have a "drop attack." Damage to the TLA, such as may be seen after a whiplash injury, puts both the brain stem and cord at risk for compression by the dens during normal flexion.

The atlantoaxial joint has the widest range of motion of all articulations in the neck. This motion at C1-C2 is limited to anterior and posterior rocking (20 degrees)[31] and rotation, without allowing any side bending. Rotation around an axis coursing through the dens is limited to 40–45 degrees to each side.[32–36] The odontoid process permits stable rotation and allows 5–10 degrees of flexion and 10 degrees of extension.[32,37] With rightward rotation, the superior articular surface of C1 translates posterior to the right articular surface of C2. The left articular surface of C1 translates anterior to the articular surface of left C2. Additionally, C1 demonstrates slight caudal translation on C2, owing to the convex–convex relationships of the C1-C2 zygapophyseal joints.

Vascular Anatomy

The vertebral artery at this level lies lateral to the atlantoaxial articulation as it travels through the C1 and C2 foraminal membrane (Figure 9-15). Its course may be variable, especially in elderly patients with severe degenerative changes. The artery is protected from the sharp osseous surfaces of the lateral joint by pericapsular soft tissue (Figure 9-16).

Loss of ligamentous stability may result in subluxation or excessive lateral and rotational instability of the C1-C2 joint (Figure 9-17). The most common cause is rheumatoid arthritis. The rheumatoid process results in destruction of synovial lined articulations, articular cartilage, and subchondral bone. The transverse and alar ligaments eventually weaken and instability progresses.

The patient may complain of neck pain with symptoms of vertebrobasilar artery insufficiency with or without head turning. These symptoms may include headache, lightheadedness, dizziness, vertigo, facial numbness, nausea, vomiting, blurred vision, diplopia, dysphagia, gait abnormalities, and tongue symptoms.[38]

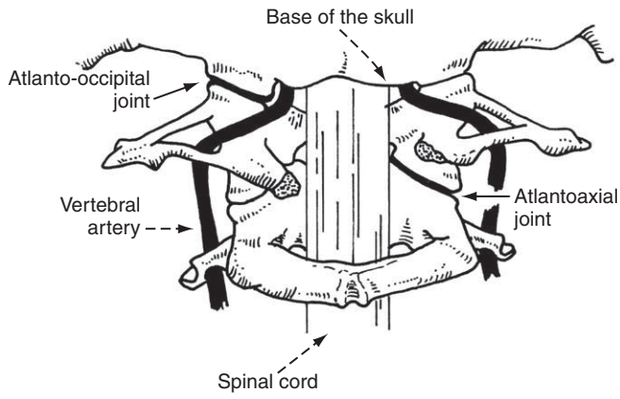


FIGURE 9–15

The anatomy of the atlantoaxial joint. Note the position of the vertebral artery, lying lateral to the atlanto-axial joint.

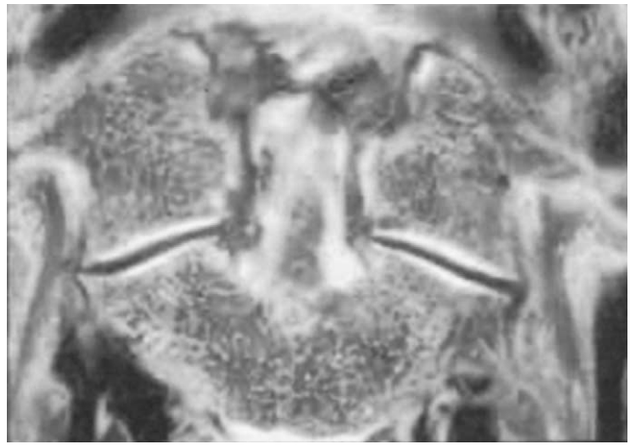


FIGURE 9–16

Specimen demonstrates coronal section through center of C1-C2 body. Note proximity of vertebral artery to lateral aspect of C1-C2 (atlantoaxial) joint. (Courtesy of Wolfgang Rauschning, MD.)

Patients suffering from any of the above symptoms must be evaluated from a neurologic standpoint prior to considering intervention. Spinal cord injury from subaxial spinal cord stenosis has also been reported from rheumatoid destruction of the C1-C2 joint.[39]

INDICATIONS

Because the goal of a joint block is to test the hypothesis that pain stems from the injected joint, the basic indication for a lateral atlantoaxial joint block is suspicion of a patient's pain arising from that joint. That suspicion should be aroused by a work-up that indicates a diagnosis of headache of unknown origin. Therefore, before a lateral atlantoaxial joint block is considered, the clinician should have excluded other possible ("red flag") causes of headache, including tumors, infection, vascular disease, and metabolic disease.

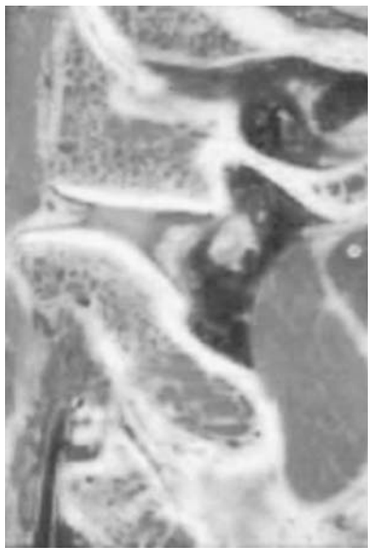


FIGURE 9–17

Specimen demonstrates sagittal section through center of O/C1 (atlanto-occipital joint) and lateral C1-C2 joints. O/C1: Condyle is convex, C1 lateral mass is concave. Vertebral artery immediately behind the joint. C1-C2: C1 and C2 articular surfaces are both convex (biconvex). C2 nerve and ganglion are behind the joint. (Courtesy of Wolfgang Rauschning, MD.)

CONTRAINDICATIONS

Absolute

Bacterial infection, systemic or localized in region of the block
Bleeding diathesis, due to hematological disease or anticoagulants
Previous surgical fusion at that level (C1-C2 arthrodiesis)
Previous cervical surgery
Pregnancy

Relative

Allergy to contrast media
Allergy to local anesthetics
Concurrent treatment with nonsteroidal anti-inflammatory medications, including aspirin, which may compromise coagulation
Inability to lie still
Arnold-Chiari syndrome
Metastasis of cervical vertebral body
Fracture of the dens
Abnormal bleeding disorders

EQUIPMENT

- 25–23-gauge 90-mm needle, 25-gauge needle for skin wheal if needle larger than 25 g
- Minimal volume tubing with T-piece connection
- 3.0-ml syringe

Bending the needle tip slightly will allow directional bevel control, which could minimize needle retraction in target acquisition.

DRUGS

Intra-articular blocks can be performed with any conventional local anesthetic. Because of the small volumes called for in the procedure, it is widely thought that high concentrations should be used to produce the most effective anesthetic effect. No more than 1.0 ml is required to adequately produce the lateral atlantoaxial joint block.

- 2.0% lidocaine or 0.5% bupivacaine
- 1.5% lidocaine for skin infiltration
- 1 ml of iohexol (Omnipaque 240) radiographic contrast solution

Corticosteroid is not used for diagnostic injection. For "therapeutic" injections, use 0.5 ml of the corticosteroid of choice. This is usually mixed in a 1:1 concentration with the local anesthetic.

PREPARATION OF PATIENT

Patient History

The patient history and physical examination should ideally result in a diagnosis of headache of unknown origin and identify any potential contraindications to the use of a lateral atlantoaxial joint block. A significant clinical feature is cervical stiffness, which the patient often describes as a "tired neck." There may be intermittent or constant pain (usually associated with head rotation toward the lesion). Associated symptoms may include visual disturbances, dizziness, nausea, tongue numbness, and ear pain.[40–43]

Practitioners should note the location and extent of the patient's pain, along with movements and activities limited or prevented by the pain (Figure 9-18).

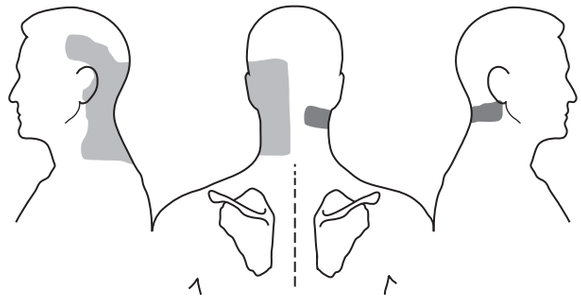


FIGURE 9–18

Atlanto-occipital (AO or C0-1) and atlantoaxial (AA or C1-2) joint referred pain diagram (Dark shade: A-A distribution. Light shade: A-O distribution). (From Dreyfuss P, Michaelsen M, Fletcher D: Atlanto-occipital and lateral atlanto-axial joint pain patterns. Spine 19: 1125–1131, 1994, with permission.)

Physical Examination

During the examination, active range of motion of the cervical spine is performed to provoke patient symptoms and evaluate limitation of motion. By testing rotation or lateral bending from a position of protraction and retraction, pain provocation can be emphasized in the upper cervical spine. Retraction will produce the greatest flexion of the C1-C2 segment; protraction will produce the greatest extension.[31] Therefore, retraction and protraction will elicit a more focused response in the area of interest.

Several clinical features have been found to be somewhat effective in indicating a patient's positive response to blocks, with a positive predictive value of 60%.[29] However, a lack of these features is not a clear sign that a patient will fail to respond to blocks.

Significant clinical features follow:

- Pain in the occipital or suboccipital region
- Tenderness (maximal or focal) in the suboccipital region
- Tenderness (maximal or focal) over the tip of the left or right transverse process of C1
- Restricted rotation of C1 on C2 on manual examination
- Aggravation of accustomed headache by passive rotation of the C1 vertebra to the left or right

Aprill et al.[29] report that patients who demonstrate four or more of these features will have 6:4 odds of a positive response to lateral atlantoaxial joint blocks.

Radiological Studies

Plain film radiographs (x-ray) may reveal disarticulation, instability, and arthropathy, among other features. Open-mouth "odontoid view" as well as lateral neutral and flexion extension views should be studied (Figure 9-19). Evaluation of joint architecture is critical, as advanced arthropathies may preclude or hamper needle entrance safely into the joint.

Early onset pain secondary to joint synovitis (assuming capsular integrity) will usually respond to joint infiltration. Elderly patients with chronic pain may reveal osteochondrotic destruction, which may appear as a partially fused joint. Typically, these patients present a more complex pain referral pattern and may respond to therapeutic injections with a corticosteroid.

Patients being possibly considered for surgical arthrodiesis should undergo diagnostic injections with the understanding that noncontainment of the injection is likely. Extravasation of injectate out of the joint must be interpreted accurately, as symptomatic changes by anesthetizing adjacent structures (i.e., atlanto-occipital joint, dura, venous, etc.) may easily confound results.

Magnetic resonance imaging, with and without contrast enhancement, will reveal craniocervical structures with exceptional detail. Pericranial and paracervical soft tissues are also revealed, which may reveal a potential cause of the patient's pain (Figure 9-20).

Obtaining craniovertebral images using computerized axial tomography scanning (CT) is ideal for demonstrating bony abnormalities. Typically, CT with 2–3 mm axial slices, sagittal and coronal views (Figures 9-21 and 9-22), are required for clear reading of the images.

Informed Consent

Lateral atlantoaxial joint blocks, like all invasive procedures, carry with them the nominal risk of infection, bleeding, and allergic reaction. Therefore, informed consent must be

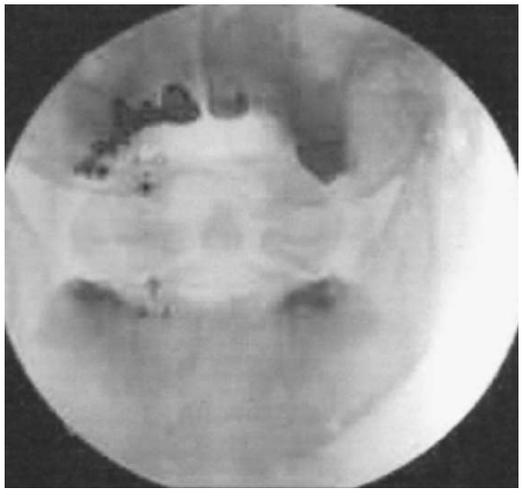


FIGURE 9–19

Odontoid view. Open-mouth frontal radiograph for visualizing the craniocervical and upper cervical segments. C1 lateral masses are centered to the dens/C0-C1 joints above and C1-C2 joints below. (Courtesy of Milton Landers, MD, and International Spine Intervention Society.)

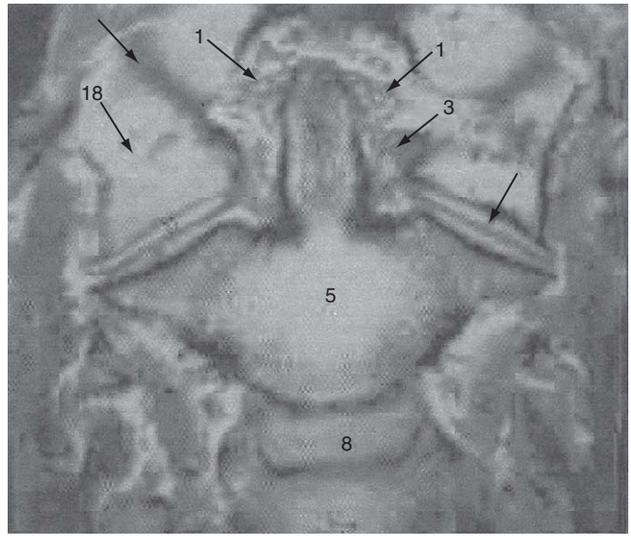


FIGURE 9–20

Coronal T1 spin echo through dens in cadaver. Structures are easily identifiable. 1, alar ligaments; 2, dens (odontoid process); 3, transverse ligament, lateral aspect; 4, atlantoaxial joint; 5, C2 vertebral body; 6, C2-C3 intervertebral disc; 7, C3 vertebral body; 8, superior articular cartilage C2; 9, inferior articular cartilage C1; 10, lateral mass C1; 11, atlanto-occipital joint. (Courtesy of International Spine Intervention Society.)

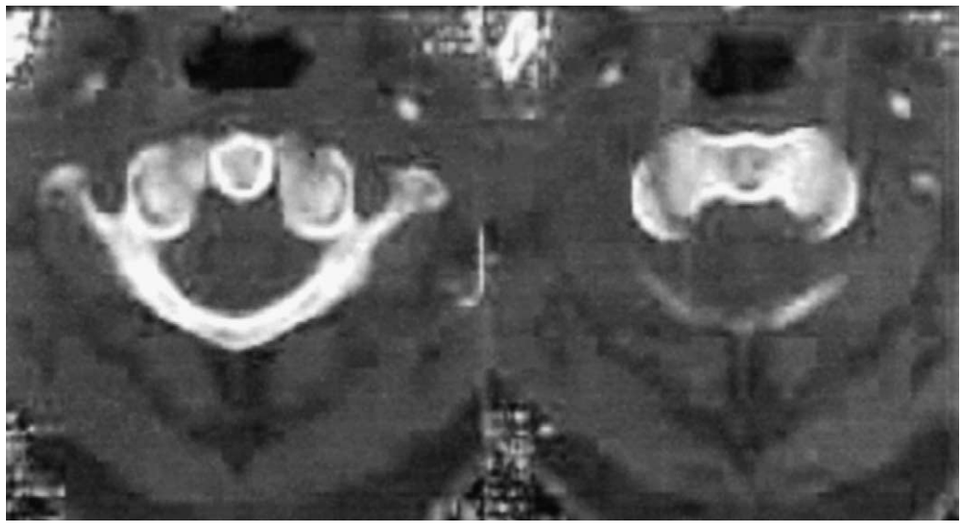


FIGURE 9–21
Computed tomography C1-C2 joints. (Courtesy of International Spine Intervention Society.)

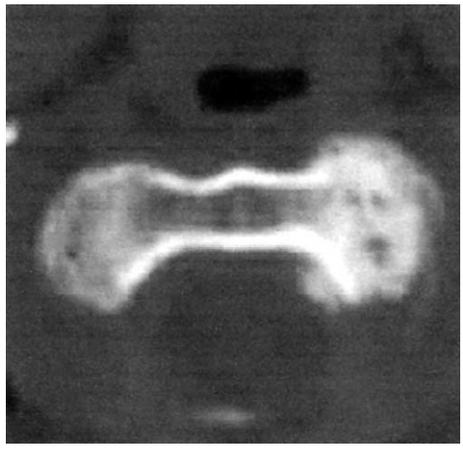


FIGURE 9–22
Axial CT at C1-C2. Hypertrophic arthropathy on left. (Courtesy of International Spine Intervention Society.)

obtained from the patient. In particular, the patient must understand the theoretical risk of high spinal anesthesia, as well as the precautions and emergency measures taken to prevent or remedy any adverse effects.

The practitioner must also be sure that the patient understands that this procedure is performed for diagnostic, and not therapeutic, reasons. The procedure may not result in any relief of the patient's pain, a possibility for which the patient must be prepared. The patient must not expect any particular amount or length of relief, but simply be prepared to monitor and record the resulting changes, if any, in their pain and function.

Laboratory Studies

No laboratory studies are required in a typical, otherwise healthy patient.

Preoperative Medication

This procedure requires no preoperative medication.

Monitoring

Sedation is not required for this procedure. If monitored anesthesia care is required, follow the standard recommendations by the American Society of Anesthesiologists (ASA).

PROCEDURE

Positioning the Patient

To begin the procedure, place the patient in a prone position with blankets under the chest to produce a slight flexion of the head. Support the patient's head and face with a cushion, but be sure to leave the mouth area open so the patient can breathe and speak. The patient may also need to open the mouth if a dental filling prevents a clear view of the target joint.

Position the fluoroscopy C-arm over the C1-C2 level in an anteroposterior direction. Then rotate the C-arm in the sagittal plane so that the beam passes from the anterosuperior aspect to the posteroinferior aspect (Figure 9-23).[32] Continue the rotation until the atlantoaxial joint is visualized.

Identifying the Target

Identify the target joint, and also identify the posterior arch of the atlas by tracing its silhouette between the two transverse processes. This is important because the posterior arch can be positioned in such a way as to prevent access to the atlantoaxial joint. To correct this problem, tilt the C-arm slightly caudally or ask the patient to flex or extend their head slightly. The patient's teeth may also be positioned so as not to block a clear view of the

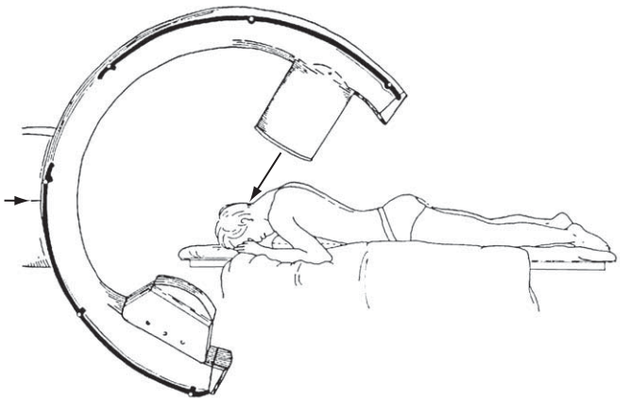


FIGURE 9–23
Illustration of the positions of the patient and the C-arm to allow for optimal viewing of the atlantoaxial joint.

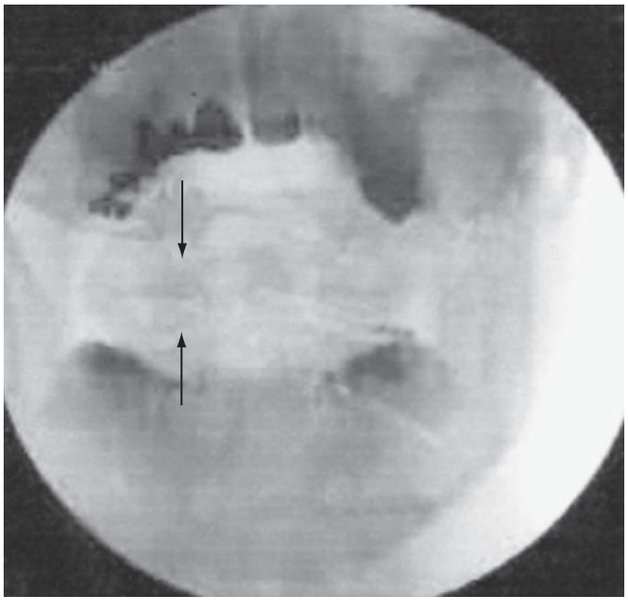


FIGURE 9–24
Open-mouth "odontoid" scout film. Note proximity of vital neurovascular structures in relation to target joint. Any miscalculation could result in severe complication resulting from inadvertent trauma/injection proximal structures. (Courtesy of Milton Landers, MD, and International Spine Intervention Society.)

joint. Correct this by having the patient turn the face slightly.

Ensure proper target joint identification by obtaining an AP view. The initial target point in this view is the lateral third of the lower end of the posterior surface of the lateral mass of the atlas. A second target point option is the lateral quarter of the upper end of the posterior surface of the superior articular process of the axis.[23]

Prior to needle placement, the patient must remain absolutely still. Discussion of reproduction of concordant pain with the patient prior to the procedure is essential in order for the familiar pain reproduction to not result in patient movement. Due to the proximity of the C2 dorsal root ganglion (DRG) to the joint (Figure 9-24), there is a possibility of irritating this structure during needle placement. If severe occipital paresthesia occurs the needle is retracted slightly. The C-arm should be rotated to a lateral position to check needle depth. If the needle is posterior to the joint, a minute quantity of contrast media is gently infiltrated under real-time imaging, which may outline the DRG. The C-arm is then rotated to AP view, which will reveal a C2 DRG neurogram. The needle is then retracted, and using the pre-bent needle with "bevel control," a safe course to the joint can be performed.

Placing the Needle

The initial target point will dictate the appropriate puncture point in the skin. Note that this point will be just above or just below the hairline. Along with proper sterilization of the upper neck for an aseptic procedure, the bases of the hair shafts along the hairline can be treated with an antiseptic solution. This generally makes shaving the patient's hair unnecessary.

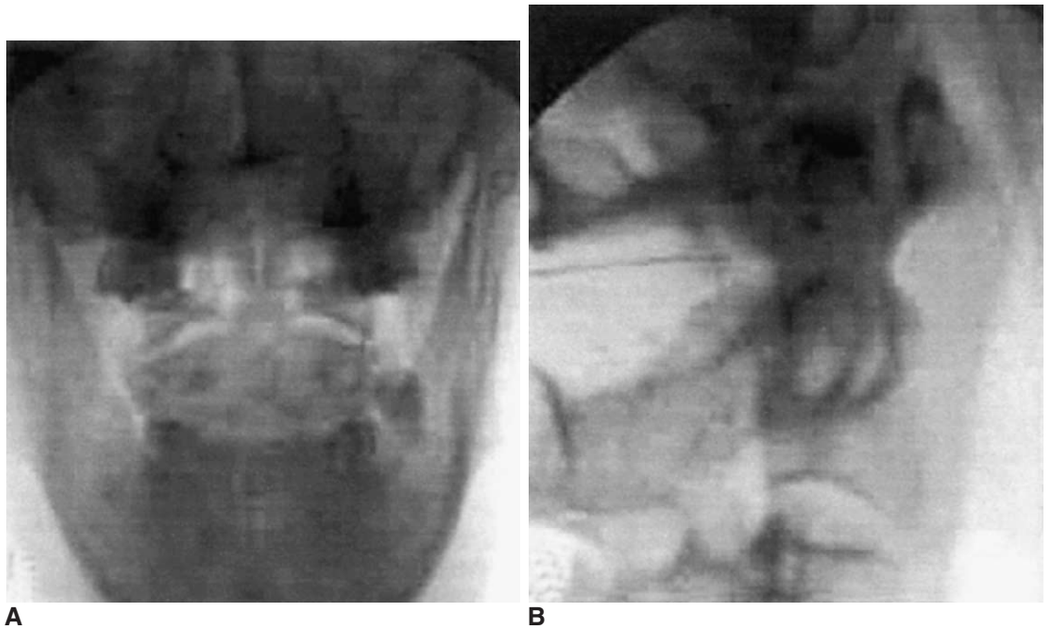
To begin, introduce a spinal needle perpendicularly through the skin at the puncture point, which is directly overlying the initial target point, advancing it slowly until the initial target point is reached. The tip of the needle must stay over the lateral quarter of the articular process that bears the target point throughout. If the needle moves lateral to the margin of the joint, it could incur the vertebral artery; if it moves to the middle of the joint, it could impinge on the C2 spinal nerve or ganglion; and if it moves beyond the center of the joint, it could touch the dural sac or the C2 nerve root sleeve.

Advance the needle slowly through the suboccipital muscles, verifying throughout that it is following a straight course. Never attempt to change course by continued forward pressure on the needle. If a deviation from the straight course is found, move needle to its last on-course position and adjust the bevel.

Proceed with the insertion of the needle until it reaches the initial target point. This can be verified by feel, with the unmistakable bony resistance. It can also be recognized by its appearance on an AP view (Figure 9-25A) and a lateral view (Figure 9-25B).

Note the depth of insertion when the needle strikes bone. Do not allow the insertions that follow to proceed more than a few millimeters past this initial depth.

Once the initial target point is reached and verified, the needle can be directed toward the joint cavity. For an initial target point on the inferior articular process of C1, adjust the needle inferomedially, following a line perpendicular to the inferior margin of the lateral mass of the atlas until it is positioned over the joint space. Then insert the needle a few millimeters farther into the joint cavity, but not toward the lateral end of the joint, which is fairly



A B

FIGURE 9–25

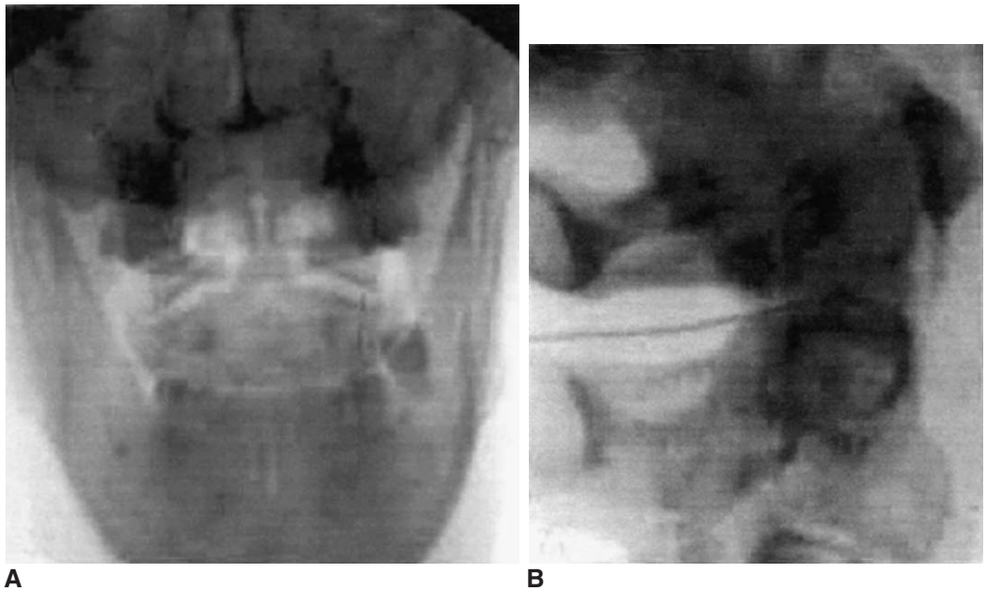
Radiographic images showing proper preliminary positioning of a needle on the back of the lateral third of the lower end of the lateral mass of the atlas. (A) Anteroposterior view. (B) Lateral view. (Courtesy of Charles Aprill, MD, and International Spine Intervention Society.)

shallow in depth at this margin. For an initial target point on the superior articular process of C2, adjust the needle upward and medially to the center of the lateral third of the joint cavity.

Confirm the passage of the needle on the AP view (Figure 9-26A), and then proceed with further insertion using the lateral view until the tip of the needle is near the junction of the first and second posterior quarters of the joint's AP diameter (Figure 9-26B).

Performing the Injection

Begin by aspirating the needle, which may have impacted a posterior meniscoid of the joint, possibly drawing blood.[44] If that has occurred, use lateral screening to



A B

FIGURE 9–26

Radiographic images showing a needle properly placed in a lateral atlantoaxial joint. (A) Anteroposterior view. (B) Lateral view. (Courtesy of Charles Aprill, MD, and International Spine Intervention Society.)

insert the needle farther into the joint and away from the meniscoid.

Once aspiration has proved to be negative, inject 0.3 ml of contrast medium to make sure that the injection is intra-articular. On the AP view, contrast medium should spread transversely across the joint cavity and fill the medial and lateral extremities of the capsule (Figure 9-27A). On the lateral view, contrast medium should spread across the joint cavity and fill the anterior and posterior extremities (Figure 9-27B). Note that an increase in the patient's occipital headache pain during the injection is not uncommon.[27,37]

Resistance to the injection could indicate that the needle is lodged in a meniscoid or in the articular cartilage. To correct the former situation, advance the needle using lateral screening toward the center of the joint until it escapes the meniscoid. To correct the latter situation, the withdrawal of the needle by 2 mm should set it free.

If, using the AP view, the contrast medium is seen to spill medially toward the median atlantoaxial joint, an arthrogram of the opposite lateral atlantoaxial joint can be obtained. Note that this spread of contrast medium is not a sign of pathology: the synovial joints in the atlantoaxial region can communicate freely back and forth. Nevertheless, the escape of contrast medium should be grounds for termination of the injection. Note the volume of contrast medium that has been injected, and do not administer a volume higher than this when injecting the local anesthetic.

After the intra-articular placement is verified by arthrography, inject the local anesthetic. Barring the escape of contrast medium and the precautionary measures cited earlier, the joint will commonly accept 1.0 ml of injectate. Monitor the injection of local anesthetic closely to make sure the joint is not filled beyond capacity with contrast medium. As the local anesthetic is injected, the intensity of the contrast medium in the joints should be observed to dilute on AP views. It should not, however, escape outside the joint. If it does, halt the injection.

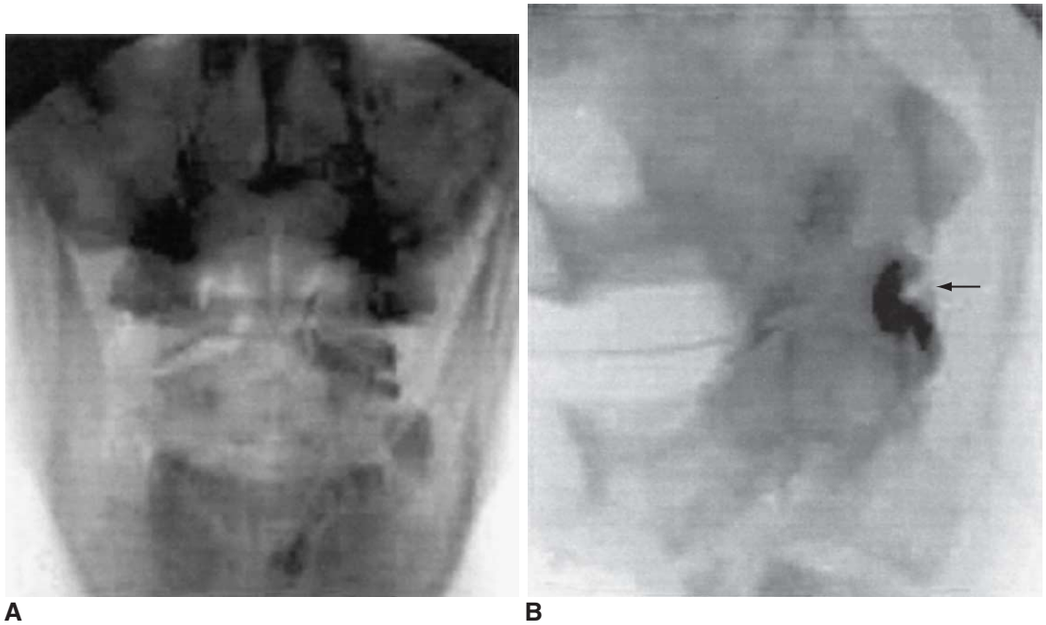
Terminate the injection if and when firm resistance is encountered and at least 0.5 ml of local anesthetic has reached the joint. Before removing the needle, it is wise to obtain AP and lateral images to document proper needle placement prior to and after injection. These images should be stored for medical and legal purposes.

Evaluating and Interpreting Results

Once the diagnostic block has been properly executed, the practitioner should be prepared to objectively evaluate and interpret the results. To overcome the problem of observer bias, the practitioner can enlist an independent observer, such as a nurse, to help evaluate the patient's response to the block.

If it is clear that there has been no relief in the patient's pain, the patient can be discharged. Patients who report relief should continue to be monitored by the observer for a specified period.

Note that a positive response to a block should result in complete relief of the patient's pain as long as the anesthetic administered continues to function. A partial reduction of pain cannot be categorized as a positive response (unless the



A B

FIGURE 9–27

Radiographic images showing an arthrogram of a lateral atlantoaxial joint. (A) Anteroposterior view. (B) Lateral view, with the contrast medium outlining a meniscoid (arrow). (Courtesy of Charles Aprill, MD, and International Spine Intervention Society.)

reported pain is actually a response to the needle-track, rather than the patient's accustomed pain). If the source of the patient's pain is not in the atlantoaxial joint, then no relief should be expected from this procedure.

POSTPROCEDURE MONITORING

After the needle is removed, the patient's skin should be cleaned and any bleeding halted by sustained pressure. Also, be alert to patient complaints of side effects of the procedure. The patient should be given detailed instructions prior to discharge, including the following:

- Orders to contact the practitioner if the patient notices any unusual symptoms or pain; an instruction sheet with contact information should be provided.
- Directions as to how the patient should record the outcome of the procedure (such as a pain diary), including the extent and duration of relief, if any. Patients should be instructed that, if relief occurs, they should attempt any movements and activities normally hindered by their accustomed pain and record the results.

COMPLICATIONS

There have been reports of patients experiencing brief periods of ataxia in the immediate postblock period.[36,37] Another possible complication, common to all blocks, is infection and allergic response to the agents injected.

CLINICAL PEARLS

This is a technically demanding procedure that requires the operator to follow a strict line from the insertion point to the target area in the joint. Operators must be able to use bevel control to restrict course deviations to a few millimeters.

EFFICACY

Because no patient-controlled, double-blind studies of lateral atlantoaxial joint blocks have been reported, only anecdotal reports of benefits are available.

CERVICAL FACET BLOCKS

HISTORY

As early as the 1940s, Hadden[45] identified the cervical facet (zygapophyseal) joints as a possible source of headache. Over the next several decades, Raney and Raney,[46] Taren and Kahn,[47] Brain and Wilkinson,[48] McNab,[49] and Mehta[50] all attempted to demonstrate that cervical facet joint pain is a source of headache. Pawl[51] explained the role of cervical facet joints in headache in 1971. Bogduk and Marsland[52,53] had completed two studies by 1986 in which a medial branch block of the C3 dorsal ramus brought complete relief of headache in 70–80% of patients. Two years later, they produced the first report of medial branch blocks at all levels of the cervical spine.[54]

By 1990, Aprill et al.[55] had demonstrated that an analysis of pain patterns could be used to predict the segmental location of painful joints. Aprill and Bogduk[56] next determined that the prevalence of cervical facet joint pain was at least 25% in patients with neck pain.

Barnsley and Bogduk[57] proved the validity of cervical medial branch blocks by demonstrating that material injected onto these nerves consistently bathed them, and did not spread beyond, countering the criticism that medial branch blocks were effective because they anesthetized nearby muscles, spinal nerves, or roots.

In 1993, in two separate published papers, Barnsley et al.[58,59] showed the need for multiple diagnostic blocks. They demonstrated that single diagnostic blocks were invalid because a large number of patients who responded to an initial block did not respond to a subsequent block.

It has been suggested that the most common causes of neck pain and nerve root irritation are whiplash injuries of the cervical spine, causing muscle ligament us sprains of the cervical facet joints with periosteal tearing. A recent study by Gibson et al.[60] found that these joints are the source of pain in 50–60% of patients with neck pain after whiplash injury. In addition, there is growing evidence that the upper cervical spine (C1-C3), including the facets, contributes significantly to the neck and head pain of cervicogenic headaches.[57]

Several studies have shown the prevalence of cervical facet–mediated pain as a significant source of pain. Using definitive epidemiologic studies, Lord and colleagues[61,62] found that the third occipital nerve was responsible for 27% of total neck pain and 53% of neck pain when headaches were the main component.

The third occipital nerve is significant because it provides the main innervation for the C2-C3 facet joint. Disruption of this joint leading to pain along the distribution of this nerve is called third occipital headache, and is the leading cause of cervicogenic headache.[62]

In a 1995 pilot study, Lord et al.[63] began assessing percutaneous radiofrequency (RF) neurotomy of the medial branches of the facet joint to more effectively treat facet pain. This led to a randomized, double-blind, placebo-controlled study, suggesting that 70% of patients could obtain complete relief of pain with this procedure.[64]

These results indicated that patients who obtained a high degree of pain relief from controlled medial branch diagnostic blocks, regardless of the duration of relief, could obtain longer-term pain relief via coagulation of those same nerves.

A recent study by McDonald et al.[65] indicated that longer relief can be achieved following neurotomy in a majority of patients, and that subsequent treatments can reinstate that relief. In that study, 64% of patients reported complete relief with an interquartile range of 223–730 days and a median duration of 421 days. The study also reported that outcomes were independent of operator skill, the use of placebo-controlled versus comparative blocks, and whether or not patients were litigants.

A subsequent study by Sapir and Gorup[66] confirmed that there was no significant difference in outcomes between litigants and nonlitigants following neurotomy. Among the 46 patients who had medial branch RF neurotomy following whiplash, at 1 year post-treatment there was a 50% (litigants) to 65% (nonlitigants) reduction in pain.

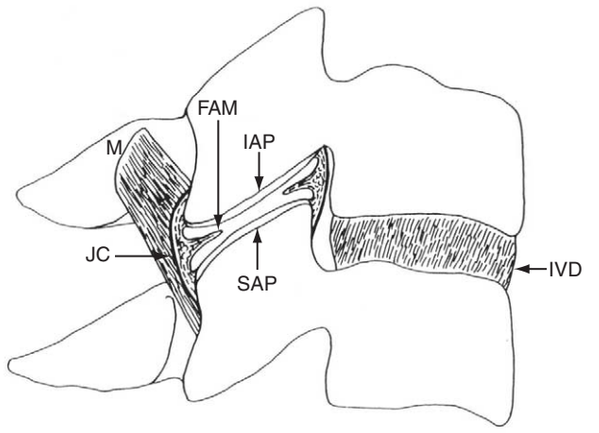


FIGURE 9–28

Illustration of a typical cervical motion segment showing structures of the facet joint. (Courtesy of Nikolai Bogduk, MD, and International Spine Intervention Society.)

ANATOMY

The facet joints are anatomically designed to limit excessive mobility and distribute axial loading over a broad area. They help resist the shearing motion produced by forward bending and the compression produced by rotation.[67] The articular facets in the cervical spine extend laterally from the junction of the lamina and pedicles and are oriented in the coronal plane to permit flexion, extension, and lateral bending.

The cervical facet joints, also known as the zygapophyseal joints (or z-jt's), can be a significant source of head, neck, and shoulder pain. The facet joints are formed by the articulation of the articular processes of adjacent vertebrae.

These sinuarthrodial joints are subject to trauma and degenerative changes such as occurs in cervical spondylosis. In the early stages of inflammation, acute synovitis of a single joint may present as a "crick" in the neck leading to severe headache (cervicogenic) recalcitrant to conservative therapies. It may also lead to chronic osteochondrosis with destruction of articular cartilage and subchondral bone, which over time may develop dynamic compensatory changes in the form of severe arthropathies, contributing to chronic nonradicular neck pain and headaches.

The superior articular facet of one vertebra and the inferior articular facet of the adjacent vertebra above (Figure 9-28) combine to form the apophyseal articulations. Hyaline cartilage covers the articular surfaces of the facets. Synovium lines the joints and tabs of synovial tissue project into the joint from the joint margins where the surfaces of the facets are not in contact.[68] Superior and inferior joint recesses, which may contain small synovial villi, are formed by the fibrous joint capsule.[69] The inferior and posterior portions of the recesses are larger, allowing a wide range of motion. Medially and anteriorly, the capsule blends with the ligament flavum and is adjacent to the neural foramen and the nerve root.

The joint capsule is richly innervated.[68,70–73] The dorsal rami each send medial branches to the facet joint at their own levels and to the levels below (Figure 9-29).

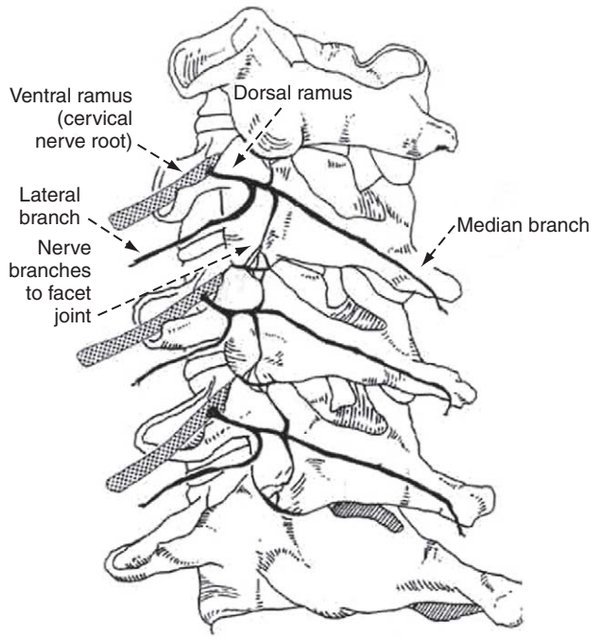


FIGURE 9–29

Oblique view of cervical spine (C3-C7). IAP, inferior articular process; SAP, superior articular process; JC, joint capsule; FAM, fibroadipose meniscoid; M, multifidus muscle; IVD, intervertebral disc.

The C2-C3 through C5-C6 facet joints are angled 35 degrees from the coronal plane. The C6-C7 joint is in transition between the orientation of the C5-C6 facet joint and that of C7-T1, which is tipped 22 degrees from the coronal plane.[72,74] All of the cervical facet joints from C2-C3 caudad to C7-T1 are angled 110 degrees from the midline posterior sagittal plane. They are much like the thoracic facet joints in orientation, yet not as close to

the vertical plane. The cervical facet joints play a physically larger role in the spinal articular tripod structure and are actually best described as the superior and inferior ends of articular pillars. Also unique to the cervical spine is the vertebral artery, which passes though the foramen of the transverse process of the C6-C7 vertebrae.[54]

The C2-C3 facet joint is unique in that it receives innervation not only from the C2 and C3 medial branches, but also the third occipital nerve.

The C3-C4 to C7-T1 facet joints are supplied by the medial branches of the cervical posterior rami at the same level and from the segmental level above.[54,75] Therefore, the C3-C4 facet is innervated by the C3 and C4 medial branch nerves. These nerves arise from the posterior primary rami in the cervical intertransverse spaces and then curve dorsally and medially to wrap around the waist of their respective articular pillars. As they begin to wrap around the articular pillars, the nerves are 2.2 mm (C3) to 1.2 mm (C7) in vertical extent. They are 7.3 mm (C3) to 5.5 mm (C7) caudad to the tip of the superior articular pillar at this location. The medial branches are bound to the periosteum by an investing fascia and are held against the articular pillars by tendons of the semispinalis capitis.[57,67,75]

The medial branches are seen on a lateral view of the cervical spine to pass through the waist of the articular pillar. Rostral and caudal branches from each nerve then pass into the joints immediately above and below. The C7 medial branch crosses the root of the C7 transverse process, and therefore lies higher on the lateral projection of the C7 articular pillar.[63] The medial branch at C7 has a different location from the other medial branches in that it courses in variable locations up the triangular silhouette of the superior articular process of C7. Care must be taken not to inadvertently anesthetize the C8 segmental nerve nor produce a C6-C7 intra-articular injection.

The C8 medial branch runs a course similar to the upper thoracic nerves. It arises from the dorsal ramus within 5 mm of the lateral margin of the intervertebral foramen of C7-T1. After passing laterally through the intertransverse space to the tip of the transverse process, it curves dorsally through that space, aiming for the lateral end of the superior border of the transverse process. Once entering the posterior compartment, it runs caudally across the surface of the transverse process.

INDICATIONS

The clinical criteria for facet syndrome are nonspecific and unreliable, and therefore an accurate diagnostic procedure is essential.

The indication of cervical medial branch blocks is to determine if the patient's pain can be relieved by anesthetizing the medial branches or the cervical dorsal rami. Complete relief of the region targeted constitutes prima facie evidence that these medial branches are mediating the patient's pain. However, the pain may be only partially relieved, which might implicate other structures as a source of the patient's pain. Additionally, steps must be taken to rule out false-positive responses.

Both intra-articular facet injections and medial branch blocks allow the practitioner to assess the facet joint as the source of pain. They test the hypothesis that the patient's pain is mediated by the nerves targeted (the medial branch of the dorsal primary rami of the respective cervical facet joint). If the patient's exact pain is produced by capsular distension of the suspected joint with documentation of its capsular integrity by arthrography, and relieved by the injection of local anesthetic into that joint, it can be inferred that that cervical facet joint is the source of the pain. Performing control medial branch blocks using stringent criteria must then yield similar results to confirm that suspicion.

The physical exam and analysis of the patient's symptoms can help identify the facets that will be subjected to diagnostic blocks. Clinical findings such as tenderness to palpation over the facet joint and the coetaneous distribution of pain help identify the facet joint to be injected. For cervical facet joints, there are distinctive segmental pain patterns, with some overlap between these patterns and those for cervical discogenic pain. If the patient has marked tenderness to palpation of a particular facet joint or if pain increases with motion or loading of the joint, trial blockade of the joint should be considered.

There are important differences between the intra-articular facet injection and the medial branch block. The cervical intra-articular facet injection is a selective block of the nerve endings in the joint capsule indicated in chronic or subchronic cases of recalcitrant facet pain. Diagnostic blocks require a small volume of anesthetic injected into the synovial capsule. Therapeutic use of the procedure is performed with the injection of corticosteroids. However, in a 1994 study, Lord et al.[76] called the long-term effectiveness of therapeutic intra-articular cervical facet blocks into serious question. Although benefits have not been definitively proven by any published studies at this time, the diagnostic procedure remains an excellent tool for localizing pain and describing the morphology of lesions.

Cervical medial branch blocks involve the direct injection of a small amount of anesthetic mixture next to the medial branch of the dorsal ramus. They have proven to be useful as both a diagnostic procedure and a key indicator for the subsequent effectiveness of RF neurotomy as a therapeutic procedure.

The medial branch block is easier to perform, theoretically safer, and more easily subject to controls. In general, medial branch blocks also have better therapeutic utility and predictive value than intra-articular facet injections because they can be followed by RF neurotomy for longer-term relief.

In patients with chronic and severe facet disease, a medial branch block is the more appropriate diagnostic

tool to test if the pain is mediated by one or more medial branches of the cervical dorsal rami.

Evidence of cervical facet arthropathy can be demonstrated with radiographs, magnetic resonance imaging, and computed tomography. However, these imaging studies are unreliable indicators of cervical facet pain. The degree of facet arthropathy imaged does not necessarily correlate with the degree of pain experienced by the patient; a patient with severe facet arthropathy on imaging may be asymptomatic, while a patient with mild cervical facet arthropathy may have severe facetogenic pain. Paracervical pain localized in a commonly recognized facet joint referral "map" region has a high likelihood of being of facetogenic origin (Figure 9-30).

While the physical exam can be very suggestive of segmental facet pain, there is overlap and fluoroscopically guided intra-articular injections are required. Aprill and colleagues[55] demonstrated that patients with C1-C2 joint pain did not differ significantly on clinical exam from patients with pain at other levels, most commonly the C2-C3 joint.

Acute sinovitis may present as posterior focal discrete pain, easily identifiable by palpation and axial loading, and referral pattern. Intra-articular facet injections can play an important role in precisely localizing the source of pain. Diagnostic arthrograms reveal capsular architecture and confirm the spread of anesthetizing medication. Medial capsular tears are more prevalent than lateral tears, and this is of clinical significance in that recognition of such tears might demonstrate communication with the adjacent epidural space. The specificity and sensitivity of diagnosing a single joint as the source of pain can be confounding. Age-related changes of the facet capsule accompanying cervical spondylosis often result in osteochondrosis and may reveal total destruction of the facet capsule. The diagnostic utility of intra-articular injections in these patients is significantly reduced and medial branch blocks would provide a more accurate diagnosis.

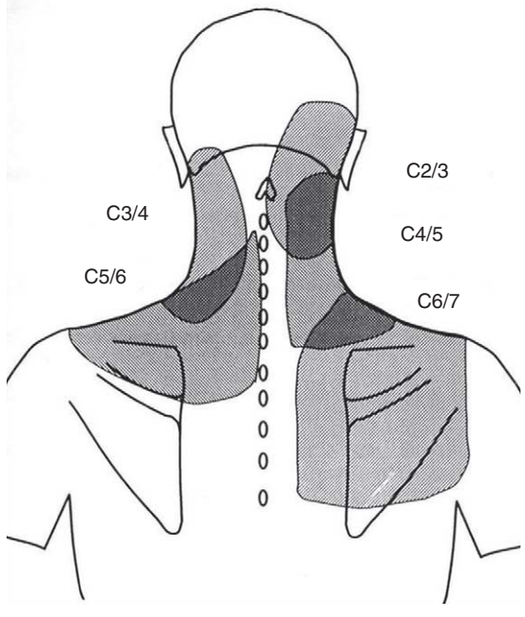


FIGURE 9–30

Illustration of a facet joint referral map, with regions of pain referral from the cervical facet joints at the segments shown. (Courtesy of International Spine Intervention Society.)

Intra-articular facet injections and medial branch blocks should be reserved for patients with no neurological deficit, when no other cause for chronic pain or headaches can be identified. Serious possible causes of neck pain and headaches, including infection, tumor, vascular, and metabolic etiologies, must be ruled out. The pain should be unresponsive to conservative therapies, such as rest and oral medications, including nonsteroidal anti-inflammatory medications.

Obviously, the most important information gained from a diagnostic block is whether the patient's typical pain is relieved by the injection. The length of pain relief is not nearly as important as the amount of relief experienced. A result of 90% pain relief or better in the region targeted is considered "positive" for that joint.

With a medial branch block, facetogenic pain is usually only relieved for the duration of the local anesthetic, making the block a primarily diagnostic tool. Barnsley et al.[59] defined the following patterns of response to cervical medial branch blocks using both bupivacaine and lignocaine as local anesthetics in patients with neck pain.

- A concordant response was considered to be one in which the patients experienced long-lasting relief when bupivacaine was administered, and short-lasting relief when lignocaine was administered, with the relief in both cases not lasting longer than the expected duration of action of the anesthetic used.
- A prolonged concordant response was similar, except that the duration of relief from the bupivacaine, the lignocaine, or both, exceeded the expected duration of action.
- A discordant response was characterized as one in which lignocaine brought longer relief than bupivacaine, but relief in either case conformed to the expected duration of action.
- A discordant prolonged response was similar, except that the relief in either case exceeded the expected duration of action.
- A discrepant response was one in which the patients did not experience relief when the same nerves were blocked on a second trial.

Given the high false-positive rate of single joint injections, even if the first block is positive, it must be confirmed with subsequent medial branch blocks to increase the reliability of the diagnosis. A minimum of two sequential blocks is often considered necessary to firmly establish the diagnosis of facetogenic pain. However, a painful and "concordant" intra-articular injection with confirmatory

medial branch block (control) may suffice in lieu of dual-control medial branch blocks.

On a cautionary note, lack of capsular integrity verified by contrast leakage (medially more than laterally) would render the injection void of diagnostic validity based on loss of target specificity. Diagnostic intracapsular joint injections require advanced skill in interpreting results as contrast medium may reveal the articular structure without stressing the synovial capsule to the point of pain provocation. In other words, if the majority of the capsule requires filling by contrast medium to provoke concordant pain, the remaining volume in that capsule then filled with local anesthetic might not be sufficient to produce an analgesic block.

Additionally, unintentional or inadvertent blocking of an adjacent facet due to lateral or medial spread of local anesthetic might occur and thus invalidate the test. It is therefore stressed that cervical medial branch blocks must be performed to further validate that the patient's pain is emanating from that joint. By performing either two separate control medial branch blocks or an intra-articular facet injection followed by dual controlled medial branch blocks, false-positive results are minimized and a decision to proceed with RF neurotomy can be made.

RADIOFREQUENCY NEUROTOMY

The essential indication for cervical medial branch RF neurotomy is a minimum of 80% relief of concordant pain following controlled diagnostic blocks of the target medial branch or successful intra-articular joint injection followed by comparative local anesthetic medial branch blocks.

The rationale for RF neurotomy is that the patient's pain can be relieved by coagulation, preventing the conduction of pain impulses down the nerve. Note that this procedure is not a treatment for the root cause of the pain; it relieves pain by anesthetizing its source.

CONTRAINDICATIONS

Contraindications for cervical intra-articular facet injections and medial branch blocks:

- Coagulopathy (INR >1.5 or platelets <50,000). Anticoagulation medication should be suspended for an appropriate period prior to the conduct of blocks.
- Systemic infection or localized infection at the puncture site.
- Severe allergy to any medications used.
- Pregnancy.
- The patient who is unable or unwilling to consent to the procedure, or who is unable to understand and cooperate with the procedure.
- Any factor that would cause inability to assess the patient's response to the procedure.
- Evidence of another source of neck pain or headaches not related to the facets.
- Motor weakness, absent reflexes, or long tract signs.
- Any anatomical derangements, surgical or congenital, that would preclude safe, successful access.

Contraindications for cervical medial branch neurotomy follow:

- Patients who have had inadequate pain relief or relief for less than 3 months following a previous neurotomy.
- When appropriate, pacemaker and ICD equipment must be deactivated prior to RF neurotomy.

EQUIPMENT

Cervical intra-articular facet injections and medial branch blocks
Metal pointer
Sterile gloves
25-gauge, 60–100-mm spinal needle
Two 3-ml Luer Lock syringes
Minimal volume extension tubing

Pre-bending the bevel is useful in obtaining efficient, minimally traumatic, target acquisition. Rotation of the hub facilitates steering without the need to withdraw the needle, creating excessive tissue trauma, pain, and ultimately confusing the results of the block with procedural pain.

Cervical medial branch neurotomy
Radiofrequency lesion generator (Figure 9-31)
Sterile, disposable RF needle (Figure 9-32)
18- or 20-gauge needles with 10-mm curved or straight active electrode tip

Cervical medial branch neurotomy at typical levels has traditionally been a two-stage procedure with oblique and sagittal insertions using a 16-gauge needle to obtain lesions

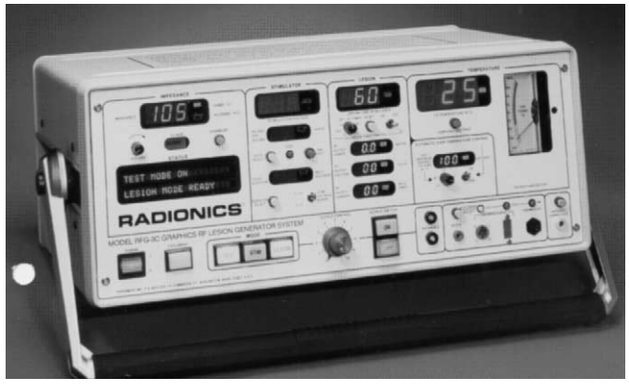


FIGURE 9–31
Radiofrequency lesion generator system. (Courtesy of Radionics Corp.)

FIGURE 9–32
Radiofrequency needle with a 10-mm active tip.

parallel to the nerve. The only validated study with consistent long-term results was demonstrated by Lord et al.,[77] who used a 16-gauge needle with six RF lesions per level. With two to three lesions produced along the anterolateral and lateral aspect of the pillar, the entire length of the nerve is coagulated using the technique while taking into account the variable positions of the nerve. This electrode is capable of producing large circumferential lesions, when performed exactly as per the protocol, thermocoagulates a zone that covers all variable neural pathways of the target branch, thus accounting for the consistent long-term pain relief.[78] At present, there are no studies validating the effectiveness of using smaller RF electrodes.

This chapter will cover the technical aspects of cervical RF neurotomy with the intent of providing the practitioner with a manner in which to integrate the principles of RF thermocoagulation using 18–20-gauge needles, as 16-gauge RAY needles are not commercially available. The described technique should hopefully cover the majority of area under which all neural pathways may exist. It is also based on the consensus of respected investigators in the field of RF ablation.

Technical success is maximized when parallel needle placement is used, larger needles are used, and multiple lesions are performed to account for variable nerve topography. Larger RF needles produce a larger lesion radius. Because lesion shapes are elliptical, with the greatest effectiveness on the sides of the electrode, needles should be placed parallel to the target nerve.[79]

DRUGS

- 1% lidocaine (skin wheal)
- 0.5–0.75% bupivacaine (prelesioning analgesia)

PREPARATION OF PATIENT

Physical Examination

Patients demonstrate marked tenderness to palpation over a particular facet joint or complain that pain increases with motion or loading of the joint.

Distinctive upper, lower, and "pancervical" neck pain syndromes have been described for cervical facet joints.[54] Upper cervical syndrome is characterized by headaches associated with neck pain (cervicogenic headache) and is often a result of whiplash injuries. Lower cervical syndrome is characterized by neck and shoulder pain (cervicobrachialgia), and midcervical syndrome is characterized by neck pain (cervicalgia). Facet pain referral patterns should be common knowledge, and will assist the practitioner in approximating the level(s) to be tested. Caution must be exercised in concluding whether pain is of facetogenic or discogenic origin since their pain referral patterns do overlap. Palpation of the suspected facet(s) combined with axial loading techniques may assist in localizing painful segments.

Deep, aching pain, extending beyond the immediate vicinity of the joint, is to some degree referred pain. Pain from the atlanto-occipital joint (C0-C2) is referred unilateral to the suboccipital area. Pain from the atlantoaxial joint (C1-C2) is unilateral, focused at the occipitocervical junction, and radiating to the postauricular region. Patients may have limited head rotation, trigger points confined to the occipital area, palpable cervical crepitus, and abnormal head position. Pain from the C2-C3 joint, commonly referred to as "third occipital headache," is located in the upper cervical region and extends at least to the occipital and sometimes into the head, toward the eye, vertex, or forehead.[55,80,81]

The C3-C4 facet joint produces pain over the posterolateral cervical region, along the course of the levator scapulae muscle. It extends craniad as far as the suboccipital region and then caudad over the posterolateral aspect of the neck without entering the region of the shoulder girdle. The C4-C5 facet joint pain involves a triangular area, with two sides consisting of the posterior midline and posterolateral border of the neck and its base running parallel to the spine of the scapula muscle. It extends craniad as far as the suboccipital region and then caudad over the posterolateral aspect of the neck without entering the region of the shoulder girdle. The C5-C6 facet joint produces pain in a triangular distribution with the apex directed toward the midcervical region posterior; the main area draped over the top of the shoulder girdle, both front and back; and the base coinciding with the spine of the scapula. The C6-C7 facet joint will refer pain over the supraspinous and infraspinous fossae, periscapular regions, and the medial aspect of the shoulder.[55,82]

Preoperative Medication and Sedation

The patient's response to cervical intra-articular facet injections and medial branch blocks is critical for the diagnosis of facetogenic pain. The patient must remain appropriately responsive in order to report any unexpected or painful symptoms experienced during the procedure; therefore sedation is not required. If excessive patient anxiety might prevent smooth performance of technique, sedation may be used judiciously. The decision to administer sedation must be tempered with the possibility that the postinjection pain diary may not reflect the true situation. A dysphoric patient may confuse physician-induced anxiolysis with pain relief. Administration of opioids is relatively contraindicated, as this would confound the practitioner's ability to determine the exact cause of postinjection pain relief. This information

must be made clear by standing order to the recovery staff, as well as the patient's family. If the goal is to access the duration of relief, the patient must be instructed to avoid pain medications until the usual pain returns.

In most cases, mild sedation and premedication are helpful. The procedure can be uncomfortable; however, most patients tolerate 18-gauge needles when prior infiltration of posterior cervical spine muscles has been adequate. Reproduction of concordant symptoms by sensory stimulation is unnecessary in accessing the patient's response. However, the patient must be able to communicate with the practitioner throughout the procedure, possibly reporting unexpected symptoms or discomfort. If sedation is used, appropriate monitoring is required.

Recommendations for conscious sedation by the American Society for Anesthesiologists (ASA) should be followed.

CERVICAL INTRA-ARTICULAR FACET BLOCK PROCEDURE

Multiple patient positions have been described for the performance of cervical intra-articular facet block. The most common are prone, supine, and lateral. As the operator's comfort zone is critical to the safe performance of the procedure, several components are optimized to provide patient comfort combined with ability to communicate with the patient. In each case, obtaining a true lateral image is essential, securing a set of sharp images with multiple views, documentation of correct needle joint access, interpretation of contrast patterns, and postinjection "washout" films must be saved.

In the lateral-oblique approach, the patient may be positioned supine, lateral, or prone. The lateral position requires placement of foam bolster under the head to maintain a neutral position of the head with the thoracic spine.

In the posterolateral approach, the patient is prone with a support or wedge under the chest enhancing flexion of the cervical spine, thus enhancing visualization of the facet joints, as well as the articular pillars. Additional helpful maneuvers such as employing a "swimmer's view" and angulating the fluoroscope to a caudo-cranial direction may help.

The neck is prepared and draped in a sterile fashion. There is no need to anesthetize the skin overlying the puncture site if a 25-gauge needle is used. Creating a 25-degree bending of the needle bevel for additional control will facilitate easier placement without removal of the entire needle for repositioning.

Technique

The facet joint is visualized under AP fluoroscopic guidance using a pillar view. The arm is then rotated 10–25 degrees oblique toward the side to be injected. This view allows for visualization of the facet joint, and further oblique manipulations of the fluoroscope may be helpful in better visualizing the joint. A metal rule is helpful in identifying the desired target.

A 25-gauge, 100-mm spinal needle is advanced toward the facet joint from a posterior or posterolateral angle (Figure 9-33). The needle is advanced until bony contact is made with either the superior articular process or the inferior articular process near the joint.

The principles of intra-articular joint access are described in the revised first edition of the guidelines of the International Spinal Intervention Society (ISIS). The reader will learn the safest and most precise aspects of this technique, and thus reading the guidelines is highly encouraged. Once entry into the outer capsule is suspected, the needle should then be walked off into the facet joint. Anteroposterior and lateral views should confirm intracapsular placement. A minute quantity of contrast may then be injected into the joint—just enough to evaluate joint morphology, unless this is a purely diagnostic injection. If the injection is therapeutic, confirmation requires just enough contrast to confirm appropriate needle placement. Filling the joint with contrast in such an injection is not desirable, because this will leave no room for local anesthetic or corticosteroids. A minimal volume of contrast medium, adequate to visualize the joint capsule, is all that is required. An intracapsular injection of 0.5–0.7 ml of local and corticosteroid anesthetic (typically in a 1:1 ratio) may be injected.

Care must be taken not to overpressurize the joint injection, as a capsular tear may be created. Spillage (or "seepage") of contrast into the epidural space is not uncommon; thus target specificity is lost. Since the diagnostic value in this case is negligible, dual-controlled medial branch blocks must be performed. It is becoming widely accepted (although debated) that a diagnostic, well-controlled, contrast-enhanced intra-articular injection is predictive of facetal pathology and may be used as the first controlled block only under the most stringent conditions. Concordant pain provocation, containment of injectate, and 80–90% pain relief in the distribution of that joint would constitute a positive initial diagnostic injection. Anything less than this should not be considered to have any diagnostic value.

If the patient states that original pain was re-created by the injection (concordant), multiple-view spot films should be saved (AP), lateral oblique. If the patient states that the injection reproduced only a fraction of original pain, adjacent levels should undergo injection until either concordant pain is entirely reproduced or discordant pain is elicited. If pain is not totally provoked, consider another source. The reader is encouraged to develop an understanding of an algorithmic treatment of the conduct of synovial joint injections for the treatment of cervical spine pain.

Documentation

After final needle placement and following injection of contrast medium, images must be saved and documented. High-resolution images, as in all procedures, must

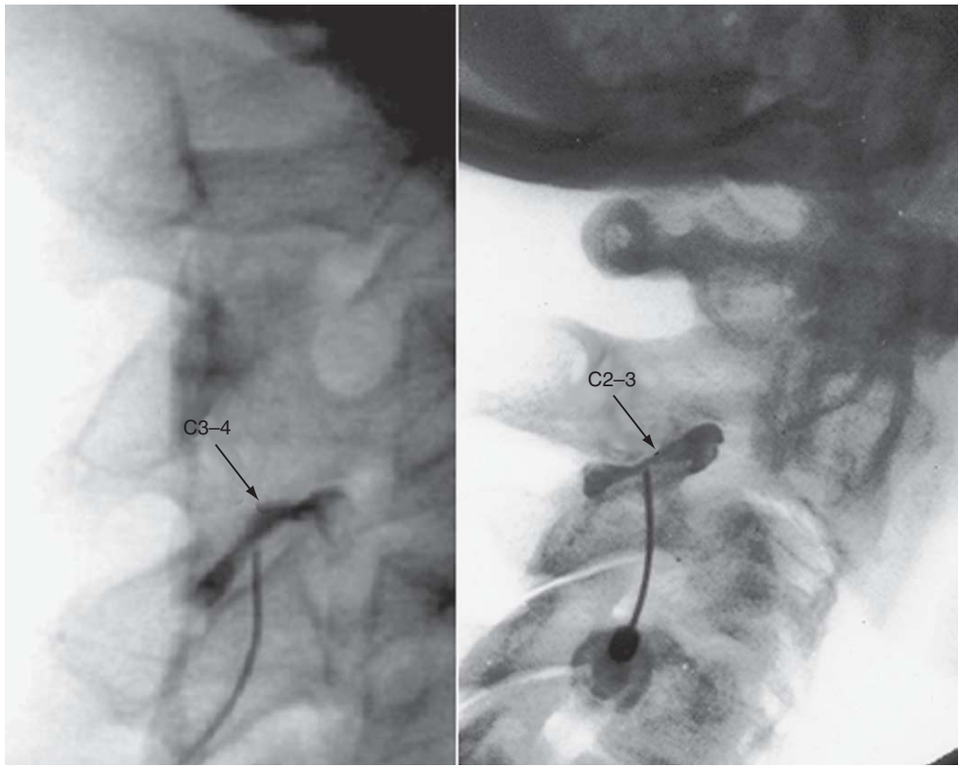


FIGURE 9–33
Cervical intra-articular facet joint arthrograms. Note posterolateral approach. (Courtesy of Paul Dreyfuss, MD.)

be saved either by quality printing and/or saving to hard drive. For cervical intra-articular facet injections, three images are required: (1) initial needle placement, (2) contrast-enhanced arthrogram, and (3) final "wash-out" film. Needle placement during RF neurotomy requires documentation in AP, sagittal, lateral, and oblique views.

CERVICAL MEDIAL BRANCH BLOCK PROCEDURE

Patient Positioning

Supine or prone positions are equally acceptable for the performance of the cervical medial branch block. Some practitioners prefer the patient in the lateral decubitus position. The key element to success in performing this block is meticulous attention to detail in that a near-perfect, true lateral view of the cervical spine is obtained.

Sterility

The skin overlying the target area must be aseptically prepared and allowed to dry to ensure sterility. Long hair must be placed under a surgical cap. A fenestrated drape or towels are placed to ensure a sterile field.

Target Identification

It is essential that the target point is at the center of the x-ray beam so as to appear on center screen. A true lateral view will eliminate any risk that the needle is aimed toward the contralateral side of the neck. This is performed by tilting the x-ray beam around the long axis of the patient until the silhouettes of the articular pillars at each segmental level are perfectly superimposed. It cannot be overstated here, as with any percutaneous injection technique, that the correct initial setup is the most important aspect in target identification.

Because the medial branches of the cervical dorsal rami vary in position at each segmental level, the practitioner must be familiar with these variations. Each joint undergoing diagnostic block will require two adjacent medial branch blocks. For example, the C4-C5 facet joint is innervated by the C4 and C5 medial branches, and would therefore require two blocks (Figure 9-34).

Needle Placement

A puncture point on the skin is selected overlying the target point. With the shaft of the needle aimed perpendicular (lateral) to the target point, a 25-gauge, 60–100-mm spinal needle is advanced under lateral fluoroscopic guidance in small increments. Any correction towards the target must

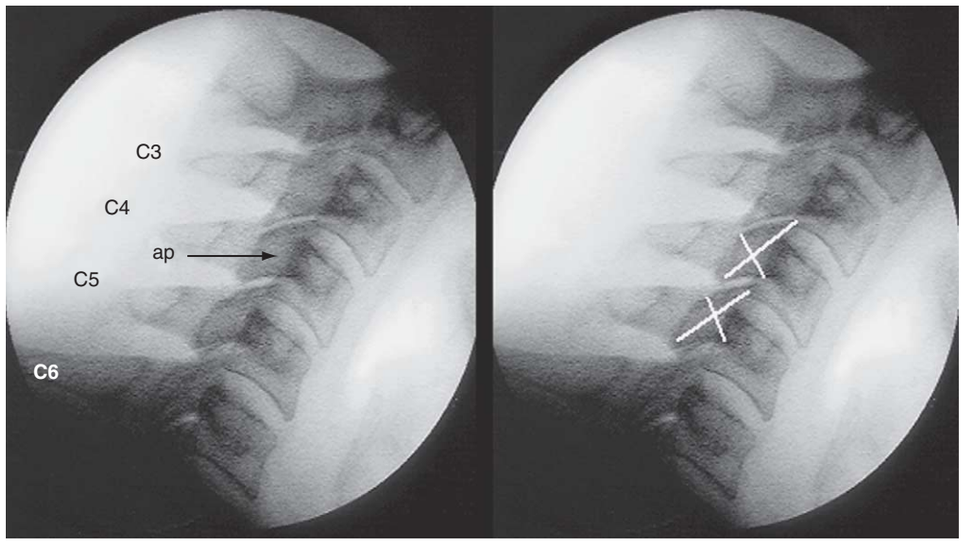


FIGURE 9–34

Lateral view of the cervical spine demonstrating the location of the articular pillars (ap). The target point anatomically coincides with the medial branch of the dorsal rami. At each of these levels, the target point is the centroid of the articular pillar with the same segmental number as the target nerve. The C4 and C5 medial branches of the dorsal rami must be adequately blocked to anesthetize the C4-C5 facet joint. (Courtesy of Newcastle Pain Management and Research Group and The International Spine Intervention Society.)

be made superficially. With periodic screening, the needle is advanced progressively and continually seen to overlie the target point. This will ensure that multiple needle insertions or overcorrections are not required. Only subtle needle corrections are then made until periosteum is contacted (Figure 9-35).

After gentle aspiration, a small volume of contrast (0.3 ml) should be injected, carefully observing for target specificity as well as possible vascular uptake.

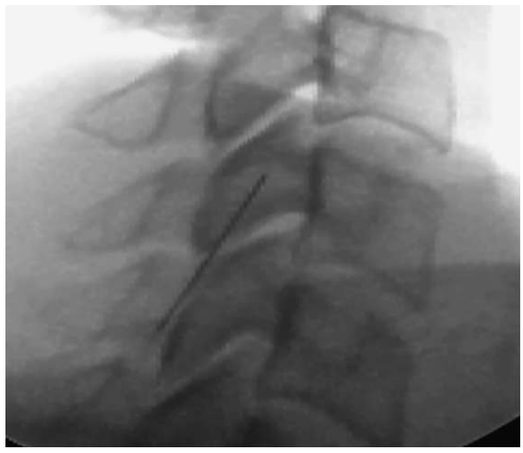


FIGURE 9–35

Spot film. Direct lateral view of midcervical spine. Articular processes are "superimposed" on one another. In this view, the processes have typical parallelogram configuration. Procedure needle is against the lateral surface of the process at the site of the C4 medial branch. (Courtesy of Charles Aprill, MD, and International Spine Intervention Society.)

Using a pillar view, this is confirmed by spread of the contrast across the lateral surface of the articular pillar, filling the concavity that lodges the target medial branch (Figure 9-36). If any venous runoff is detected, the needle should be repositioned. If, for example, venous uptake is substantial and the needle not repositioned, then one must assume that subsequent injection of local anesthetic would be reduced by a similar amount, which might yield a false-negative response leading to an incomplete investigation and an erroneous diagnosis (Figure 9-37).

Once proper positioning is confirmed by contrast, anesthetize the target nerve with 0.3 ml of 0.5% bupivacaine or 2% lidocaine. The needle should be held in place, against bone, during the injection. Note that some patients have multiple joint involvement, usually C5-C6 with C6-C7.

At C7, the goal is to place the needle onto the lateral aspect of the superior articular process near its apex (Figure 9-38). Utilizing a sagittal view, the superior articular process is contacted.

A lateral view should be obtained to confirm that the needle tip lies against the lateral margin of the superior articular process. Contrast medium is then injected to confirm target acquisition prior to injecting local anesthetic. Care must be taken to ensure that the contrast injected covers the intended target. As can be seen from this contrast enhanced image, there is no question that unintentional epidural spread has occurred. In such situations, it may be wise to re-schedule the block as further injection of contrast medium may be difficult to visualize. Perhaps digital subtraction angiography may be applicable if the

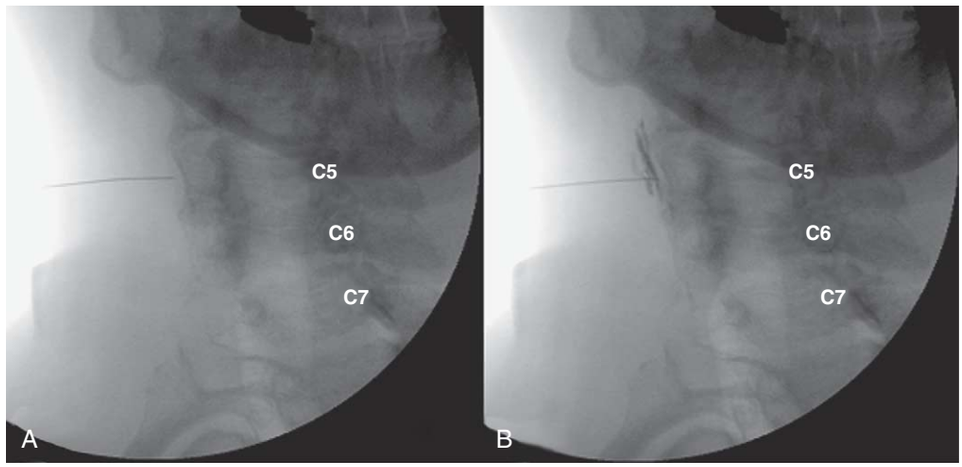


FIGURE 9–36
Needle placed in mid-articular pillar for C5 medial branch block. Note contrast medium filling articular pillar. (Courtesy of Paul Dreyfuss, MD, and International Spine Intervention Society.)

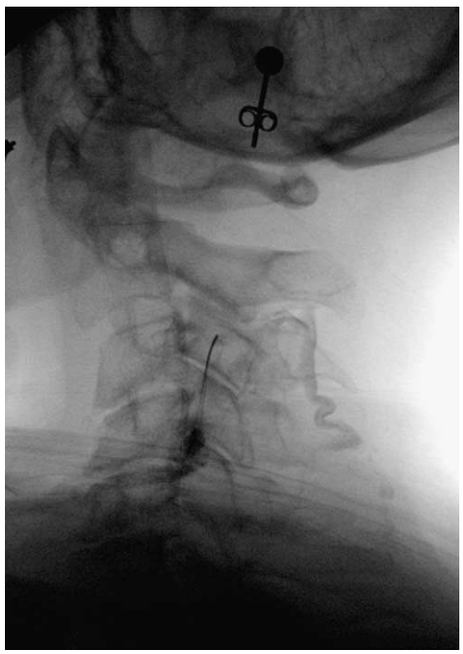


FIGURE 9–37
Venous runoff prior to medial branch block. (Courtesy of Paul Dreyfuss, MD.)

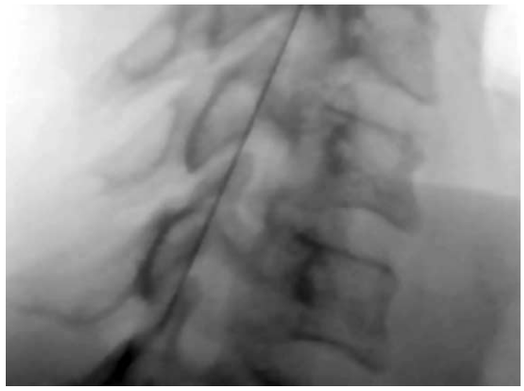


FIGURE 9–38
C7 medial branch block. (Courtesy of Charles Aprill, MD, and International Spine Intervention Society.)

operator is comfortable with this advanced imaging technique (Figure 9-39).

Due to the anatomical variance of the C7 medial branch, an additional injection should be performed to ensure infiltration of the nerve. Withdraw the needle 4 mm and inject an additional 0.3 cc of local anesthetic. In addition, if the C7 superior articular process is tall, an additional block at the junction of the transverse process with the superior articular process may be necessary.

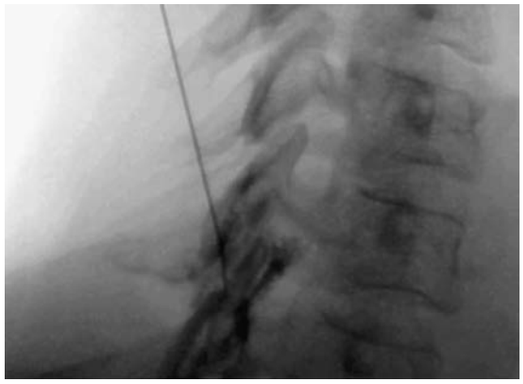


FIGURE 9–39
Contrast-enhanced C7 medial branch block. (Courtesy of Charles Aprill, MD, and International Spine Intervention Society.)

To block the C7-T1 facet, the C8 medial branch must be blocked. The C8 medial branch courses around the superior and posterolateral aspect of the transverse process of the first thoracic transverse process at T1. A needle is advanced to this position down the beam of the x-ray to its target point, which will be the dorsal surface of the transverse process, opposite the lateral end of its superior border. The point is not the superior lateral corner of the transverse process, but lies medially to it. Contrast medium is injected under continuous AP screening to ensure spread across its location on the transverse process and to rule out venous uptake. Once the needle is confirmed in the correct position, 0.3 ml of local anesthetic is injected.

Given the high false-positive rate found with cervical medial branch blocks, if the patient reports relief of 90% or more of typical pain, repeat the procedure. If a second block produces similar results, treatment with RF medial branch neurotomy is indicated.

POSTPROCEDURE MONITORING

Patients must be aware that they will need to report pain responses accurately. Ideally, an independent examiner who is "blinded" to the procedure performed and drug injected would perform pre- and post-injection pain and functional assessments. Pre-VAS (visual analogue scale) pain scores are compared to postinjection VAS, as well as range of motion, among other features. Independent outcome assessment tools are available from many sources, and the reader is encouraged to validate their interventions by postprocedure assessment.

The patient should be instructed to keep a postprocedure pain diary to meticulously document progress after injection. In the diary the patient must note any immediate change in symptoms; he or she must be instructed to keep track of any change in pain in the first 24 hours postprocedure. The physician must insist that prior to any such diagnostic injection, the patient's pain must be of a significant VAS number in order that any postinjection VAS number has changed enough to allow for at least an 80% pain relief, which is critical to the determination to proceed with a second comparative block or RF medial branch neurotomy. A telephone interview is acceptable following the proper conductance of the postinjection assessment.

RADIOFREQUENCY MEDIAL BRANCH NEUROTOMY

Principles

Cervical medial-branch RF neurotomy aims to destroy the afferent nerve supply to the facet joints in recalcitrant cases. The procedure uses RF current, with lesions performed at 85°C for 60–90 seconds. Nerve regeneration is assumed to occur in 9–12 months, and therefore pain may recur. However, the procedure may be performed again.

Following are several advantages in using RF neurotomy:

- Controlled lesion size
- Good monitoring of lesion temperature
- Precise placement of electrode with electrical stimulation
- Rapid recovery
- Low incidence of morbidity
- Ability to repeat lesion if neural pathway regenerates

Radiofrequency electrocoagulation involves the placement of an insulated electrode with an uninsulated tip into nervous tissue. Electrical current is then delivered to the tissue, and heat is generated as a result of current flow through the resistance of the tissue. Charged molecules (mostly proteins) oscillate with the rapid changes in alternating current; this friction in the tissue produces heat.

Between 42.5°C and 44°C, neural function temporarily stops.[83] Early cytotoxic temperature for nervous tissues is 50°C. It is recommended that temperatures of at least 70–80°C be used to create irreversible lesions.[74,84] Boiling or carbonization of tissues can occur when temperatures rise beyond 90°C.[85]

Larger RF needles produce a larger lesion radius (Figure 9-40). At least 60 seconds, and not more than 90 seconds, are required to control the appropriate and adequate lesion radius.

Radiofrequency neurotomy is used to denervate a facet joint. This procedure should only be performed after the appropriate diagnostic medial branch blocks have yielded positive results.

The medial branches are small targets with variable locations up along the articular pillar. The third occipital nerve measures some 1.5 mm in diameter and the medial branches 1.0 mm in diameter, being displaced from the bone of the articular pillar between 1 and 2 mm[77]; therefore, it is not necessary that the RF needle be in direct contact with the osseous surface of the articular pillar.

The practitioner must have prior knowledge of variations in medial branch locations to perform this procedure adequately. It is highly recommended that the practitioner develop an intimate understanding of the locations of medial branches at various segmental levels. Lord's[77] composite tracings of these nerves based on cadaver studies outline their paths (Figure 9-41). It is suggested that his diagram should be readily available to student practitioners as an intra-operative guide so that lesions are performed at correct locations along the articular pillars. The course of the cervical medial branch wraps around the curved articular pillar and requires both sagittal and oblique approaches to coagulate the maximal length of the nerve. A maximal length of the medial branch must be coagulated along the lateral and anterolateral sector of the pillar, as it will take longer for neural regeneration (with subsequent return of pain) if a longer length of the nerve is coagulated.

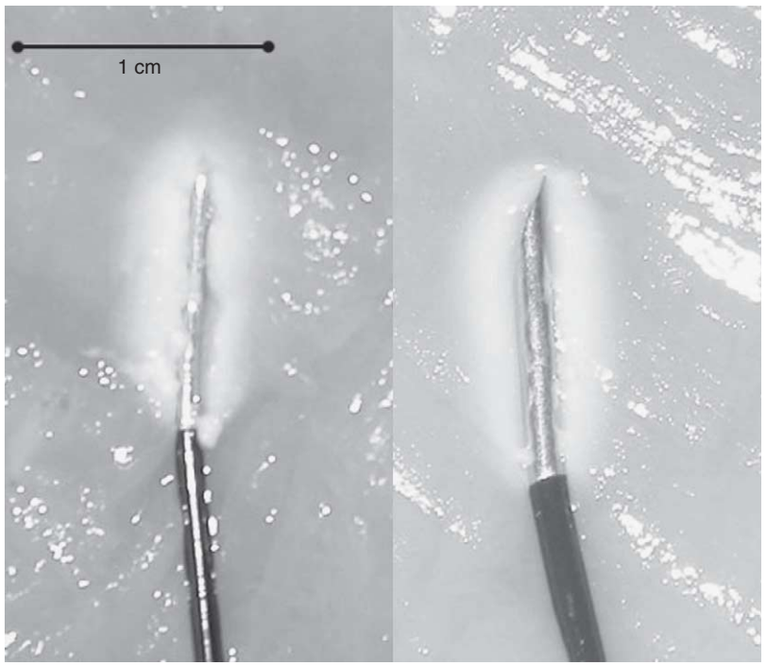
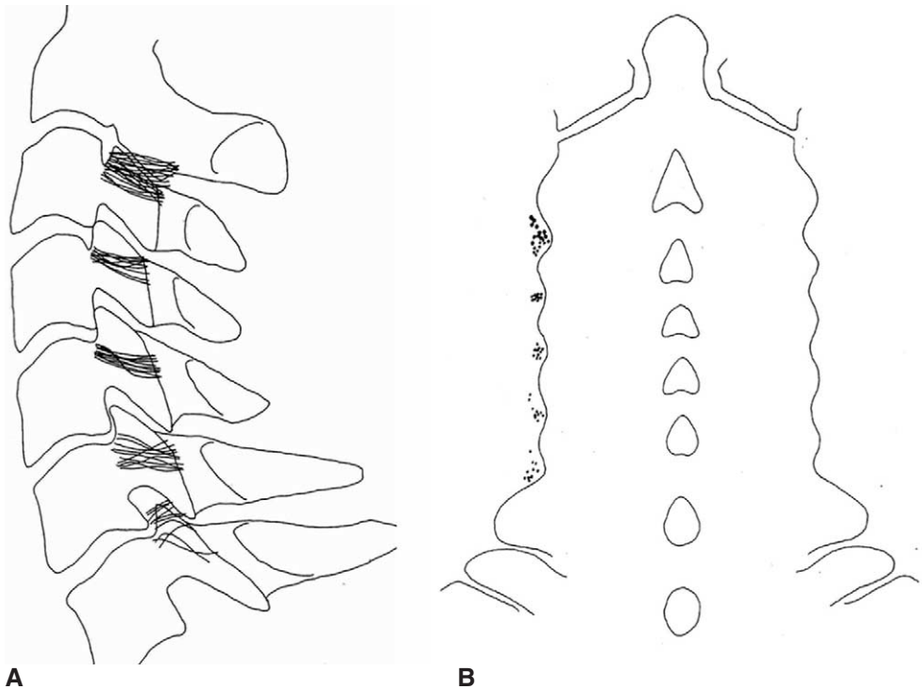


FIGURE 9–40

Note different-sized lesion produced with 20-gauge electrode (left) versus 18-gauge electrode (right). The 20-gauge RF needle with 10-mm active electrode tip produces an average lesion width of 4.8 mm. The 18-gauge RF needle with 10-mm active electrode tip produces an average width of lesion of 5.8 mm. (Courtesy of Paul Dreyfuss, MD.)



A B

FIGURE 9–41

Illustration of composite tracings of radiographs with wires applied to the medial branches of the cervical dorsal rami. These are marked to show the variation in their location. (A) Lateral view. At the C3 level the locations of the C3 deep medial branch are shown. It will be noted that the C5 medial branches are generally located over the middle fifth of the C5 articular pillar, whereas the medial branches are located increasingly higher on their respective articular pillars at levels increasingly removed from the C5 level. (B) Anteroposterior view. Nerves are depicted as dots passing from front to back. (From Lord SM, McDonald GJ, Bogduk N: Percutaneous radiofrequency neurotomy of the cervical medial branches: a validated treatment for cervical zygapophyseal joint pain. *Neurosurg Q* 8:288–308, 1998, with permission.)

This principle is what guides the rationale for a two-needle approach utilizing sagittal and oblique passes (Figure 9-42). The geometry of an RF lesion dictates that for optimal coagulation, the electrode should be placed parallel to the nerve. Approximately 80% effective coagulation is achieved by dual-pass lesioning. Consequently, a single pass using a straight electrode will coagulate 60% as opposed to a curved electrode of similar length, which will coagulate 65% of the nerve (Figure 9-43).

Features of the geometry of coagulation have special implications. Consider that the radial lesion coagulates tissues 1.6–2.3 electrode widths (standard deviation 0.3–0.4). These standard deviations indicate 97.5% confidence in coagulating one electrode width radially (SMK) and distal to the tip of 0.6 electrode width. Therefore, there is no reliable coagulation distal to the electrode tip. In order to coagulate a wide volume of tissue thoroughly, electrode placement must be parallel with additional lesions one electrode-width apart. Routinely, three lesions are performed at each level. The lesion created is elliptical surrounding the electrode with the zone of coagulation 1–1.5 times the diameter of the needles and does not extend beyond the tip of the probe.[79,86]

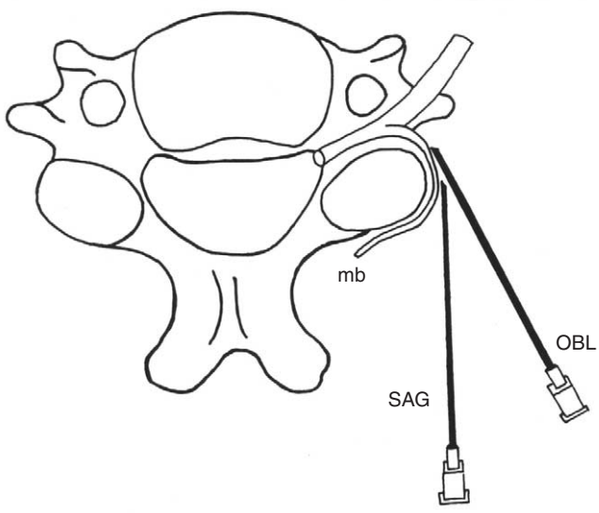


FIGURE 9–42

Illustration using axial view to demonstrate a lateral (sagittal) pass where the needle lies over the 9 o'clock sector of the articular pillar and an anterolateral (oblique) pass lies over the the anterolateral sector. (Courtesy of International Spine Intervention Society.)

The electrodes must be placed parallel to each other, separated by no more than the distance of the diameter of each electrode.[79] This ensures that the entire path of the nerve is exposed to an RF coagulation, therefore obtaining a higher likelihood of success.

The size of the lesion depends on certain variables, including tissue impedance and duration of thermocoagulation. To achieve adequate lesion size, at least 60 seconds at 80–90°C is required. It is recommended that a rise from body temperature of 1°C per second will provide the largest lesion. This is to avoid irregular-sized and -shaped lesions due to a too-rapid increase in temperature, which may cause tissue cavitation, boiling, (Figure 9-44).

An alternative and perhaps more practical means of ensuring maximum lesion size requires continual observation of tissue impedance as it pertains to temperature increase. In this manner, the lesion can be created more efficiently and cavitation can be avoided. A sudden change in temperature or fluctuations in impedance should then alert the practitioner to the presence of heat-absorbing tissue or faulty equipment.

Patient Positioning

The patient lies prone or in the lateral position. The prone position is most frequently employed for patient stability and for the ease of C-arm rotation to obtain all

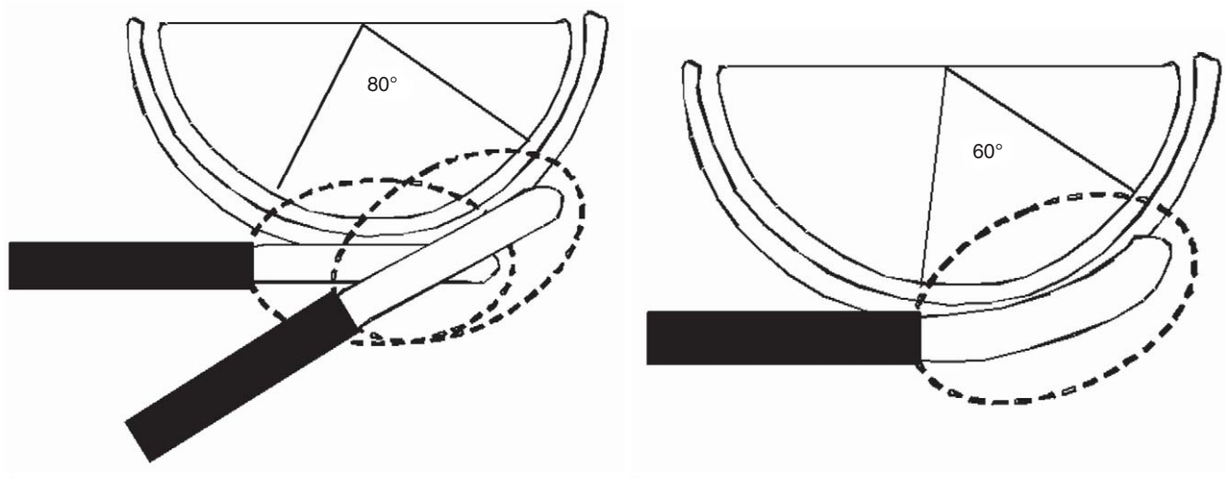


FIGURE 9–43

Use of combined oblique and sagittal passes, with overlapping lesion zones (A), will produce 33% more coagulation than a single curved pass (B), even though in each case the pass is 60 degrees. (Courtesy of International Spine Intervention Society.)

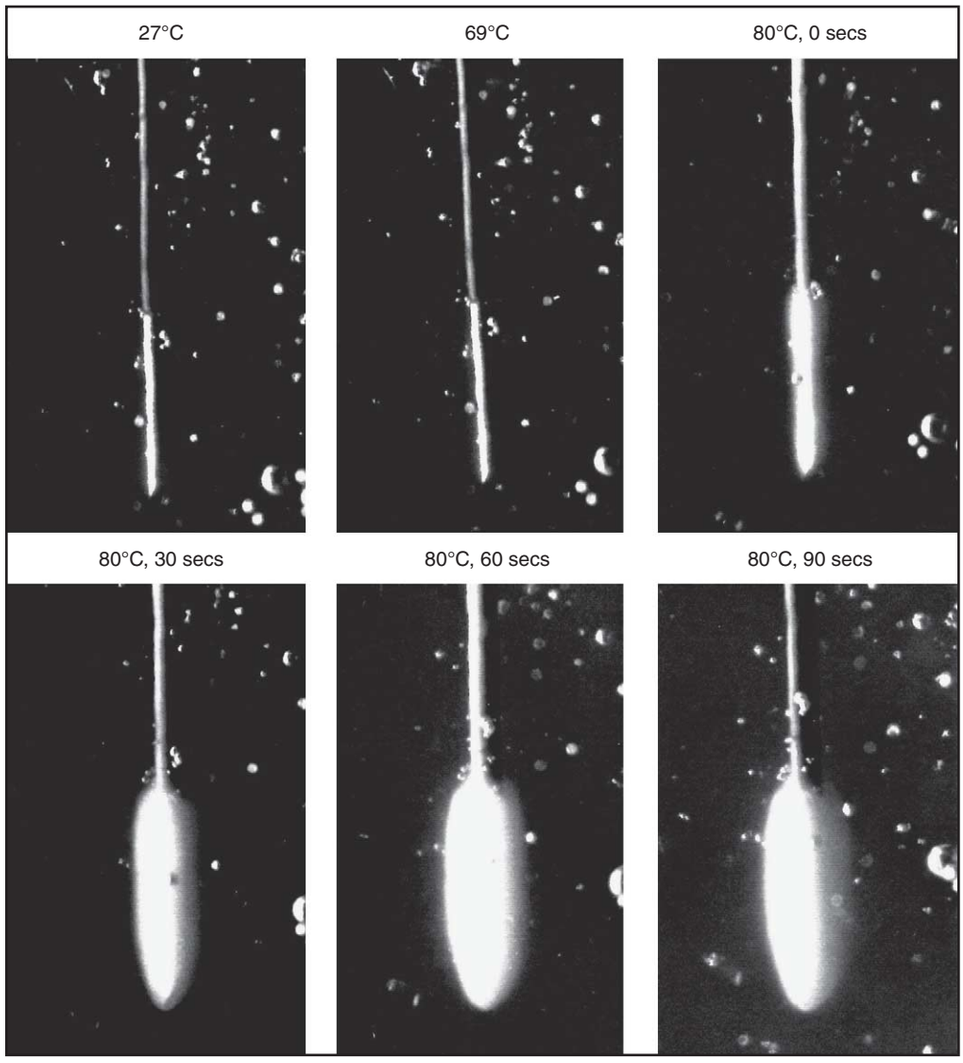


FIGURE 9–44

Lesions created with the SMK electrode in egg whites. Note the lesion expands radially with time. The size of the lesion at a constant temperature of 80 degrees demonstrates at 30 seconds 85% maximum, 60 seconds 94% maximum, and 90 seconds 100% maximum. (Courtesy of International Spine Intervention Society.)

views. A pillow or foam wedge is placed under the patient's chest, and a small support is placed under the forehead. This allows for greater flexion of the cervical spine while allowing patient comfort and adequate ventilation. The arms are placed by the patient's side and a Velcro wrap is placed around the patient for stability, pulling the shoulders caudad so as to not obstruct fluoroscopic visualization. Performing the neurotomy in the lower cervical levels (C6, C7) may require additional maneuvers in order to clearly observe exact location of the electrodes and ensure parallel positioning, with electrode tips posterior to the intervertebral foramen. Such maneuvers may include a "swimmer's view" and a 10–15-degree contralateral oblique view. Patients with short necks may also require this special positioning to obtain an unobstructed view.

IMAGING

Target Identification

This is a two-step technique. Using an AP view without rotation, a metal rule is placed over the facet joint caudal to the medial branch to be lesioned. For example, if the target branch is C4, the rule is placed just lateral to the silhouette of the C4-C5 facet joint (Figure 9-45).

Without changing the pointer location, the C-arm is then adjusted to obtain a pillar view and rotated approximately 10–15 degrees oblique. Following this maneuver, the rule is now overlying the waist of the articular pillar of C4 to target the lateral aspect of the articular pillar (Figure 9-46).

A sterile metal rule is used to identify the target, and the overlying skin is then marked with a sterile

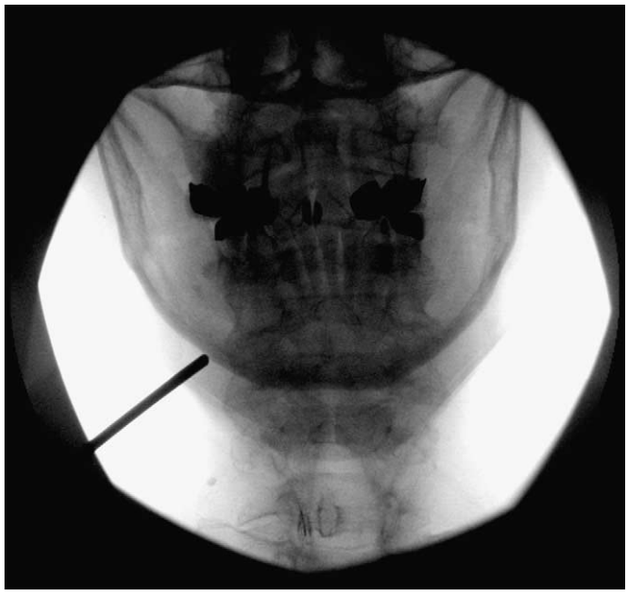


FIGURE 9–45

Trajectory from skin. Anteroposterior, nonpillar view. Note metal rule at the "flange" of the facet joint just caudal to the midpillar target. (Courtesy of Paul Dreyfuss, MD.)

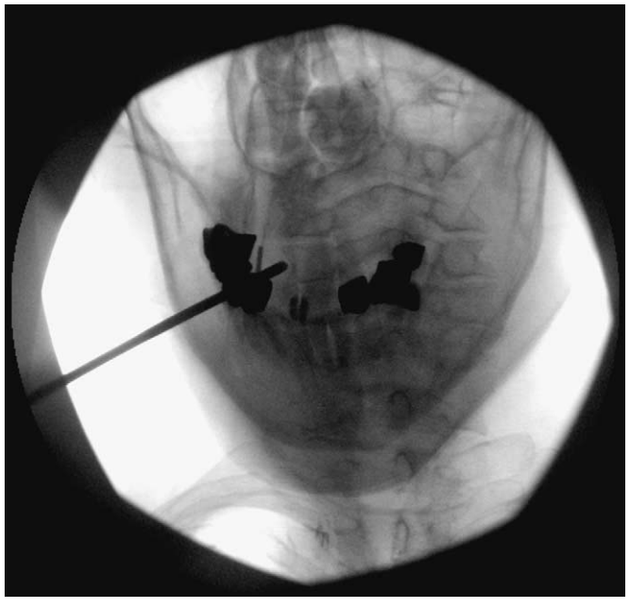


FIGURE 9–46

Without changing pointer location, the C-arm is maneuvered to obtain a pillar view and 10–15 degree oblique. The pointer now lies in the midpillar position. (Courtesy of Paul Dreyfuss, MD.)

marking pen. Subcutaneous infiltration with local anesthetic along the intended needle track using a 25-gauge, 1-1/2-inch needle is then performed. To facilitate smooth passage of the RF needle, an 18-gauge, 1-1/2-inch needle can be advanced using a "gun barrel" technique.

Needle Placement

A single-pass technique with a commercially available needle (18–20 gauge) is described here. Using three parallel needle passes to the anterior and anterolateral aspect of the target points on the articular pillars should provide adequate coagulation on the appropriate sectors of the pillars while ensuring maximal length coagulation of the medial branch. Although this technique has not been verified with Lord's study in terms of outcomes, its performance relies heavily on the physics, principles and unique anatomy of the cervical pillars, as well as possible neural pathways at typical and atypical (C2-C3) levels.

During performance of the procedure, it is critical that consecutive images on split screen are identical to the previous ones. Even the slightest movement of the patient or C-arm will change the appearance of target structures on the monitor. Therefore, prior to the procedure all necessary steps should be taken to reduce this by ensuring patient cooperation, as well as communicating the importance of this to the radiographer.

Multiple needle passes are required to effectively coagulate all the territories in which the nerve might lie. Smaller needle gauges will require more lesions to coagulate a similar target area than larger needles. The electrodes must be inserted according to how they conform to the cervical articular pillars to which the cervical medial branches are related. A cephalad-anterior slope or angle (seen as a "pillar" view under fluoroscopy) is used for needle placement. This will avoid needle contact with the lateral flange and any osteophytes that would displace the needle laterally, away from the waist of the articular pillar.

The needle is advanced along the anesthetized and dilated path to make osseous contact with the midposition (waist) of the pillar, just medial on the upper or posterior aspect of the pillar (Figure 9-47). The needle is then advanced under true lateral fluoroscopy to the middle third of the articular pillar while constantly maintaining osseous contact. Subsequent adjustments to the anterior third of the pillar are then made under lateral view. The needle should lie snugly against the anterolateral margin of the articular pillar (Figure 9-48). The last image or view obtained prior to stimulation is oblique. This will confirm that the needle has not strayed too far anterior, thus ensuring that the electrode tip remains posterior to the intervertebral foramen (Figure 9-49).

Stimulation

Familiarity with the anatomical landmarks and the safe radiographic interpretation in regard to needle placement and RF lesioning is critical to the safe and proper performance of this technique.

Sensory/motor stimulation may be a useful adjunct to the performance of radiological imaging. There are no studies, however, to validate the effectiveness of sensory stimulation as related to outcomes of cervical RF neurotomy. In fact, very convincing views on sensory stimulation

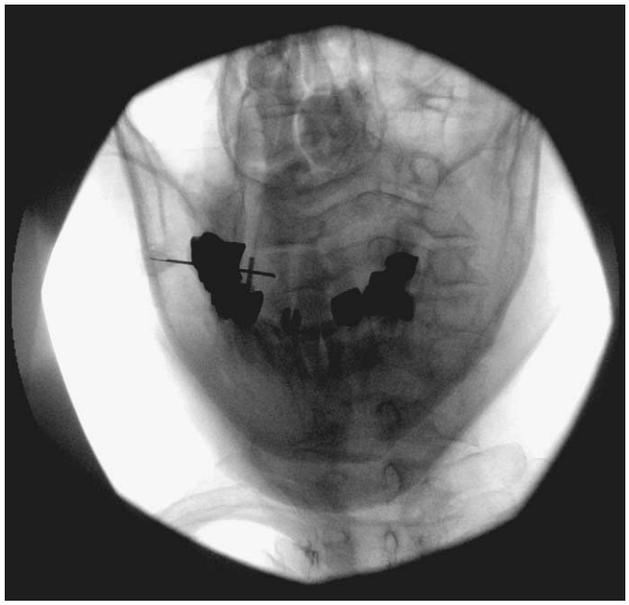


FIGURE 9–47

The radiofrequency needle advanced to touch the medial and posterior aspect of the articular pillar. The needle is then advanced slightly along the midposition (waist) of the pillar, always making contact with bone. (Courtesy of Paul Dreyfuss, MD.)

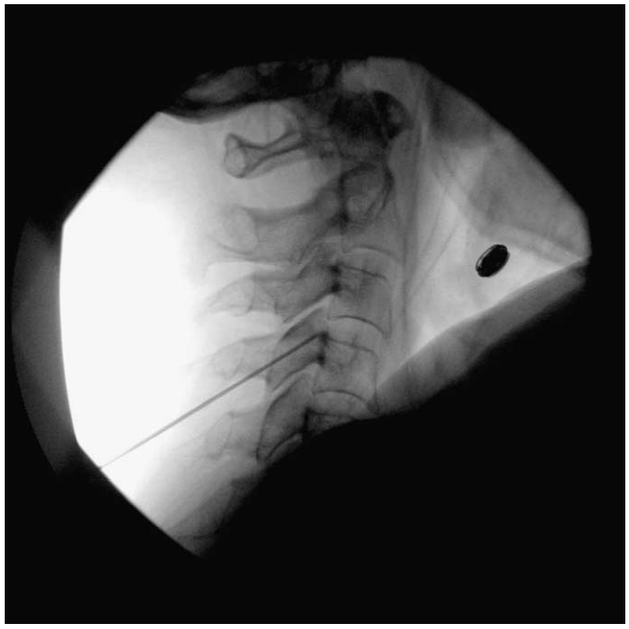


FIGURE 9–48

Under true lateral view, the needle is advanced along the midpillar to its anterolateral border. (Courtesy of Paul Dreyfuss, MD.)

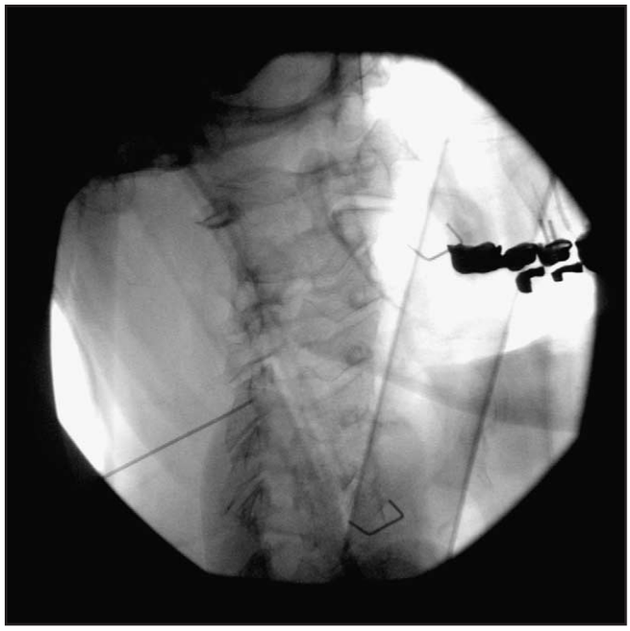


FIGURE 9–49

Oblique view. Confirmation of safe distance of electrode tip from the intervertebral foramen. (Courtesy of Paul Dreyfuss, MD.)

that point to the contrary are discussed in the ISIS *Practice Guidelines: Spinal Diagnostic and Treatment Procedures*.

For the beginning practitioner of RF neurotomy, it is recommended that sensory stimulation be performed for the following reasons. There are many factors that may contribute to wrong needle placement. The slightest movement of the patient or C-arm could easily result in small yet potentially harmful radiographic misinterpretation. This is especially important to the student who is in the early stages of learning this technique. Stereotactic needle localization combined with sensory stimulation is an excellent learning tool as the beginner must be cognizant of how the needle location changes with different movements of the fluoroscope. Recognition of electrodes in the various locations of the articular pillar combined with stimulation might challenge the practitioner to a steeper learning curve. An increase in one's "comfort zone" in that the practitioner feels she or he has an extra "safety net" should not be underestimated.

For sensory stimulation, the generator should be set at 50 Hz, and the output slowly increased by small increments up to 0.3 V. Paresthesia in the cervical area corresponding to the level being stimulated should be noted by the patient. The needle may require repositioning if this is not the case.

Motor stimulation is helpful and confirmatory that a safe distance exists between electrode tip and ventral ramus. Motor stimulation is performed with the generator set at 2 Hz and a maximum output of 2 V. An output of 0.75 V will ensure no arm twitch in the patient.

Motor contraction may be seen in the paraspinous muscle (multifidus) in the neck, but no stimulation should be felt by the patient down the upper extremity or in the shoulder. A sensory stimulation should be greater than 0.3 V to avoid nerve root lesioning.

Once acceptable stimulation is achieved, local anesthetic (2.0 ml of 2% lidocaine, 0.5% bupivacaine) is injected prior to the first lesion. At least 2 minutes should be allowed prior to lesioning for analgesia to take effect. Two consecutive adjacent lesions are then performed. It must be understood that the local anesthetic administered will block further attempts at stimulation. Therefore, sensory and motor stimulation are performed only prior to the initial lesion.

Obtaining split-screen images of needle placement will allow the practitioner to ensure parallel needle positioning while "walking" the needle up the articular pillar to perform subsequent lesions. Oblique views should be utilized to determine that electrode depth is at a safe distance from the intervertebral foramen. Spot films for each needle placement are saved for documentation.

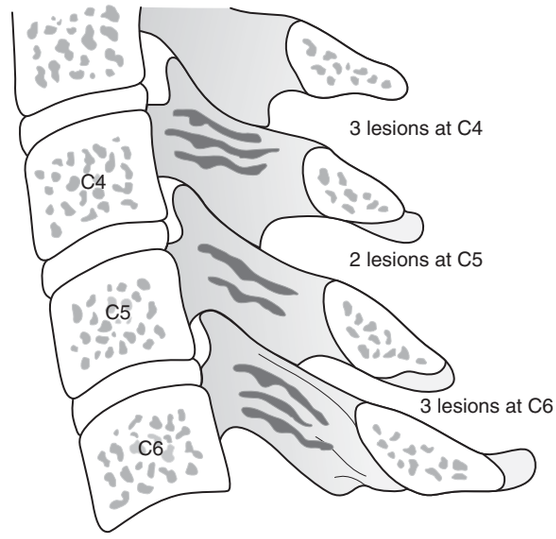


FIGURE 9–50

Typical midcervical medial branches and overlying needle/electrode positioning in parallel. As much of the area as possible under the suspected nerve locations should be coagulated. (Courtesy of Paul Dreyfuss, MD.)

RADIOFREQUENCY NEUROTOMY

Following performance of the previous steps (imaging, target location, needle placement, stimulation), the needle should be in the appropriate position for the production of the first thermal lesion.

At all times during an RF lesion, the patient must be monitored for pain. If the patient complains of pain, the lesion must then be immediately terminated and the nature of symptoms interpreted. If symptoms are localized to the tip of the electrode, additional local anesthetic may be given. If radicular pain occurs, the electrode must be repositioned and the position of the electrode tip must be determined by oblique imaging to determine that it is a safe distance from the foramina.

Once proper positioning is reconfirmed, coagulation is resumed. If symptoms cannot be eradicated by the above methods, the procedure should be terminated and the patient reassessed.

The C4-C6 medial branch nerves have the most consistent locations at the mid-aspect of the articular pillar as seen on lateral imaging. The locations of the medial branches in adjacent articular processes (i.e., C3, C7) ride higher or in a more cephalad position on the articular pillar.

For C3, the most cephalad lesion may share a similar location on the superior articular pillar as the third occipital nerve. Due to variations in the course of that nerve, sensory testing will differentiate the two. Sensory testing of the third occipital nerve would send paresthesia to territories innervated by that nerve that will be clinically distinct from C3 and perhaps not recognized as "typical" or concordant by the patient.

Three RF lesions are required at levels C3, C4, and C6. This should cover the anterior third and potentially the midpillar. For C5 nerves, two or three lesions are needed to cover the anterior third and hopefully the middle third of the pillar (Figure 9-50). Therefore, if the facet joint to undergo denervation is C5-C6, at least five to six lesions are required—two to three lesions at C5 and three lesions at C6. For C7, four lesions are required to effectively coagulate the anterior and potentially the middle third of the pillar as well (Figures 9-51 and 9-52).

The placement (position) of the first needle will dictate where adjacent needles will be placed. The variability of location of medial branches must be taken into account as the goal is to coagulate the highest percentage of the target area in order that maximum neural tissue destruction is accomplished.

Adjacent needle placement is performed in the same manner, with needles placed no farther apart than one electrode width. The second electrode is placed to lie over the adjacent anterior and anterolateral aspect of the appropriate location on the articular process. The third electrode is placed farther cephalad on the pillar. To catch the nerves that run high on the pillar, the second and third electrodes are placed farther cephalad from the initial needle and are "walked up" the pillar using split-screen imaging to assist in confirming correct needle placement.

Once the first lesion is complete, the electrode is withdrawn slightly and readjusted to each of any subsequent positions required above or below the initial position. To ensure that contact with the pillar is maintained, the electrode should not be withdrawn farther than the posterior margin of the articular pillar. At each subsequent position, the same protocol should be followed to check, confirm, and record correct electrode placement.

Following the initial neurotomy, the same steps are taken to coagulate the adjacent medial branch. Therefore, six RF lesions are required to successfully denervate a single facet joint.

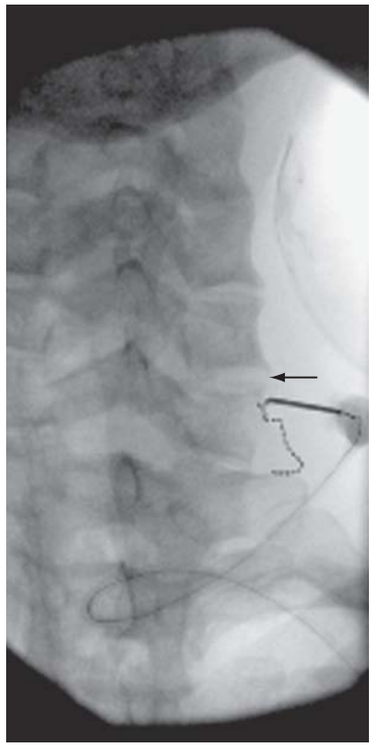


FIGURE 9–51

C7 medial branch neurotomy. Initial placement. The anterior and then middle third of the C7 pillar will require four lesions to effectively coagulate all possible locations of the nerve. (Courtesy of International Spine Intervention Society.)

Radiofrequency Neurotomy of C7

Under AP view, the electrode is inserted initially toward the back of the C7 superior articular process until it strikes bone. This step ensures that the needle has not been advanced anteriorly into the intervertebral foramen. The electrode is advanced 2–3 mm and is then redirected to slip onto the lateral surface of the superior articular process (Figure 9-53). A lateral view is then required in order to advance the electrode to the apex of the lateral aspect of the superior articular process. The depth of insertion should be determined under a 10–15-degree oblique view when possible. This will ensure that the electrode tip lies posterior to the intervertebral foramen of C6-C7 (Figure 9-54).

Prior to generating a lesion, electrode position must be checked, confirmed, and recorded on AP, lateral, and if necessary, oblique views. The same precautions and protocols that apply to lesioning at typical cervical levels must be adhered to in this case.

Radiofrequency Neurotomy of C8

Medial-branch RF neurotomy of C8 is similar to that of the upper thoracic segments. This technique should be based upon contemporary anatomical knowledge. Various clinical techniques have been described in textbooks and by practitioners without validation. Variations include sensory and/or motor stimulation for localization of the target nerve with single lesioning to overlapping lesions of the

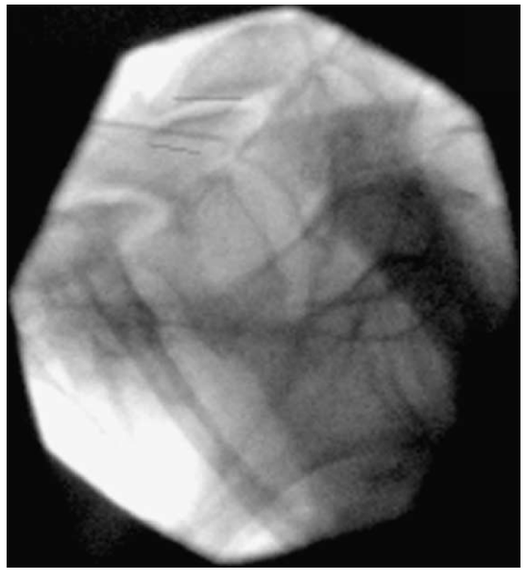


FIGURE 9–52

C7 medial branch neurotomy. Oblique view with borders of lesion zone. Four RF lesions are required, no greater than one needle-width apart. (Courtesy of International Spine Intervention Society.)

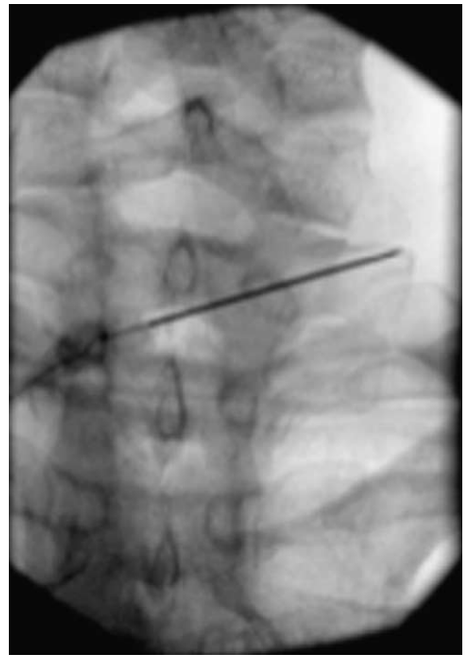


FIGURE 9–53

C8 radiofrequency neurotomy. Following sensory stimulation, three needle passes from the contralateral side are performed along the posterolateral and superior aspect of the first thoracic transverse process. (Courtesy of Paul Dreyfuss, MD.)

territory where the nerve may reside. A contralateral approach to the target nerve is suggested to coagulate the target area. This geometrical approach utilizes greater electrode length combined with parallel lesions to ensure that the nerve does not escape coagulation (Figure 9-55).

POSTPROCEDURE MONITORING

Because patients may experience unsteadiness following this procedure, they should be escorted and given support while transferring to recovery. Patients should not be allowed to transport themselves home and should only be discharged into the care of a responsible adult. At discharge patients should be instructed to apply cold packs to the site for a day or two, to administer simple analgesia when required, and to notify the practitioner of any unusual sensations that may indicate an infection of the operation site.

COMPLICATIONS

Transiently increased pain is the most common complication following cervical facet blocks and RF, with an incidence of 2%, lasting from several weeks to 8 months maximum.[87] On the other hand, spinal anesthesia has been reported in intra-articular lumbar facet joint injections.[88] This is one reason that medial branch blocks and radioneurotomy are preferred over intra-articular injections.

Chemical meningism, which has been noted after medial branch blocks,[89,90] could also be caused by inadvertent dural puncture. It is important to confirm needle placement in more than one view (particularly in the lateral view) to ensure that the needle tip is posterior to the neural foramen.

Constant vigilance for the maintenance of sterile technique is essential. Failure to do so may result in less severe but still troublesome complications. For example, paraspinous tissue infections may result in abscesses.[54,91,92]

Intra-articular injections, particularly within the vertebral artery, are a risk. If the needle tip during a supine approach is too anterior, this could result in vertebral artery puncture.

Neuritis, with RF of the median branches, presents as new-onset burning pain caused by the RF needle being too close to a large nerve root. This uncommon complication is self-limited, lasts less than 2–3 weeks, and responds to conservative therapy and systemic steroids.

Inadvertent lesioning of nontargeted tissue may occur if there is a break in the needle's insulation. If the patient experiences sudden burning pain or pain down the arm, the cycle should be stopped immediately and needle position checked or the procedure aborted. The use of fluoroscopy is essential to guarantee accurate needle placement and patient safety. In the cervical region, an inappropriately placed needle could result in devastating spinal cord injury. Ataxia, particularly after upper cervical blocks, is a temporary side effect.

Complications specific to cervical RF neurotomy include:

- Dyasthenias and numbness in the cutaneous territory of one of the coagulated nerves (19–29%)
- Vasovagal syncope
- Neuritis
- Dermoid cyst
- Kobner's phenomenon

CLINICAL PEARLS

It is important to note that significant degenerative disease of the spine or prior spine surgery (particularly fusion) will proportionately increase the difficulty of the procedure and decrease the success rate. In patients who have had previous surgery, the pain may be caused by a scar or bony entrapment of nerves. The procedure can be extremely demanding, and it may be wiser to abandon the procedure than to place the patient at additional risk.

Although the median branches of the dorsal rami innervate both joints and muscle, pain that is relieved by medial branch block can be assumed to arise from the facet joint instead of overlying muscles or ligaments.

The small-volume diagnostic injections used for median branch nerves, although fairly specific for assessing facet pain, have been reported to produce false-negative results 8% of the time in the lumbar spine. This occurs when the injectate is inadvertently delivered to the vessels accompanying the median branch nerves. The injectate is either carried away or diluted by a small hematoma, and the pain is not relieved. The practitioner may then falsely assume that that facet joint is not the cause of the pain.

The procedure requires that the practitioner is highly skilled in precision spinal diagnostic techniques. This procedure should therefore only be performed by practitioners with extensive experience in treating pain of cervical spine origin. Intra-articular injections are helpful diagnostic tools; however, the proper performance of the medial branch block is essential. Only local anesthetic is used and must be placed directly to ensure that the target nerve is blocked. Minimal to no sedatives and the understanding that opioids as well as other antinociceptive medications (i.e., tramadol) are to be administered during the course of postprocedural assessment. The diagnostic utility of the block is the critical aspect as to whether the patient would be an appropriate candidate to receive long-term relief by RF neurotomy. Failure to adhere to strict criteria will decrease the likelihood of success.

These procedures are time-consuming, and there is no room for error. The patient's understanding and cooperation along with quality high-resolution imaging are essential.

The RF neurotomy of the cervical medial branch requires patience and skill. A single facet joint requires six discrete needle positions followed by production of the lesion. A reasonable amount of time (block time) must be allowed and it is not uncommon for the practitioner to

spend 30–45 minutes to perform a single facet joint denervation. It is recommended that no more than two to three facet joints be treated at one session. This is not a technique for patients suffering from bilateral multilevel cervical spondylytic related pain. Careful patient selection and technique are critical to ensure the best possible outcomes.

EFFICACY

Well-controlled studies indicate that RF neurotomy provides predictable long-term pain relief in patients with cervical facet joint pain secondary to trauma.[64] Lord showed the efficacy of cervical medial branch neurotomy to be 80%, with good-to-excellent relief over 1 year when using meticulous patient selection and technique. The most comprehensive study regarding efficacy of cervical medial branch neurotomy was done by Lord and associates in 1995. Among the seven series included, 37–89% of patients had greater than 40% relief for more than 2 months. Even though these studies were flawed due to technical and anatomical errors, their results yield encouraging evidence that medial branch radioneurotomy could be beneficial in well-selected patients.[93]

In 1996, the same authors compared a group of control patients to a neurotomy group. The neurotomy patients demonstrated significantly longer pain relief (median time 263 days) in comparison with the control group. The authors reported that 71% of patients undergoing denervation by RF neurotomy had a good response when those patients were chosen by double diagnostic blocks.[64]

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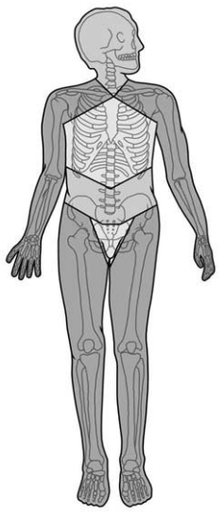
C H A P T E R



10

Myofascial Blocks of the Head and Neck

LELAND LOU AND P. PRITHVI RAJ

CERVICAL MUSCLE INJECTIONS

Neck and shoulder pain are among the most common musculoskeletal disorders, with neck pain being perhaps the second most common reason for workplace and motor-vehicle injury and disability claims, following low back pain.[1] In evaluating neck and shoulder pain, it is critical to identify the pathophysiological and anatomical substrates involved so that appropriate and effective therapies can be implemented rapidly.

HISTORY

Over the past two decades, Travell's call for improved recognition and treatment of myofascial trigger points has begun to be answered.[2] Relatively rapid changes in our knowledge have begun to accrue, particularly since the establishment of a standardized nomenclature and the usage of pain diagnostic tools.

These advances have led to a major exploration of the epidemiology, pathogenesis of, and therapeutic options for, pain states caused by muscular trigger points, which are now specifically known as *myofascial pain syndromes*.

The delineation of chronic benign intractable pain syndromes of the neck and back as being myofascial pain syndromes is of particular importance. These chronic benign intractable pain syndromes constitute a large proportion of the chronic pain population, who, because the diagnosis of chronic benign intractable pain syndromes carried considerable psychological overtones, were frequently exposed to psychologically based therapies, which usually had little effect on pain reduction. Thus the finding that these patients have a physical cause for their pain is of great significance as it allows for treatments that target the tissue and reflexes that cause and maintain their pain.

A Danish study of 1504 randomly selected people aged 30–60 years found that 37% of males and 65% of females had localized myofascial pain.[3] An American study of 100 male and 100 female Air Force personnel (average age 19) determined that 45% of males and 54% of females had focal neck muscle tenderness (latent trigger points).[3]

In another study, 269 female student nurses were examined. Forty-five percent had trigger^{o} points in the masseter and 35% in the trapezius, and 28% had myofascial pain at the time of examination. In a patient group's community pain medical center of 96 patients studied by a neurologist, 93% had at least part of their pain caused by myofascial trigger points; and in 74% of the patients, myofascial trigger points were considered to be the primary source of pain.[3]

In a comprehensive pain center study with 283 consecutive admissions to a comprehensive pain center, the diagnosis made independently by a neurosurgeon and a psychiatrist based on physical examination as described by Travell and Simons[2] assigned a primary organic diagnosis of myofascial pain in 85% of the cases.

ANATOMY

Bony Anatomy of Cervical Spine

The cervical portion of the human spine is composed of seven bony segments, C1–C7, with cartilaginous disks between each vertebral body. The neck supports the weight of the head and protects the nerves that travel from the brain down to the rest of the body. In addition, the neck is highly flexible and allows the head to turn and flex in all directions. From top to bottom the cervical spine is gently curved in a convex-forward position (Figure 10-1).

Surface Anatomy

In the middle line below the chin, the body of the hyoid bone can be felt; just below is the prominence of the thyroid cartilage, which is better marked in men than in women. Still lower, the cricoid cartilage is easily felt, while

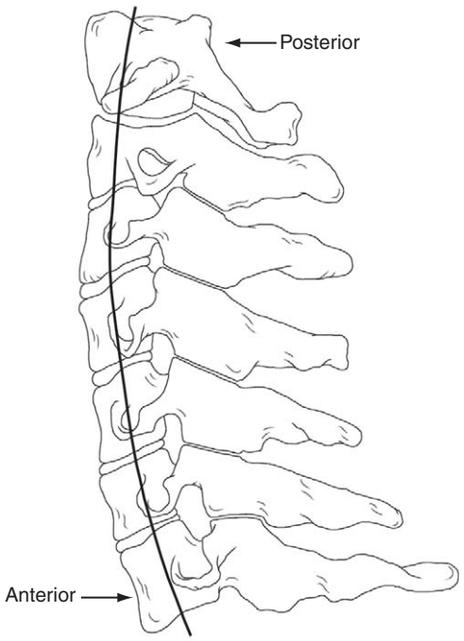


FIGURE 10–1
Bony anatomy of the cervical spine.

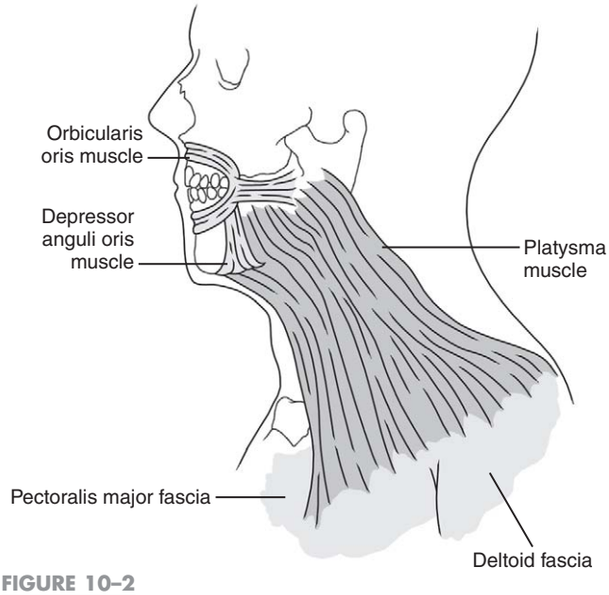


FIGURE 10–2
Origin and insertion of platysma muscle.

between this and the suprasternal notch, the trachea and isthmus of the thyroid gland can be made out. At the side the outline of the sternomastoid muscle is the most striking mark; it divides the anterior triangle of the neck from the posterior. The upper part of the former contains the submaxillary gland, also known as the parotid glands, which lie just below the posterior half of the body of the jaw. The line of the common and the external carotid arteries may be marked by joining the sternoclavicular articulation to the angle of the jaw.

The 11th or spinal accessory nerve corresponds to a line drawn from a point midway between the angle of the jaw and the mastoid process to the middle of the posterior border of the sternomastoid muscle, and then across the posterior triangle to the deep surface of the trapezius. The external jugular vein can usually be seen through the skin; it runs in a line drawn from the angle of the jaw to the middle of the clavicle, and close to it are some small lymph glands. The anterior jugular vein is smaller, and runs down and half an inch from the middle line of the neck. The clavicle or collarbone forms the lower limit of the neck, and laterally the outward slope of the neck to the shoulder is caused by the trapezius muscle.

Superficial Cervical Muscles

The superficial fascia of the neck is a thin lamina investing the *platysma*, and is hardly demonstrable as a separate membrane (Figure 10-2). The platysma is a broad sheet arising from the fascia covering the upper parts of the pectoralis major and deltoid. Its fibers cross the clavicle and proceed obliquely upward and medial-ward along the side of the neck. The anterior fibers interlace below and behind the symphysis menti, with the fibers of the muscle of the opposite side; the posterior fibers cross the mandible, some being inserted into the bone below the oblique line, and others into the skin and subcutaneous tissue of the lower part of the face. Many of these fibers blend with the muscles about the angle and lower part of the mouth. Sometimes fibers can be traced to the zygomaticus, or to the margin of the orbicularis oculi. Beneath the platysma, the external jugular vein descends from the angle of the mandible to the clavicle.

The *trapezius* is a flat, triangular muscle, covering the upper and back part of the neck and shoulders (Figure 10-3). It arises from the external occipital protuberance and the medial third of the superior nuchal line of the occipital bone, from the ligamentum nucha, the spinous process of the seventh cervical, and the spinous processes of all the thoracic vertebra, and from the corresponding portion of the supraspinal ligament. From this origin, the superior fibers proceed downward and lateral-ward, the inferior upward and lateral-ward, and the middle horizontally; the superior fibers are inserted into the posterior border of the lateral third of the clavicle; the middle fibers into the medial margin of the acromion, and into the superior lip of the posterior border of the spine of the scapula; the inferior fibers converge near the scapula, and end in an aponeurosis, which glides over the smooth triangular surface on the medial end of the spine, to be inserted into a tubercle at the apex of this smooth triangular surface. At its occipital origin, the trapezius is connected to the bone by a thin fibrous lamina, firmly adherent to the skin. At the middle it is connected to the spinous processes by a broad semielliptical aponeurosis, which reaches from the sixth

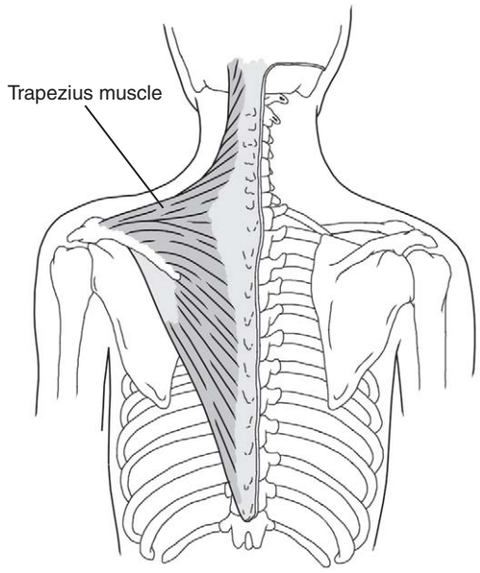


FIGURE 10–3
Orgin and insertion of trapezius muscle.

cervical to the third thoracic vertebra and forms, with that of the opposite muscle, a tendinous ellipse. The rest of the muscle arises by numerous short tendinous fibers. The two trapezius muscles together resemble a trapezium, or diamond-shaped quadrangle: two angles correspond to the shoulders; a third to the occipital protuberance; and the fourth to the spinous process of the 12th thoracic vertebra.

The attachments to the dorsal vertebra are often reduced and the lower ones are often wanting; the occipital attachment is often wanting; separation between cervical and dorsal portions is frequent. Extensive deficiencies and complete absence occur. The clavicular insertion of this muscle varies in extent; it sometimes reaches as far as the middle of the clavicle and occasionally may blend with the posterior edge of the sternocleidomastoideus or overlap it.

The *rhomboideus major* arises by tendinous fibers from the spinous processes of the second, third, fourth, and fifth thoracic vertebra, and the supraspinal ligament, and is inserted into a narrow tendinous arch, attached above to the lower part of the triangular surface at the root of the spine of the scapula, and below to the inferior angle, the arch being connected to the vertebral border by a thin membrane. When the arch extends, as it occasionally does, only a short distance, the muscular fibers are inserted directly into the scapula.

The *rhomboideus minor* arises from the lower part of the ligamentum nucha and from the spinous processes of the seventh cervical and first thoracic vertebra. It is inserted into the base of the triangular smooth surface at the root of the spine of the scapula and is usually separated from the rhomboideus major by a slight interval, but the adjacent margins of the two muscles are occasionally united.

The vertebral and scapular attachments of the two muscles vary in extent. A small slip from the scapula to the occipital bone close to the minor occasionally occurs, called the *rhomboideus occipitalis muscle*.

The *levator scapula* (levator anguli scapula) is situated at the back and side of the neck. It arises by tendinous slips from the transverse processes of the atlas and axis and from the posterior tubercles of the transverse processes of the third and fourth cervical vertebra. It is inserted into the vertebral border of the scapula, between the medial angle and the triangular smooth surface at the root of the spine.

The number of vertebral attachments varies; a slip may extend to the occipital or mastoid, to the trapezius, scalene or serratus anterior, or to the first or second rib. The muscle may be subdivided into several distinct parts from origin to insertion. The *levator clavicula* from the transverse processes of one or two upper cervical vertebra to the outer end of the clavicle corresponds to a muscle of lower animals, which more or less unites with the serratus anterior.

The rhomboidei are supplied by the dorsal scapular nerve from the fifth cervical. The levator scapula is innervated by the third and fourth cervical nerves, and frequently by a branch from the dorsal scapular.

The movements effected by the preceding muscles are numerous, as may be conceived from their extensive attachments. When the entire trapezius is in action, it retracts the scapula and braces back the shoulder; if the head is fixed, the upper part of the muscle will elevate the point of the shoulder, as in supporting weights; when the lower fibers contract they assist in depressing the scapula. The middle and lower fibers of the muscle rotate the scapula, causing elevation of the acromion. If the shoulders are fixed, the trapezii, acting together, will draw the head directly backward; or if only one acts, the head is drawn to the corresponding side.

Lateral Cervical Muscles

The fascia colli (deep cervical fascia) (Figure 10-4) lies under cover of the platysma and invests the neck. It also forms sheaths for the ca rotid vessels and for the structures situated in front of the vertebral column.

The investing portion of the fascia is attached behind to the ligamentum nucha and to the spinous process of the seventh cervical vertebra. It forms a thin investment to the trapezius, and at the anterior border of this muscle is continued forward as a rather loose areolar layer, covering the posterior triangle of the neck, to the posterior border of the sternomastoid muscle, where it begins to assume the appearance of a fascial membrane. Along the hinder edge of the sternocleidomastoideus it divides to enclose the muscle, and at the anterior margin again forms a single lamella, which covers the anterior triangle of the neck, and reaches forward to the middle line, where it is continuous with the corresponding part from the opposite side of the neck. In the middle line of the

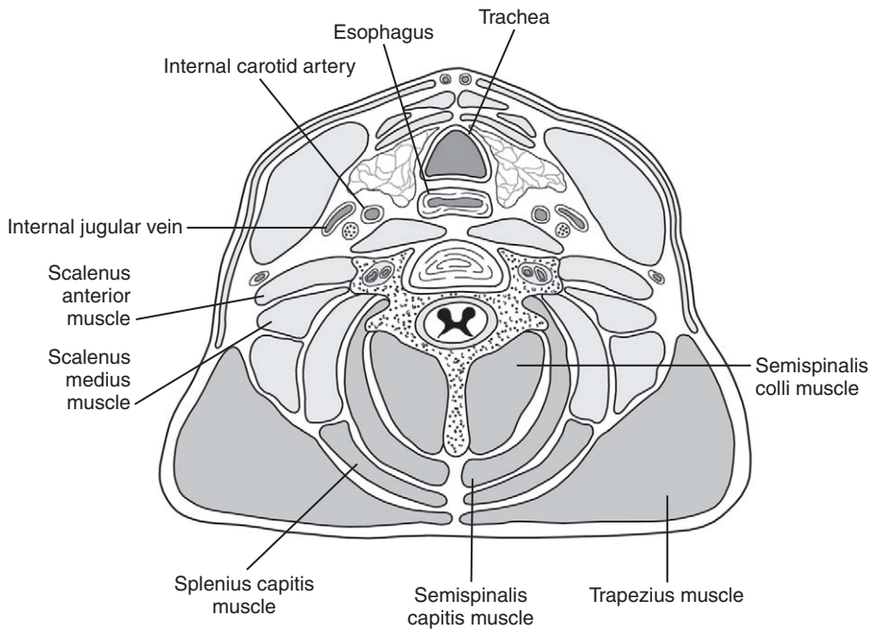


FIGURE 10–4
Transverse section of the neck showing the compartments of deep fascia of the neck.

neck, it is attached to the symphysis menti and the body of the hyoid bone.

The fascia is attached above to the superior nuchal line of the occipital, to the mastoid process of the temporal, and to the entire length of the inferior border of the body of the mandible. Opposite the angle of the mandible the fascia is very strong, and binds the anterior edge of the sternomastoid muscle firmly to that bone. Between the mandible and the mastoid process, it ensheathes the parotid gland; the layer that covers the gland extends upward under the name of the parotideomasseteric fascia and is fixed to the zygomatic arch. From the part that passes under the parotid gland, a strong band extends upward to the styloid process, forming the stylomandibular ligament. Two other bands may be defined: the sphenomandibular and the pterygospinous ligaments. The pterygospinous ligament stretches from the upper part of the posterior border of the lateral pterygoid plate to the spinous process of the sphenoid. It occasionally ossifies, and in such cases, between its upper border and the base of the skull, a foramen is formed that transmits the branches of the mandibular nerve to the muscles of mastication.

The fascia is attached below to the acromion, the clavicle, and the manubrium sterni. Some little distance above the last it splits into two layers, superficial and deep. The former is attached to the anterior border of the manubrium, and the latter to its posterior border and to the interclavicular ligament. Between these two layers is a slit-like interval, the suprasternal space; it contains a small quantity of areolar tissue, the lower portions of the anterior jugular veins and their transverse connecting branch, the sternal heads of the sternocleidomastoidei, and sometimes a lymph gland.

The fascia, which lines the deep surface of the sternomastoid muscle, gives off the following processes: a process envelops the tendon at the omohyoid, and binds it down to the sternum and first costal cartilage. A strong sheath, the carotid sheath, encloses the carotid artery, internal jugular vein, and vagus nerve. The prevertebral fascia extends medial-ward behind the carotid vessels, where it assists in forming their sheath, and passes in front of the prevertebral muscles. It forms the posterior limit of a fibrous compartment, which contains the larynx and trachea, the thyroid gland, and the pharynx and esophagus. The prevertebral fascia is fixed above to the base of the skull, and below is continued into the thorax in front of the longus colli muscles. Parallel to the carotid sheath and along its medial aspect, the prevertebral fascia gives off a thin lamina, the buccopharyngeal fascia, which closely invests the constrictor muscles of the pharynx. It continues forward from the constrictor pharyngis superior on to the buccinator. It is attached to the prevertebral layer by loose connective tissue only, and thus an easily distended space, the retropharyngeal space, is found between them. This space is limited above by the base of the skull, while below it extends behind the esophagus into the posterior mediastinal cavity of the thorax. The prevertebral fascia is prolonged downward and lateral-ward behind the carotid vessels and in front of the scaleni, and forms a sheath for the brachial nerves and subclavian vessels in the posterior triangle of the neck. It is continued under the clavicle as

the axillary sheath and is attached to the deep surface of the coracoclavicular fascia. Immediately above and behind the clavicle, an areolar space exists between the investing layer and the sheath of the subclavian vessels, and in this space are found the lower part of the external jugular vein, the descending clavicular nerves, the transverse scapular and transverse cervical vessels, and the inferior belly of the omohyoideus muscle. This space is limited below by the fusion of the coracoclavicular fascia with the anterior wall of the axillary sheath. The pretracheal fascia extends medially in front of the carotid vessels, and assists in forming the carotid sheath. It is continued behind the depressor muscles of the hyoid bone, and, after enveloping the thyroid gland, is prolonged in front of the trachea to meet the corresponding layer of the opposite side. Above, it is fixed to the hyoid bone, while below it is carried downward in front of the trachea and large vessels at the root of the neck, and ultimately blends with the fibrous pericardium. This layer is fused on either side with the prevertebral fascia, and with it completes the compartment containing the larynx and trachea, the thyroid gland, and the pharynx and esophagus.[4]

Sternomastoid Muscle

The sternomastoid muscle passes obliquely across the side of the neck, is thick and narrow at its central part, but broader and thinner at either end. It arises from the sternum and clavicle by two heads. The medial or sternal head is a rounded fasciculus, tendinous in front and fleshy behind, which arises from the upper part of the anterior surface of the manubrium sterni, and is directed upward, laterally, and backward. The lateral or clavicular head, composed of fleshy and aponeurotic fibers, arises from the superior border and anterior surface of the medial third of the clavicle. It is directed almost vertically upward. The two heads are separated from one another at their origins by a triangular interval but gradually blend below the middle of the neck into a thick, rounded muscle that is inserted by a strong tendon into the lateral surface of the mastoid process, from its apex to its superior border, and by a thin aponeurosis into the lateral half of the superior nuchal line of the occipital bone.

The sternocleidomastoid muscle varies much in the extent of its origin from the clavicle; in some cases, the clavicular head may be as narrow as the sternal, and in others it may be as much as 7.5 cm in breadth. When the clavicular origin is broad, it is occasionally subdivided into several slips separated by narrow intervals. More rarely, the adjoining margins of the sternocleidomastoideus and trapezius have been found in contact. The supraclavicularis muscle arises from the manubrium behind the sternocleidomastoideus and passes behind the sternocleidomastoideus to the upper surface of the clavicle (Figure 10-5).

The sternomastoid muscle divides the quadrilateral area of the side of the neck into two triangles, an anterior and a posterior. The boundaries of the anterior triangle

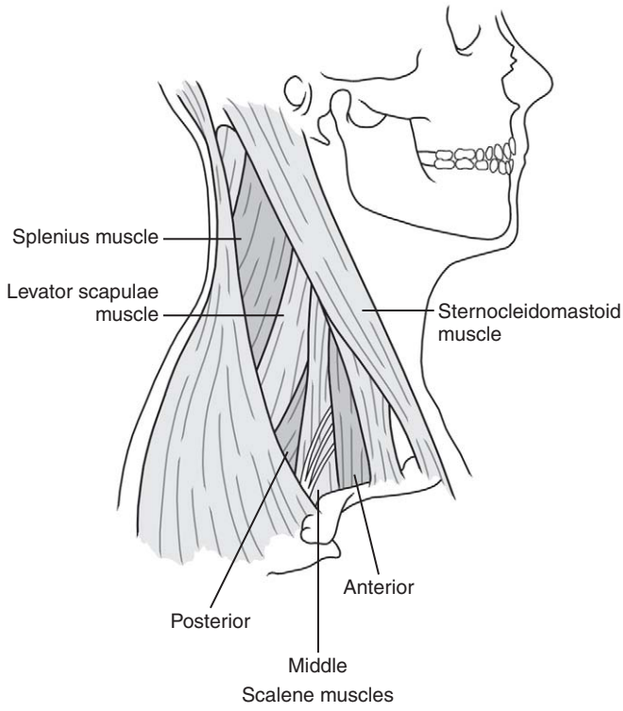


FIGURE 10–5

Shows posterior triangle of the neck with scalene, sternocleidomastoid, levator scapulae, and splenius capitis muscle.

follow: in front, the median line of the neck; above, the lower border of the body of the mandible, and an imaginary line drawn from the angle of the mandible to the sternocleidomastoideus; and behind, the anterior border of the sternocleidomastoideus. The apex of the triangle is at the upper border of the sternum. The boundaries of the posterior triangle are in front, the posterior border of the sternocleidomastoideus; below, the middle third of the clavicle; and behind, the anterior margin of the trapezius. The apex corresponds with the meeting of the sternocleidomastoideus and trapezius on the occipital bone.

The sternomastoid muscle is supplied by the accessory nerve and branches from the anterior divisions of the second and third cervical nerves.

When only one sternomastoid muscle acts, it draws the head toward the shoulder of the same side, assisted by the splenius and the obliquus capitis inferior of the opposite side. At the same time, it rotates the head so as to carry the face toward the opposite side. Acting together from their sternoclavicular attachments, the muscles will flex the cervical part of the vertebral column. If the head is fixed, the two muscles assist in elevating the thorax in forced inspiration.

Deep Muscles of the Neck

The *longus colli* is situated on the anterior surface of the vertebral column, between the atlas and the third thoracic vertebra (Figure 10-6). It is broad in the middle, narrow, and pointed at either end, and consists of three

portions—superior oblique, inferior oblique, and vertical. The superior oblique portion arises from the anterior tubercles of the transverse processes of the third, fourth, and fifth cervical vertebra and, ascending obliquely with a medial inclination, is inserted by a narrow tendon into the tubercle on the anterior arch of the atlas. The inferior oblique portion, the smallest part of the muscle, arises from the front of the bodies of the first two or three thoracic vertebra; and, ascending obliquely in a lateral direction, is inserted into the anterior tubercles of the transverse processes of the fifth and sixth cervical vertebra. The vertical portion arises below, from the front of the bodies of the upper three thoracic and lower three cervical vertebras, and is inserted into the front of the bodies of the second, third, and fourth cervical vertebra.

The *longus capitis* (rectus capitis anticus major), broad and thick above and narrow below, arises by four tendinous slips, from the anterior tubercles of the transverse processes of the third, fourth, fifth, and sixth cervical vertebra, and ascends, converging toward its fellow of the opposite side, to be inserted into the inferior surface of the basilar part of the occipital bone.

The *rectus capitis anterior* (rectus capitis anticus minor) is a short, flat muscle, situated immediately behind the upper part of the longus capitis. It arises from the anterior surface of the lateral mass of the atlas, and from the root of its transverse process, and passing obliquely upward and medial-ward, is inserted into the inferior surface of the basilar part of the occipital bone immediately in front of the foramen magnum.

The *rectus capitis lateralis*, a short, flat muscle, arises from the upper surface of the transverse process of the atlas, and is inserted into the under-surface of the jugular process of the occipital bone.

The rectus capitis anterior and the rectus capitis lateralis are supplied from the loop between the first and second cervical nerves; the longus capitis, by branches from the first, second, and third cervical; and the longus colli, by branches from the second to the seventh cervical nerves.

The longus capitis and rectus capitis anterior are the direct antagonists of the muscles at the back of the neck, serving to restore the head to its natural position after it has been drawn backward. These muscles also flex the head, and from their obliquity, rotate it, so as to turn the face to one or the other side. The rectus lateralis, acting on one side, bends the head laterally. The longus colli flexes and slightly rotates the cervical portion of the vertebral column.

Lateral Vertebral Muscles

The *scalenus anterior* lies deeply at the side of the neck, behind the sternocleidomastoideus (Figure 10-7). It arises from the anterior tubercles of the transverse processes of the third, fourth, fifth, and sixth cervical vertebra, and descending, almost vertically, is inserted by a narrow, flat tendon into the scalene tubercle on the inner border of the first rib, and into the ridge on the upper surface of the rib in front of the subclavian groove.

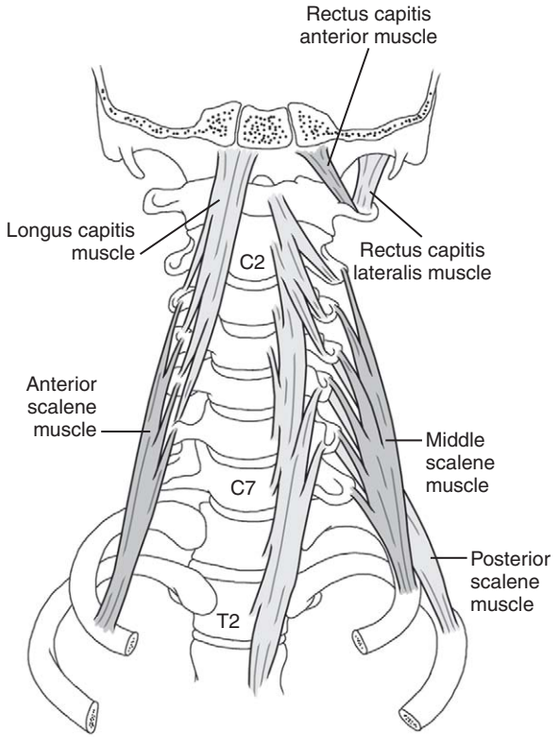


FIGURE 10–6
Deep muscles of the neck shown anteriorly.

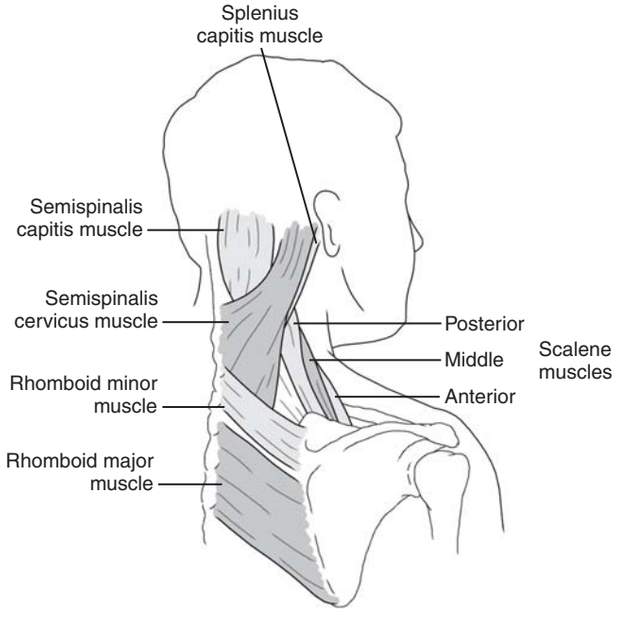


FIGURE 10–7
Origin and insertion of lateral vertebral muscles of the neck.

The *scalenus medius*, the largest and longest of the three scaleni, arises from the posterior tubercles of the transverse processes of the lower six cervical vertebra, and descending along the side of the vertebral column, is inserted by a broad attachment into the upper surface of the first rib, between the tubercle and the subclavian groove.

The *scalenus posterior*, the smallest and most deeply seated of the three scaleni, arises, by two or three separate tendons, from the posterior tubercles of the transverse processes of the lower two or three cervical vertebra, and is inserted by a thin tendon into the outer surface of the second rib, behind the attachment of the serratus anterior. It is occasionally blended with the scalenus medius.

The scaleni muscles vary considerably in their attachments and in the arrangement of their fibers. A slip from the scalenus anticus may pass behind the subclavian artery. The scalenus posticus may be absent or extend to the third rib. The scalenus pleuralis muscle extends from the transverse process of the seventh cervical vertebra to the fascia supporting the dome of the pleura and inner border of first rib.

The scaleni are supplied by branches from the second to the seventh cervical nerves.

When the scaleni act from above, they elevate the first and second ribs and are, therefore, inspiratory muscles. Acting from below, they bend the vertebral column to one or other side; if the muscles of both sides act, the vertebral column is slightly flexed.

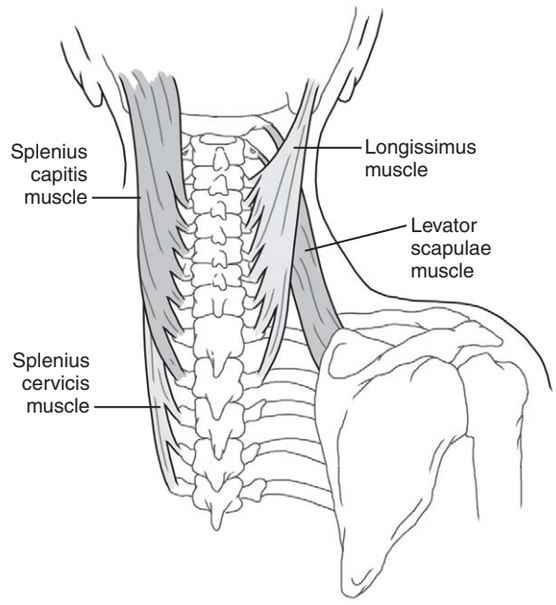


FIGURE 10–8

Intrinsic deep muscles of posterior part of the neck. Identifying the location and course of the splenius capitis, longissimus, splenius cervicis, and levator scapulae.

Intrinsic Muscles of the Back of the Neck

The *splenius muscles* are bandage-like muscles (G. *sphenion*, bandage) and are applied to the sides and back of the neck, somewhat like spiral bandages (Figure 10-8). They ascend from the medial plane of the neck and the transverse processes of the superior cervical vertebrae to the base of the skull. Each muscle is divided into a cranial portion, splenius capitis, and a cervical portion, the splenius cervicis. They originate in the inferior half of the ligamentum nuchae and the spinous processes of T1 to T6 vertebrae. The splenius capitis inserts into the lateral aspect of the mastoid process and the lateral third of the superior nuchal line of the occipital bone (deep to the sternocleidomastoid muscle). The splenius cervicis insets into the posterior tubercles of the transverse processes of C1 to C4 vertebrae (posterior to the levator scapulae muscle). These muscles are innervated through dorsal rami of the inferior cervical nerves. Acting alone, the splenius muscles laterally flex and rotate the head and neck to the same side. Acting together, they extend the head and neck.

Intermediate Layer of Deep Back Muscles

The longissimus muscle is the intermediate column of the erector spinae muscle. It arises from the common origin and is attached to the transverse processes of the thoracic and cervical vertebrae, and the mastoid process of the temporal bone of the skull. The muscle thus has a herringbone appearance.

The longissimus can also be divided into three parts according to the regions it traverses. The longissimus thoracis inserts into the tips of the transverse processes of all of the thoracic vertebrae, and into the tubercles of the inferior nine to ten ribs; the longissimus cervicis extends from the superior thoracic transverse processes to the cervical transverse processes; and the longissimus capitis arises in common with the cervical part and attaches to the mastoid process of the temporal bone.

The *levator scapulae* originates from the transverse processes of C1–C4, is inserted into the superior angle of the scapula. Its function is to elevate the scapula, and in extension, laterally flexes the head. It is innervated by cervical plexus C3, C4, and dorsal scapula nerve, C5.

Other Significant Muscles for Cervical Muscle Injection

The rhomboid major originates from spinous processes of T2-T5 and the supraspinous ligament (Figure 10-9). It inserts into the medial scapula from the scapular spine to the inferior angle. Its action is to retract the scapula. It is innervated by the dorsal scapula nerve.

The *rhomboid minor* originates from the spinous processes of C7 and T1, ligamentum nuchae, and supraspinous ligament. It is inserted into the medial margin of the scapula at the medial angle. Its action is to retract the scapula. It is innervated by C5 and occasionally also by C4.

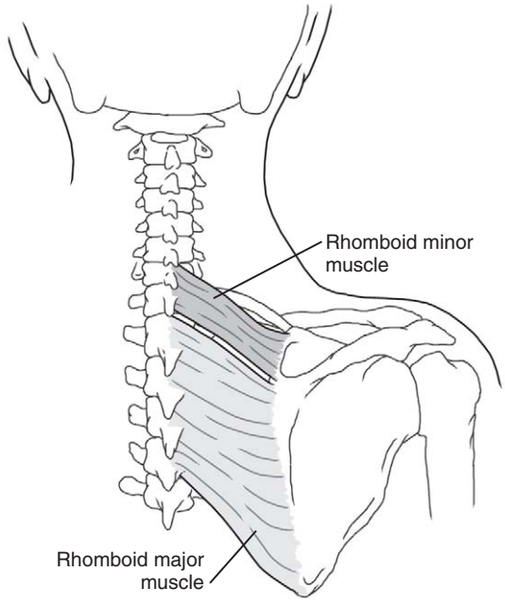


FIGURE 10–9
The figure shows the origin and insertion of rhomboids major and minor.

INDICATIONS FOR CERVICAL MUSCLE INJECTIONS

Myofascial pain of the neck caused by:
Advancing age with arthritis
Overuse of muscles
Trauma to neck
Pain secondary to:
Dystonia
Spasticity
Whiplash injury
Spondylosis
Cervical facet syndrome
Torticollis

CONTRAINDICATIONS

Generalized and local infection
Coagulopathy
Anatomic anomalies secondary to:
Cancer
Surgery

EQUIPMENT

- 25- or 22-gauge 1-1/2-inch needle
- 10-ml syringe
- Iohexal (Omnipaque)
- Portable EMG
- Necessary needles

DRUGS

For diagnostic and acute situations:
Local anesthetic (xylocaine 1% or bupivacaine 0.125% or 2%, or ropivicaine 0.5%) or
Long-acting steroid agents
Dextomethasone or equivalent or
Depomedrol or equivalent or
Triamcenolone acetate
For neurolytic procedures
3% phenol or
50% alcohol
Other drugs that prolong action
Botulinum toxin A
Botulinum toxin B

MYOFASCIAL PAIN SYNDROMES

CLINICAL FEATURES

There is history of spontaneous pain associated with acute overload or chronic overuse of the muscle. The mildest symptoms are caused by latent myofascial trigger points that cause no pain but cause some degree of functional disability. More severe involvement results in pain related to the position or movement of the muscle. The most severe level involves pain at rest and is considered to be caused by active trigger points.

Assessment

Subjective
Visual Analogue Scale (VAS)
McGill Pain Questionnaire
Pain diagram
Objective
Pressure threshold algometry
Differential local anesthetic blocks
Thermography, EMG, etc.

Pathophysiology

Pain attributed to muscle and its surrounding fascia has been termed *myofascial pain*. The diagnosis of this syndrome is clinical, with no confirmatory laboratory tests available. Thus, myofascial pain in any location is characterized on examination by the presence of trigger points located in skeletal muscle. In the cervical spine, the muscles most often implicated in myofascial pain are the trapezius, levator scapulae, rhomboids, supraspinatus, and infraspinatus. A trigger point is defined as a hyperirritable area located in a palpable taut band of muscle fibers. According to Hong and Simons's[5] recent review on the pathophysiology and electrophysiologic mechanisms of trigger points, the following observations help to define them further:

- Trigger points are known to elicit local pain and/or referred pain in a specific recognizable distribution.
- Palpation in a rapid fashion (i.e., snapping palpation) may elicit a local twitch response (LTR), a brisk contraction of the muscle fibers in or around the taut band. The LTR also can be elicited by rapid insertion of a needle into the trigger point.
- Restricted range of motion and increased sensitivity to stretch of muscle fibers in a taut band are frequently noted.
- The muscle with a trigger point may be weak because of pain. Usually, no atrophic change is observed.
- Patients with trigger points may have associated localized autonomic phenomena (e.g., vasoconstriction, pilomotor response, ptosis, hypersecretion).
- An active myofascial trigger point is a site marked by generation of spontaneous pain or pain in response to movement. This phenomenon is in contrast to the case of latent trigger points, which may not produce pain until they are compressed.

In the United States, myofascial pain is thought to occur commonly in the general population. As many as 21% of patients seen in general orthopedic clinics have myofascial pain. Of patients seen at specialty pain management centers, 85–93% have a myofascial pain component. No studies clarify whether racial/ethnic differences exist in frequency of cervical myofascial pain. While fibromyalgia occurs more commonly in women than in men, cervical myofascial pain occurs in both sexes, also with predominance among women. Myofascial pain seems to occur more frequently with increasing age until mid-life. Incidence declines gradually after middle age.

SYNDROMES THAT CAUSE CERVICAL MYOFASCIAL PAIN

CERVICAL DYSTONIA

Cervical dystonia is the most common focal dystonia. There are intermittent or continuous spasms of the sternomastoid muscle, trapezius, and other cervical muscles, usually more prominent on one side than on the other. About 70% of patients with cervical dystonia report pain as a principal complaint.

SPASTIC DISEASE STATES

Two other subgroups of pain that patients detailed in this review are those in whom there is a known cause of spasticity, tracing its origin to either the peripheral or central nervous systems (CNS). CNS dysfunction may lead to the sometimes-painful spasticity in certain patients with cerebral palsy, multiple sclerosis, stroke, and traumatic brain injury, while peripheral lesions may cause myofascial pain syndrome.

Patients experiencing acute or longstanding insults and degenerative processes of the CNS may display a wide variety of signs that together constitute the upper motor neuron syndrome. Spasticity, a velocity-dependent increase in muscle tone characterized by hyperactive stretch reflexes, is but one sign. Additional positive and negative signs characterize the syndrome. Among those classed as positive are such signs as hyperactive tendon reflexes, increased resistance to passive movement, flexed posture in the arm and extension in the leg, excessive contraction of antagonistic muscles, and stereotypic movement synergies; negative signs include weakness, lack of dexterity, and paresis.[6]

Until recently, spasticity was viewed as a consequence of overactive muscle spindles or fusimotor fibers, resulting from disruption of descending inhibitory tracts, the corticospinal and corticobulbar tracts, and sensory afferents.[6] O'Brien[7] suggests that this view is no longer entirely accurate. Spastic paresis or spastic dystonia may be better understood as an imbalance of inhibition and excitation occurring at the motor neuron level of the spinal cord, not unlike focal hypertonia with dystonic features.[8,9] The most fundamental component of this sequence is the abnormal intraspinal response to sensory input. Modulation of local spinal cord activity occurs via the descending pathways, such as the rubrospinal tract.[10,11] In general, positive symptoms such as hyper-reflexa are caused by the disinhibition of local cord excitatory circuits. Negative symptoms, such as paresis or loss of dexterity, reflect dysfunction of corticospinal pathways. The positive signs of spasticity interfere with the activities of daily living, can cause fractures or contractures, increase the frequency of pressure sores, and are often associated with pain.[12] Though they can interfere with rehabilitation, they are also more amenable to clinical intervention than are negative signs.

Spasticity as described earlier is a prominent clinical feature of several important afflictions of the CNS, including, stroke, cerebral palsy, multiple sclerosis, Parkinson's disease, and traumatic brain injury. For chronic or degenerative states, the management of spasticity is an ongoing task, which is best begun with conservative measures and accelerated as needed.[13–15] Initially, physical therapeutic modalities should be tried, such as avoidance of noxious stimuli, passive movement exercises, thermal agents, vibratory treatment, and serial inhibitive casting.[16] Oral medications can be tried in conjunction with physical measures or alone, but neither neural depressants (e.g., oral or intrathecal baclofen, benzodiazepines, clonidine, and tizanidine) nor muscle relaxants (e.g., dantrolene) have proved very satisfactory by reason of limited efficacy and intolerable side effects.[17–19] For the more seriously affected or unresponsive patient, invasive procedures such as phenol and alcohol nerve blocks[20,21] spinal cord stimulation, rhizotomies, and intrathecal baclofen administration have been implemented.[12,22–26]

The cost of prolonged care and relative lack of benefit of conservative management have lead to the suggestion that botulinum toxins be used in managing spasticity. Botulinum toxins can be used therapeutically to produce a reversible, partial, chemical denervation when injected directly into a contracted muscle. Because of its potentially pronounced paralytic action, botulinum toxins can be as effective as certain surgeries presently in use for managing spasticity, yet it has the advantage of being reversible and generally repeatable as needed, in accord with the fluctuating state of the patient.

WHIPLASH

Whiplash is defined as a medical condition where the soft tissues of the neck have been injured after a sudden jerking (whipping) of the head, which results in a strain on the muscles and ligaments of the neck when it is moved beyond the normal range of motion, causing a sprain-type injury.[27] Whiplash is a description of the movement that causes injury but has become synonymous with the soft tissue injury which occurs.[28]

Cause

A whiplash injury can be the result of impulsive stretching of the spine, often the result of a rear-end collision between cars or trucks. When a vehicle stops suddenly or is hit from behind and the occupants are wearing seat belts their bodies are prevented from being thrown forward, but their heads can snap forward and then back again causing a whiplash injury.[27] The Insurance Institute for Highway Safety defines whiplash as "a range of neck injuries that are related to sudden distortions of the neck. It takes about 100 milliseconds for an occupant's body to catch up to the car when it is hit, and it is during this time that the damage occurs. Whiplash injury can result from a rear-impact collision, front-impact collision, lateral (side) impact or rollover."[28]

Whiplash can be caused by any motion similar to a rear-end collision in a motor vehicle, such as may take place on a roller coaster or other rides at an amusement park, sports injuries such as skiing accidents, other modes of transportation such as airplane travel, or from being hit or shaken.[29] Shaken baby syndrome can result in a whiplash injury.[27]

Symptoms

Symptoms reported by sufferers include ringing or whistling in the ear, headache, deafness, memory loss, dizziness, depression, jaw joint pain, and difficulty in swallowing. Symptoms can appear directly after the crash or hours or days afterwards.[28]

Diagnosis

Reliably diagnosing a whiplash injury or disorder is not difficult for a trained doctor. If a patient cannot achieve full motion or has excessive range of motion, the probable ultimate cause is the whiplash motion.

The Québec Task Force (QTF) was sponsored by Société d'assurance automobile du Québec (SAAQ), the public auto insurer in the province of Quebec, Canada. The QTF submitted a report on whiplash-associated disorders (WADs) in 1995, which made specific recommendations on prevention, diagnosis, and treatment of WADs. The recommendations have become the basis for *Guideline on the Management of Claims Involving Whiplash-Associated Disorders*,[30] a guide to classifying WADs and guidelines on managing the disorder. The full report titled "Redefining 'Whiplash'," was published in the April 15, 1995 issue of *Spine*.

Severity Grades of Whiplash Injury

Four grades of whiplash were defined by the Quebec Task Force on WADs:

- Grade 1: complaints of neck pain, stiffness, or tenderness only, but no physical signs are noted by the examining physician.
- Grade 2: neck complaints and the examining physician finds decreased range of motion and point tenderness in the neck.
- Grade 3: decreased range of motion plus neurological signs such as decreased deep tendon reflexes, weakness, insomnia, and sensory deficits.
- Grade 4: neck complaints and fracture, dislocation, or injury to the spinal cord.[30]

The consequences of whiplash range from mild pain for a few days, to severe disability caused by restricted head movement or of the cervical spine, sometimes with persistent pain.

CERVICAL SPONDYLOSIS

Cervical spondylosis is a chronic degenerative condition of the cervical spine that affects the vertebral bodies and intervertebral disks of the neck (e.g., disk herniation, spur formation), as well as the contents of the spinal canal (nerve roots and/or spinal cord). Some authors also include the degenerative changes in the facet joints, longitudinal ligaments, and ligamentum flavum.

Spondylosis progresses with age and often develops at multiple interspaces. Chronic cervical degeneration is the most common cause of progressive spinal cord and nerve root compression. Spondylotic changes can result in spinal canal, lateral recess, and foraminal stenosis. Spinal canal stenosis can result in myelopathy, whereas the latter two can cause radiculopathy.

Pathophysiology

Intervertebral disks lose hydration and elasticity with age, and these losses lead to cracks and fissures. The surrounding ligaments also lose their elastic properties and develop traction spurs. The disk subsequently collapses as a result of

biomechanical incompetence, causing the annulus to bulge outward. As the disk space narrows, the annulus bulges, and the facets override. This change, in turn, increases motion at that spinal segment and further hastens the damage to the disk. Annulus fissures and herniation may occur. Acute disk herniation may complicate chronic spondylotic changes.

As the annulus bulges, the cross-sectional area of the canal is narrowed. This effect may be accentuated by hypertrophy of the facet joints (posteriorly) and the ligamentum flavum, which becomes thick with age. Neck extension causes the ligaments to fold inward, reducing the anteroposterior diameter of the spinal canal.

As disk degeneration occurs, the uncinate process overrides and hypertrophies, compromising the ventrolateral portion of the foramen. Likewise, facet hypertrophy decreases the dorsolateral aspect of the foramen. This change contributes to the radiculopathy associated with cervical spondylosis. Marginal osteophytes begin to develop. Additional stresses such as trauma or long-term heavy use may exacerbate this process. These osteophytes stabilize the vertebral bodies adjacent to the level of the degenerating disk and increase the weight-bearing surface of the vertebral endplates. The result is decreased effective force on each of these structures.

Degeneration of the joint surfaces and ligaments decreases motion and can act as a limiting mechanism against further deterioration. Thickening and ossification of the posterior longitudinal ligament also decrease the diameter of the canal.

The blood supply of the spinal cord is an important anatomic factor in the pathophysiology. Radicular arteries in the dural sleeves tolerate compression and repetitive minor trauma poorly. The spinal cord and canal size are also factors. A congenitally narrow canal does not necessarily predispose a person to myelopathy, but symptomatic disease rarely develops in individuals with canals larger than 13 mm.

Etiology

In the United States, cervical spondylosis is a common condition that is estimated to account for 2% of all hospital admissions. It is the most common cause of spinal cord dysfunction in patients older than 55 years. On the basis of radiologic findings, 90% of men older than 50 years and 90% of women older than 60 years have evidence of degenerative changes in the cervical spine.[31]

Internationally, investigators in a study involving Ghanaians reported, "out of 225 patients who carried loads on their head, 143 (63.6%) had cervical spondylosis, and of the 80 people who did not carry load on their head, 29 (36%) had cervical spondylosis."[32]

The course of cervical spondylosis may be slow and prolonged, and patients may either remain asymptomatic or have mild cervical pain. Long periods of non progressive disability are typical, and in a few cases, the patient's condition progressively deteriorates. Morbidity ranges from chronic neck pain, radicular pain, diminished cervical range of motion, headache, myelopathy leading to weakness, and impaired fine motor coordination to quadriparesis and/or sphincteric dysfunction (e.g., difficulty with bowel or bladder control) in advanced cases. The patient may be eventually chair-bound or bedridden.

No apparent correlation between race/ethnicity and cervical spondylosis exists. Both genders are affected equally. Cervical spondylosis usually starts earlier in men than in women. Symptoms of cervical spondylosis may appear in those as young as 30 years and are most commonly in those aged 40–60 years. Radiologic spondylotic changes increase with patient age, as 70% of asymptomatic persons older than 70 years have degenerative cervical spine changes in one form or another. Cervical spondylosis usually starts earlier in men than in women. When cervical spondylosis develops in a young individual, it is almost always secondary to a predisposing abnormality in one of the joints between the cervical vertebrae, probably as the result of previous mild trauma.

Cervical Pain Due to Spondylosis

- Chronic suboccipital headache may be present. Mechanisms include direct nerve compression; degenerative disk, joint, or ligamentous lesions; and segmental instability.
- Pain can be perceived locally, or it may radiate to the occiput, shoulder, scapula, or arm.
- The pain, which is worse when the patient is in certain positions, can interfere with sleep.

EXAMINATION OF PATIENT WITH CERVICAL MYOFASCIAL PAIN

Typical findings reported by the patient with cervical myofascial pain may include the following:

- The patient may present with a history of acute trauma associated with persistent muscular pain. In contrast, myofascial pain also manifests insidiously, without a clear antecedent accident or injury. It may be associated with repetitive tasks, poor posture, stress, or cold weather.
- Cervical spine range of motion is often limited and painful.
- The patient may describe a lumpiness or painful bump in the trapezius or cervical paraspinal muscles.
- Massage is often helpful, as is superficial heat.
- The patient's sleep may be interrupted because of pain. The cervical rotation required for driving is difficult to achieve.
- The patient may describe pain radiating into the upper extremities, accompanied by numbness and tingling, which makes discrimination from radiculopathy or peripheral nerve impingement difficult.

- Dizziness or nausea may be a part of the symptomatology.
- The patient experiences typical patterns of radiating pain referred from trigger points.

On examination common findings noted upon physical examination may include the following:

- Patients with cervical myofascial pain often present with poor posture. They exhibit rounded shoulders and protracted scapulae.
- Trigger points frequently are noted in the trapezius, supraspinatus, infraspinatus, rhomboids, and levator scapulae muscles.
- The palpable taut band is noted in the skeletal muscle or surrounding fascia. A lateral trigger point often can be reproduced with palpation of the area.
- Cervical spine range of motion is limited with pain reproduced in positions that stretch the affected muscle.
- While the patient may complain of weakness, normal strength in the upper extremities is noted on physical examination.
- Sensation typically is normal when tested formally. No long tract signs are observed on examination.

Causes

Cervical myofascial pain is thought to occur following either overuse or trauma to the muscles that support the shoulders and neck. Common scenarios are that the patient recently was involved in a motor vehicle accident or has performed repetitive upper extremity activities. Trapezoidal myofascial pain commonly occurs when a person with a desk job does not have appropriate armrests or must type on a keyboard that is too high. Other issues that may play a role in the clinical picture include endocrine dysfunction, chronic infections, nutritional deficiencies, poor posture, and psychological stress.

Laboratory Studies

Myofascial pain traditionally does not produce abnormalities in the results of the patient's lab work. Simons and colleagues[33] describe a study looking at lactate dehydrogenase (LDH) isoenzymes. A shift was noted in distribution of the isoenzymes with higher levels of LDH1 and LDH2, while the total LDH remained within normal limits. In clinical practice, myofascial pain is diagnosed by way of a thorough physical examination in conjunction with an adequate medical history.

Depending on the clinical presentation, it may be reasonable to check for indicators of inflammation, assess thyroid function, and perform a basic metabolic panel to rule out a concomitant medical illness.

Imaging Studies

Imaging studies often reveal nonspecific change only and typically are not helpful in making the diagnosis of cervical myofascial pain; however, x-rays and a cervical spine magnetic resonance imaging (MRI) may be helpful in ruling out other pathology that may be present at the same time.

Other Tests

Several research articles have attempted to identify changes on electromyograms/nerve conduction velocity studies that may be unique to patients with myofascial pain. The research has been somewhat contradictory, with some studies finding no real electromyographic activity and others finding nonspecific electrical activity. Studies by Simons[33] and by Hubbard and Berkoff[34] describe low-amplitude action potentials recorded at the region of the myofascial trigger point. Spontaneous electrical activity apparently can be detected using high-sensitivity recordings at the site of the trigger point. The spontaneous electrical activity may be a type of endplate potential.

TREATMENT OBJECTIVES

Effective treatment and management of primary and secondary myofascial pain syndrome in the neck should (1) strive to relieve pain, (2) return normal muscular function and range of motion, and (3) eliminate perpetuating factors.[35,36] This can be accomplished through a multidisciplinary approach to pain management, especially if the myofascial pain is chronic in nature.[37,38] For this reason, the physician should maintain constant contact with other specialists throughout the management process, such as anesthesiologists, physical therapists, and clinical psychologists.[36] Initial pain treatment, however, should focus on interrupting the reflexive pain cycle created by myofascial trigger points.[35,36,38] This is accomplished by eliminating the myofascial trigger points through one of several modalities, including trigger-point injection, "stretch and spray," dry needling (acupuncture), massage/trigger-point pressure release, exercise, and pharmacological agents.[36–49]

PREPARATION OF PATIENT FOR INJECTION

The patient should be reassured and communicated with throughout the procedure to promote relaxation. The clinician will already have located the myofascial trigger points during the diagnostic phase and marked them with a skin pencil. The presence of multiple myofascial trigger points is common, and the physician should inject them in order beginning with the most symptomatic. The skin around each insertion site is washed and sterilized to avoid infection. The myofascial trigger point is then confirmed through one of the three palpation techniques, while wearing surgical gloves to retain

sterility. For flat palpation, the myofascial trigger point can be pinned for injection midway down the fingertips to prevent movement during the injection. Deep palpation, commonly used for identifying myofascial trigger points in cervical sternomastoid muscle, is used to identify and note the area of maximum tenderness. The injection will take place in the exact location of finger placement and be directed to the point of maximum tenderness. The injection site may be anesthetized with vapocoolant or a pre-injection block to prevent discomfort and muscle tension. The needle is then inserted until it encounters the myofascial trigger point. Local and/or referred pain may be experienced, in addition to a local twitch response. Once located, the myofascial trigger point is then injected with local anesthetic. Injections should consist of 0.5% procaine, 0.25–0.5% lidocaine, or 0.125–0.25% bupivacaine. Epinephrine should *never* be used to treat myofascial trigger points. This technique is repeated until all identified myofascial trigger points in the affected muscle have been treated.

IDENTIFICATION OF INJECTION SITES IN CERVICAL MUSCLES

The most important muscles in the neck are identified with the trigger points (Figure 10-10A and B). Other options of identifying the site of injection: Even though trigger point injection is common in clinical practice for myofascial pain and much literature is available to identify these trigger points,[38] some clinicians prefer to identify the required muscle by portable EMG (Figure 10-11). The conformation is obtained by identifying the typical sound of entering the muscle in spasm.

With myofascial pain syndrome, most investigators have injected active trigger points directly or used a grid pattern (Lang's method)[50] around them to get more diffuse spread through the involved muscle. Scalene injections under fluoroscopic guidance can be used with success to target adjacent muscles. Some clinicians routinely identify the muscles for injection by real-time CAT scan.

DRUGS USED FOR MYOFASCIAL INJECTIONS

Diagnostic
Local anesthetic and/or long-acting steroids
For prolonged therapeutic action
Botulinum toxins A or B

TECHNIQUE

Several treatment options for cervical myofascial pain are discussed in the literature. Trigger-point injection probably is one of the most accepted means of treating myofascial pain besides physical therapy and exercise. Injection is performed most commonly with local anesthetic, although dry needling has been shown to be equally effective.

Palpate the trigger point in the taut band, and place the muscle in a slightly stretched position to prevent it from moving. Hold the trigger point between two fingers while injecting with the other hand (Figure 10-12). Then redirect the needle in the area to ensure widespread infiltration of the anesthetic. Instruct the patient to be aggressive about compliance with stretching protocols, as they increase effectiveness of the injection. Production of a local twitch response helps confirm the diagnosis. Hong and Simons's[5] article describes a fast-in fast-out method as more successful in eliciting the local twitch response. This approach, therefore, generally is the most helpful technique for reducing myofascial pain. Muscle identification during drug injection is based on the clinician's anatomical knowledge and surface landmarks.

DOSING CONSIDERATIONS OF BOTULINUM TOXIN

Once the decision is made to consider botulinum toxin for the treatment of myofascial pain syndrome or headache, the key questions are which patient will best benefit from this therapy, what dose to administer (in what concentration and in what diluents), and how to do it. Unfortunately, the answers to these questions are still uncertain. Until more studies are performed, only general guidelines are available from the currently available literature.

As with any new therapy, especially one that is expensive, it makes sense to use botulinum toxins only in more refractory cases until the treatment becomes established and pharmacoeconomics data are supportive. In myofascial pain syndrome, the potential for significant reduction in medication use and complete resolution of symptoms in a substantial portion of refractory cases is a strong argument in support of botulinum toxin use. In both conditions, quality of life and functional improvement can be measurably improved.

Cervical and VIIth nerve dystonia data have been used as a starting point for botulinum toxin dose calculations with adjustments depending upon the size of the muscle and degree of spasm. Clinical experience with botulinum $toxin_A$ would seem to support this extrapolation to myofascial pain syndrome/cervical pain, but with botulinum $toxin_B$ it will be important to be cautious and start at a maximum of 2500 to 5000 units and move upward depending on clinical response until data from current studies provides dose–response information.

The total maximum dose per visit for botulinum toxin (Botox®) typically should not exceed the 300–400 unit range (although many have gone as high as 600–700 units safely for numerous involved muscles as in diffuse spasticity/dystonia), and intervals between doses should be no more frequent than every 3 months. Following these general guidelines will reduce adverse events (primarily

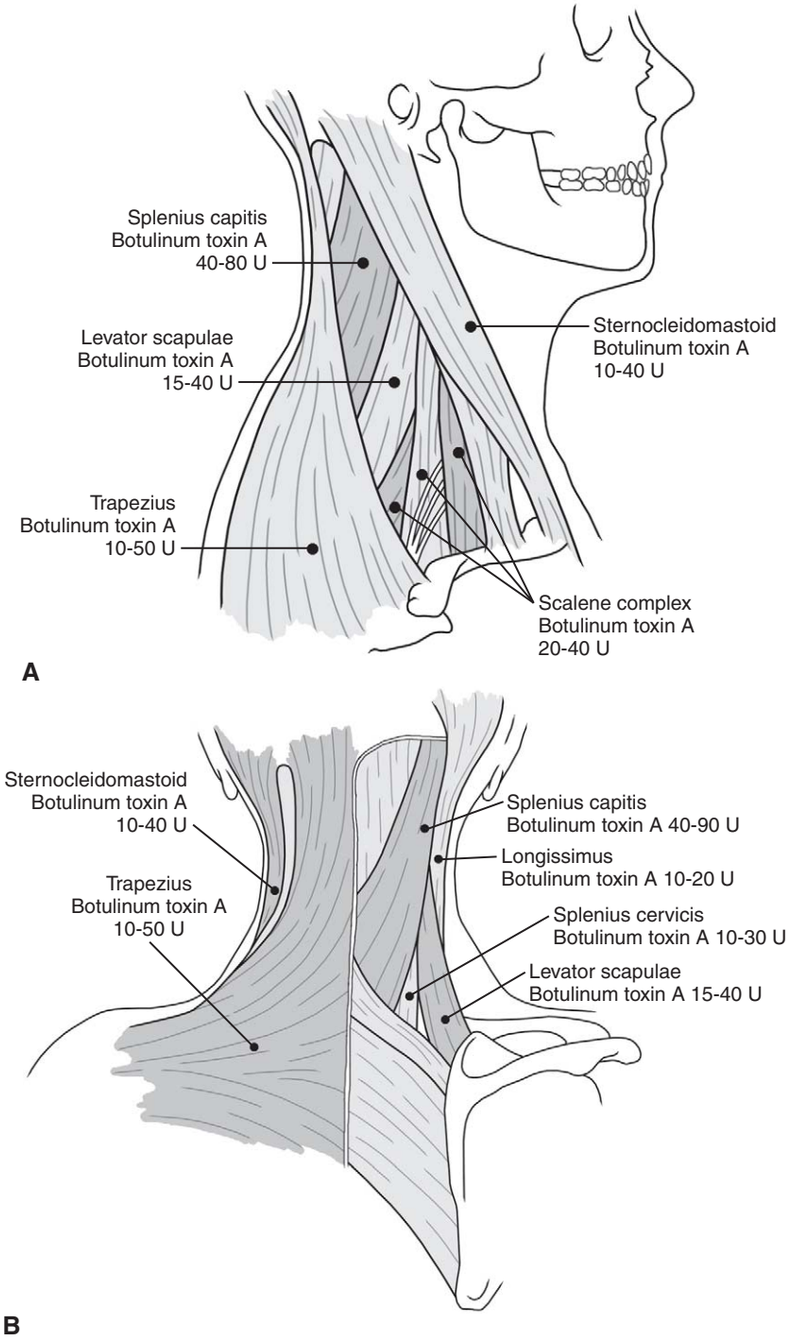


FIGURE 10–10

(A and B) The common important muscles to be injected for cervical myofascial pain, especially for dystonia. The dots on each muscle represent the site of injection.

weakness) and antibody formation. Little data are available to help one decide on botulinum $toxin_B$ dosing outside of cervical dystonia. It appears to be about 40 to 50 times less potent than botulinum toxin, with very few patients having received doses at or above 20,000 units, although these doses appear to be well tolerated. In the cervical dystonia data, botulinum $toxin_B$ produced duration of effect between 12 and 16 weeks.

EFFICACY

The first double-blind, placebo-controlled study showing a positive effect of botulinum $neurotoxin_A$ in 21 patients with spasmodic torticollis was published in 1986 by Tsui et al.[51] Injection of 100 mouse units (50 mouse units in each of two injection sites per muscle) resulted in both subjective and objective improvements 6 weeks after the injections. Scores

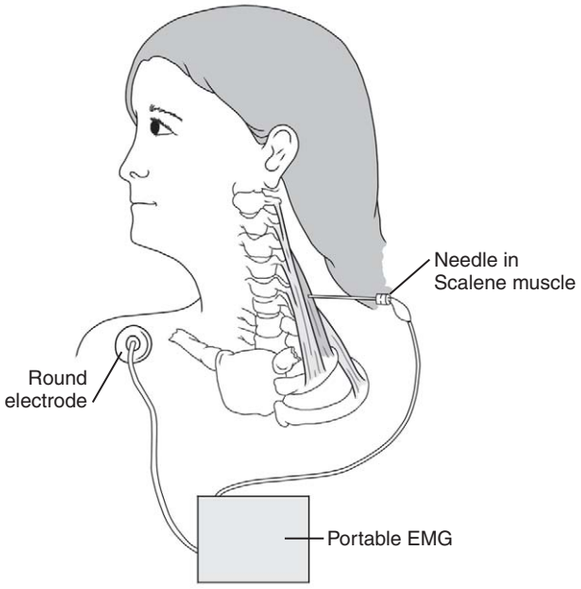


FIGURE 10–11
Portable EMG.

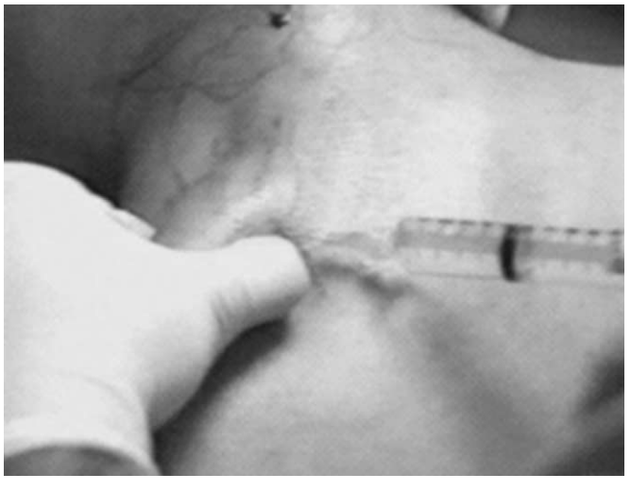


FIGURE 10–12
The left hand holding the muscle firm while the injection is given by the right hand.

on a scale to assess range-of-neck movement, duration of involuntary contractions, and degree of shoulder elevation and tremor (Tsui scale), as well as amount of pain, indicated improvement in 63% of these patients. The reported adverse effects of neck weakness or stiffness were of brief duration (1–2 days) and occurred with similar frequency in the botulinum neurotoxin$_A$ and control groups.

Subsequent studies have revalidated the efficacy of botulinum neurotoxin$_A$ as a treatment of choice for cervical dystonia. In a double-blind, placebo-controlled study of 55 patients with idiopathic cervical dystonia, Greene and Fahn[52] reported pain reduction in 63% of treated patients and improvements in functional state, head turning at rest, and walking in 61% of treated patients. Efficacy remained apparent at 2 weeks postinjection, but the best results were seen at 6 weeks. Repeat injections were performed 3 months after the first injection. In the open-phase arm of this study, higher doses of botulinum neurotoxin$_A$ (~240 units per patient) were the most efficacious (74% of patients).[50] There was no significant difference in the occurrence of immediate and transient adverse effects, such as local pain, between botulinum neurotoxin$_A$- and placebo-treated patients. Other reported adverse effects associated with botulinum neurotoxin$_A$ were swallowing difficulties, neck or generalized weakness, spasms, and transient paresthesias, ranging in frequency from 3 to 10%.

In a series of 19 patients treated with botulinum neurotoxin$_A$ (50 mouse units per muscle, two to four muscles), 74% showed improved movement, and 80% noted a significant reduction in neck pain.[53] Again, these effects were apparent within 2–10 days, became maximal at 4–6 weeks, and lasted for 9–11 weeks. Adverse effects were mild and consisted of either local pain at the injection site for 1–2 days (63%%) or transient neck weakness (16%).

Using a British preparation of botulinum neurotoxin$_A$ (Dysport,® Ipsen Biopharm Ltd, Wrexham, UK), the German Dystonia Study Group found a dose–response effect on the subjective improvement and Tsui scale scores in 58 (79%) of 73 patients with cervical dystonia.[54] Maximal improvement was seen at 4–8 weeks. Data from the same group also provided information about the long-term efficacy and safety of botulinum neurotoxin$_A$.[55] A total of 303 patients received an average of 10.2 injections each over a mean duration of 3.2 years (range 1.3–5.9 years). Improvement was measured using subjective scores and the Tsui scale. Patients typically improved within 1 week, and the effect lasted 11 weeks. Maximal improvement occurred after the first injection and remained stable after the sixth injection. Adverse effects were reported in 22% of patients. Of those who reported adverse reactions, 77% manifested mild to moderate dysphagia starting 9.7 days following injection and lasting an average of 3.5 weeks. Adverse effects were generally mild and transient, and decreased with an increasing number of treatment sessions. Secondary lack of response to treatment occurred in approximately 5% of patients, typically after the sixth injection. More than half of these secondary nonresponders tested positive for neutralizing antibodies.[55]

A randomized, multicenter, double-blind, placebo-controlled study of botulinum neurotoxin$_A$, organized by Allergan (unpublished data, Allergan, Inc., Irvine, CA), assessed the efficacy of botulinum neurotoxin$_A$ in 214 patients with cervical dystonia using the Cervical Dystonia Severity Scale (CDSS), and physician and global assessment scores of pain severity and functional disability. The CDSS measures head position in terms of the degree of deviation from normal, in 5-degree increments.[56] Botulinum neurotoxin$_A$ injections reduced the intensity and frequency of pain, as well as head deviation from midline. It also improved physical functioning and had an acceptable

adverse effect profile. Reported adverse effects were not significantly different from those in the placebo group, the only exception being rhinitis, which occurred more frequently in the botulinum neurotoxin$_A$-treated group (6.8% of patients). A separate study noted that the beneficial effects of botulinum neurotoxin$_A$ in patients with cervical dystonia also have a positive effect on patients' quality of life.[57]

Comparisons of the original preparation (25 ng of protein/100 units) and the current bulk preparation of botulinum neurotoxin$_A$ (5 ng of protein/100 units) showed similar efficacy and safety profiles for the two preparations.[58,59] Interestingly, at the end of a 14.5-month treatment period during which 119 patients received an average of four injections, each of the current botulinum neurotoxin$_A$ preparation, none of the patients tested positive for neutralizing antibodies. This suggests that the lower protein concentration of the current botulinum neurotoxin$_A$ preparation may render it less immunogenic.[52]

BOTULINUM NEUROTOXIN$_F$ FOR CERVICAL DYSTONIA

Greene and Fahn[52] reported the effects of botulinum neurotoxin type F in patients with CD who initially responded to botulinum neurotoxin$_A$ but developed secondary resistance. Of nine seronegative botulinum neurotoxin$_A$ secondary nonresponders, five reported subjective improvement with botulinum neurotoxin$_F$, but this improvement lasted only up to 1 month, the same benefit reported by seropositive patients.

BOTULINUM NEUROTOXIN$_B$ FOR CERVICAL DYSTONIA

The first open-label, dose-escalation study of botulinum neurotoxin$_B$ with positive results in managing cervical dystonia was conducted in 1995 by Tsui and colleagues as cited in Brashear.[60] The efficacy of botulinum neurotoxin$_B$ in idiopathic cervical dystonia was subsequently tested by Lew et al.[61] in a randomized, multicenter, double-blind, placebo-controlled study of 122 patients with idiopathic cervical dystonia of 1–10 years. To assess the possible role of botulinum neurotoxin$_B$, the investigators closely studied 26 patients previously treated with botulinum neurotoxin$_A$ who subsequently developed resistance. These patients were given 2500–10,000 units of botulinum neurotoxin$_B$ per session, with follow-up injections every 1–4 months. Botulinum neurotoxin$_B$ injections improved outcomes, as assessed by the Toronto Western Spasmodic Torticollis Rating Scale (TWSTRS-Total scale) and subscales, which grade severity of symptoms, disability, and pain. Maximal effects were seen 1 month after injection. Overall, 58–77% of all patients had responded to botulinum neurotoxin$_B$ at 4 weeks. Efficacy was still apparent but significantly reduced during the following 2 months, and best effects were seen only with the highest doses (5000 to 10,000 units/session). Disability also improved at all doses, but significant reductions in pain were noted only at the highest dose. Among the 26 patients who initially responded and subsequently became resistant to botulinum neurotoxin$_A$, 14 clearly improved with botulinum neurotoxin$_B$. Dry mouth was recorded in a significant number of patients (3–33%) and was considerably more frequent at higher doses. Dysphagia was also noted in 10–27% of the patients. Other adverse effects, ranging in frequency from 3 to 19%, included headache, nausea, and other pain.

Following these encouraging results, the same investigators pursued further phase III clinical trials in 1999 aimed at testing the efficacy of botulinum neurotoxin$_B$ in both botulinum neurotoxin$_A$-resistant and botulinum neurotoxin$_A$-responsive patients with cervical dystonia. In the first arm of the study, two doses of botulinum neurotoxin$_B$ (5000 and 10,000 units) were tested in 109 patients with cervical dystonia who were still responsive to botulinum neurotoxin$_A$.[62] Both doses resulted in improvement in the TWSTRS-Total scores at 4 and 8 weeks following injection, compared with placebo. At 4 weeks, improvement was also seen in symptom severity and pain. Almost 80% of the patients reported at least one adverse effect, usually mild, and commonly dry mouth (14–24% of patients in the active-treatment groups) and dysphagia (11–22% of patients).

In the second arm of this study, 77 patients with resistance to botulinum neurotoxin$_A$ were randomized to treatment with 10,000 units of botulinum neurotoxin$_B$ or placebo, and prospectively followed at 2, 4, 8, 12, and 16 weeks.[63] Significant improvements again were noted in the TWSTRS-Total score at all time points (4–12 weeks following injection), as well as in the severity, disability, and pain scoring subscales at 4 weeks. Eighty-four percent of patients treated with placebo and all patients treated with botulinum neurotoxin$_B$ reported at least one adverse effect, generally dry mouth (44% of patients), dysphagia (28% of patients), pain at the injection site (18% of patients), and nausea (15% of patients).

The long-term safety and efficacy of botulinum neurotoxin$_B$ injections in 29 patients with cervical dystonia treated for an average of 20 months were recently reported in the proceedings of the International Congress of Parkinson's Disease and Movement Disorders.[64] Of these patients, 22 were botulinum neurotoxin$_A$-resistant and 9 had undergone prior denervation therapy. Overall 16 patients experienced significant improvement with botulinum neurotoxin$_B$, 5 withdrew, 3 did not respond at all, and 3 developed resistances to treatment. Two botulinum neurotoxin$_B$–resistant patients were also botulinum neurotoxin$_A$-resistant. Among all 36 patients who received botulinum neurotoxin$_B$ injection for cervical dystonia or other conditions, the most frequent adverse effect was dry mouth; less frequent adverse effects were dysphagia, injection site pain, and neck weakness. While botulinum neurotoxin$_B$ may be an effective therapeutic alternative for patients who develop resistance

to botulinum neurotoxin$_A$, the exclusion of patients with cervical dystonia shown to be primary nonresponders to botulinum neurotoxin$_A$ does not yet allow researchers to draw conclusions about which serotype is more potent. It appears, however, that botulinum neurotoxin$_B$-treated patients may experience more adverse effects.

BOTULINUM NEUROTOXIN$_A$ FOR POSTSTROKE SPASTICITY IN PECTORAL GIRDLE

In a randomized, controlled clinical trial, Bakheit et al.[65] studied the effect of the British formulation of botulinum neurotoxin$_A$, Dysport, in poststroke upper limb spasticity in 59 patients. Injections of 1000 units of Dysport were administered in five upper limb muscles. Assessments of muscle tone using the Modified Ashworth Scale (MAS), joint range of motion, Barthel Index, pain score, goalattainment, and subjective evaluation of benefit were performed at baseline and at 4, 8, 12, and 16 weeks following injection. At 4 weeks, only the MAS showed significant improvement. However, at 16 weeks, approximately 90% of botulinum neurotoxin$_A$-treated patients had improved both globally and in range of motion at the elbow. The most frequent adverse effects were accidental injury, urinary tract infections, and muscle pain.

BOTULINUM NEUROTOXIN$_A$ FOR REFRACTORY CERVICOTHORACIC MYOFASCIAL PAIN

The first pilot study of six patients with myofascial syndrome appeared in 1994.[66] In this double-blind, placebo-controlled trial, botulinum neurotoxin$_A$ or saline injections were administered at trigger points, and botulinum neurotoxin$_A$ was shown to separate significantly from placebo in pain, palpable muscle firmness, and pressure pain thresholds. In a later randomized, double-blind study of 33 patients with cervicothoracic myofascial syndrome, patients received injections of placebo or 50 or 100 mouse units of botulinum neurotoxin$_A$.[67] Evaluation at 4-month intervals to assess pain, disability, and pressure algometer readings initially showed no statistically significant differences between groups. Approximately half the patients in each group proceeded to a second injection of 100 mouse units of botulinum neurotoxin$_A$. This significantly increased the percentage of asymptomatic patients in both groups compared with placebo.

In a follow-up, open-label retrospective study, Wheeler and Goolkasian[67] found that 35 of 44 patients with myofascial syndrome showed marked reductions in pain with botulinum neurotoxin$_A$ injections. The majority (59%) of these patients received multiple injections (50–150 mouse units). Other authors, including Lang,[50] also obtained excellent results using botulinum neurotoxin$_A$ for this syndrome. Additional studies are required to further document these promising preliminary results.

BOTULINUM NEUROTOXIN$_A$ FOR WHIPLASH-ASSOCIATED DISORDER AND PAIN

In a randomized, placebo-controlled, pilot study, 28 patients with chronic grade II whiplash–associated disorder received either botulinum neurotoxin$_A$ injections (100 units of Botox) or placebo, bilaterally under EMG guidance in one or more of the following muscles: splenius capitis, rectus capitis, semispinalis capitis, and trapezius.[68] Statistically significant improvements in all parameters measured were noted at 4 weeks and persisted for 3 months after injection. The response rate was 64%. The same authors also found significant improvements in pain associated with whiplash injury at 2 and 4 weeks after botulinum neurotoxin$_A$ injections into the neck musculature.[69] Following up on their initial study, the same group reported that injections of a total of 150 units of botulinum neurotoxin$_A$ into 10 separate sites on the same neck muscles were also efficacious in managing whiplash-associated disorder.[69] With regard to adverse effects, this cohort reported no significant neck weakness and only a few episodes of what was termed "late-day fatigue."

COMPLICATIONS

When local injection and/or steroids are injected, side effects such as pain after injection, hematoma, or infection are well described in the literature. Use of 3% phenol or 50% alcohol have also been described by early workers as pain following injection and fibrosis at the site and painful nodule formation.

Botulinum toxins have early effects of (1) "feeling under the weather," (2) dysphagia, and (3) dysphonia and transient paresis. Botulinum toxin$_B$ seems to have greater spread from the site of injection, shortness of breath, and dysphagia.

CONCLUSIONS

Neck and shoulder pain are a major cause of morbidity and disability among adults. Both cervical dystonic and nondystonic neck pain disorders are frequently associated with headache. As is evident from this review, chemical denervation of the neck muscles using botulinum neurotoxin provides considerable improvement in many aspects of functioning, including range of motion; disability scores; head, neck, and shoulder pain; and activities of daily living. Additional indirect benefits accrue to the psychological and social aspects of these diseases. In addition, botulinum neurotoxins are effective treatments for many other focal dystonias. Both botulinum neurotoxin$_A$ and botulinum neurotoxin$_B$ have been used successfully to treat all conditions reviewed here, but there is substantially more clinical experience with botulinum neurotoxin$_A$. Success rates range from 60 to 90% with the

condition. Botulinum neurotoxin$_B$ has been shown to be effective in both botulinum neurotoxin$_A$ responders and secondary botulinum neurotoxin$_A$ nonresponders and may thus be a useful alternative to botulinum neurotoxin$_A$. Further prospective studies should directly compare the efficacy and safety profiles of both serotypes in a botulinum neurotoxin$_A$-naive population. Existing data demonstrate a safer adverse-effect profile for botulinum neurotoxin$_A$, and botulinum neurotoxin$_B$ is associated with more diffuse adverse effects, such as dry mouth and dysphagia. With both serotypes, the effects of treatment appear as early as 1–2 weeks, are maximal at 4–6 weeks, and may last at least 3–4 months, necessitating repeat injections every 3 months. The greatest improvement occurs after the first injection, and this improvement is maintained following subsequent injections, suggesting an enduring effect.

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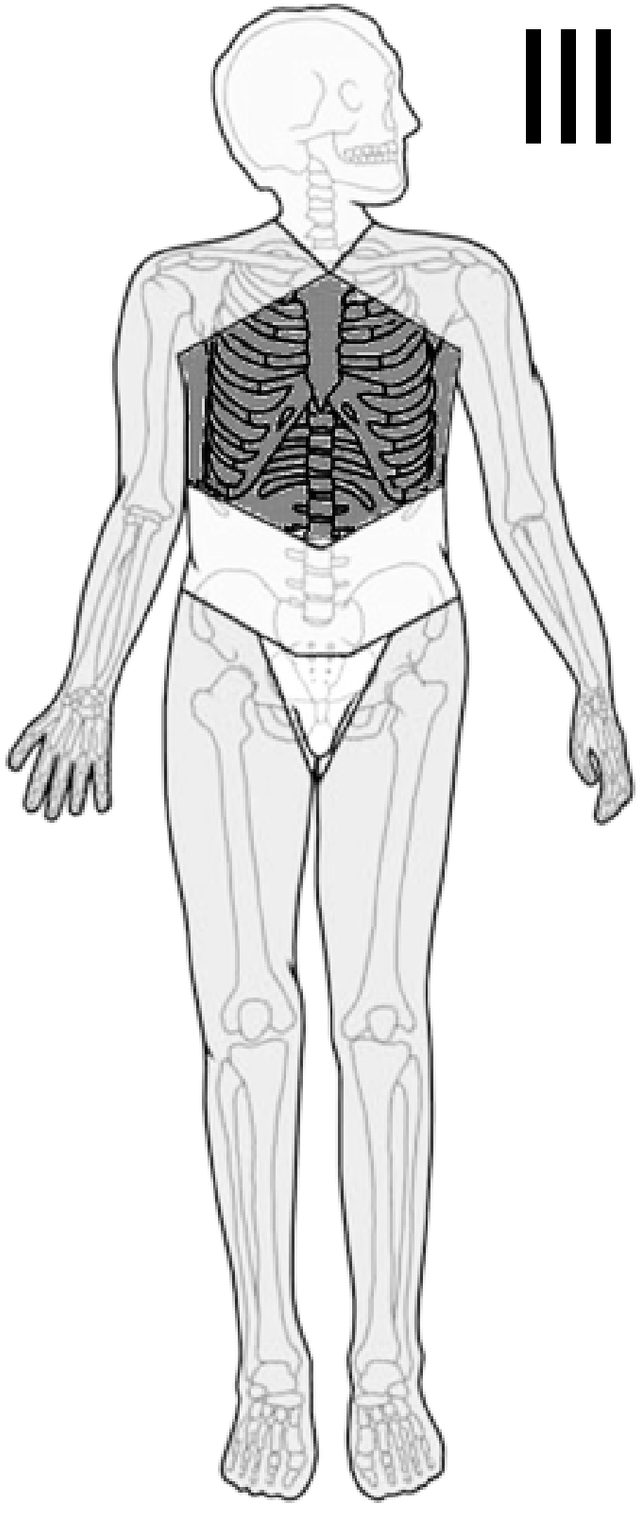
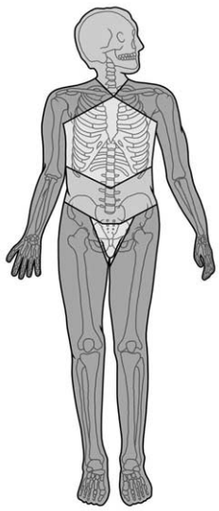
SECTION

III



THORAX

C H A P T E R



11

Somatic Blocks of the Thorax

STEVEN D. WALDMAN

INTERCOSTAL NERVE BLOCK

HISTORY

The history of the use of intercostal nerve block roughly parallels the development of regional anesthesia. First with cocaine and later with procaine, which was introduced into clinical practice in 1909, surgeons blocked the chest wall and upper abdomen with large volumes of dilute local anesthetic to perform a variety of surgeries of the chest and abdomen. It was the early advocates of regional anesthesia such as Labat[1,2] who shifted the focus of regional anesthesia to the blocking of specific nerves with more concentrated local anesthetics to obtain surgical anesthesia. The intercostal nerves were ideally suited to such an approach. In 1922, Labat,[1,2] in his classic text on regional anesthesia, provided clinicians with a concise description of intercostals block and presented a technique that is little changed today.

ANATOMY

The intercostal nerves arise from the anterior division of the thoracic paravertebral nerve.[3] A typical intercostal nerve has four major branches (Figure 11-1). The first branch is the unmyelinated postganglionic fibers of the gray rami communicantes, which interface with the sympathetic chain. The second branch is the posterior cutaneous branch, which innervates the muscles and skin of the paraspinal area. The third branch is the lateral cutaneous division, which arises in the anterior axillary line. The lateral cutaneous division provides the majority of the cutaneous innervation of the chest and abdominal wall. The fourth branch is the anterior cutaneous branch supplying innervation to the midline of the chest and abdominal wall. Occasionally, the terminal branches of a given intercostal nerve may actually cross the midline to provide sensory innervation to the contralateral chest and abdominal wall. The 12th nerve is called the subcostal nerve and is unique in that it gives off a branch to the first lumbar nerve, thus contributing to the lumbar plexus.

INDICATIONS

Intercostal nerve block is useful in the evaluation and management of pain involving the chest wall and the upper abdominal wall.[4] Intercostal nerve block with local anesthetic can be used as a diagnostic tool when performing differential neural blockade on an anatomical basis in the evaluation of chest and abdominal pain. If destruction of the intercostal nerve is being considered, this technique is useful as a prognostic indicator of the degree of motor and sensory impairment that the patient may experience. Intercostal nerve block with local anesthetic may be used to palliate acute pain emergencies, including rib fractures, acute herpes zoster, and cancer pain, while waiting for pharmacologic, surgical, and antiblastic methods to become effective. It is also useful prior to placement of percutaneous thoracotomy and nephrotomy tubes. Intercostal nerve block with local anesthetic and steroid is also useful in the treatment of postthoracotomy pain, cancer pain, rib fractures, metastatic lesions of the liver, and postherpetic neuralgia.[5]

Destruction of the intercostal nerve is indicated for the palliation of cancer pain, including invasive tumors of the ribs and the chest and upper abdominal wall.[6] Given the desperate nature of many patients suffering from aggressively invasive malignancies, blockade of the intercostal nerve using a 25-gauge needle may be carried out in the presence of coagulopathy or anticoagulation, albeit with an increased risk of ecchymosis and hematoma formation.

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represents a relatively strong contraindication to the performance of intercostal nerve block. Some clinicians will

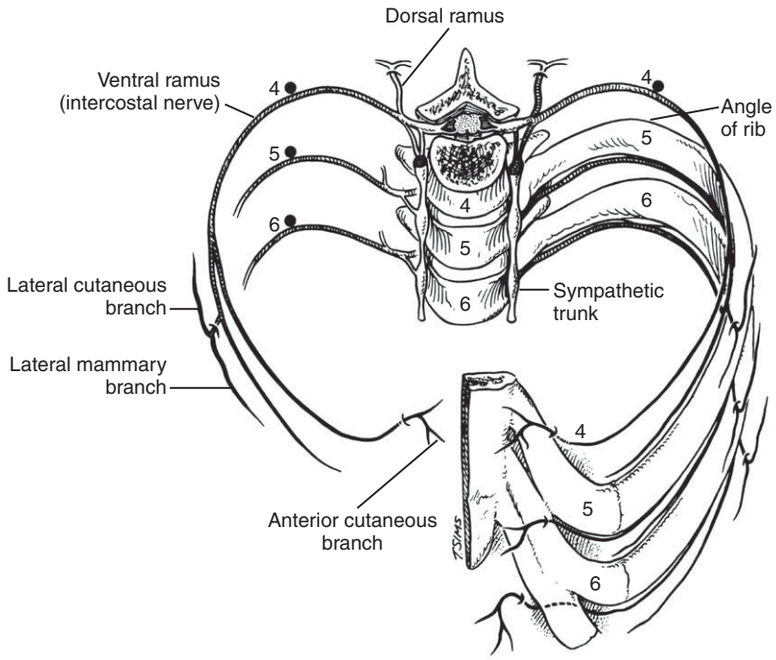


FIGURE 11–1
Anatomy of the intercostal nerve and its distribution with its branches along the ribs.

consider performing intercostal block with a 25-gauge needle in the setting of life-threatening pulmonary compromise secondary to multiple rib fractures or in patients with metastatic disease involving the ribs in whom pain is uncontrolled by systemic analgesics.[4]

EQUIPMENT

- Peripheral nerve block tray
- 22-gauge, 1-1/2-inch needle
- 25-gauge, 1-1/2-inch needle (if anticoagulated or coagulopathy present)
- 12-ml sterile syringe

DRUGS

- 1% preservative free lidocaine (for diagnostic or prognostic block)
- 0.5% preservative free bupivacaine (for therapeutic block)
- Depot methylprednisolone (for therapeutic block)
- 6.5% aqueous phenol (for chemical neurolytic block)

PROCEDURE

Patient Positioning

The patient is placed in the prone position with the arms hanging loosely off the side of the cart. Alternatively, this block can be done in the seated or lateral position.

Site of Needle Entry

The rib to be blocked is identified by palpating its path at the posterior axillary line. The index and middle fingers are then placed on the rib bracketing the site of needle insertion. The skin overlying the rib is then marked with a sterile marker and is then prepared with antiseptic solution. The fluoroscopic C-arm is then centered over the vertebral body and the levels to be blocked are then confirmed. The C-arm is then rotated ipsilaterally to follow the path of the affected intercostal nerve as it travels posteriolaterally beneath the rib (Figure 11-2).

A 22-gauge, 1-1/2-inch needle is attached to a 12-ml syringe and advanced perpendicular to the skin, aiming for the middle of the rib in between the index and middle fingers. The needle should impinge on bone after being advanced approximately 3/4 inch. After bony contact is made, the needle is withdrawn into the subcutaneous tissues, and the skin and subcutaneous tissues are retracted with the palpating fingers inferiorly. This allows the needle to be walked off the inferior margin of the rib (Figure 11-3). As soon as bony contact is lost, the needle is slowly advanced approximately 2 mm deeper. This will place the needle in proximity to the costal grove, which contains the intercostal nerve as well as the intercostal artery and vein. Placement may be confirmed with the C-arm if the adequacy of needle placement is in doubt (Figure 11-4). After careful aspiration reveals no blood or air, 3–5 ml of preservative-free local anesthetic is injected. If there is an inflammatory component to the pain, the local anesthetic is combined with 80 mg of methylprednisolone and injected in incremental doses. Subsequent daily nerve blocks are carried out in a similar manner, substituting 40 mg of methylprednisolone for the initial

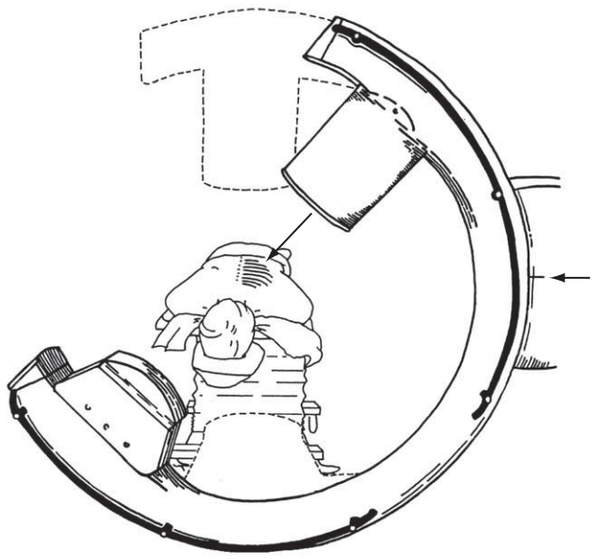
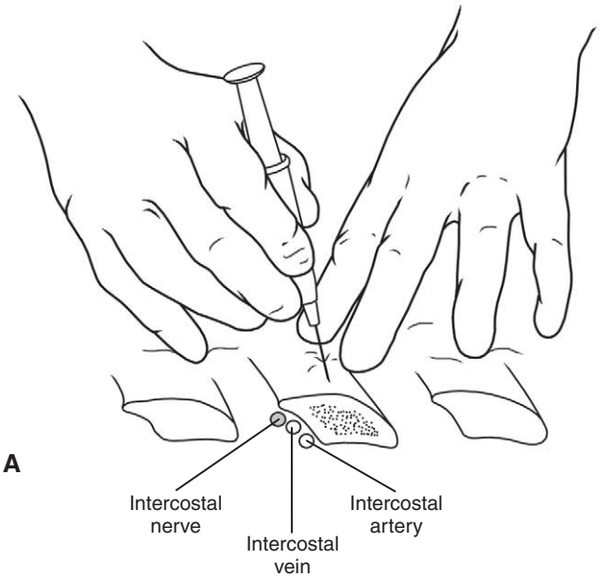


FIGURE 11–2

Patient is lying in the prone position beneath the fluoroscopic C-arm. After identifying the appropriate levels for blockade, the C-arm is rotated ipsilaterally to enhance the posterolateral ribs.



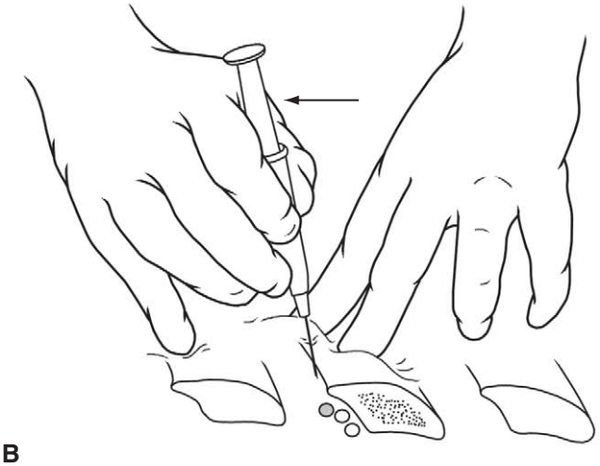


FIGURE 11–3

(A) The needle is advanced until it impinges on the periosteum of the rib. (B) The needle is walked off the inferior margin of the rib.

80-mg dose. 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 have to be blocked. Water-soluble contrast medium may be added to the local anesthetic to confirm appropriate needle placement and the spread of the local anesthetic along the intercostal groove containing the intercostal nerve to be blocked (Figure 11-5).

COMPLICATIONS

Given the proximity of the pleural space, pneumothorax after intercostal nerve block is a distinct possibility. The incidence of the complication is less than 1%, but it occurs with greater frequency in patients with chronic obstructive pulmonary disease.[7] Because of the proximity to the intercostal nerve and artery, the pain management specialist should carefully calculate the total milligram dosage of local anesthetic administered because vascular uptake via these vessels is high.[4] Although uncommon, infection remains an ever-present possibility, especially in the immunocompromised cancer patient. Early detection of infection is crucial to avoid potentially life-threatening sequelae.

HELPFUL HINTS

Intercostal nerve block is a simple technique that can produce dramatic relief for patients suffering from the previously mentioned pain complaints. Intercostal block with local anesthetic before placement of chest tubes provides a great degree of patient comfort and should routinely be used. Intercostal block with local anesthetic and steroid is useful in the palliation of the pleuritic pain secondary to lung tumors and liver tumors that are irritating the parietal peritoneum. Neurolytic block with small quantities of phenol in glycerin or by cryoneurolysis or radiofrequency lesioning has been shown to provide long-term relief for patients suffering from post-thoracotomy and cancer-related pain who have not responded to more conservative treatments. As mentioned earlier, the proximity of the intercostal nerve to the pleural space makes careful attention to technique mandatory.

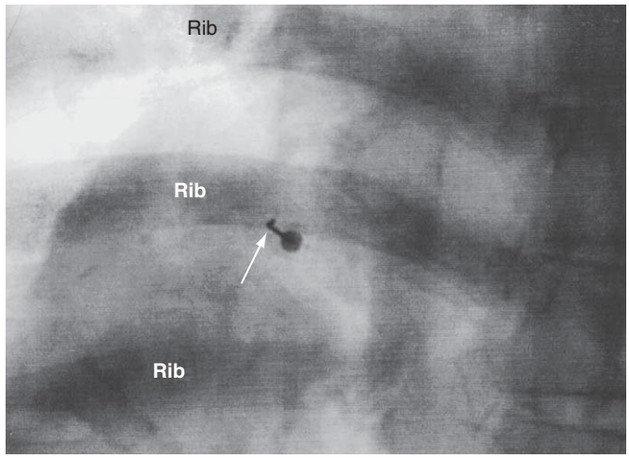


FIGURE 11–4
Intercostal nerve block, anteroposterior view. The arrow indicates where the needle touches and stops below the rib.

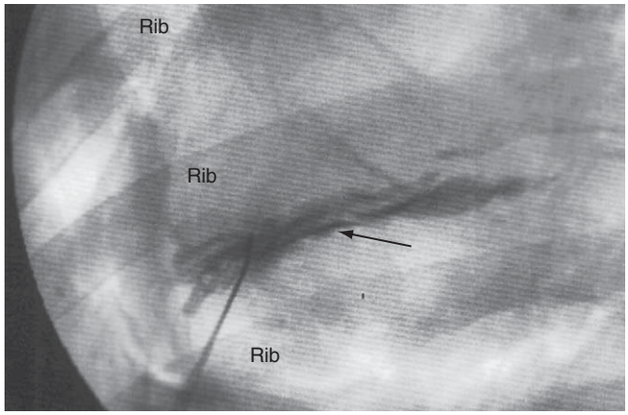


FIGURE 11–5
Intercostal nerve block with contrast medium, anteroposterior view. The arrow indicates the spread of contrast in the intercostal groove.

SUPRASCAPULAR NERVE BLOCK

HISTORY

The early use of suprascapular nerve block was focused primarily on its utility as a regional anesthetic technique to render the shoulder joint relatively insensate to allow manipulation under anesthesia. Treatment of streetcar man's shoulder, which was a form of adhesive capsulitis and calcific tendonitis of the shoulder, were among its main uses. The introduction of sodium pentothal and later methohexital sodium (Brevitol) made brief general anesthetics more feasible and safer, and the use of suprascapular nerve block fell into disuse. More recently, there has been a resurgence in interest in this regional anesthetic technique to provide postoperative pain relief following shoulder surgery and to treat suprascapular nerve entrapment syndrome (see later).

ANATOMY

The suprascapular nerve is formed from fibers originating from the C5 and C6 nerve roots of the brachial plexus with some contribution of fibers from the C4 root in most patients.[8] The nerve passes inferiorly and posteriorly from the brachial plexus to pass underneath the coricoclavicular ligament through the suprascapular notch. The suprascapular artery and vein accompany the nerve through the suprascapular notch (Figure 11-6). The suprascapular nerve provides much of the sensory innervation to the shoulder joint and provides innervation to two of the muscles of the rotator cuff, the supraspinatus and infraspinatus.

INDICATIONS

The primary indications for suprascapular nerve block technique are to provide postoperative pain relief following shoulder surgery and to treat suprascapular nerve entrapment syndrome.[8,9] Suprascapular nerve entrapment syndrome is caused

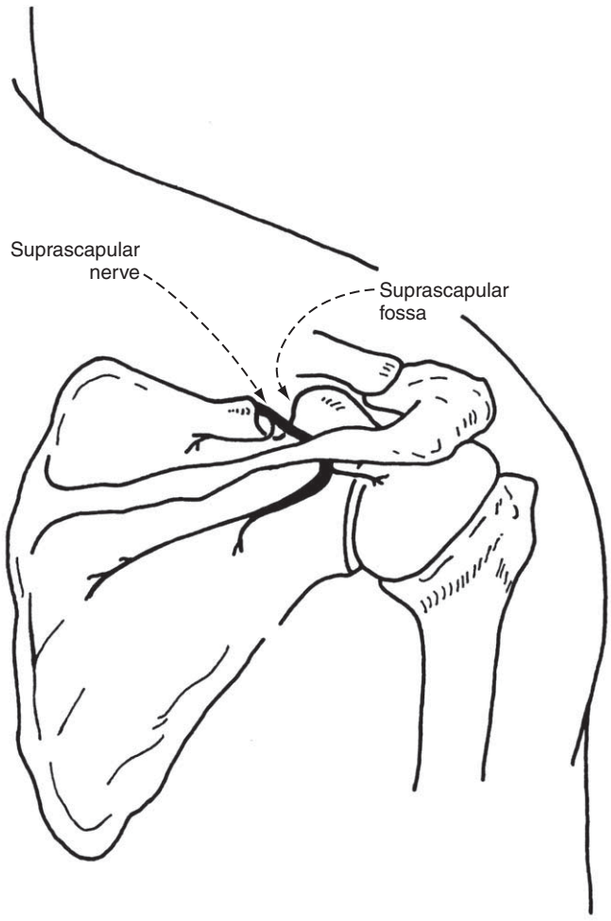


FIGURE 11–6
Drawing of the patient in the prone position with the fluoroscope slightly lateral to midline at the T2-T3 level with a slight cephalocaudad tilt.

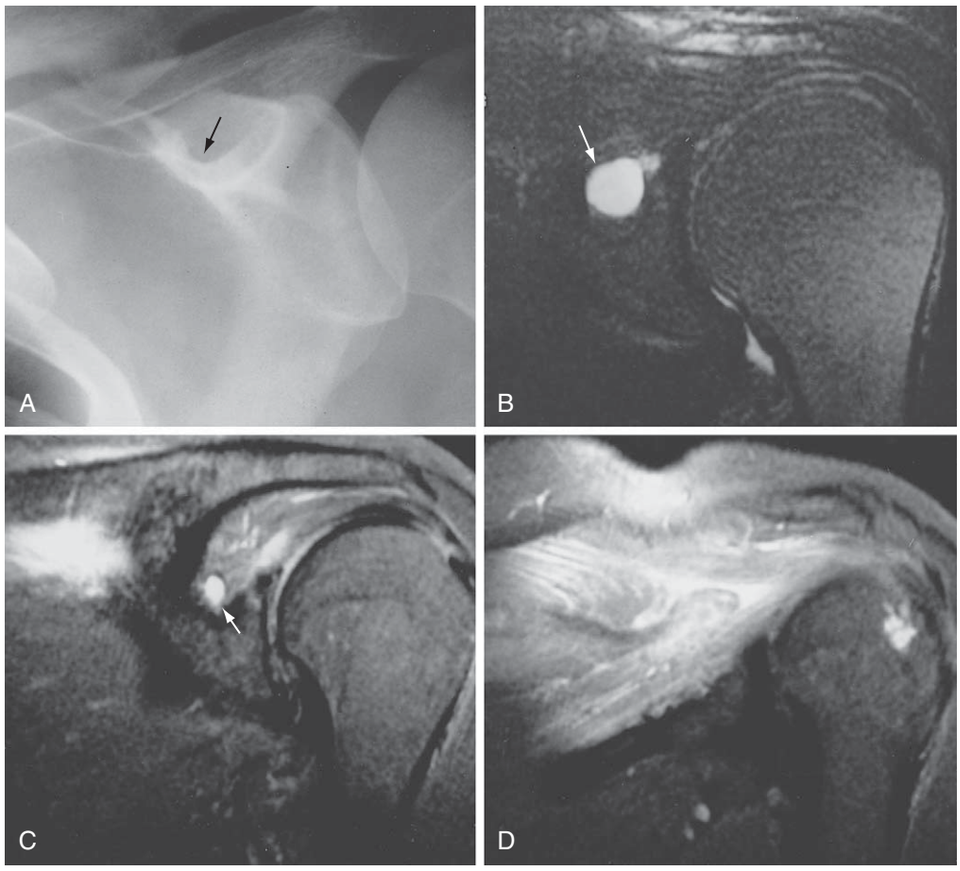


FIGURE 11–7

Entrapment of the suprascapular nerve: (A) Routine radiograph reveals erosion of bone (arrow) at the spinoglenoid notch of the scapula. (B) An oblique coronal fat-suppressed fast spin echo (TR/TE, 3250/105) MRI shows a ganglion cyst (arrow) in the spinoglenoid notch. (C–D) Two oblique coronal fat-suppressed fast spin echo (TR/TE, 2650/48) MRI reveal a ganglion cyst (arrow) in the spinoglenoid notch with intense signal in the infraspinatus muscle related to denervation. Also note an undersurface tear of the infraspinatus tendon and a humeral cyst. (From Resnick D: *Diagnosis of Bone and Joint Disorders*, 4th ed. Philadelphia, Saunders, 2002, figure 71-65 A–D, p. 3532, with permission.)

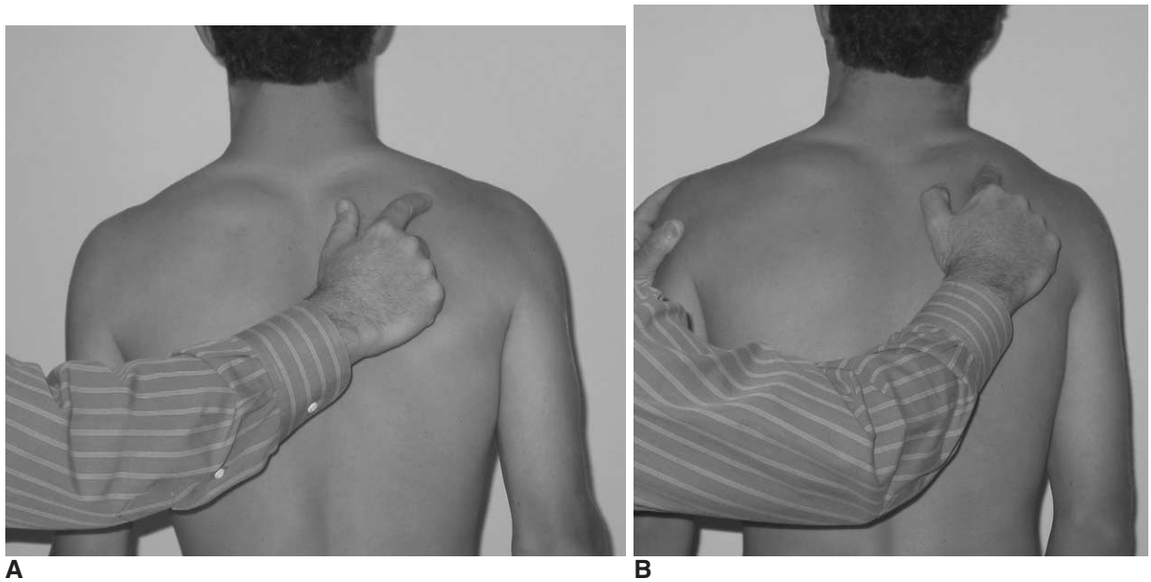
FIGURE 11–8

Mechanism of injury in compression of the suprascapular nerve. (From Waldman SD: *Atlas of Uncommon Pain Syndromes*. Philadelphia, Saunders, 2003, figure 16-1, p. 64, with permission.)

by compression of the suprascapular nerve as it passes through the suprascapular notch (Figure 11-7).[9] The most common causes of compression of the suprascapular nerve at this anatomic location include the prolonged wearing of heavy backpacks and direct blows to the nerve such as occur in football injuries and in falls from trampolines (Figure 11-8). This entrapment neuropathy presents most commonly as a severe, deep, aching, pain which radiates from the top of the scapula to the ipsilateral shoulder. Tenderness over the suprascapular notch is usually present (Figure 11-9).[10] Shoulder movement, especially reaching across the chest, may increase the pain symptomatology. Untreated, weakness and atrophy of the supraspinatus and infraspinatus muscles will occur.

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represents a relatively strong contraindication to the performance suprascapular nerve block. Some clinicians will consider performing suprascapular nerve block with a 25-gauge needle in the setting of patients with primary tumor or metastatic disease involving the shoulder joint in whom pain is uncontrolled by systemic analgesics. Local infection involving the area of the suprascapular nerve is also a contraindication to the performance of suprascapular nerve block.



A B

FIGURE 11–9

A-(B) Eliciting the suprascapular notch sign for suprascapular nerve entrapment syndrome. (From Waldman SD: *Physical Diagnosis of Pain: An Atlas of Signs and Symptoms.* Philadelphia, Saunders, 2005, figure 101-2 A(B) p. 206, with permission.)

EQUIPMENT

- Peripheral nerve block tray
- 22-gauge, 1-1/2-inch needle
- 25-gauge, 1-1/2-inch needle (if anticoagulated or coagulopathy present)
- 12-ml sterile syringe

DRUGS

- 1% preservative-free lidocaine (for diagnostic or prognostic block)
- 0.5% preservative-free bupivacaine (for therapeutic block)
- Depot methylprednisolone (for therapeutic block)
- 6.5% aqueous phenol (for chemical neurolytic block)

PROCEDURE

Patient Positioning

The patient is placed in the prone position with the arms hanging loosely off the side of the cart. Alternatively, this block can be done in the seated or lateral position.

Site of Needle Entry

A total of 10 ml of local anesthetic and 40 mg of methylprednisolone are drawn up in a 20-ml sterile syringe. The spine of the scapula is identified, and the clinician then palpates along the length of the scapular spine laterally to identify the acromion. At the point at which the thicker acromion fuses with the thinner scapular spine, the skin is prepped with antiseptic solution. At this point, the skin is marked with a sterile skin marker and the position is confirmed with the C-arm. Identification of the suprascapular notch can be made easier by moving the C-arm from an upright to slightly cephalocaudal tilt (Figure 11-10). After the affected suprascapular notch has been properly identified, the skin and subcutaneous tissues are anesthetized utilizing a 1-1/2-inch needle. After adequate anesthesia is obtained, a 3-1/2-inch 25-gauge needle is inserted in an inferior trajectory toward the body of the scapula. The needle should make contact with the body of the scapula at a depth of about 1 inch (Figure 11-11). The needle is then gently walked superiorly and medially until the needle tip walks off the scapular body into the suprascapular notch.[8] If the notch is not identified, the same maneuver is repeated directing the needle superiorly and laterally until the needle tip walks off the scapular body into the suprascapular notch. Use of the C-arm to track the position of the needle relative to the suprascapular notch may be beneficial if difficulty in needle placement is encountered. A paresthesia is often encountered as the needle tip enters the notch and the patient should be warned of such. If a paresthesia is not elicited after the needle has entered the suprascapular notch, advance the needle an additional 1/2 inch to place the needle tip beyond the substance of the coricoclavicular ligament. The needle should never be advanced deeper or pneumothorax is likely to occur.

After paresthesia is elicited or the needle has been advanced into the notch as described above, gentle aspiration

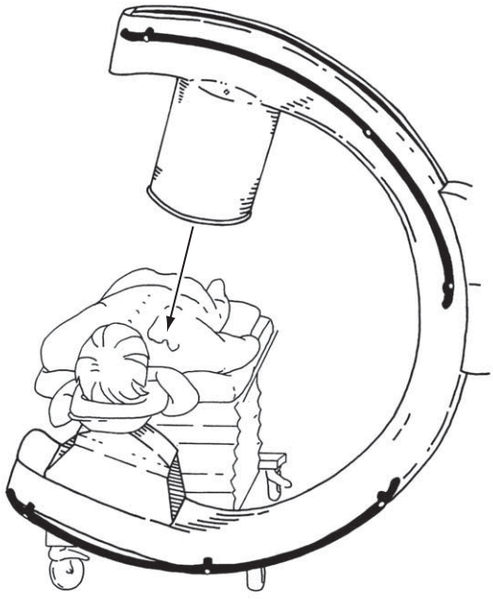


FIGURE 11–10
Anatomy of the suprascapular nerve.

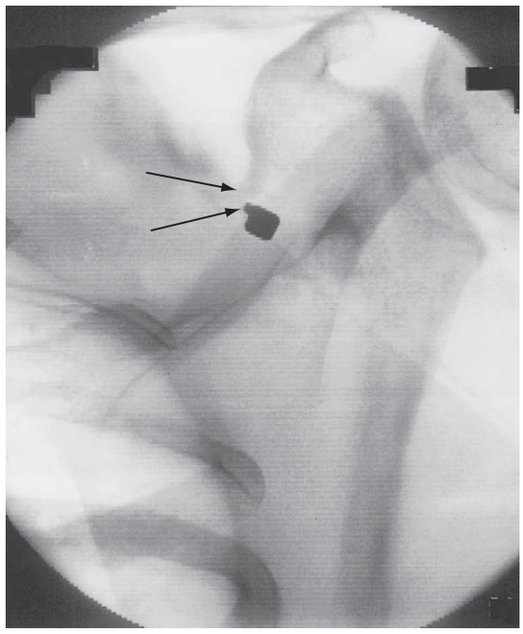


FIGURE 11–11
Suprascapular nerve block with needle in place contacting bone just below the suprascapular nerve. (A) Suprascapular notch. (B) Curved blunt needle tip at the notch.

is carried out to identify blood or air. If the aspiration test is negative, the local anesthetic and/or steroid is slowly injected, with the patient being monitored closely for signs of local anesthetic toxicity. The addition of small amounts of water soluble contrast medium will help confirm spread of the injectate into the suprascapular notch as suprascapular fossa if there is a question regarding the adequacy of needle placement (Figure 11-12).

COMPLICATIONS

The proximity to the suprascapular artery and vein suggests the potential for inadvertent intravascular injection and/or local anesthetic toxicity from intravascular absorption. The clinician should carefully calculate the total milligram dosage of local anesthetic that may be safely given when performing this injection technique. Due to proximity of the lung should the needle be advanced too deeply through the suprascapular notch, pneumothorax is a possibility.

CLINICAL PEARLS

This injection technique will render the shoulder joint insensate. Therefore, it is important that the clinician be sure that the physical and occupational therapists caring for the patient who has undergone suprascapular nerve block understand that not only the shoulder girdle but the shoulder joint has been rendered insensate following this injection technique. This means that deep heat modalities and range of motion exercises must be carefully monitored to avoid burns or damage to the shoulder.

Suprascapular nerve entrapment syndrome is often misdiagnosed as bursitis, tendonitis, or arthritis of the shoulder. Cervical radiculopathy of the C5 nerve root may also mimic the clinical presentation of suprascapular nerve entrapment syndrome. Parsonage-Turner syndrome, which is idiopathic brachial neuritis, may also present as sudden onset of shoulder pain and can be confused with suprascapular nerve entrapment. Tumor involving the superior

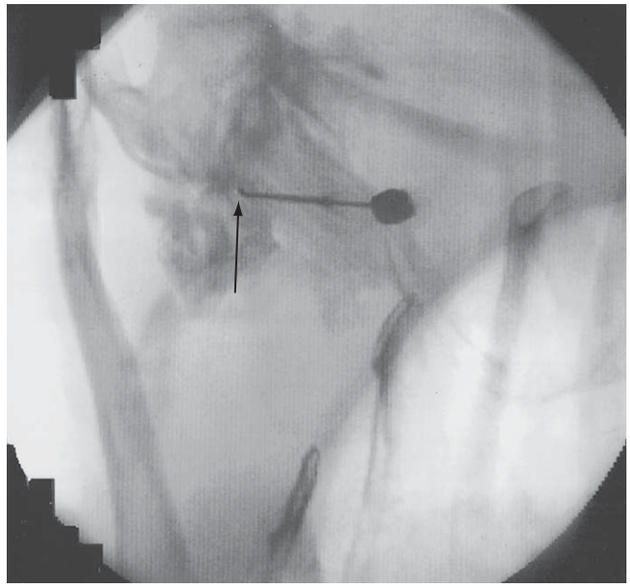


FIGURE 11–12
Suprascapular nerve block with contrast medium filling the suprascapular fossa (arrow).

scapular and/or shoulder should also be considered in the differential diagnosis of suprascapular nerve entrapment syndrome.

Electromyography will help distinguish cervical radiculopathy and Parsonage-Turner syndrome from suprascapular nerve entrapment syndrome. Plain radiographs are indicated in all patients who present with suprascapular nerve entrapment syndrome to rule out occult bony pathology. Based on the patient's clinical presentation, additional testing including complete blood count, uric acid, sedimentation rate and antinuclear antibody testing may be indicated. MRI scan of the shoulder is indicated if primary joint pathology or a space occupying lesion is suspected.

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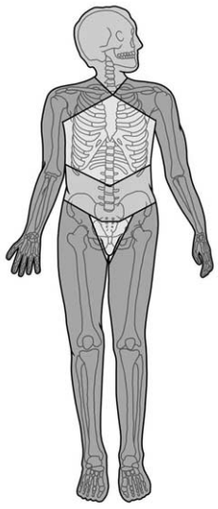
C H A P T E R



12

Sympathetic Blocks of the Thorax

SERDAR ERDINE AND GUL KOKNEL TALU

T2 AND T3 SYMPATHETIC BLOCK AND NEUROLYSIS

HISTORY

Thoracic sympathectomies have been used for the past 80 years to manage painful conditions and vascular insufficiencies of the upper extremities. Indications for sympathectomies at the thoracic ganglia level are for treatment of CRPS I (reflex sympathetic dystrophy), CRPS II (causalgia), arterial occlusions leading to ischemia, drug-resistant Raynaud's disease, Buerger's disease, and frost injuries of the upper extremities.[1]

In 1916, Jonnesco[2] was the first surgeon to promote the stellate ganglion for the treatment of angina pectoris. It soon was recognized that the ablation of the stellate ganglion function also provided pain relief in addition to vasodilation in patients with Raynaud's disease. Jonnesco[1] further advocated stellate ganglionectomy for vascular diseases (Raynaud's) of the upper extremities, in 1921. This was performed via the supraclavicular approach. In reality, it did not give prolonged relief of vasospasticity, and sympathetic tone would eventually return. In 1927, Kuntz[3] observed that nerves from the T2 and T3 sympathetic ganglia would connect to the brachial plexus in 20% of the population, bypassing the stellate ganglion. This fact contributed to the explanation of lack of success noted in stellate ganglionectomy alone. This new information led to multiple procedures to remove or block the T2 and T3 sympathetic ganglia.

The first operative procedure was the modification of the supraclavicular approach described above, which went through the neck down to T2 and T3 retropleurally.[4] Other approaches[5] included the transthoracic axillary[6] and anterior.[7] In 1954, Kux[8] devised a transthoracic approach in which an endoscope was used for electrocoagulation. This approach was largely forgotten for 25 years and then rediscovered recently by a group of surgeons.[9,10]

During the time when these surgical procedures were being developed, percutaneous blocks were also explored. The posterior paravertebral approach was first suggested by Kappis[11] in 1919, and is further described by Labat[12] and Adriani.[13] In 1925, Leriche and Fontaine[14] first used a paravertebral approach for sympathetic blockade with procaine on patients with severe pain due to angina pectoris, causalgia, and reflex sympathetic dystrophy. In 1926, Mandl[15] described paravertebral blocks for diagnosis and treatment of visceral pain and anginal pain. That same year, Swetlow[2] used 85% alcohol with the same technique. The patient was placed in a lateral decubitus position with the side to be injected up. The knees were flexed to the abdomen and the head bowed down. The ribs were used as landmarks, and the intercostal space to be injected was carefully palpated. The skin was marked over the spinous processes. At a 4-cm mark from the spinous processes, the skin was anesthetized with procaine hydrochloride. This was used as a marker, and to numb the skin. The needle was introduced perpendicular to the rib, just above the injection would be performed. The needle was advanced to touch the rib and then withdrawn in order to redirect the needle. At this juncture, the needle was advanced caudal, medial, and anterior at a 45-degree angle, and then advanced 2 cm from the lateral border of the rib. The needle point was between the internal and external intercostal muscles. It was then attached to a water manometer to determine whether it was in the pleural cavity. The absence of extensive oscillation with respiratory movement indicated that the needle was not in the pleural cavity. Then 2.5 cc of 85% alcohol was injected.[1,2] White and White[16] noted that in their experience, hyperesthesia occurred for 2–4 weeks in all of the patients injected with 85% alcohol over the area of the injected nerve. Leriche and Fontaine,[14] in 1932, published a case of inadvertent tracing of alcohol through the root sleeve into subarachnoid space, causing paraplegia.

Mandl[15] noted that the sympathetic ganglia at the thoracic level lie so close to the intercostal nerves that alcohol infiltrated around the sympathetic chain bathed the intercostal nerve trunks. At first the patients became paralyzed, but anesthesia disappeared within 14 days, and at approximately 4 weeks, hyperesthesia to the chest wall increased and commonly persisted for several months. This procedure was thus rejected by many because of the frequency of neuritis that occurred in these patients. Many abandoned chemical interruption of sympathetic fibers and looked for surgical methods because of intercostal neuritis, as well as other side effects, caused by percutaneous alcohol infection.

In 1979, Wilkinson[17] devised a technique for radiofrequency thermocoagulation (RFTC) via percutaneous needle placement for ablation of the T2 and T3 blocks with minimal complications.

ANATOMY

The preganglionic fibers of the thoracic sympathetics exit the intervertebral foramen along with respective thoracic paravertebral nerves. After exiting the intervertebral foramen, the thoracic paravertebral nerve gives off a recurrent branch that loops back through the foramen to provide innervation to the spinal ligaments, meninges, and its corresponding vertebra. The thoracic paravertebral nerve also interfaces with the thoracic sympathetic chain via the myelinated preganglionic fibers of the white rami communicantes and the unmyelinated postganglionic fibers of the gray rami communicantes. At the level of the thoracic sympathetic ganglia, preganglionic and postganglionic fibers synapse, and some of the postganglionic fibers return to their respective somatic nerves via the gray rami communicantes. These fibers provide sympathetic innervation to the vasculature, sweat glands, and pilomotor muscles of the skin. Other thoracic sympathetic postganglionic fibers travel to the cardiac plexus and course up and down the sympathetic trunk to terminate in distant ganglia.

The first thoracic ganglion is fused with the lower cervical ganglion to help make up the stellate ganglion. As the chain moves caudad, it changes its position, the upper thoracic ganglia lying just beneath the rib and the lower thoracic ganglia moving farther anterior to rest along the posterolateral surface of the vertebral body. The pleural space lies lateral and anterior to the thoracic sympathetic chain. Given the proximity of the thoracic somatic nerves to the thoracic sympathetic chain, the potential exists for both neural pathways to be blocked during blockade of the thoracic sympathetic ganglion.[18]

Yarzebski and Wilkinson[19] noted the discrepancies in the description of the location of sympathetic chain ganglia in anatomy textbooks. This led them to study the location of T2 and T3 sympathetic ganglia in 24 freshly embalmed adult cadavers. They noted that the locations of T2 and T3 vary. In the dorsoventral location, the T2 ganglia on the right side had a median location of 19 mm (range 12–31 mm) dorsal to the ventral surface of the vertebral body, and the left side had a median location of 17 mm (range 6–27 mm) dorsal to the ventral surface. The right-sided T3 median location was 20 mm (range 9–31 mm) dorsal to the ventral surface of T3 vertebral body. The left-sided T_3 median was 19.5 mm (range 9–30 mm) to the ventral surface (Figure 12-1A).

The relationship of the ganglia caudad and cephalad to the vertebral bodies was more constant. The median location of the T2 ganglia was 2-mm rostral to the midpoint of the T2 vertebral body on the right side (range 1–7 mm), between the head of the ribs. The left median was 1.5 mm (range 1–2 mm) rostral to the midpoint of vertebral body. The T3 ganglia were located 2 mm (range 2–3 mm) rostral to the midpoint of the T3 vertebral body bilaterally (Figure 12-1B).[19]

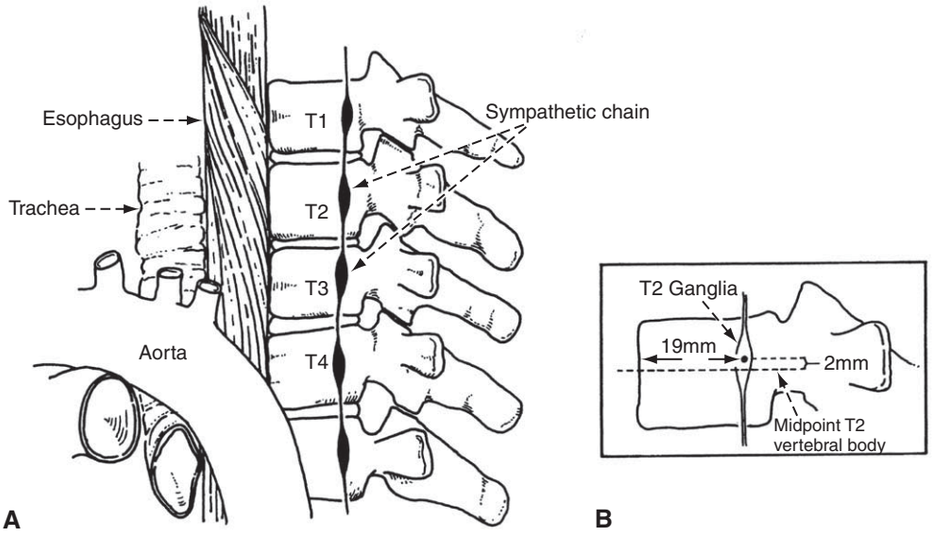


FIGURE 12–1

In this drawing, the thoracic sympathetic chain is approximately 20 mm dorsally from the anterior vertebral body. For practical purposes, this is equivalent to the posterior third of the thoracic vertebrae in the lateral view. (B) This close-up view shows the slightly (2-mm) rostral location of the thoracic sympathetic ganglion in relation to the midpoint of the vertebrae.

Sympathetic ganglion cell bodies that supply the upper limbs are in the intermediolateral horn of the spinal cord from the T2 to T8 levels. Preganglionic fibers run to the sympathetic chain via white rami communicantes. Here, they ascend cephalad and synapse with postganglionic fibers, primarily in T2, but also in T3, in the stellate ganglia, and in the middle cervical ganglia. By blocking T2 and T3, which are the "key" synaptic stations, all the synaptic nerves destined for the upper limbs can be blocked.[20]

INDICATIONS

The T2 and T3 sympathetic block is considered for patients who have sympathetically maintained pain or upper extremity vascular disease.

Indications for sympathectomies at the thoracic ganglia level are for treatment of CRPS I, CRPS II, neuropathic pain in thorax, chest wall, thoracic viscera, upper abdominal viscera, herpes zoster, postherpetic neuralgia, and phantom breast pain after mastectomy.[18] Other possible indications may include arterial occlusions leading to ischemia, drug-resistant Raynaud's disease, Buerger's disease, and frost injuries of the upper extremities.[1]

Sympathetic nerve blocks in this area can also provide analgesia of the intrathoracic viscera that may be involved in neoplastic or other painful processes. Neurolytic block of the sympathetic chain from T2 to T8 can be used in patients with severe intractable pain caused by cancer of the esophagus, heart, bronchi, trachea, lung, pleura, or by some other chronic pathologic process of the upper two-thirds of the esophagus. Destruction of this chain is indicated for palliation of pain syndromes that have responded to thoracic sympathetic blockade with local anesthetics.[1,18]

CONTRAINDICATIONS

Absolute
Local infection
Systemic infection
Coagulopathy
Relative
Thoracic aortic aneurysm
Respiratory insufficiency

EQUIPMENT

- 10-cm, curved blunt R-F RFTC needle, or 23-gauge SMK needle, 5-mm active tip
- 1-1/4-inch, 20-gauge IV introducer catheter
- IV T-piece
- Metal marking clamp
- 3-cc syringe
- 10-cc syringe
- 1-1/2-inch, 19-gauge needle
- 3/4-inch, 25-gauge needle

DRUGS

- Radiographic contrast: iohexol
- Local anesthetic (lidocaine, bupivacaine or ropivacaine), 5–10 ml
- Depot steroids (triamcinolone), 40 mg

PREPARATION OF PATIENT

Prior to the procedure, the patient should be cleared for any bleeding diathesis by obtaining prothrombin time, partial thromboplastin time, and bleeding time values, bleeding and coagulation times. This reduces the risk of hematoma formation into the chest cavity. Patients on any anticoagulants are discontinued 7 days prior to the procedure. Patients should be evaluated with respect to any anatomical distortions of the thorax prior to surgery.

Laboratory Studies

- CBC with platelets
- PT, PTT, platelet function studies
- Anteroposterior chest x-ray plain film

Preprocedure Medication

Use the standard recommended protocol for conscious sedation by the American Society of Anesthesiologists.

PROCEDURE

Position of Patient/Physician

The patient must be positioned prone decubitus with pillow under chest on a fluoroscopic-compatible table. The back is prepped and draped in a sterile fashion. Vertebral bodies C7-T1-T2-T3 must be visualized prior to the procedure. The fluoroscope is then used to identify the T2 vertebral body in an anteroposterior view. The fluoroscope is then obliqued approximately 20 degrees toward the ipsilateral side. The fluoroscope is then rotated in a cephalocaudad direction approximately 20 degrees. This helps open up the intervertebral space and "squares up" the T2 vertebral body (Figure 12-2).

Kelly's forceps are used to identify the point of skin entrance at the lateral edge of the T2 vertebral body just cephalad to the third rib. The skin is anesthetized using a 25-gauge, short, beveled needle with 1.5% lidocaine. A 16-gauge, 2-inch angiocatheter is advanced toward the lateral border at T2 above the third rib, in a tunnel view, with the aid of fluoroscopy.

A tunnel view is maintained to keep straight orientation in deeper tissues. The stylet is removed from the angiocatheter at about 1-inch deep. A 20-gauge, 10-cm, blunt curved, 10-mm active-tip RFTC needle is inserted. The needle is advanced using "direction-depth-direction" technique with the fluoroscope to confirm placement of the needle (Figure 12-3).

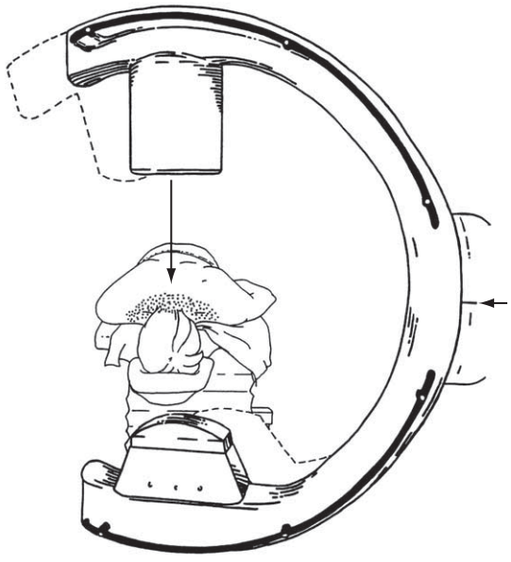


FIGURE 12–2

The drawing shows the arrangement of the patient and fluoroscope in an anteroposterior view to first identify the appropriate vertebral level. The lateral stippling demonstrates the rotation of the fluoroscope to create a tunneled view for radiographic needle placement.

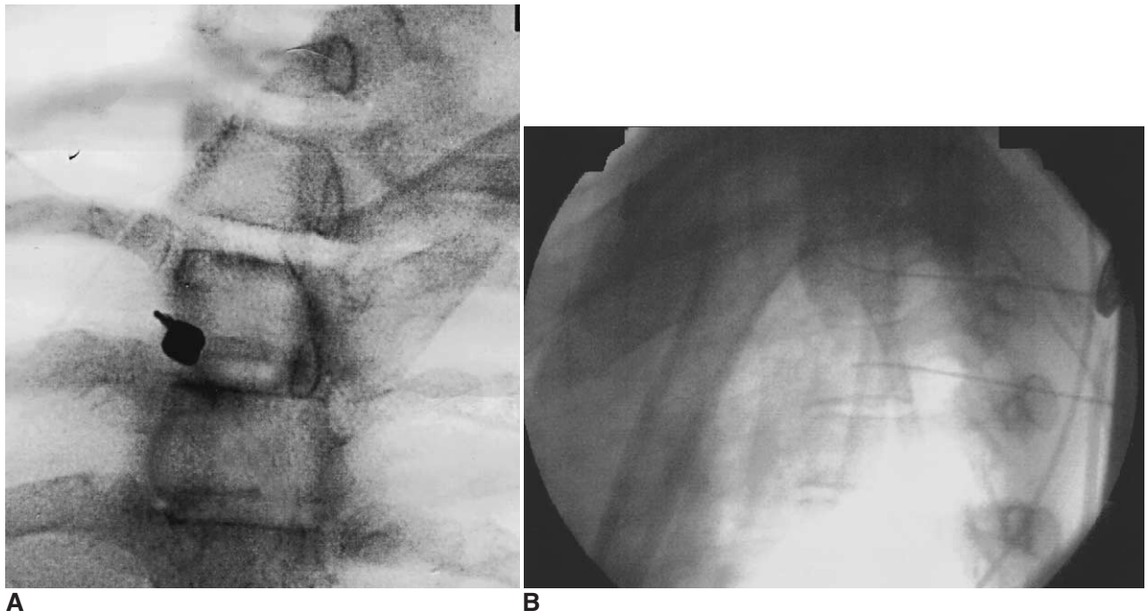
The needle is advanced, hugging the lateral edge of the T2 vertebral body. A lateral view confirms the posterior half of the T2 thoracic vertebral body (Figure 12-4A). An anteroposterior view will demonstrate the needle "hugging" the T2 vertebra at approximately the level of the pedicle (Figure 12-4B). Iohexol 240 dye (approximately 2 cc) is then injected. The dye spread is up and down the thoracic vertebral column, and unilateral placement is confirmed if the spread follows the dome of the lung, the needle is more lateral than the parietal pleura. The needle needs to be redirected medially.

If this is a diagnostic block, local anesthetic and steroid solutions are injected. Approximately 6–8 cc of a 1:1 mixture 0.5% ropivacaine and 2% lidocaine with 40 mg/cc of triamcinolone are injected. This volume is generally sufficient to block both the T2 and T3 sympathetic ganglia. This can be confirmed by watching the spread of dye before and after injection of local anesthetic.

Prior to the procedure, temperature probes are placed on each hand. A baseline temperature is noted and compared with postprocedural temperature. Fentanyl, midazolam, and lidocaine all produce vasodilation and affect sympathetic function for hours after the procedure. Bilateral temperature probes will help avoid confusion. The patient is then followed for 1 month after the diagnostic block. The duration of the block and the percentage of pain relief are noted.

Radiofrequency Thermocoagulation of T2 and T3 Sympathetic Ganglion

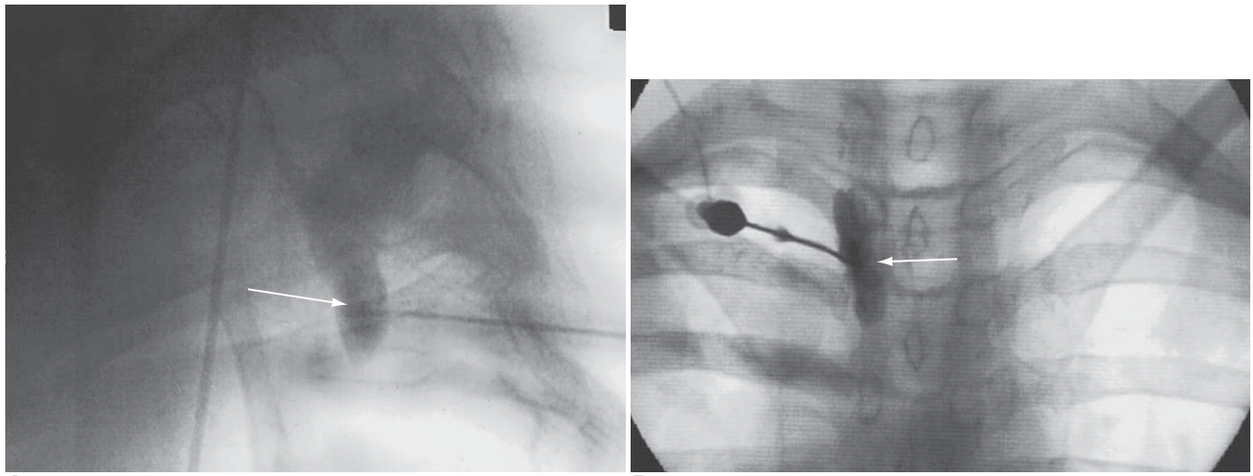
If the block was beneficial, the patient is then scheduled for an RFTC of the T2 and T3 sympathetic ganglia. The needle is placed as mentioned above at the T2 sympathetic ganglion. Iohexol dye is injected (Figure 12-5). Stimulation at 50 Hz and 2 V is used to record any stimulation of intercostal nerves. Prior to lesioning and after stimulation, local anesthetic and steroids are injected.



A B

FIGURE 12–3

(A) Oblique fluoroscope view of T2 and T3 vertebrae. The curved blunt needle is shown in a "tunnel" view at the T2 vertebral level (arrow). (B) Lateral view. The needle tip is at the midpoint to the posterior third of the thoracic vertebral body.



A B

FIGURE 12–4

(A) Lateral fluoroscopic view of T2 and T3 vertebrae. The contrast medium is seen over the vertebral body (arrow) without tracking of the contrast into the neuroforamina. (B) Anteroposterior fluoroscopic view. The contrast medium is seen "hugging" the thoracic vertebral body (arrow).

Thirty seconds is usually enough time to wait before lesioning occurs.

Lesioning occurs at 80°C for 90 seconds. When lesioning is complete, the needle hub is directed in a medial-caudad direction. The tip of the RFTC needle is curved approximately 15 degrees, and this gives a larger area of burn when rotated, increasing the chance of burning the ganglia. The process is repeated again for the T3 ganglia. At the end of lesioning, the needle and catheter are removed. The back is cleansed. A triple antibiotic ointment and bandage are placed over the injection site(s).

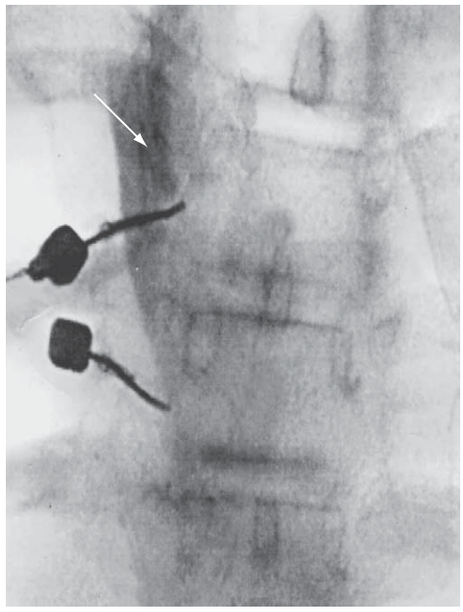


FIGURE 12–5

Anteroposterior fluoroscopic view. The two radiofrequency needles can be seen in place at the T2 and T3 vertebral levels. The arrow indicates the correct position of the needle tip on the T2 andT3 sympathetic chain.

COMPLICATIONS

The patient is transported to the recovery room. A chest radiograph is ordered to rule out pneumothorax, and the pain and temperature are rechecked. A quick evaluation is done to rule out any neurologic deficits. The patient is recovered in 45 minutes and sent home. The patient is advised of late-occurring pneumothorax, and instructed that if he or she has increased shortness of breath or chest pain, to go to the emergency room for evaluation.

Another side effect of this procedure is intercostal neuritis. Wilkinson[21] noted neuritis on approximately 40% of his procedures. This too can be minimized by meticulously performing sensory and motor stimulation prior to lesioning. If no dysesthesias or muscle contractions occur in the somatic nerve (intercostal) distribution, then one could deduce that the needle tip is at least 1 cm away from the nerve root. The data of the incidence of neuritis by this procedure are still scant and need to be further evaluated.

CLINICAL PEARLS

The lack of significant side effects in our group can be attributed to the use of a blunt, curved-tip needle, which seems to push nerves and arteries out of its pathway rather than injuring them. Staying within 4 cm of the spinous process is another safety factor that contributes to the success of the technique and alleviates pneumothoraces. During RF lesioning, intercostal stimulation during motor testing can be resolved by advancing the needle 2–3 mm anteriorly.

EFFICACY

Retrospective data on T2 and T3 were reviewed, using the Current Procedural Technology coding for T2 and T3 sympathetic block or RFTC. Charts that were on

microfilm were ignored, and charts that did not have a radiograph report or a dictated physician's note excluding a pneumothorax were also ignored. A total of 42 patients had 110 percutaneous thoracic sympathetic blocks or RFTC performed. Of these patients, 27 had CRPS I of the upper extremities; 1 had a brachial plexus injury; 1 had postmedication neuritis of the brachial plexus; 2 had phantom limb pain, 1 had Arnold-Chiari syndrome and deafferentation pain following dorsal root entry zone lesioning; and 4 had chest wall pain due to multiple reasons (1 following breast removal, 1 had costochondritis, 1 had status post–chest-tube placement, and 1 was unknown).

From these 110 procedures, 36 of the procedures were diagnostic blocks, 73 were T2 and T3 sympathetic RFTC blocks, and one was electromagnetic pulse frequency. A T4 block was done in eight of the blocks, T1 in one block, and T4 and T9 in another. A total of 193 needle punctures into the thoracic cavity were placed.

From the 110 procedures, there were two pneumothoraces. One was on a 57-year-old female with diffuse reflex sympathetic dystrophy. This was a diagnostic block and it caused a 10% apical pneumothorax. It was not realized at the time the radiograph was read by the anesthesiologist, but was confirmed by the radiologist at a later time. No treatment was necessary and the pneumothorax resolved on its own. The second pneumothorax occurred in a patient with deafferentation pain in the right upper extremity. This also was a diagnostic block at T2. The patient experienced a 20% pneumothorax on the right side. The patient's oxygen saturations were normal and no chest pain was noted. No chest tube was placed to treat the pneumothorax. It resolved on its own. The incidence of pneumothoraces in this group was 1.82%. Wilkinson[21] reported six pneumothoraces in 247 procedures, or an incidence of 2.4%. These were symptomatic pneumothoraces and required brief chest tube placement.

SPLANCHNIC NERVE BLOCKS

HISTORY

The first anterior percutaneous approach was when Kappis introduced splanchnic anesthesia in 1914[22] and followed it up in 1918[23] with the publication of a series of 200 cases. The recognition that splanchnic nerve block may provide relief of pain in a subset of patients who fail to obtain relief from celiac plexus block, has led to a renewed interest in this technique.

Interest in this technique has been regenerated by the introduction of the computed tomography (CT)–guided approach, and, recently, by the use of RF-produced lesions. Raj and associates reported good outcome with RF lesioning using the Racz-Finch curved blunt needles.[24]

The technique for splanchnic nerve block differs little from the classic retrocrural approach to the celiac plexus, except that the needles are aimed more cephalad in order to ultimately rest at the anterolateral margin of the T12 vertebral body.[25] It is imperative that both needles be placed medially against the vertebral body to reduce the incidence of pneumothorax. Abram and Boas[26] described a technique for splanchnic nerve block that used a paravertebral transthoracic approach. The needle was advanced to rest against the anterolateral aspect of the T11 vertebral body. In the Boas technique, the needles are bilaterally advanced 6 cm lateral to the midline of T11 intercostal space contacting vertebral body.

Despite neurolytic agents having been used widely for splanchnic blockade, Raj defined RF lesioning for more selective cases with fewer side effects. The predictable relationship of the splanchnic nerves to other structures allows for accurate needle placement and hence a low risk of iatrogenic damage. Other authors had different results in the application of splanchnic nerve blockades via various methods.

ANATOMY

There are three bilateral splanchnic nerves. The great splanchnic nerves arise from the roots T5/T6-T9/10. The lesser splanchnic nerves arise from T10/T11. The least splanchnic nerves arise from T11/T12. The great splanchnic nerves run paravertebrally through the thorax, crus of the diaphragm and enter the abdominal cavity ending in the celiac ganglion. The lesser splanchnic nerves pass parallel to the great splanchnic nerves and end in the celiac ganglion. The least splanchnic nerves pass through the diaphragm to the celiac ganglion. All three splanchnic nerves are preganglionic.[27]

The splanchnic nerves transmit the majority of nociceptive information from the viscera. These nerves are contained in a narrow compartment made up by the vertebral body medially and the pleura laterally, the posterior mediastinum ventrally, and the pleural attachment to the vertebra dorsally (Figure 12-6). This compartment is bounded caudally by the crura of the diaphragm. Abram and Boas[26] have determined that the volume of this compartment is approximately 10 ml on each side.

INDICATIONS

Indications for celiac plexus and splanchnic nerve local anesthetic blocks have been used as diagnostic tools to determine whether flank, retroperitoneal, or upper abdominal pain are sympathetically mediated with acute pancreatitis in chronic benign abdominal pain syndromes, such as chronic pancreatitis.[28] Most investigators report a lower success rate with this procedure for patients suffering from chronic nonmalignant abdominal pain than when treating abdominal pain of the neoplastic orgin.[29] The

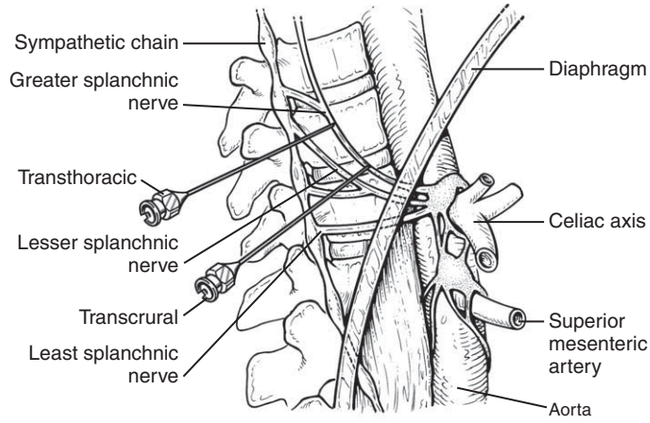


FIGURE 12–6

Drawing of the anatomic origin of the splanchnic nerves and its relation to other structures. (From Waldman SD, editor: *Interventional Pain Management*, 2nd ed. Philadelphia, Saunders, 2001, p. 503, with permission.)

most common indication is pain management of upper abdominal malignancies. Recently splanchnic nerve/nerves blockades have been applied solely or in conjunction with celiac blockade in patients with malignant or benign upper abdominal pain.

CONTRAINDICATIONS

Absolute
Local infection
Sepsis
Coagulopathy
Relative
Tumor distorting anatomy
Abdominal aortic aneurysm
Respiratory insufficiency, such as when unilateral pneumothorax adversely affects sustaining life
Pleural adhesions

EQUIPMENT

- RF machine
- 15-cm curved RF needle with 15-mm electrode tip
- 14-gauge, 4-cm extracath (for skin entry prior to RF needle insertion)
- One extension set to help manipulate needle and easy injection of solutions

DRUGS

- Two 10-ml plastic syringes with local anesthetic
- One 10-ml syringe with iohexol (contrast solution) to confirm correct placement of needle tip
- One 2-ml syringe with local anesthetic for skin infiltration
- Neurolytic agents: 75–100 % alcohol or 10% phenol

PREPARATION OF PATIENT

Laboratory Studies

- CBC with platelets
- PT, PTT
- Platelet function test or bleeding time
- Urinalysis
- Laxative to clear bowels
- Plain anteroposterior chest x-rays
- Abdominal/thorax CT when necessary

Preprocedure Medication

For preoperative medication, use the standard recommendations for conscious sedation by the American Society of Anesthesiologists.

All patients should have an intravenous catheter inserted in a large vein and securely anchored. A 500-ml solution of dextrose-Ringer's lactate should be started with at least 200 ml of solution infused prior to the lesion. Vital signs should be monitored throughout the procedure. Intravenous analgesic drugs should be available for use as needed. Sedation is used to relax the patient on an as needed basis, taking into account the physical status of the patient. The patient needs to be kept awake and should be able to answer reliably during the testing of sensory and motor stimulation.

PROCEDURE

The techniques used for splanchnic nerve block are (1) classical approach, (2) RF lesioning, (3) CT-guided anterior approach, and (4) transdiscal approach.

Patient Positioning

The patient lies prone in a comfortable position, taking care of the head and feet in particular. The position on the table should be such that the C-arm could

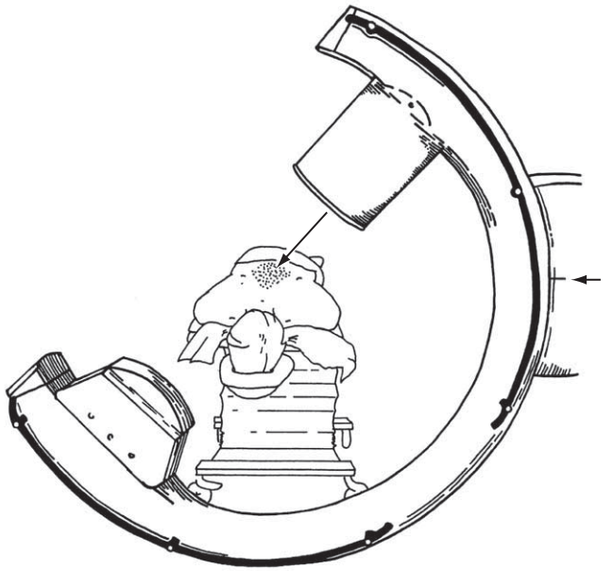


FIGURE 12–7
Drawing shows the fluoroscopic C-arm in oblique view for "tunnel" placement of the needle at the left T12 vertebral level.

be rotated to visualize the T10 to L3 level without difficulty (Figure 12-7).

Site of Needle Entry

In the prone position, the T12 vertebral body is identified in the posteroanterior view of the fluoroscope. Keeping a mark on the T12 or T11 vertebra, the C-arm is moved to an oblique position (Figure 12-8) (about 45 degrees). The edge of the diaphragm lateral to the vertebral body is viewed. Its movement during inspiration and expiration are noted. If the diaphragm shadows T12 vertebra and its rib, then the T11 rib is identified. The point of entry for both levels is at the junction of the rib and vertebra. Skin infiltration is made at this point.

Technique of Needle Entry

With the oblique fluoroscopic view still in place, a 14-gauge, 5-cm extracath is inserted, such that the catheter traverses toward the target as a pinhead. When two-thirds of the extracath is inserted, the stylet is removed and the radiofrequency needle is inserted. The oblique view of the fluoroscope is maintained. An extension tubing is attached to the needle. With short thrusts of 0.5 cm at a time (Figures 12-9), the tip of the needle is advanced anteriorly, keeping in mind that the needle stays hugging the lateral aspect of the T11 or T12 vertebral body (Figures 12-10), close to the costovertebral angle. After advancing 1–1.5 cm anteriorly, the lateral fluoroscopic view is taken. In the lateral view, the needle is advanced until it reaches the junction of anterior one third and posterior two-thirds of the lateral aspect of the vertebral body, then aspirated for fluid, which could be blood, cerebrospinal fluid. Views are taken to confirm the final position of the curved needle on the vertebral body (Figure 12-11). Iohexol is injected to note that the solutions in anteroposterior and lateral views hug the spine (Figure 12-12).

Neurolytic Block

Smaller volumes (12 to 15 ml) of absolute alcohol are recommended for single-needle procedures.[30] Many investigators believe that alcohol, as a neurolytic agent, is superior to phenol in duration of neural blockade; however, alcohol has the disadvantage of producing transient severe pain on injection.[31]

Some clinicians have recommended the use of 6–10% phenol for splanchnic nerve block.[11] An advantage of phenol over alcohol is that it can be combined with contrast solution. The combination allows radiographic documentation

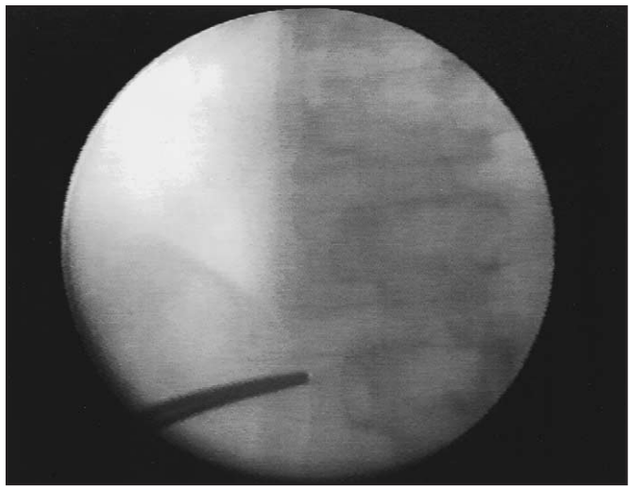


FIGURE 12–8
Radiographic oblique image shows the marker on the skin entry site for the "tunnel" view approach.

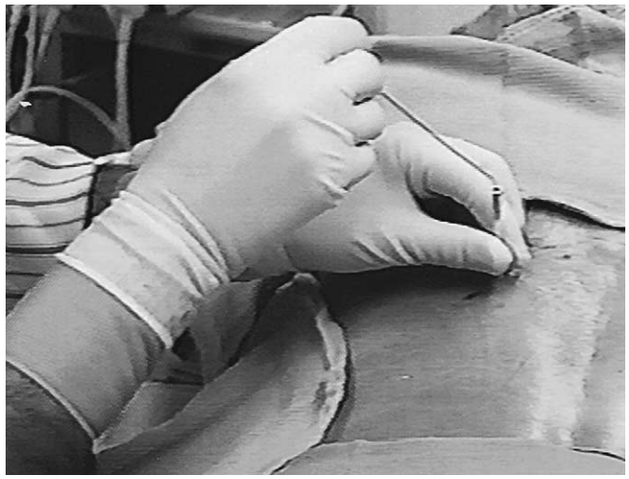


FIGURE 12–9
Insertion of a radiofrequency needle through the angiocatheter.

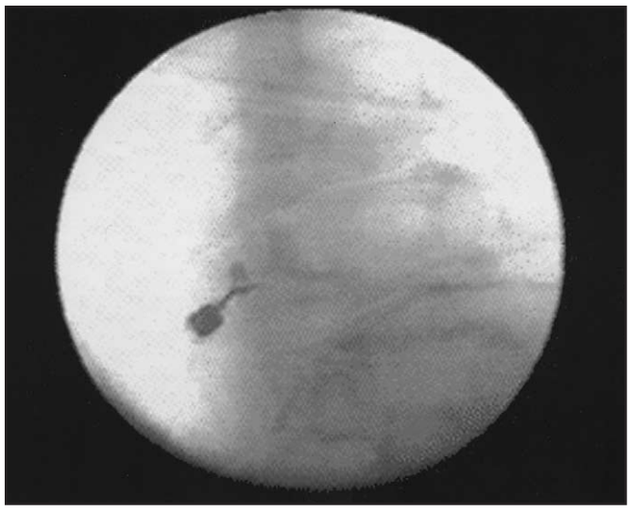


FIGURE 12–10
Oblique radiographic image of the radiofrequency needle in position.

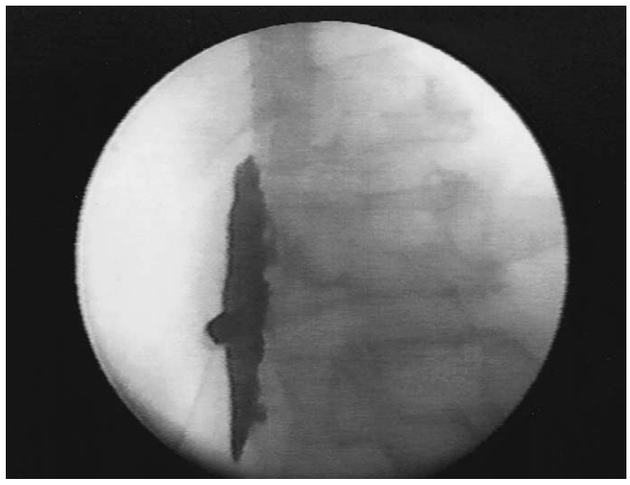


FIGURE 12–12
Anteroposterior radiographic view shows the needle in position and spread of the contrast medium.

of the distribution of the neurolytic solution. Mixtures of 10% phenol and iodinated contrast medium (iohexol) remain stable for up to 3 months.[32] The fact that phenol is not commercially available and must be prepared for each patient by a pharmacist is a disadvantage. The apparent greater affinity of phenol for vascular rather than neurologic tissue also represents a theoretic disadvantage, in view of the vascularity of the region surrounding the celiac plexus and splanchnic nerves.[33] Some investigators believe that phenol produces a block of shorter duration than alcohol, making it a less desirable agent for the intractable and progressive pain of malignant origin. It is important to note that comparative studies between alcohol and phenol are not available.

Radiofrequency Lesioning of Splanchnic Nerves

Because splanchnic nerves are contained in a narrow compartment, it is accessible for RF lesioning. To produce a lesion of the splanchnic nerve, the needle needs to lie on the mid-third portion of the lateral side of the T11 or T12 vertebral body (Figures 12-14 and 12-15). To approach this region, a curved 15-cm needle with a 15-mm lesion tip is recommended. The needle should remain retrocrural and posterior to the descending aorta; hence, safely away from the aorta. Theoretically then, it is possible to produce a safe and reliable RF lesion of splanchnic nerves.

Test Stimulation

Once the needle is in place, a 15-cm electrode is introduced through the RF needle (Figure 12-13). The electrical circuit is tested. The impedance is noted. It should be below 250 ohms. At 50 Hz, the sensory stimulation is conducted up to 1 V. The patient may report that he or she feels stimulation in the epigastric region. This is typical and satisfactory. If the stimulation is in a girdle-like fashion

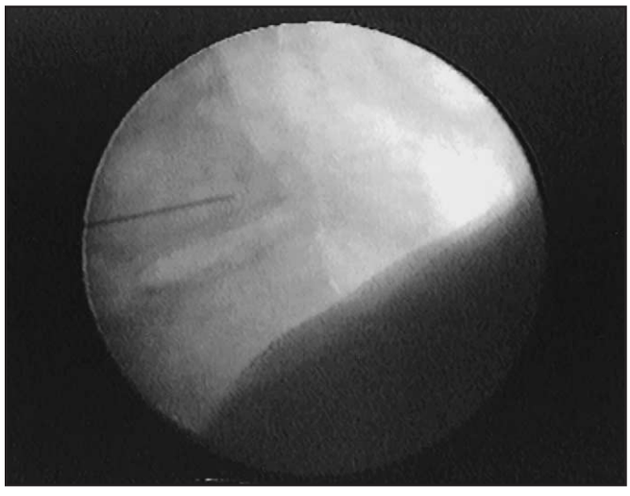


FIGURE 12–11
Lateral radiographic view of the needle against the T12 vertebral body.

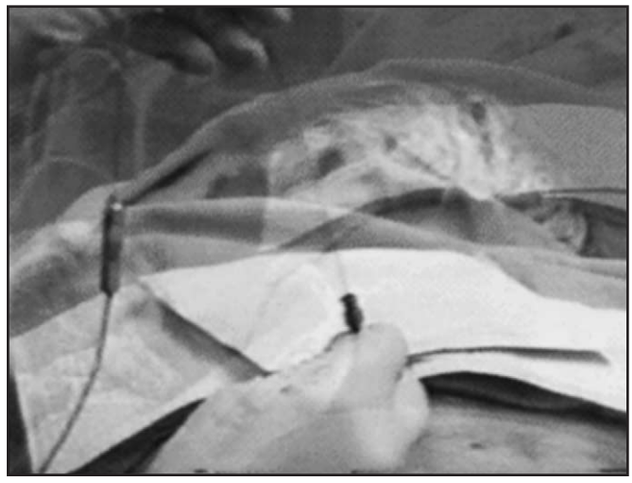


FIGURE 12–13
An oblique radiographic view shows insertion of the angiocatheter using the "tunnel" approach.

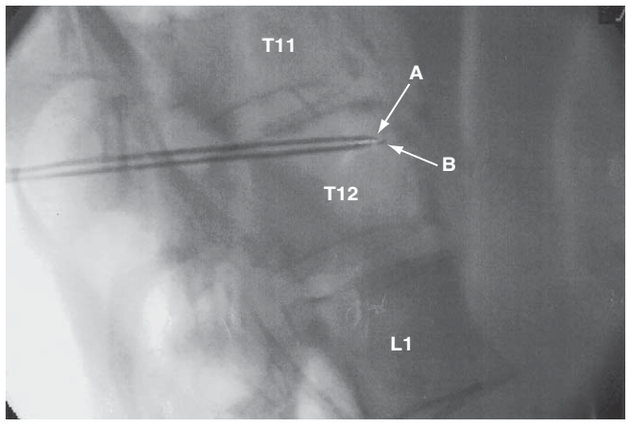


FIGURE 12–14

Lateral radiographic view should confirm correct needle placement for bilateral T12-level radiofrequency thermocoagulation. Arrows A and B indicate tips of the radiofrequency needles. Note the active part of RF needle is over the middle 43 of the lateral position of the vertebral body.

around the intercostal spaces, the needle needs to be pushed anteriorly. At 2 Hz, motor stimulation is done up to 2 V. One tries to palpate or see the intercostal muscle contraction. If this is negative, then test stimulation is satisfactory. The next step is to produce the RF lesioning. Two ml to 5 ml of local anesthetic (ropivacaine 0.5%) with steroid—40 mg of triamcinolone—is injected through the RF needle.

After waiting 1–2 minutes, the physician creates an RF lesion with settings set for 90 seconds at 80°C. The second lesion at the same setting is done turning the RF needle 180 degrees. If the procedure is for the bilateral neurolysis, then the same procedure of testing and lesioning is done on the opposite site.

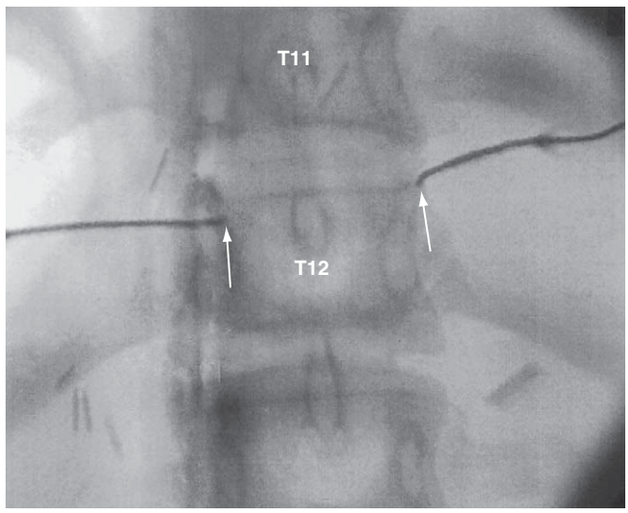


FIGURE 12–15

An anteroposterior view of the radiofrequency thermocoagulation needles against the vertebral body confirms its correct position. Arrows indicate tips of the radiofrequency needles.

CLINICAL PEARLS

The tip of the curved RF needle should face laterally initially until it passes the foramen. Then the tip can be turned medially once it reaches the lateral surface of the vertebral body. This ensures the needle remains medial to the interpleural surface and in close contact with the vertebral body. Watch for spread and dispersion of contrast material, especially in a blood vessel. A fluoroscopic oblique view ensures medial direction of the needle. A lateral view ensures that the needle stays posterior to the aorta and anterior to the foramen.

Prior to lesioning, the injection of a local anesthetic helps in reducing the discomfort due to the RF lesioning and decreases pain immediately after the procedure. Steroids help in treating the occasional occurrence of neuritis by reducing edema and inflammation of the lesioned structures.

CT-Guided Anterior Approach

Mercadante[34] defined the anterior approach. In the supine position, CT images of the abdomen (5-mm slice thickness) were obtained at the T12-L1 level. The skin is draped sterilely, and the site of puncture is infiltrated with lidocaine 2%. A 20-cm needle with a stylet is inserted and CT images are obtained to assure correct needle direction passing through the aorta-vena cava space to reach the retrocrural area on the right. On the left, the best trajectory is chosen to avoid organ perforation and to place the tip of the needle in the left retrocrural area. Two milliliters of contrast medium is injected through both needles to confirm the proper spread.

With CT scanning (5-mm slice thickness), the celiac trunk is localized. One needle is inserted perpendicular. Kidneys and adrenal glands are avoided. Half a milliliter of nonionic contrast medium is applied, and, after controlling the position of the contrast medium, 20 ml of neurolytic solution is injected (1 ml contrast, 12 ml alcohol 96%, and 6 ml of lidocaine 0.5%). CT scan always provides an accurate localization, even with a tumor mass in front or a very large cystic liver. Passage of visceral structures is not associated with relevant complications.

Transdiscal Approach

The patient was placed in the prone position with a pillow beneath the chest/abdomen to facilitate opening of the interdiscal space. The T11-T12 interdiscal space was identified under fluoroscopy. Next the fluoroscopy is placed in an oblique fashion and angled at 15–20 degrees or more for obtaining the best image of the disc to align the inferior endplates. In order to do so, cephalocaudal trajectory is needed. The entry point was approximately 3–5 cm from the midline.

After local anesthetic infiltration of the skin and the subcutaneous tissues with 2% lidocaine, a 22-gauge, 10-cm needle was introduced by tunnel vision lateral to the inferior aspect of the facet joint. The needle was advanced through

the disc. While entering the disc, 0.5 ml of contrast solution was administered to verify needle position within the disc by lateral and anteroposterior views. The needle was then advanced further under lateral fluoroscopic control, and a 5-ml syringe with saline was attached for loss of resistance. When the needle passed outside the T11-T12 interdiscal space, 3 ml of contrast was administered to verify its final position. The dye was spread with a direct line image (in a vertical plane) at that position (Figures 12-7 and 12-8).

Five milliliters of 10% aqueous phenol is given through the needle followed by 0.5 ml of air before drawing back to prevent the spread of the neurolytic solution within the disc material.

While further drawing back the needle, cephazolin 50 mg in 1 ml was administered to the disc to prevent discitis. One gram of cephazolin as a prophylactic antibiotic had been given intravenously 30 minutes before the procedure.

COMPLICATIONS ASSOCIATED WITH CELIAC PLEXUS AND SPLANCHNIC BLOCKS

Complications of splanchnic and celiac plexus blocks can be regarded as minor, moderate, or severe. Those that are relatively common (e.g., hypotension, gastrointestinal hypermotility, pain during injection) is of minor significance and is readily reversible. Complications of a moderate nature (e.g., pneumothorax) occurs infrequently, and although generally reversible, entail management that is more demanding, in that they may require hospitalization and additional procedures. It is important that patients should be re-evaluated with anteroposterior/lateral chest x-rays after the procedure to rule out pneumothorax.

Complications of the most severe nature (e.g., paraplegia, damage to blood vessels) are rare, but are rarely associated with recovery. The risks of the splanchnic nerve block are similar to those of the celiac plexus block. Apart from the common risks associated with celiac and splanchnic nerve blocks, the rates of pneumothorax, thoracic duct injury, and inadvertent spread of the injected drug to the somatic nerve roots are higher for the splanchnic nerve block than for the celiac plexus block. Serious complications rarely occur from either nerve block. Because of the close proximity of vital structures coupled with the use of large volumes of neurolytic drugs, side effects and complications may occur.[24,35]

EFFICACY

Since the definition of splanchnic nerve blockade, various techniques and drugs have been evaluated for complications, quality of life, and drug consumption. Garcia[36] studied 10 patients with RF of splanchnic nerves taking into consideration of the pain levels, anxiety, quality of life, and mood. Although the patient number is small, all the parameters associated with long-term debilitating chronic pain were improved. Ozyalcin at al.[37] compared the survival rate and quality of life in patients with pancreatic cancer treated either with celiac plexus blockade or splanchnic nerve blockade. They found splanchnic nerve blockade with neurolytics superior to celiac plexus blockade on the basis of survival rates, quality of life, and side effects. Phan et al.[38] studied the correlation of splanchnic nerve block efficacy and cancer staging. They found that splanchnic nerve block effectively helped control pain in patients with pancreatic and GI malignancies, producing significant decreases in pain and MEDD. However, staging of cancer did not significantly predict procedure efficacy. Mercadante et al.[34] defined the anterior approach to splanchnic nerve block under CT guidance with neurolytics where there is distorted anatomy in patients to increase affectivity and decrease side effects.

Plancarte-Sánchez[39] used percutaneous transdiscal splanchnic nerve blockade under tomographic control in 64 patients, aiming to reduce possible complications due to nerve blockade. Side effects included dyspnea, 5%; hypotension, 26.7%; nausea, 31.7%; and diarrhea, 83.3%. Neither morbidity (which was minor) nor efficacy (70–80% immediate success and 60–75% persistence of effect until death) correlated with anatomic technique. Splanchnic nerve block maintains a deservedly meaningful role in the armamentarium of the contemporary pain specialist. Despite a dearth of scientifically determined outcome data, even the most critical observer is nearly certain to acknowledge the therapeutic value of these techniques in patients with viscerally mediated abdominal and/or back pain or neoplastic origin, especially early in the course of established disease. For patients with longer life expectancies, the role of celiac/splanchnic neural blockade is increasingly recognized as modest, on other than a diagnostic basis. Despite daunting logistic and ethical methodological barriers, there is a pressing need to design and undertake collaborative controlled trials aimed at better determining the relative value of various technical approaches.

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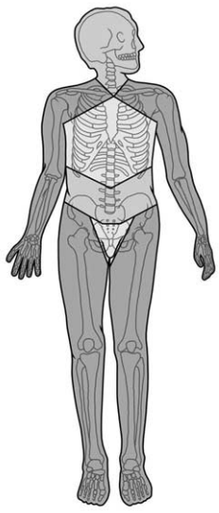
C H A P T E R



13 Spinal Neuroaxial Blocks

RICARDO RUIZ-LOPEZ, CARMEN PICHOT, AND P. PRITHVI RAJ

THORACIC EPIDURAL INJECTION AND CATHETER PLACEMENT

HISTORY

Accessing the epidural space was first described in 1921. The initial reports mostly described epidural catheter placement for the management of flailed chest, post-CABG pain, and post-thoracotomy pain. Chronic pain management via epidural space access was reported for epidural steroid injections and dorsal column stimulation, among other procedures.

ANATOMY

The thoracic epidural space extends from the lower margin of the C7 vertebra to the upper margin of L1. The vertebral column in the thoracic area normally has a kyphotic curvature with its apex at approximately T6. Slight scoliosis to the right can occur, even in normal persons. Significant scoliosis is associated with the rotation of the vertebral column, which can produce significant technical difficulty in performing this block. The inclination of the spinous processes is different at different levels of the thoracic vertebral column. The vertebrae from T1-T4 have very little inclination, whereas those of T5-T8 tilt significantly downward, making a midline approach to the epidural space practically impossible. The T9-T12 spines point dorsally without significant inclination, so the midline approach is possible. The ligamentum flavum is not as thick as it is in the lumbar spine, and, occasionally, the epidural space can be entered without encountering much resistance. The attachment of the ligamentum flavum to the lower margin of the lamina on its inner aspect reduces the size of the epidural space, whereas the space is wider at the upper margin of the lamina, because the ligamentum flavum is attached to the outer aspect of the upper margin of the lower lamina (Figures 13-1, 13-2, and 13-3). The epidural space is 3–4 mm wide in the thoracic area. The thoracic epidural space, just like the rest of the epidural space, contains loose areolar tissue, fat, and vertebral venous plexus.[1–5]

INDICATIONS

- Surgical
- Postoperative analgesia
- Herpes zoster and post herpetic neuralgia
- Epidural steroids
- Acute pain secondary to trauma
- Angina
- Cancer pain
- Spinal cord stimulation
- Management of acute pancreatitis[1–6]

CONTRAINDICATIONS

Absolute
Local infection in the area of needle insertion
Coagulopathies
Uncorrected hypovolemia
Relative
Septicemia
Distorted anatomy

EQUIPMENT

- Tuohy epidural needle or similar
- When applicable, special needles for epidural andelectrical stimulation catheter
- 25-gauge, 3/4-inch infiltration needle
- 3-cc syringe
- 10-cc syringe
- Loss-of-resistance (LOR) syringe

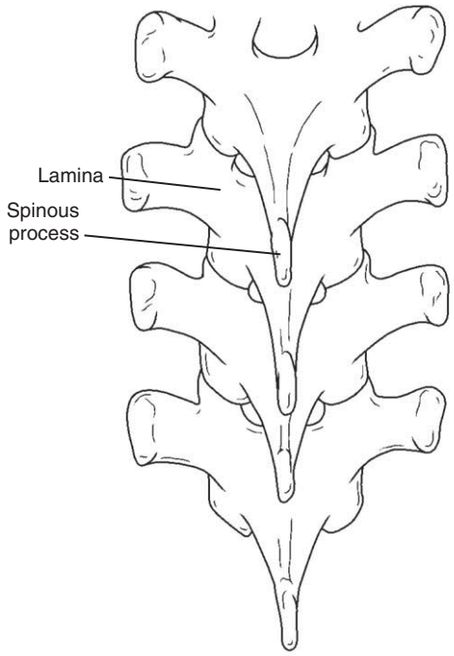


FIGURE 13–1
The posterior view of the thoracic spine showing the vertebra close to each other with large spinous processes overlapping the vertebra below.

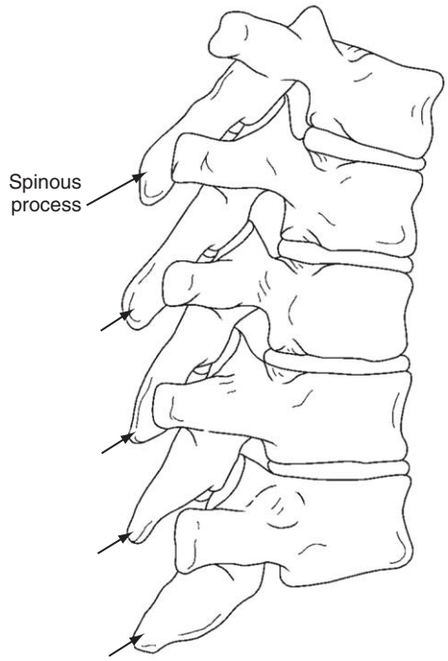


FIGURE 13–2
The lateral view of the thoracic spine showing the vertebra discs, foramina, and the vertebral canal surrounded posteriorly by the lamina and the spinous processes.

- When applicable, epidural catheters and electrodes
- IV T-piece extension

DRUGS

- 1.5% lidocaine
- 2% lidocaine
- 0.25–0.5% bupivacaine and ropivacaine
- Steroids
- Preservative-free normal saline (PFNS)

PREPARATION OF PATIENT

Physical Examination

Examining the local area for infections and anatomy-distorting factors such as previous surgery provides guidance.

Laboratory Studies

- CBC with platelets
- Prothrombin time and partial thromboplastin time
- Platelet function studies/bleeding times

Preprocedure Medication

Use the standard recommended protocol for conscious sedation by the American Society of Anesthesiologists.

PROCEDURE

Positioning of Patient

Placement of the thoracic epidural catheter can be done with the patient sitting or in the lateral decubitus position. The sitting position provides better alignment of the skin midline to the spine and facilitates identification of landmarks. The procedure can also be done with the patient prone (Figure 13-4).

Midline Approach

The midline approach (Figure 13-5) is applicable in the upper part of the thoracic spine between C7 and T5 and in the lower part, including T9-L1, since the spinous processes project directly posteriorly and are horizontal. The level of the spinous process corresponds to the level of the vertebra. The epidural technique is similar to that used in the lumbar areas, with a 90-degree approach, starting at the lower part of the interspace, just above the lower spine, so that the needle is angled cephalad. This facilitates insertion and advancement of the catheter. The desired level of entry is determined using fluoroscopy and a radiographic marker in the anteroposterior (AP) view. After choosing the desired intralaminar level, the ideal skin entry site is about 1 to 1-1/2 levels more caudal. Dermal infiltration with lidocaine is performed using a short 25-gauge needle. Injection of a local anesthetic with a slightly longer needle, such as a 1.5-inch, 22-gauge needle, into the paraspinal muscles on either side of the spine provides significant

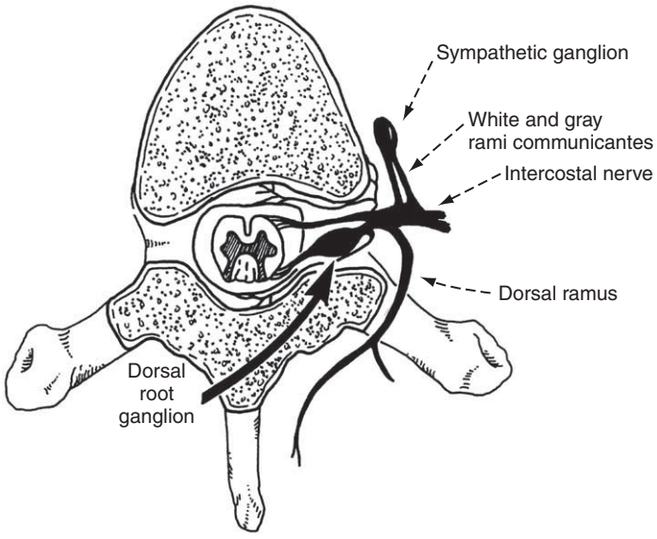


FIGURE 13–3
Drawing of the coronal cross-section of thoracic vertebra demonstrating its shape and contents.

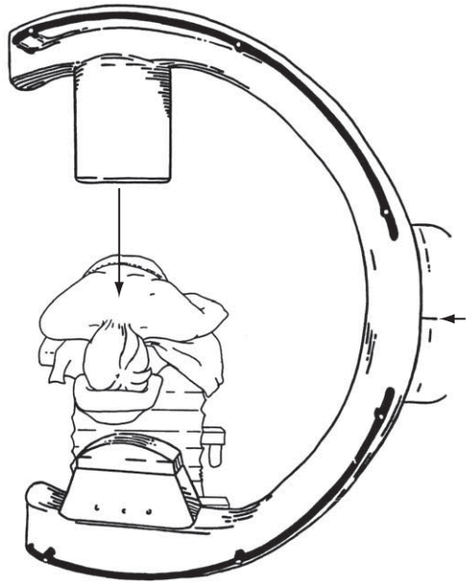


FIGURE 13–4
Drawing of the C-arm in position for anteroposterior view with the patient in the prone position.

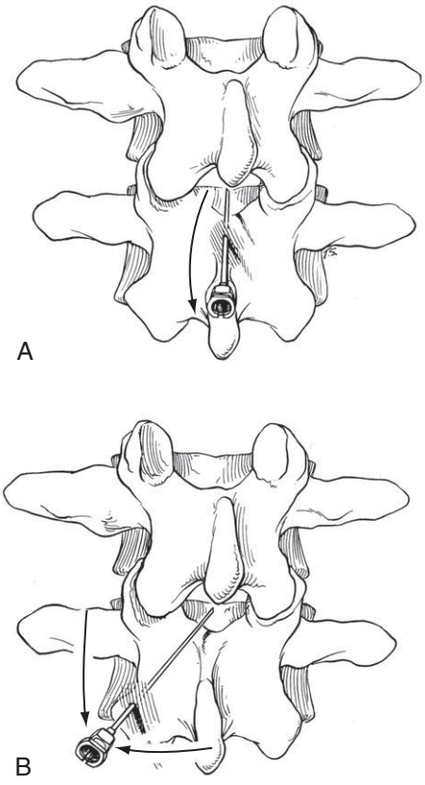


FIGURE 13–5
Angle and the direction of the needle for the median (A) and paramedian (B) approaches for spinal and epidural techniques.

analgesia for the procedure by blocking the nerve fibers as they come from lateral areas toward the midline. The 16- or 18-gauge, 3-1/2-inch Tuohy needle is advanced with the bevel cephalad so that the smooth part of the needle will bounce off the lamina. The needle is advanced through the skin, subcutaneous tissue, supraspinous ligament, and intraspinous ligament, in the AP view. As the interlaminar space is approached, the C-arm is switched to a lateral view, posterior to the bony canal (Figure 13-6).

The loss-of-resistance technique uses an air- or a fluid-filled syringe that contains a small bubble of air to allow compression (since a liquid is not compressible) (Figure 13-7). If a liquid is used, the authors prefer 0.9% saline without preservatives. The hanging-drop technique has been used, especially in the thoracic area, because of the significant negative pressure. Despite a low incidence of dural puncture, the drop is sucked in only 88% of the time. Since both hands are used to slowly advance the

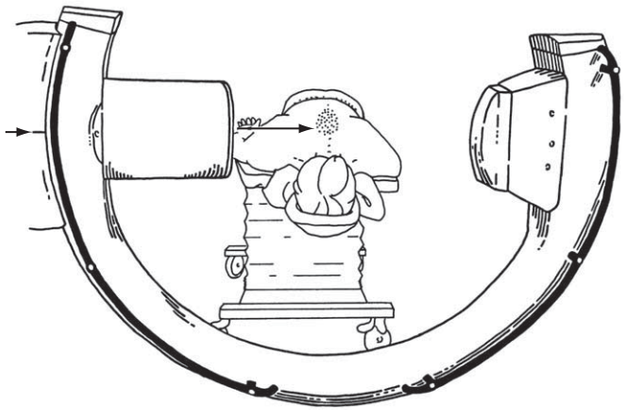


FIGURE 13–6
Drawing of the C-arm in position for lateral view with the patient in the prone position.

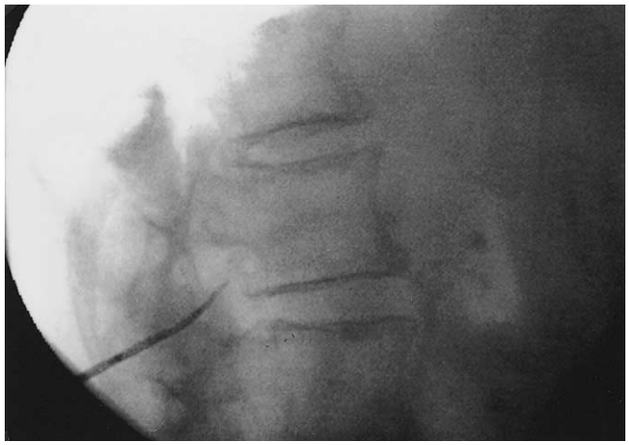


FIGURE 13–8
Radiographic imaging showing lateral view of the spine, confirming that the needle has entered the epidural space.

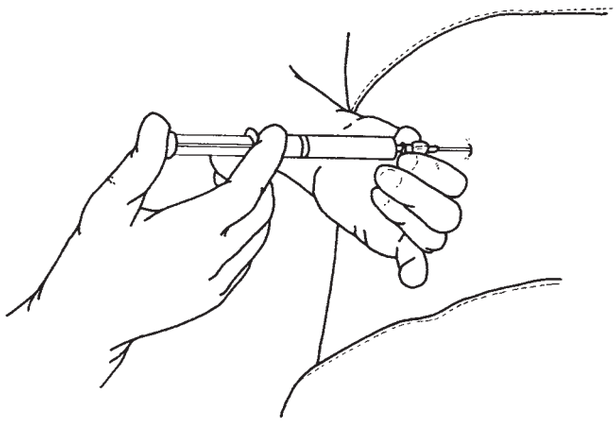


FIGURE 13-7
Line drawing showing the loss-of-resistance technique to enter the epidural space.

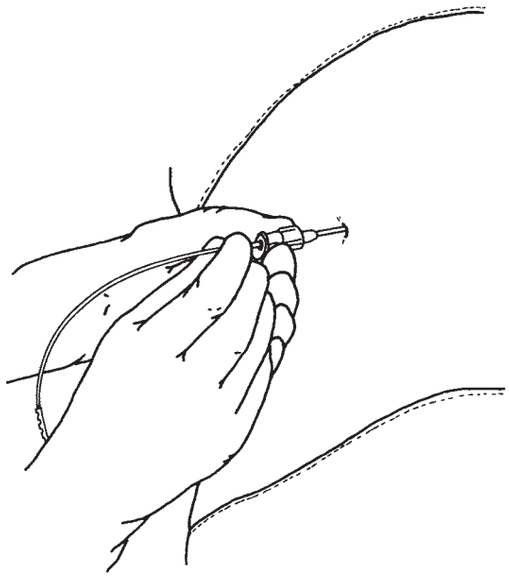


FIGURE 13–9
Line drawing showing placement of the catheter through the needle in the epidural space.

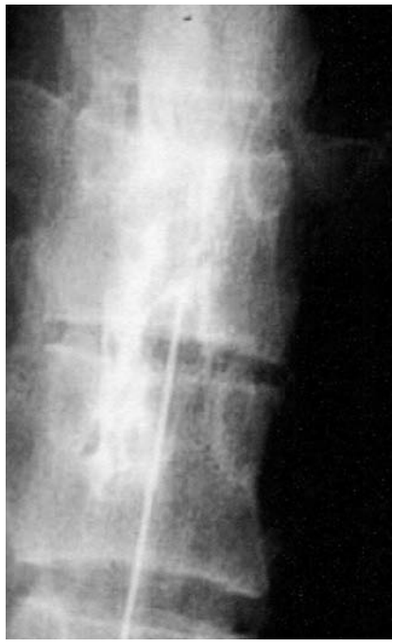
needle, entry into the epidural space is recognized even when the drop is not sucked in.

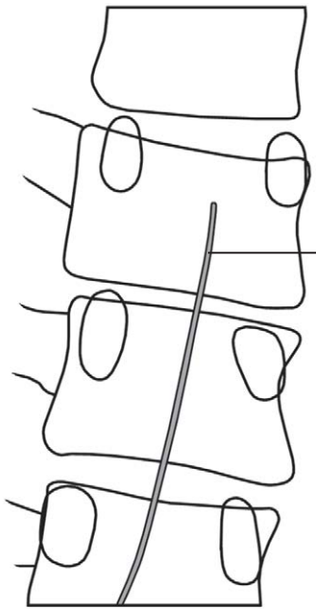
After the epidural space is entered, 1–2 ml of nonionic water-soluble radiographic contrast is injected to confirm epidural entry and to avoid intravascular or intrathecal injection (Figure 13-8). A mixture consisting of 40–80 mg of triamcinolone diacetate or methylprednisolone, 1 ml of 0.25% levobupivacaine, and 2 ml of preservative-free normal saline can be injected. After completion of the bolus injection, the needle is removed and a bandage applied over the skin entry site.

Catheter Placement

Once entry into the epidural space is confirmed, a catheter is advanced 3–4 cm for continuous infusions or phenol injection (Figure 13-9). As in any epidural technique, the catheter should not be withdrawn after it passes the tip of the needle, as the catheter may be sheared off. Inserting the catheter too far may result in migration through the intervertebral foramen, epidural vein, or true knot formation. To be absolutely sure that the catheter is in the epidural space, anteroposterior and lateral radiographic images are taken (Figures 13-10 and 13-11). Tunneling the catheter for 5 cm using another epidural needle reduces the risk of catheter migration in long-term infusions.[7]

The technique described by Raj[8] for taping the catheter, using Steri-Strips, Mastisol, and Tegaderm, can be employed instead of suturing. This technique reduces the

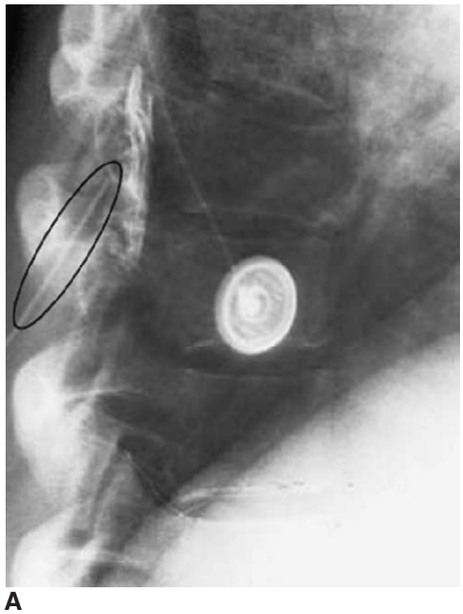


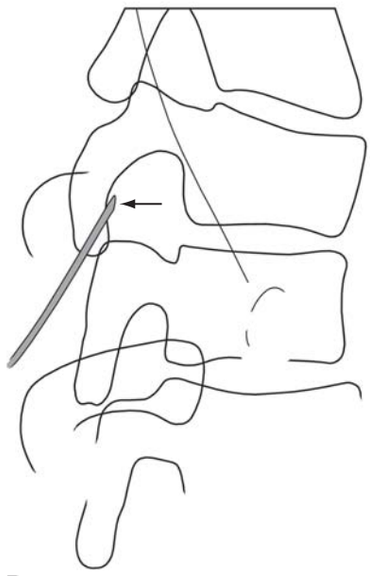


A B

FIGURE 13–10

(A) Radiographic image (courtesy of M. Furman) and (B), line drawing of the anteroposterior view of the thoracic spine showing placement of the catheter in the thoracic epidural space.





A B

FIGURE 13–11

(A) Radiographic image (courtesy of M. Furman), and (B) line drawing of the lateral view of the thoracic spine showing placement of the catheter in the thoracic epidural space.

possibility of catheter dislodgment and facilitates maintaining the catheter for a longer period of time. The catheter is connected to an adapter, a filter, and an injection site and then taped over the infraclavicular area to afford easy access for reinjection.

Single-Shot Epidural Injection

This is indicated for surgical procedures of 2–3 hours duration for thoracoabdominal surgery. Local anesthetics (lidocaine and/or bupivacaine or ropivacaine) are commonly used. A single-shot injection of local anesthetic steroid (bupivacaine or ropivacaine with steroids) has commonly been used for patients suffering from acute herpes zoster in the thoracic dermatomes.

Continuous Infusion via Thoracic Catheter

This is indicated for postoperative management of thoracic surgical patients and is very widely used. It is also used for chronic pain conditions such as chest wall pain secondary to trauma, pancreatitis, terminal cancer patients, and refractory

postherpetic neuralgia. It is important to remember that the catheter is always kept sterile, well anchored, and the delivery of the drug is in a closed circuit (Figure 13-12A). The dermatomes that have analgesia are shown in Figure 13-12B. The current technique of patient-controlled analgesia has become very popular for such cases. Local anesthetics and narcotics are mixed commonly in much lower concentrations than single-shot injections. Continuous infusion drugs seem to be synergistic, and hence continuous monitoring is required to prevent weakness and anesthesia in days following the start of the infusion.

COMPLICATIONS

The complications of the thoracic epidural technique are similar to those of the lumbar epidural block–infection, epidural hematoma, injury to the nerve roots, intravascular injection, respiratory depression, and subdural and subarachnoid injection, among others. But the presence of the spinal cord in the thoracic vertebral canal brings in the possibility of spinal cord damage. The incidence of spinal cord damage due to attempted thoracic epidural analgesia is not known. There are very few reports of this complication. In one series of 1071 postoperative patients, no long-term serious complications were reported.[9] In a study of 4185 patients, absence of serious neurologic complications was documented.[10] A case of accidental pleural puncture and placement of the catheter in the pleural cavity has been documented.[11] Although uncommon, this complication can be life threatening if not recognized. Many studies document safety and absence of infection.[12,13]

CLINICAL PEARLS

1. Since the spinal cord is present in the thoracic vertebral level, a thoracic epidural technique should be attempted only by an operator who has extensive experience doing lumbar epidural blocks. Bromage[4] recommended that a person who performs a thoracic epidural block should have done at least 50 consecutive lumbar epidural blocks without a dural puncture or a complication.
2. Because of the inclination of the spine in the mid-thoracic area, the technique could be technically difficult, although it can be mastered with some practice.
3. Because the nerve roots contain the sympathetic nerves to the heart, a block of these fibers can produce significant bradycardia and hypotension.

Intercostal muscle weakness resulting from thoracic epidural block can produce significant difficulty, especially in obese patients and those with respiratory impairment. In a person with impaired function of the diaphragm, chronic obstructive lung disease, or obesity, intercostal paralysis can significantly contribute to respiratory impairment.

EFFICACY

No double-blind, random controlled studies can be found. On an individual basis, it has shown itself to be useful.

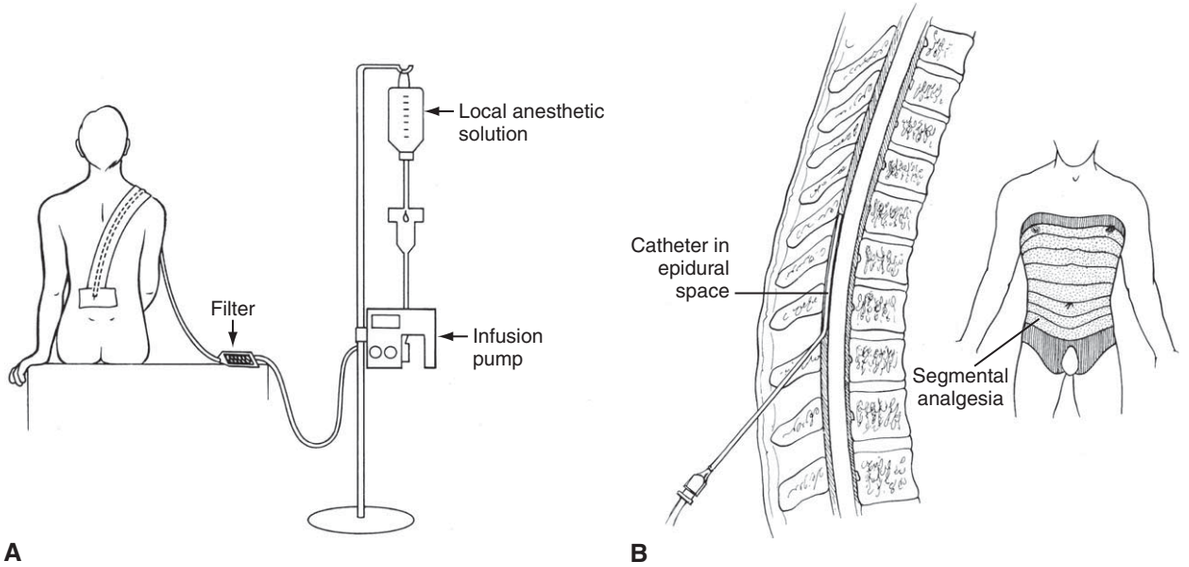


FIGURE 13–12

(A) Diagram showing an arrangement used for continuous epidural analgesia. (B) Drawing of the thoracic spine the catheter in place and the extent of dermatomes that can be anesthetized using a thoracic epidural approach.

DORSAL ROOT ENTRY ZONE LESIONING

HISTORY

Brachial plexus injuries are followed by chronic pain in the deafferentated area in 30–90% of patients. In groups of patients with predominantly preganglionic lesions, that is, root avulsions, as many as 90% had severe pain.[14] The incidence of central disabling pain after spinal cord and/or cauda equina injuries varies among the various reported series from 1 to 25% of patients.[15]

In 1967, Loeser and Ward[16] reported that neurons in the dorsal horn became hyperactive when deafferentated. The surgical procedure was designed to selectively destroy the nociceptive fibers grouped in the lateral bundle of the dorsal rootlet, but in practice the lesions destroy not only the substantia gelatinosa (SG) but also the excitatory medial part of Lissauer tract (LT) and the deafferentated hyperactive neurons of the posterior and lateral funiculi of the dorsal horn. Thus, these were termed dorsal root entry zone (DREZ) lesions. The DREZ operation entails making a series of lesions aimed at the substantia gelatinosa Rolandi and the surrounding fiber tracts. Several techniques of DREZ lesioning have been developed for treating neuropathic pain.

The microsurgical DREZotomy procedure based on microsurgical incisions and bipolar coagulations was introduced by Sindou in early 1970s.[17] This was also performed by Nashold and coworkers[18] in 1974 in a patient with severe deafferentation pain secondary to brachial plexus avulsion. By 1989, Nashold's group reported a total of 550 operations for various conditions. Almost 500 additional operations have been reported by other authors, and many other neurosurgeons are performing this ablative procedure. The same procedure was applied initially in patients suffering from trigeminal neuralgia, postherpetic neuralgia, or painful spasticity after spinal cord trauma.[15,19–25]

ANATOMY

The DREZ operation involves the destruction of dorsal horn neurons and perhaps the axons traveling in juxtaposition to the gray matter, particularly those segments that correspond to the patient's reported area of pain (Figure 13-13).

An understanding of anatomy of the spinal cord and exact identification of the DREZ remains essential to carry out a successful DREZ operation with a minimal rate of complications.

The dorsal horn can be segmented on a cytoarchitectonic basis into six laminae (Rexed laminae) (Figure 13-14). The five most superficial are clearly involved in the transmission of nociceptive information from the periphery and can play an essential role in some deafferentation or central pain states.

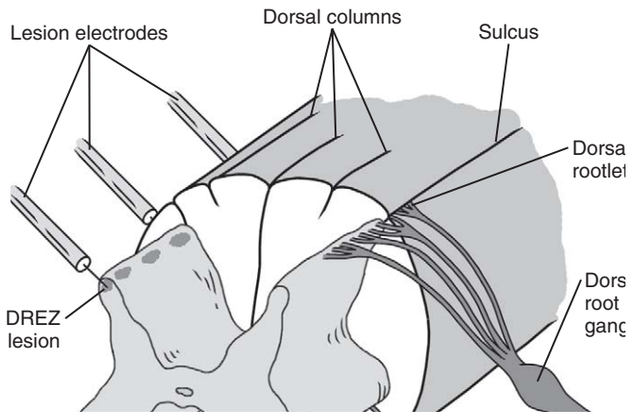


FIGURE 13–13

Drawing of the spinal cord showing the area of lesioning for the DREZ operation.

Not only are the synaptic connections from peripheral nociceptors localized in laminae I through V, but studies have demonstrated major concentrations of opiate receptors, substance P, and other biologically active peptides in these regions. Indeed, many believe that the "gate," as described by Wall and Melzack,[26] resides in this region of the spinal cord. Because the dendrites of the spinoreticulothalamic cells make synaptic connections with the primary afferent fibers inside the SG layers, the SG exerts a segmental modulating effect on the nociceptive input. When the lemniscal afferents in peripheral nerves or dorsal roots are altered, there is a reduction in the inhibitory control of the dorsal horn.

The procedure is presumed to preferentially destroy the nociceptive fibers grouped in the lateral bundle of the dorsal rootlets, as well as the excitatory medial part of the LT. The upper layers of the dorsal horn are also destroyed if microbipolar coagulations are made inside the dorsal horn. Thus, a combination of classic neurophysiology and modern pharmacology points to the region as being critical in the processing of sensory information (Figure 13-15).

INDICATIONS

- Brachial plexus avulsion lesion
- Brachial plexus destructive lesions
- Sacral root avulsion
- Postparaplegic pain

The procedure has also been used, although with a lower success rate, in the treatment of phantom limb pain, stump pain, post-thoracotomy pain, postherpetic neuralgia, peripheral mononeuropathy, spinal cord tumor, multiple sclerosis, causalgia, and postrhizotomy pain.

The indications for DREZ lesions for the treatment of chronic pain include an established diagnosis and failure of medical-pharmacological management. In addition, it is

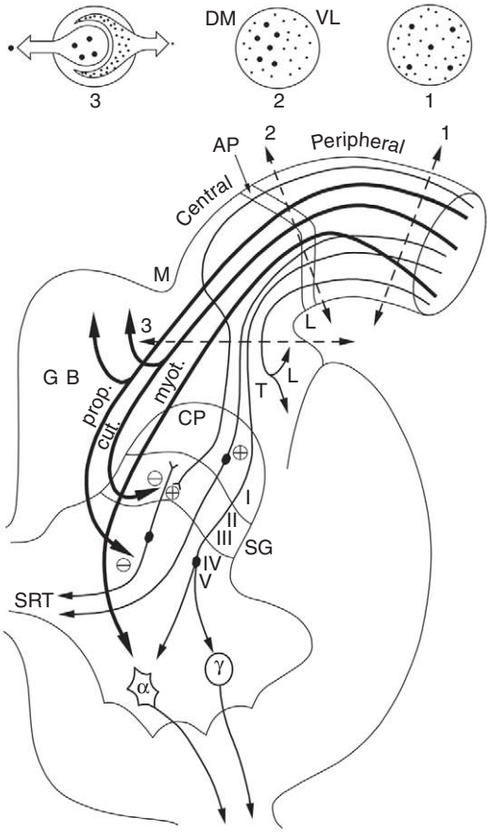


FIGURE 13–14

Cytoarchitecture of the dorsal horn showing six dorsal horn laminae and the fiber course in the dorsal horn. Note the segregation of small fibers laterally and large fibers medially at the level of the Pia. This is the anatomic basis for the DREZ lesion introduced by Sindou.

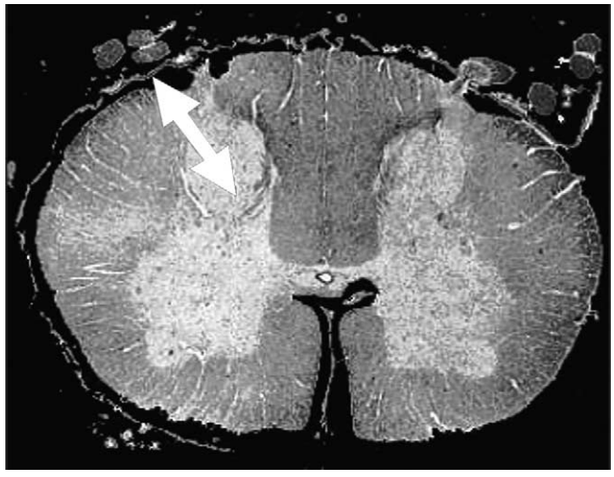


FIGURE 13–15

Cross-section of the spinal cord. The arrow shows the site of DREZ lesion. (Adapted from Wall PD, Melzack R: Pain mechanisms: a new theory. *Science* 150:971–979, 1965.)

important that the patient have an understanding of alternative strategies, risks, and potential benefits. The diagnoses listed above are those that have thus far been considered to be appropriate for DREZ lesions. The number of patients treated, however, is currently limited in some of those diagnostic groups.

CONTRAINDICATIONS

Contraindications relate to the patient's general health and ability to withstand a major surgical procedure, including such factors as infection, resistance to wound healing, blood and coagulation problems, and poor cardiopulmonary status. Patients who have a significant emotional component to their pain are rarely good surgical candidates, although the negative effects of chronic pain can alter a patient's judgments and emotions.

EQUIPMENT

- Surgical equipment to perform a laminectomy
- Operating microscope
- Radiofrequency generator
- Temperature-monitoring radiofrequency (RF) electrode 0.25 mm in diameter and 2 mm long

PROCEDURE

DREZ lesions are performed under general anesthesia and require a laminectomy over each segment to be lesioned. While in a state of general anesthesia with tracheal intubation and short-lasting curarization, the patient is placed in the prone position and the neck flexed in the so-called Concorde position with the head maintained in a three-pin head holder.

Through a median cutaneo-aponeurotic posterior incision and a unilateral paravertebral muscle division, a hemilaminectomy with preservation of the spinous processes is performed ipsilaterally to the avulsion and extended according to the injured cord segments. For brachial plexus avulsion involving C5 to T1 dorsal roots, it is necessary to do a C4 through T1 laminectomy. In the sacral segments, the anatomy of the conus permits extensive lesioning with a more limited laminectomy.

The length of the surgical lesioning is established on the basis of pain topography, which generally corresponds with the avulsed segments, as well as the altered adjacent rootlets.

After performing the laminectomy, the dura is longitudinally opened and the spinal cord is visualized. The dorsolateral sulcus is identified; the operating microscope is an important adjunct. Some neurosurgeons section the dentate ligaments so that the spinal cord can be rotated to orient the dorsal horn ventrally. When the dorsal roots have been avulsed, the dorsolateral sulcus has a series of microcysts and gliosis, and scar tissue is a common finding. It is essential to see the intact dorsal roots rostral and cau-

dal to the avulsion to identify the dorsolateral sulcus positively. Injury to nerves in the periphery or herpes zoster often leads to atrophic dorsal roots.

Nashold and coworkers' careful studies of this operation procedure[27] should be required reading for any surgeon who wants to use DREZ lesions. His original RF electrode made too large a lesion and was apparently responsible for the high incidence of postoperative dorsal column and pyramidal tract dysfunction (Figure 13-16). The neurological complication rate has fallen dramatically since Nashold shifted to a smaller thermistor-controlled electrode, which is 0.25 mm in diameter with a 0.5-mm insulated stainless steel electrode and with a tapered noninsulated 2-mm tip for DREZ lesions in the spinal cord (Radionics, Inc.) (Figure 13-17).

The spinal cord anatomy in brachial plexus avulsion is often distorted by injury. Using an operative microscope is necessary in accurately identifying the DREZ. A very useful technique in guiding the placement of the thermocoagulation lesions is localization of the DREZ by evoked potentials. Several techniques of evoked potentials recording have been described in literature.

The procedure consists of a longitudinal incision of the dorsolateral sulcus ventrolaterally at the entrance of the rootlets into the sulcus and then of microbipolar coagulations preformed continuously—in a dotted manner—inside the sulcus, down to the apex of the dorsal horn, along all the spinal cord segments selected for surgery. The average lesion is 2–3 mm deep and is made in the axis of the dorsal horn, at a 30-degree angle medially and ventrally for the cervical, 35 degrees for the thoracic, and 45 degrees for the lumbar and sacral segments of the spinal cord.

An RF coagulation series is made under a current of 35–40 mA (not over 75°C) for 10–15 seconds. RF thermocoagulation is performed in the rostrocaudal direction, with entry into the DREZ along the edge of the intermediolateral sulcus. Lesions are made at approximately 2- to 3-mm intervals along the longitudinal extent of the dorsolateral sulcus, with great care to avoid disrupting the small vessels over the surface of the spinal cord or brainstem. Using these parameters, each lesion in the spinal cord will measure 2.0 × 1.5 mm, which is adequate to destroy laminae I through V of the dorsal horn.

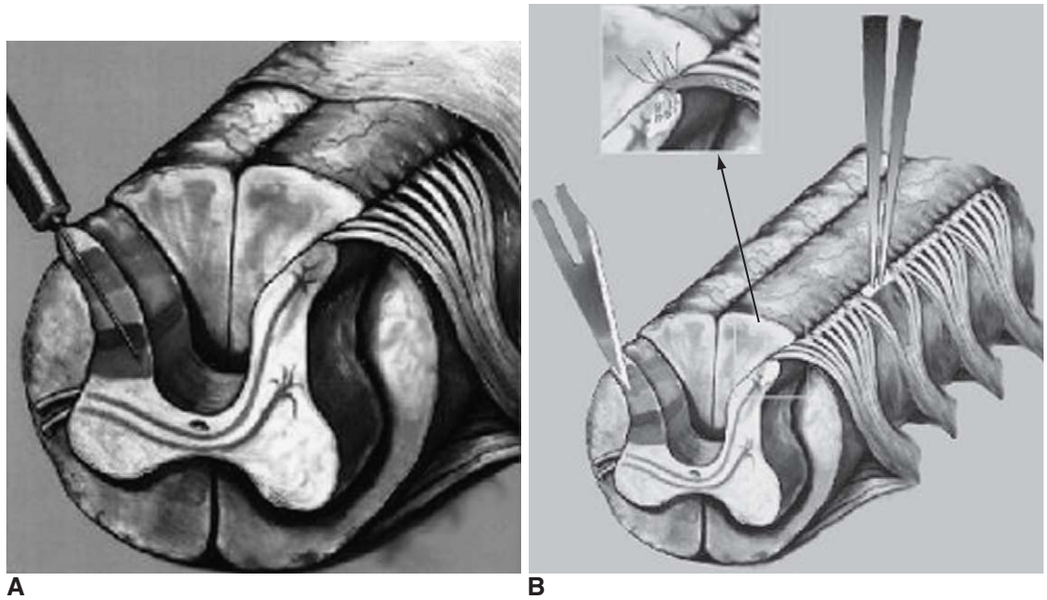
The standard spinal cord lesion is performed through the entire length of the affected painful dermatomes. Impedance measuring during insertion of the electrode is helpful since relatively high values indicate possible penetration into posterior columns and relatively low values suggest possible penetration into a cyst or syrinx. The standard impedance measurement is usually less than 1200 ohms in damaged spinal cord, reaching normal levels of 1500 ohms in normal parenchyma tissue.

Sweet and Poletti[28] have described the deliberate use of a larger lesion in a small number of cases, and others have used variations of lesion making.

After irrigation and hemostasis, the dura is then closed in a watertight fashion, and the muscular and soft tissues are closed in a standard manner.

RADIOFREQUENCY VERSUS LASER LESIONS

The advent of the laser as a surgical tool has led to the use of both CO_2 and argon lasers to lesion the DREZ.[29,30] In research on cats regarding the DREZ procedure,[31] the lesions in the dorsal horn produced by the RF probe were compared with those produced by CO_2 laser. Histologic examination revealed that depths of the laser and RF were



A B

FIGURE 13–16

Two methods of DREZ. (A) Nashold method. (B) TWMU method. (Adapted from Nashold BS Jr: Neurosurgical technique of the dorsal root entry zone operation. *Appl Neurophysiol* 51:136–145, 1988, with permission.)

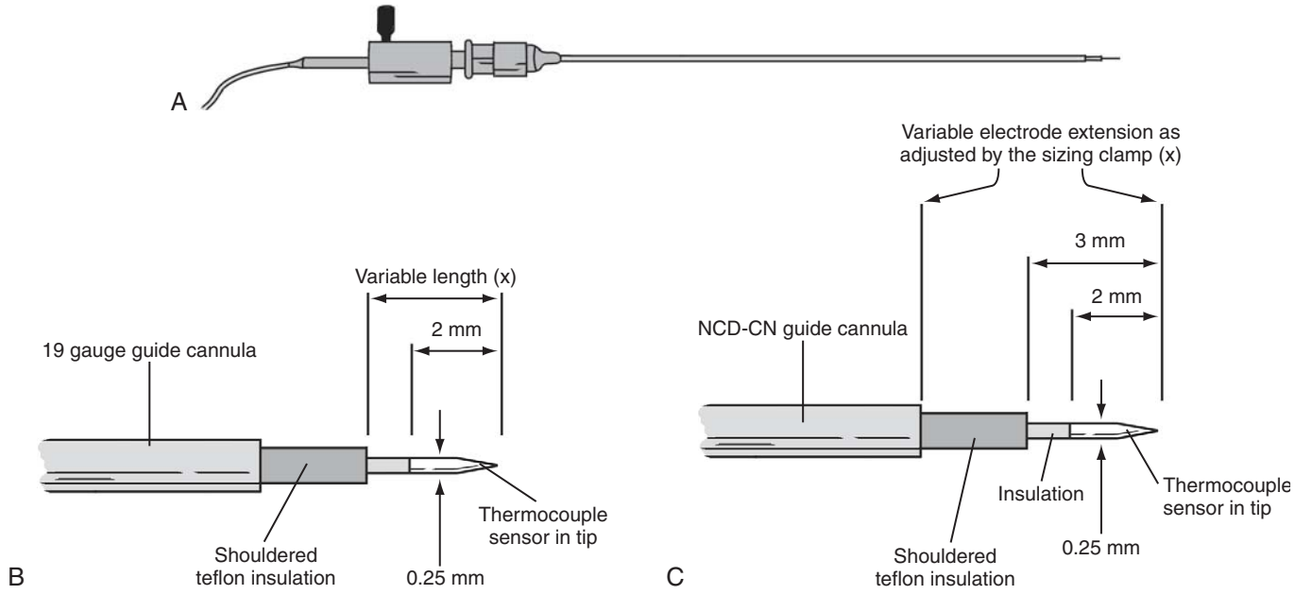


FIGURE 13–17

Thermistor-controlled electrode is 0.25 mm in diameter (A) with a 0.5-mm insulated stainless steel electrode (B) and with a tapered noninsulated 2-mm tip (C) for DREZ lesions in the spinal cord. (Courtesy of Radionics, Inc.)

similar but the RF lesions showed more lateral spread. Laser lesions comprised 4.4% (± 1.6%) of the cross-sectional area of the spinal cord, whereas the RF lesions occupied 22.8% (± 4%), demonstrating that the CO_2 laser produces smaller lesions than the RF electrode. Although the laser technique seems to be less traumatic to the spinal cord, it has not gained widespread use, possibly because the smaller lesions might not be as effective and/or the use of laser equipment is less manageable as compared to RF equipment. Another surgical technique is to coagulate the pia and vessels over the dorsolateral sulcus and to use a sickle knife to incise into the dorsal horn, followed by a ball dissection to destroy the dorsal horn under microscopic control.

SPINAL CORD STIMULATION

A major concern with regard to the placement of DREZ lesions is proper localization for placement of these lesions. The dorsolateral sulcus is varyingly discernible: when the dorsal roots have been avulsed, the dorsolateral region can be densely adherent to the arachnoid or to the dura. The dorsal horn is obliquely oriented, and the electrode, knife, or laser beam must be angled or the spinal cord rotated to create the desired lesion and avoid damage to the pyramidal tract or the dorsal column. When roots have been avulsed or the spinal cord damaged, it is helpful to expose the levels rostral and, when feasible, caudal to the proposed operative area so that normal dorsal roots can be identified. Evoked potentials are very useful to localize the superficial tracts in the spinal cord and assist in the placement of lesions.[32]

Almost all centers monitor motor-evoked potentials (MEPs) concurrently with spinal sensory–evoked potentials (SEPs). The monitoring of MEPs is the most appropriate technique to assess the functional integrity of descending motor pathways in the spinal cord. Transcranial electrical brain stimulation with brief pulse trains is predominantly used for MEP monitoring.

EFFICACY

Brachial Plexus Avulsion

The most common and most successful application of DREZ lesions is for the relief of pain of brachial plexus avulsion. This traumatic lesion is most common in young men who ride motorcycles. An overall 83% success rate has been reported with many follow-ups of longer than 5 years.[14,19,21,23,33–40] A few patients with sacral root avulsions have been included in these series. No other diagnosis has as high a likelihood of success, and no other operation is as likely to relieve this type of deafferentation pain. The efficacy is likely to diminish with long-term follow-up.

Postparaplegic or Postquadriplegic Pain

Postparaplegic or postquadriplegic pain has also been relieved by DREZ lesions, particularly pain occurring at the transition between normal and anesthetic skin. The long-term success rate is 54%, but the duration of follow-up has been variable. Some of the reported patients had drainage of post-traumatic syringomyelia at the same time; it is

unclear whether DREZ lesions alone are responsible for the pain relief.

Postamputation Pain

Another type of central pain that has responded to DREZ lesions is postamputation pain (REF). Included in this category are two different types of pain syndromes: stump pain and phantom limb pain. The overall results for postamputation pain are 39% of success (11 patients) in a group of 28 patients. In the series by Saris and coworkers,[41] phantom limb pain was highly likely to respond (six of nine patients), whereas stump pain was never relieved (none of six patients). When phantom limb pain and stump pain were both present, good results were noted in two of seven patients, but only the phantom pain responded regularly. None of the other reports clearly discriminated between phantom limb pain and stump pain.

Because of the aforementioned failures, DREZ lesion is not highly recommended for stump pain.

Postherpetic Neuralgia and Other Disorders

In the earlier reports on the use of the DREZ lesion for postherpetica neuralgia, Nashold and associates[42] and Friedman and Nashold[43,44] reported that 10 of 17 patients (59%) had good pain relief accompanied by a complication rate of 35%. In patients who had postherpetic pain for 6 months to 11 years and were followed 6 months to 6 years postoperatively, 29 of 32 (91%) had immediate pain relief. At 6 months, however, the figure dropped to 17 (53%), and at 18 months and thereafter, only eight (25%) had persistent relief of postherpetic neuralgia involving the spinal nerves.[30] Thirty-one patients with various myelopathies and neuropathies have also been treated with DREZ lesions (REF). Approximately two-thirds had good results, with follow-ups of 6–19 months and a complication rate of 10–20%.

Subnucleus Caudalis Dorsal Root Entry Zone Lesions

Since 1989, there have been at least 46 reported operations for facial pain using a two-electrode technique for subnucleus caudalis DREZ lesions. The overall pain relief was noted as "excellent" in 34% of patients and "good" in another 40%. The best results were obtained in patients with postherpetic pain involving one or more divisions of the trigeminal nerve. Pain resulting from facial trauma or dental surgery was not improved. In general, deafferentation pain responds to lesioning, but pain of a peripheral origin does not.

COMPLICATIONS

Misplaced or a too large thermocoagulation lesion may cause undesirable neurological deficit because of the proximity to the lateral corticospinal tract and dorsal column to the site of DREZ lesioning. The complication rate has been reduced by improved electrocoagulation electrodes with better control over the size of lesion.

Review of the literature showed that reported complication rate varied between 0 and 60%.[45] The major complication of the spinal DREZ operation is weakness in the ipsilateral leg caused by injury to the corticospinal tract, which is seen in 5–10% of patients. This complication occurs most frequently after thoracic DREZotomy. There may also be ipsilateral loss of sensation. Loss of bladder control can occur but is less common. To prevent neurological complications, the neurosurgeon must pay strict attention to the technical details of the surgery, including an understanding of the anatomy of the spinal cord, the use of precise lesion parameters, and the careful exposure of the damaged spinal cord using the operating microscope. Misplaced lesions may be related to an inappropriate angle of penetration of the coagulation needle and/or lack of anatomical landmarks in the spinal cord with avulsed roots.

CONCLUSION

DREZ lesions are effective in alleviating brachial plexus avulsion originating intractable chronic pain and are a valuable method in treating certain pain syndromes following spinal cord injury. Disabling neurological injuries following DREZ lesions are rare, being more prevalent in patients suffering postherpetic chronic pain refractory to other therapies, due to the advanced age of this subpopulation. Despite the efforts carried out with this method as a therapy for other chronic pain conditions, its place in therapy remains to be defined.

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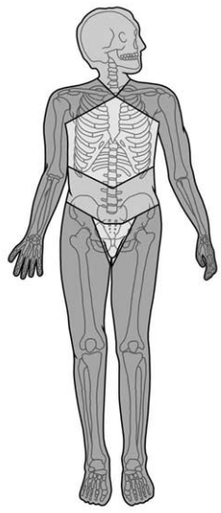
C H A P T E R



14

Thoracic Facet Joint Blocks and Neurotmy

JOHN W. NELSON

THORACIC FACET AND MEDIAL BRANCH BLOCK

HISTORY

The facet joints of the spine may be otherwise known as the apophyseal joints. The Greek word apophysis means "an offshoot," and the anatomical definition of the word is a natural outgrowth or process on a vertebra or other bone.[1] The degenerative changes and associated muscle spasm that develop when a facet joint is involved in a sprain from a forceful or violent twisting motion were termed the "facet syndrome" by Ghormley in 1933.[2] The intra-articular facet joints at all levels are subject to trauma. Pain emanating from various structures of the spine is a major cause of chronic pain problems.[3,4] Linton et al.[5] estimated the prevalence of spinal pain in the general population as 66%, with 44% of patients reporting pain in the cervical region, 56% in the lumbar region, and 15% in the thoracic region. Manchikanti et al.[6] reported similar results. Despite the high prevalence of spinal pain, it has been suggested that a specific etiology of back pain can be diagnosed in only about 15% of patients with certainty based on clinical examination alone.[7–11]

Bogduk and McGuirk[11] noted that a reductionist approach to chronic low back pain requires an anatomical diagnosis. Bogduk[12] identified four factors necessary for any structure to be deemed a cause of back pain: a nerve supply to the structure, the ability of the structure to cause pain similar to that seen clinically in normal volunteers, the structure's susceptibility to painful diseases or injuries, and demonstration that the structure can be a source of pain in patients using diagnostic techniques of known reliability and validity.

The facet or zygapophyseal joints of the spine are well innervated by the medial branches of the dorsal rami.[13–17] Facet joints have been shown capable of causing pain in the neck, upper and mid back, and low back, with pain referred to the head or upper extremity, chest wall, and lower extremity in normal volunteers.[18–27] They also have been shown to be a source of pain in patients with chronic spinal pain using diagnostic techniques of known reliability and validity.[28–45] Conversely, the reliability of physical examination in diagnosing the specific cause of back pain has been questioned.[46]

ANATOMY

The thoracic facet joint lies between the pedicles of the segment above and below and at the level of the disk of the segment. The T1-T2 facet joint is angled 66 degrees from a transverse plane, with the cephalad end more anterior than the caudad end.[47,48] The angle steepens to 75 degrees by the T3-T4 facet joint and then remains constant to the T11-T12 facet joint. The T1-T2 to the T11-T12 facet joints are uniformly angled 110 degrees from the midline posterior sagittal plane. The thoracic facet joints are thus mostly vertically oriented and almost parallel to the coronal plane. The transition from the thoracic to the lumbar facet joints occurs primarily at the T11-T12, and T12-L1 joints. There is some variability at the T11-T12 facet joint, but it is essentially oriented vertically (perpendicular to the sagittal plane) and faces directly anterior (parallel to the coronal plane). The T12-L1 facet joint assumes the more lumbar orientation and is approximately 25 degrees oblique to the sagittal plane from the midline posteriorly. Medial branch nerves from two segmental levels innervate the joint at the same level plus the joint below. Below the T3 level this pattern is consistent, but it seems that the C7 and C8 nerves may travel caudad as far as the T2 and T3 levels[49] (Figure 14-1). To establish the anatomical basis for thoracic medial branch neurotomy, an anatomical study was undertaken. Using an X40 dissecting microscope, a total of 84 medial branches from seven sides of

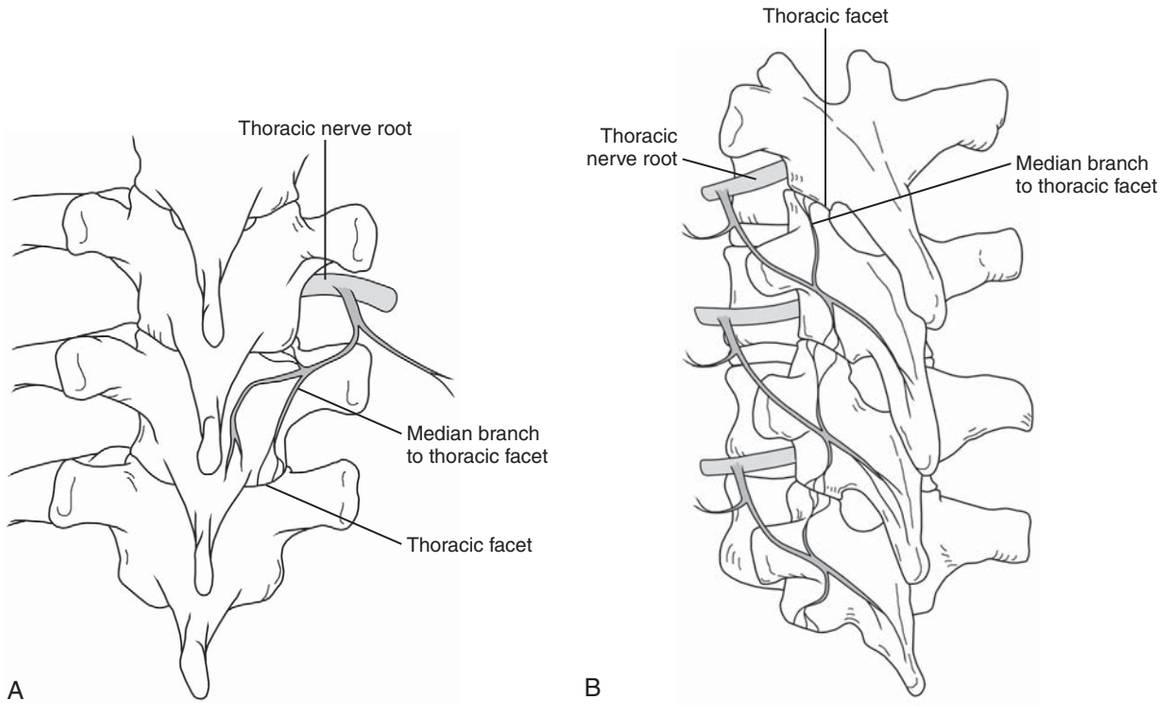


FIGURE 14–1

These line drawings illustrate the course of the median nerve as it traverses to the thoracic facet from the posterior primary rami. (A) Posterior view. (B) Oblique view.

four embalmed human adult cadavers were studied (Figure 14-2).

The medial branches of the thoracic dorsal rami were found to assume a reasonably constant course. Upon leaving the intertransverse space, they typically crossed the superolateral corners of the transverse processes and then passed medially and inferiorly across the posterior surfaces of the transverse processes before ramifying into the multifidus muscles. Exceptions to this pattern occurred at midthoracic levels (T5-T8). Although the curved course remained essentially the same, the inflection occurred at a point superior to the superolateral corner of the transverse process.

At no time during the dissection were nerves encountered crossing the junctions between the superior articular processes and transverse processes, which have been the target points advocated for thoracic facet denervation. Rather, the results of this study indicate that the superolateral corners of the transverse processes are more accurate target points.

INDICATIONS

The major indications for facet joint injection include (1) focal tenderness over a facet joint, (2) chronic back pain with or without radiation but with a normal radiographic evaluation, and (3) back pain with evidence of disk disease and facet arthritis.[50,51]

The findings on conventional radiographs correlate poorly with the clinical symptoms.[52,53] There may be better correlation between symptoms and findings on CT scans.[54,55] Facet arthrography with injection of local anesthetic and an anti-inflammatory agent is a diagnostic procedure that is often therapeutic, with relief of symptoms lasting much longer than expected from the pharmacological effects of the injected agents.[22,56–58]

CONTRAINDICATIONS

The only absolute contraindication to facet joint block is infection in the overlying soft tissues. A relative contraindication is allergy to injecting agents. Facet joint block can be accomplished, however, without injection of contrast, and the newer nonionic contrast agents also decrease the risk in allergic individuals.

EQUIPMENT

For injection
22-gauge, B-bevel needle 1-1/2-inch
25-gauge skin infiltration 3/4-inch needle
3-cc syringe
10-cc syringe
IV T-piece extension

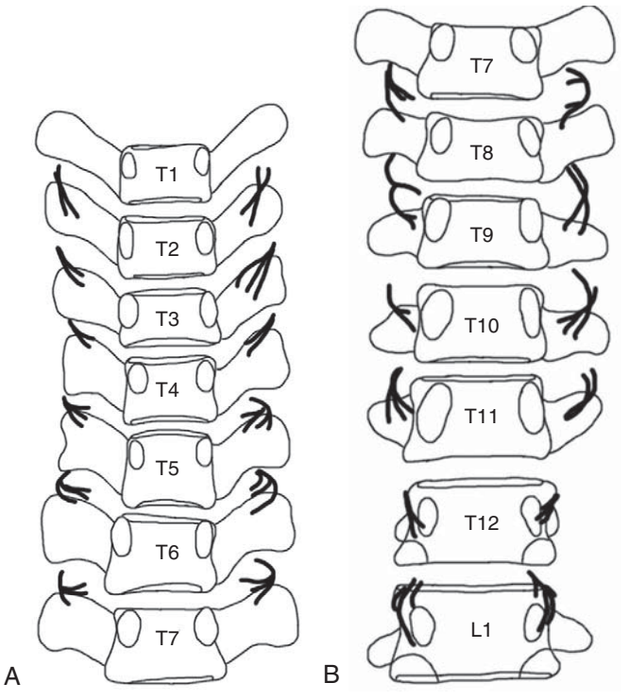


FIGURE 14–2

This figure represents the course of the median nerves as seen from the posterior view (A) from T1-T7, and (B) T7-L1 (From Ref. 22, with permission.

Metal clamp for radiographic marker
For RFTC lesioning
20-gauge, 3/4-inch angiocath
10 cm, with 10-mm, active tip, Racz-Finch RFTC needle
RFTC electrode and connecting cables

DRUGS

- Radiographic contrast solution (iohexol [Omnipaque or equivalent])
- Local anesthetic agent (1.5% lidocaine, 0.2% ropivacaine, or 0.25% bupivacaine)
- Steroids (methylprednisolone, triamcinolone, or dexamethasone)

INCLUSION CRITERIA FOR THORACIC FACET OR MEDIAL BRANCH INJECTION

Patients should have had pain for at least 6 months, and nonspecific rather than radicular pain. Disc-related pain with radicular symptoms should be excluded in patients based on radiologic or neurologic testing, lack of a neurological deficit, and no radicular symptoms or pain that involves predominantly the upper back and extremity. All patients should have failed conservative management, which includes physical therapy, chiropractic manipulation, exercises, drug therapy, and bed rest.

Work-up should include a comprehensive history, physical examination, and evaluation of the results of prior procedures and investigations. In one study of 585 potentially eligible patients, 500 patients agreed to participate in the study after the nature of the study and the potential hazards of the procedures were explained.[59] This study evaluated patients with chronic, nonspecific spinal pain involving all three regions of the spine–cervical, thoracic and lumbar spine. Painful cervical facets were identified in 55% of patients with neck pain, 42% of patients with thoracic pain, and 31% of patients with low back pain. False-positive rates after single injections were 63%, 55%, and 27% for cervical, thoracic, and lumbar facet joint blocks, respectively. Overall, of the 500 patients with chronic spinal pain evaluated in this study, 28% had painful cervical facets, 6% painful thoracic facets, and 25% painful lumbar facets. At least one region was involved in 43% of patients; at least two regions in 15% of patients, and 2% of patients had painful facets in all three regions of the spine. Depending on the regions involved, most patients had two or three symptomatic facet joints.

Although single diagnostic blocks appear unreliable, with a relatively high false-positive rate, true-positive results obtained by performing two sets of diagnostic blocks on separate occasions indicate that facet joints are a cause of chronic spinal pain in nearly half of all patients with chronic spinal pain presenting to an interventional pain management clinic. Because these patients typically have failed conservative management, including physical therapy, chiropractic treatment, and analgesics, patients with chronic spinal pain may benefit from specific interventions designed to identify and eliminate facet joint pain.

PREPARATION OF PATIENT

History and Physical Examination

The thoracic facet joint syndrome is similar to that of the cervical facet syndrome, resulting from a sudden twisting motion, twisting while lifting overhead, or an unguarded rotating motion of the thoracic spine. The resultant pain may be mild, dull, and aching, with radiation encircling the chest, or it may be sharp, pleuritic-type pain that can affect functional vital capacity or become overwhelming to the patient. There is usually decreased motion in the portion of the spine involved. Examination of the patient may reveal a loss of the thoracic curve or muscle spasms, causing localized scoliosis.

Nonetheless, pain referral patterns from injection into normal joints have been described[6,15] (Figure 14-3A and B). Distension of normal joints was not painful in 27.5% of volunteers, but, when it was painful, the referral patterns were always unilateral and reproducible for most of the thoracic spine. In all subjects for the T2-T3 to T11-T12 levels, the area of most intense pain was one segment inferior and slightly lateral to the involved joint and never crossed the midline. Although significant

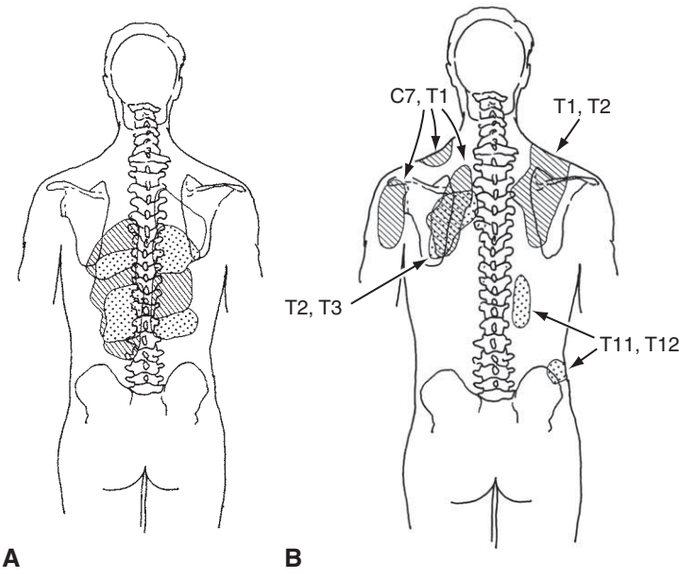


FIGURE 14–3

(A) The drawing shows the skin reference zones for the T3-T4 to T10-T12 facet joints (adapted from Dreyfuss P, Tibiletti C, Dreyer S: Thoracic Zygoapophyseal Joint Pain Patterns, Spine 19: 807-811, 1994.) (B) The drawing shows the skin reference zones for the C7-T3 and T11-T12 facet joints. (Adapted from Fukui S, Ohseto K, Shiotani M: Patterns of pain induced by distending the thoracic zygapophyseal joints. Reg Anesthes 22:332-336, 1997.)

overlap occurred, pain was not referred more than 2-1/2 segments inferior to the joint injected. At the C7-T1 joint, pain was felt in the paravertebral area over the injected point, which extended superiorly toward the superior angle of the scapula and inferiorly toward the inferior angle of the scapula. The pain was sometimes also referred toward the shoulder joint and suprascapular region. At the T1-T2 joint, pain was felt in the paravertebral region over the joint, below the inferior angle of the scapula, and sometimes into the suprascapular region. Because of the considerable overlap, the pain maps for the C7-T1 through T2-T3 joints were not felt to be reliable enough to identify the joint of origin.

Laboratory Investigations

The usual lab work for any interventional procedure should be performed, including appropriate lab work-up for patients with physical disability.

Preprocedure Medication

Use the standard recommended protocol for conscious sedation by the American Society of Anesthesiologists.

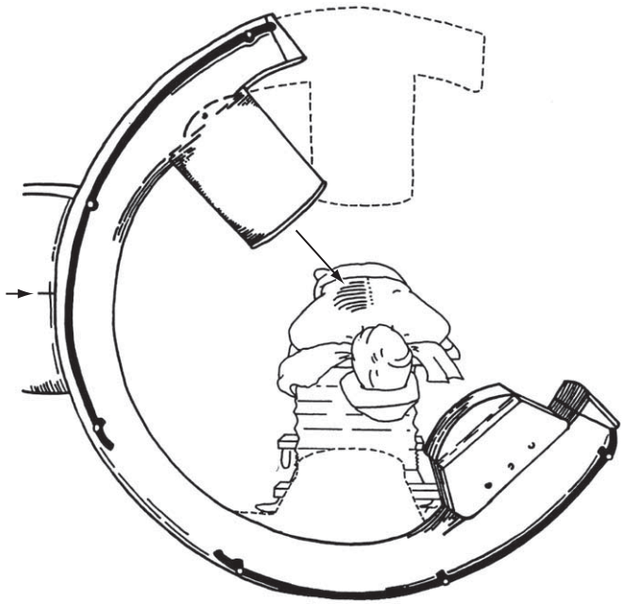


FIGURE 14–4

The fluoroscope is rotated into an oblique position to create a "Scottie dog" image of the posterior thoracic vertebrae components. The "eye of the Scottie dog" will be on the same side as the lateral rotation of the C-arm.

PROCEDURE

Intra-Articular Thoracic Facet Injection

The patient is put in the prone position with C-arm guidance. For the articular injection technique, the patient is positioned prone. The joint to be blocked is identified by counting the ribs from T1 caudad and from T12 cephalad (Figure 14-4). Once the joint is identified, a radiopaque marker on the skin over the joint may help in needle localization. Because of the steep angle of the thoracic facet joints, the skin entry point may need to overlie the pedicle one or two segments caudad. After sterile preparation and draping, local anesthetic is injected into the skin and tissues along the needle path. A 20- or 22-gauge, 10-cm spinal needle is then directed steeply cephalad toward the joint. Using the skin marker and a combination of anteroposterior and lateral fluoroscopic images, the needle is then advanced into the joint. As in the lumbar spine,

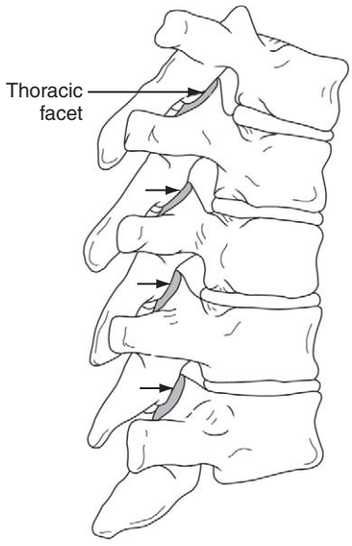


FIGURE 14–5

A line drawing showing the lateral view of the thoracic vertebrae. Note the arrows pointing to the thoracic facet (dark shade).

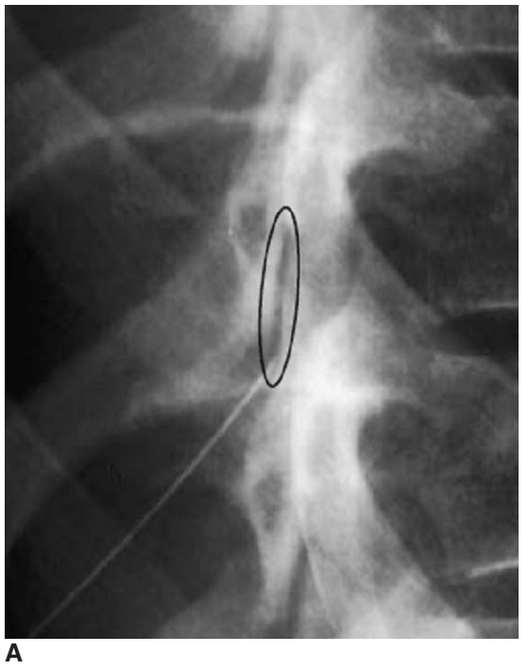
a combination of dye injection and needle "feel" confirms proper needle placement in the thoracic facet joint (Figure 14-5). The thoracic facet joints are very small and can hold only 0.4–0.6 ml of injectate (Figure 14-6A and B). Therefore, the mixture of dye, local anesthetic, and steroid should not exceed this total joint volume. Additional solutions may be injected periarticularly for therapeutic purposes.

Thoracic Median Branch Injection

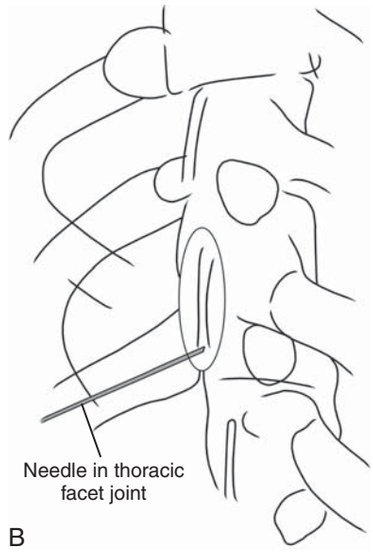
For the median branch blocks, the C-arm is rotated cephalocaudad until the end-plate of the disk above the target is squared off and the distal superior aspect of the transverse process is identified (Figure 14-7). The needle is then placed on bone on the superior lateral corner of the transverse process at T1 through T4 and T10 and at the junction of the superior articular process and transverse process at T11 and T12 (Figure 14-8A and B). After negative aspiration, a small volume of myelographic nonionic contrast solution is injected to be sure there is no intravascular or intraneural injection prior to the therapeutic injection of 0.3–0.5 ml of local anesthetic. For the T5–T8 medial branch blocks, the needle is placed below the rib above at the same depth of the transverse process, and injection is done in a similar fashion.

Management During and After the Procedure

The patient should not be given large doses of narcotics or sedatives, as these may make the interpretation of the diagnostic blocks unreliable. The patient should be reexamined in the recovery room, preferably by a neutral observer, and the patient should repeat the maneuvers that normally aggravates or reproduces their pain to determine whether there is at least an 80–100% reduction of their pain in order to consider the injection diagnostic. As in the cervical and lumbar spine, two comparative blocks with long-acting and short-acting local anesthetics should be done to rule out false positives prior to considering radiofrequency ablation.



A



B

FIGURE 14–6

(A) This radiographic image is taken in an oblique view to optimize the thoracic facet joint (note the arrow pointing to it). (B) Line drawing of the same view.

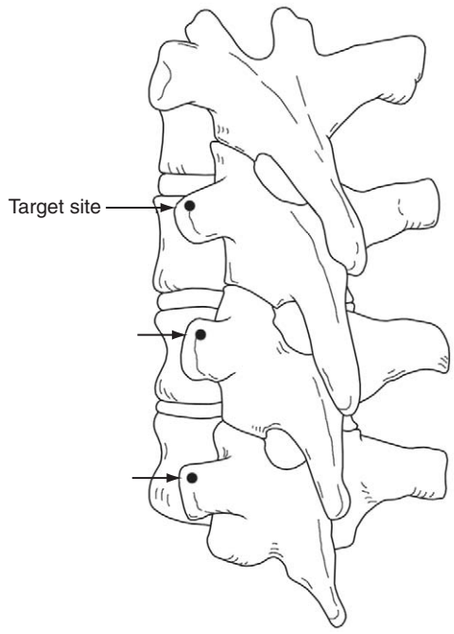


FIGURE 14–7
This line drawing shows the arrows pointing to the target site for the median branch block.

Radiofrequency Thermocoagulation of Thoracic Median Branches

For patients who do not get meaningful long-term relief from diagnostic injections, but who meet the criteria for thoracic facet–mediated pain, then radiofrequency ablation is an option. In order to place the active tip of the radiofrequency cannula parallel to the course of the median branches, the needle should be injected from the midline, slightly caudal to the level of the transverse process, and the radiofrequency cannula angled cephalad and laterally until the tip of the radiofrequency needle just lies at the tip of the superior corner of the transverse process (Figure 14-9A and B).

Sensory stimulation is then carried out to try to reproduce pain at 0.3–0.5 volts, although in the study done by Dreyfuss et al.[22], with medial branch radiofrequency in the lumbar spine, there was no significant correlation between sensory thresholds and efficacy with radiofrequency ablation, although they were using a large 18-gauge electrode.

For radiofrequency ablation of the T5–T8 branches, one may actually need to be above the transverse process and below the rib in order to identify the medial branch. Once sensory stimulation has been carried out, a superthreshold stimulation can be performed up to 1.5 volts to be sure that the active electrode is not near an intercostal nerve, which would be very unlikely as long as the depth of the needle placement is at the level of the tip of the transverse process. A small amount of local anesthetic is then injected, and radiofrequency lesions are made at 80 to 85°C for 70–90 seconds. A curved radiofrequency needle is recommended, and the needle can be rotated 90 degrees cephalad and 90 degrees caudad from neutral to create a bigger lesion.

Gauci[60] recommends doing further lesions by pulling the electrode back along the course of the nerve toward the midline, although once the local anesthetic has been injected distally, it may theoretically be impossible to get accurate stimulation as one pulls back medially.

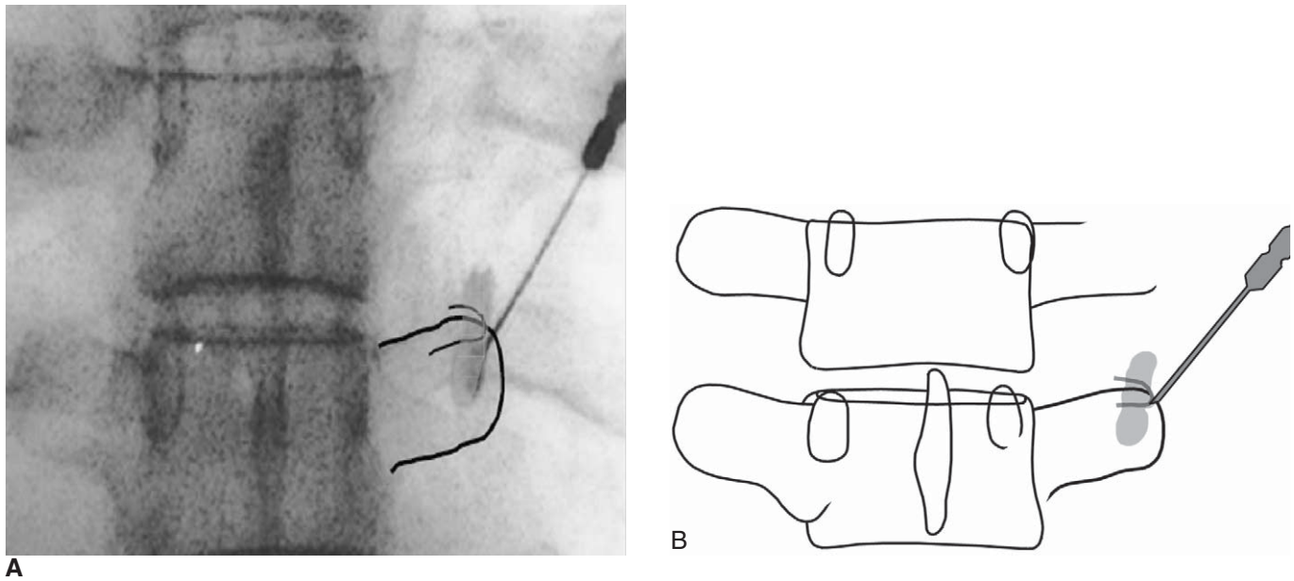


FIGURE 14–8
(A) Radiographic image showing the placement of the needle for the median branch block and spread of 1 ml of contrast solution over the transverse process (anteroposterior view). (B) Line drawing showing the needle placement on the median branch (From Ref. 22, with permission).

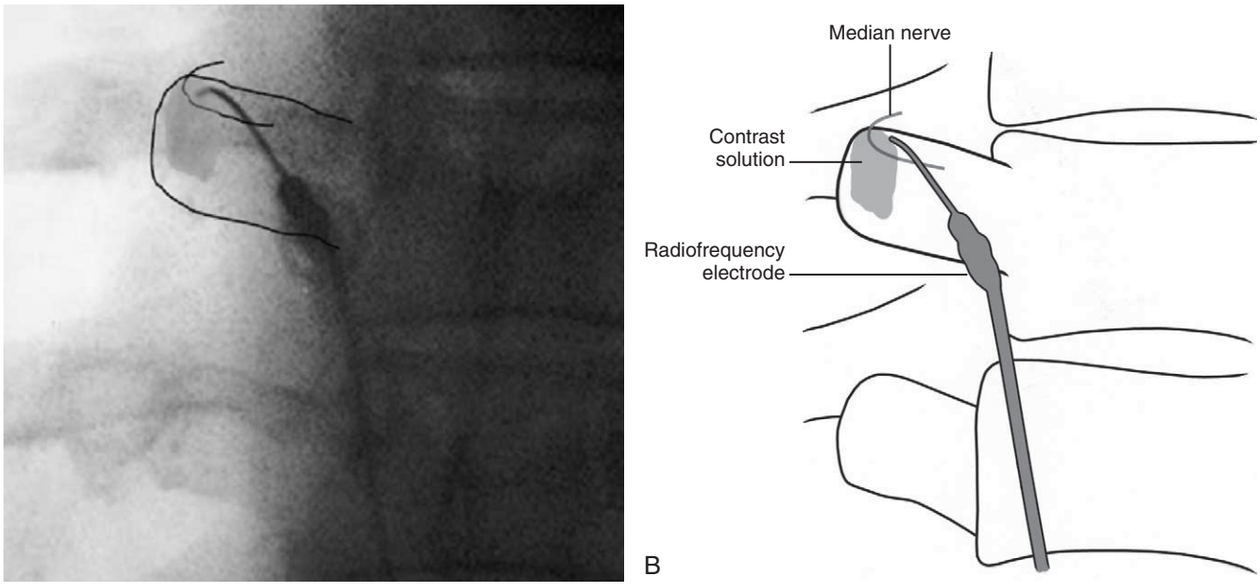


FIGURE 14–9

(A) Radiographic image (anteroposterior view) showing the placement of radiofrequency electrode at the superolateral aspect of the transverse process where the median nerve traverses. (B) Line drawing of the same view (From Ref. 22, with permission).

EFFICACY

Only a few studies of thoracic facet denervation have been conducted in the past. Unfortunately, the results are unreliable because the anatomical location of the medial branch nerves in the thoracic spine has since been shown to be 12 mm away from the site of denervation. Therefore, until a reliable technique is developed, the only injection for thoracic facets is either intra-articular or periarticular. These may be useful therapeutically, and perhaps also diagnostically.

In a recent study, Manchikanti and colleagues[59] evaluated the efficacy of thoracic facet and medial branch blocks in terms of pain relief with a minimum 1-year follow-up of 55 patients. Once the diagnosis was confirmed, the patient underwent either medial branch blocks or interarticular facet injections. Twenty percent of the patients had two joints injected, 65% had three joints injected, 16% had four joints injected, and 60% had bilateral injections. Each injection provided relief for an average of 4–6 months. The percentage of patients with greater than 50% relief was 71% at 3–6 months, 76% at 12 months, 71% at 24 months, and 69% at 36 months.

Most patients experience little or no pain during injection of the facet joints. If the injected facet is the cause of the pain, frequently dramatic relief of pain immediately follows the injection. The patient is questioned concerning any immediate change in symptoms, and is instructed to keep track of any change in pain over the next 24 hours as well as during the following weeks.

COMPLICATIONS

Complications following facet blocks are rare, but include infection, allergic reaction, transient radicular pain, and pneumothorax. Theoretically, the subarachnoid space can be entered during a facet block. It is important to aspirate before any injection to ensure that there is no return of cerebrospinal fluid. Placement of the needle under fluoroscopic visualization and proper technique are safeguards to prevent this possibility.

CLINICAL PEARLS

Neuritis, subarachnoid injection, and pneumothorax risks are minimal if the needle is kept posterior and on the "eye of the Scottie dog." To ensure accuracy of needle placement, incremental corrections and measured advancement of the needle minimize imprecise placement.

SUMMARY

Facet joints have been shown to be a source of chronic spinal pain by means of diagnostic techniques of known reliability and validity. Blocks of facet joints can be performed to test the hypothesis that the target joint is a source of the patient's pain. Facet joints can be anesthetized with intra-articular injections of local anesthetic or by anesthetizing the medial branches of the dorsal rami that in-

nervate the target joint. If pain is not relieved, the joint cannot be considered the source of pain. The true source may be another facet joint or some other structure. True-positive responses are determined by performing controlled blocks, either in the form of placebo injections of normal saline or comparative local anesthetic blocks on two separate occasions, when the same joint is anesthetized using local anesthetics with different durations of action. The value and validity of medial branch blocks and comparative local anesthetic blocks in the diagnosis of facet joint pain have been demonstrated.[29,34,46] Further, controlled blocks are the only reliable tool in diagnosing chronic spinal pain because there are no clinical features or diagnostic imaging studies that can determine whether a facet joint is painful or not.[5,7–9,11,27,29,32,34,44,54,61]

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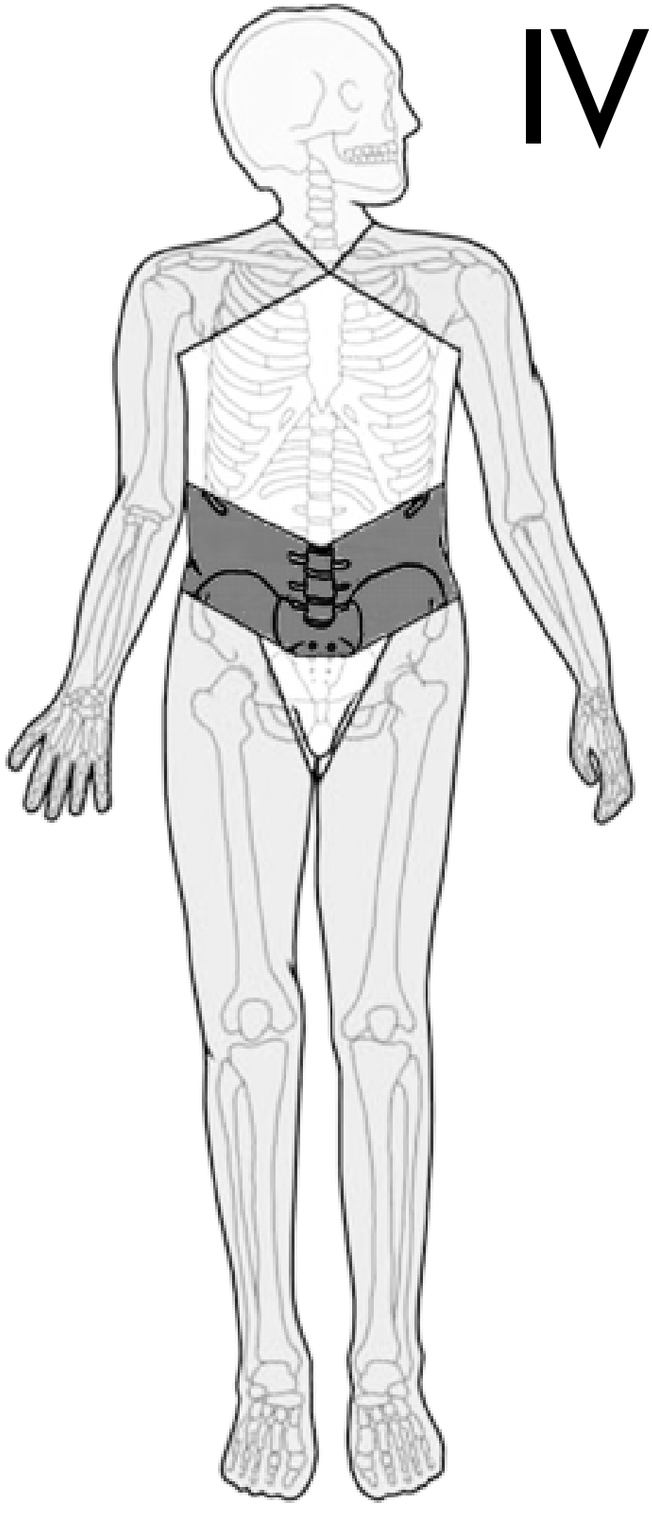
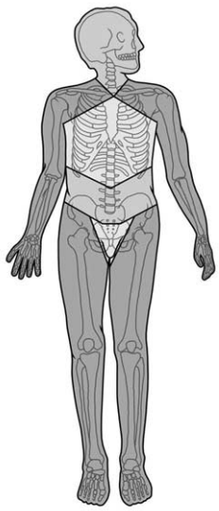
SECTION

IV



Lumbar Region

C H A P T E R



15 Lumbar Somatic Blocks

STEVEN D. WALDMAN

HISTORY

The use of regional anesthesia to perform surgical procedures on the groin and lower extremity evolved in tandem from two basic realities: (1) unlike the upper extremity where blockade of a single nerve can render a reasonably large anatomic area insensate, most surgeries on the lower extremity and groin require blockade of more than one nerve, and (2) the fact that spinal and epidural anesthesia can easily and more consistently render the groin and lower extremity insensate. A resurgence of interest in somatic nerve blocks of the groin and lower extremities is the result of using regional anesthesia to provide both acute, chronic, and postoperative pain management following surgical procedures in these regions.

LUMBAR PLEXUS BLOCK

ANATOMY

The lumbar plexus lies within the substance of the psoas muscle (Figure 15-1). The plexus is made up of the ventral roots of the first four lumbar nerves and, in some patients, a contribution from the 12th thoracic nerve.[2] The nerves lie in front of the transverse processes of their respective vertebrae; as they course inferolaterally, they divide into a number of peripheral nerves. The ilioinguinal and iliohypogastric nerves are branches of the L1 nerves, with an occasional contribution of fibers from T12. The genitofemoral nerve is made up of fibers from L1 and L2. The lateral femoral cutaneous nerve is derived from fibers of L2 and L3. The obturator nerve receives fibers from L2-L4, and the femoral nerve is made up of fibers from L2-L4. The pain management specialist should be aware of the considerable interpatient variability in terms of the actual spinal nerves that provide fibers to make up these peripheral branches. This variability means that differential neural blockade on an anatomical basis must be interpreted with caution.

The rationale behind lumbar plexus block using the psoas compartment technique is to block the nerves that compose the lumbar plexus because they lie enclosed by the vertebral bodies medially, the quadratus lumborum laterally, and the psoas major muscle ventrally. Solutions injected in this "compartment" flow caudally and cranially to bathe the lumbar nerve roots just as they enter the psoas muscle.

INDICATIONS

The lumbar plexus nerve block via the psoas compartment technique is used primarily for surgical anesthesia of the lower extremity. It is occasionally used in the area of pain management during treatment of pain secondary to inflammatory conditions of the lumbar plexus such as idiopathic lumbosacral plexitis or when tumor has invaded the tissues subserved by the lumbar plexus or the plexus itself.[2,3] Lumbar plexus nerve block via the psoas compartment technique with local anesthetic is occasionally used diagnostically during differential neural blockade lower extremity and groin pain. If destruction of the lumbar plexus is being considered, this technique is useful as a prognostic indicator of the degree of motor and sensory impairment that the patient may experience. Lumbar plexus nerve block via the psoas compartment technique with local anesthetic may be used to palliate acute pain emergencies, including groin and lower extremity trauma or fracture, acute herpes zoster, and cancer pain, while waiting for pharmacologic, surgical, and antiblastic therapies to become effective. This technique along with local anesthetic and steroids is also useful in the treatment of lumbar plexitis secondary to virus or diabetes. For most surgical and pain management applications, epidural or subarachnoid block is a better alternative, although one should expect fewer cardiovascular changes with lumbar plexus block compared with epidural or subarachnoid

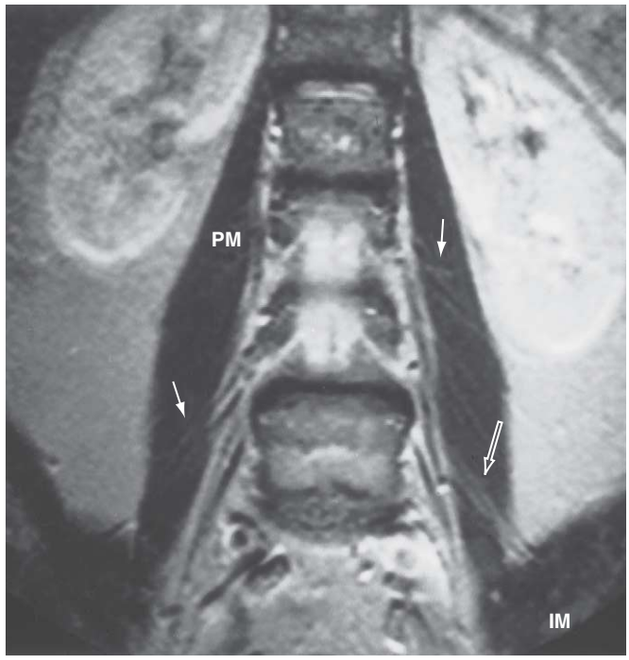


FIGURE 15–1

Anatomic location of the lumbar plexus. (From Stark DD, Bradley WG: *Magnetic Resonance Imaging*, 3rd ed. St. Louis, Mosby, 1999, p. 1908, with permission.)

techniques. Destruction of the lumbar plexus is indicated for the palliation of cancer pain, including invasive tumors of the lumbar plexus and the tissues that the plexus innervates. More selective techniques such as radiofrequency lesioning of specific lumbar paravertebral nerve roots may cause less morbidity than lumbar plexus neurolysis.

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represents a strong contraindication to the performance of the lumbar plexus block. Local infection involving the area of the lumbar plexus is also a contraindication to the performance of lumbar plexus block.

EQUIPMENT

- Peripheral nerve block tray
- 22-gauge, 13-cm styletted needle
- 25-gauge, 1-1/2-inch needle
- 18-gauge, 1-1/2-inch needle
- 12-ml sterile syringe

DRUGS

- 1% preservative-free lidocaine (for diagnostic or prognostic block)
- 0.25% preservative-free bupivacaine (for therapeutic block)
- Depot methylprednisolone (for therapeutic block)
- 6.5% aqueous phenol (for chemical neurolytic block)

PROCEDURE

The patient is placed in the prone position with the lumbar spine flexed. The superior iliac crest is identified, and the spinous process is palpated in a direct line medially with the crest. This is the spinous process of the L4 vertebra in the vast majority of patients. Counting down one level, the L5 spinous process is identified by palpation and then confirmed by posteroanterior and lateral fluoroscopy through the vertebral bodies (Figures 15-2, 15-3, and 15-4). At a point 1-1/2 inches lateral to the L5 spinous process, the skin is prepared with antiseptic solution.[2] After adequate anesthesia of the skin and subcutaneous tissues is obtained, a 22-gauge, 13-cm styletted needle is advanced perpendicular to the skin, aiming for the middle of the transverse process. The needle should impinge on bone after being advanced approximately 1-1/2 inches (Figure 15-5). After bone is contacted, the needle is withdrawn into the subcutaneous tissues and redirected superiorly and "walked off" the superior margin of the transverse process. As soon as bony contact is lost, the stylet is removed and a 5-ml, well-lubricated syringe filled with sterile preservative-free saline is attached. The syringe and needle are slowly advanced in a manner analogous to the loss-of-resistance technique used for identification of the epidural space, with constant pressure being placed on the plunger of the syringe (Figure 15-6). At a depth of 2–2-1/2 inches, a sudden loss of resistance is encountered as the needle exits the quadratus lumborum muscle and enters the psoas compartment (Figure 15-7).

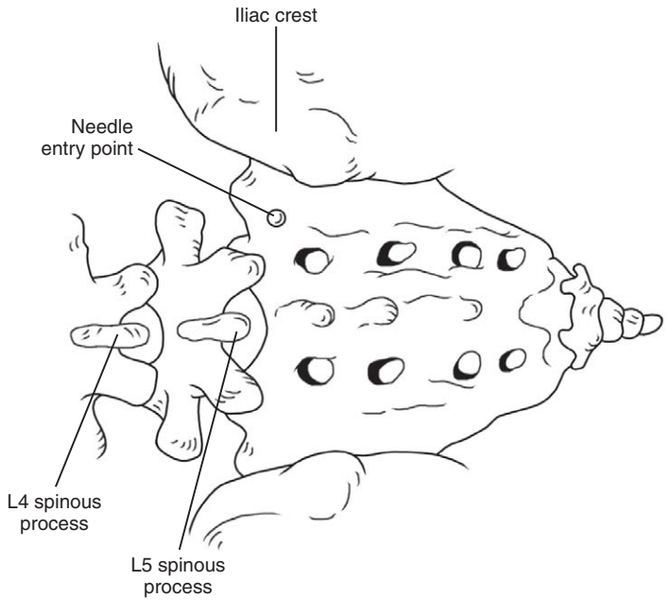


FIGURE 15-2

Point of entry for lumbar plexus block.

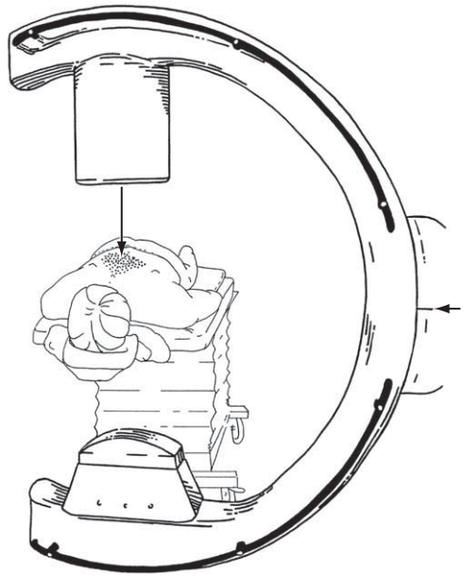
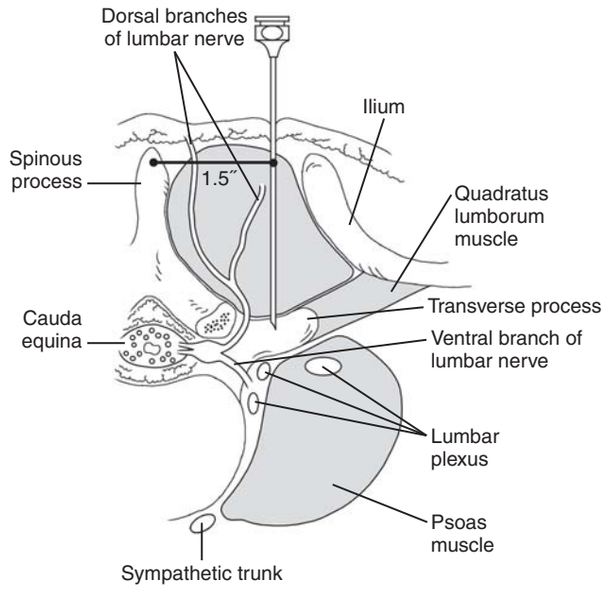


FIGURE 15–3
The patient lies in the prone position. The fluoroscope is positioned initially in the posteroanterior position to view the L3, L4, and L5 vertebrae.



FIGURE–15-5
Relationship between the spinous process and the point of needle entry.

If careful aspiration reveals no blood or cerebrospinal fluid, 25–30 ml of 1.0% preservative-free lidocaine containing dilute water-soluble contrast is slowly injected in incremental doses under fluoroscopic guidance to ensure proper spread of the local anesthetic and contrast. Care must be taken to observe the patient for signs of local anesthetic toxicity as the drugs are being injected. If the pain has an inflammatory component, the local anesthetic is combined with 80 mg of methylprednisolone and is injected in incremental doses. Subsequent daily nerve blocks are carried out in a similar manner, substituting 40 mg of methylprednisolone for the initial 80-mg dose.

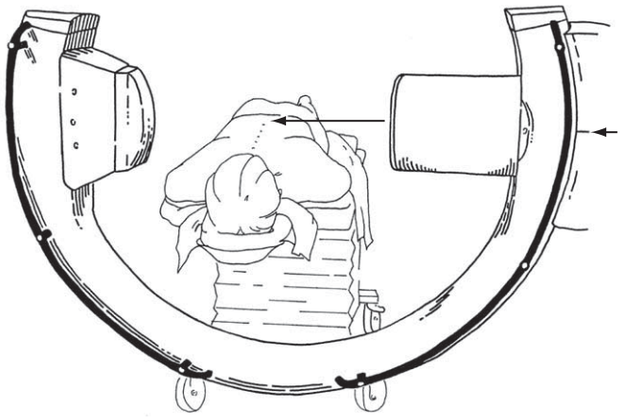
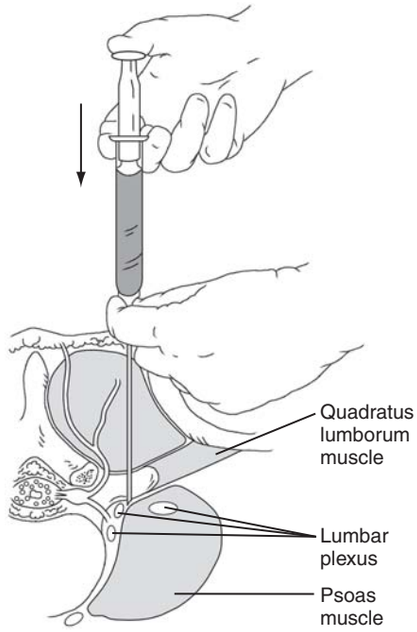


FIGURE 15–4
The position of the C-arm for viewing the lateral aspect of the lumbar spine.

COMPLICATIONS

The proximity to the spinal cord and exiting nerve roots makes it imperative that this procedure be performed only by those well versed in the regional anatomy and experienced in interventional pain management tech-



FIGURE–15-6
Constant pressure is maintained on the plunger of the syringe as the needle is advanced with the opposite hand.

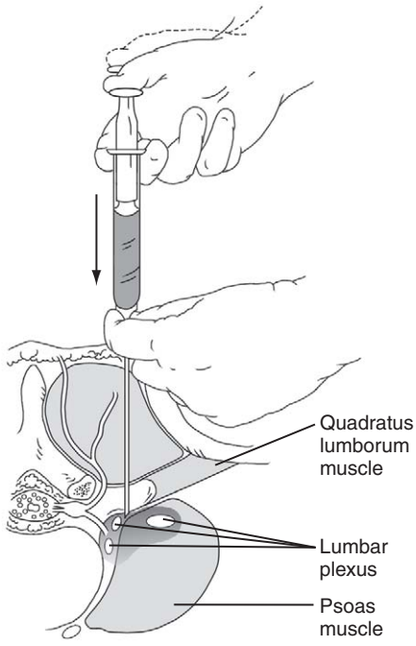


FIGURE 15–7
The operator will appreciate a sudden loss of resistance as the needle tip enters the psoas compartment.

niques. Needle placement that is too medial may result in epidural, subdural, or subarachnoid injections, or trauma to the spinal cord and exiting nerve roots. Placing the needle too deep between the transverse processes may result in trauma to the exiting lumbar nerve roots. Although uncommon, infection remains an ever-present possibility, especially in the immunocompromised cancer patient. Early detection of infection is crucial to avoid potentially life-threatening sequelae. Post-block back pain from trauma to the paraspinous musculature is not uncommon after lumbar plexus block using the psoas compartment technique.

CLINICAL PEARLS

Lumbar plexus nerve block via the psoas compartment technique is simple for those who understand the regional anatomy and have mastered the loss-of-resistance technique. Unfortunately, most of the things that can be done with lumbar plexus block can be done more easily with epidural or spinal techniques, which may be more acceptable to the surgeon and pain specialist alike. Neurolytic block with small quantities of phenol in glycerin or with absolute alcohol has been shown to provide long-term relief for patients suffering from cancer-related pain in whom more conservative treatments have been ineffectual. As mentioned earlier, the proximity of the lumbar paravertebral nerve to the neuraxis necessitates careful attention to technique.

ILIOHYPOGASTRIC NERVE BLOCK

ANATOMY

The iliohypogastric nerve is a branch of the L1 nerve root with a contribution from T12 in some patients.[4] The nerve follows a curvilinear course that takes it from its origin of the L1 and occasionally T12 somatic nerves to inside the concavity of the ilium. The iliohypogastric nerve continues anteriorly to perforate the transverse abdominis muscle to lie between it and the external oblique muscle. At this point, the iliohypogastric nerve divides into an anterior and a lateral branch. The lateral branch provides cutaneous sensory innervation to the posterolateral gluteal region. The anterior branch pierces the external oblique muscle just beyond the anterior superior iliac spine to provide cutaneous sensory innervation to the abdominal skin above the pubis (Figure 15-8). The nerve may interconnect with the ilioinguinal nerve along its course, resulting in variation of the distribution of the sensory innervation of the iliohypogastric and ilioinguinal nerves.

INDICATIONS

Iliohypogastric nerve block is useful in the evaluation and management of groin pain thought to be subserved by the iliohypogastric nerve, including the pain associated with iliohypogastric neuralgia.[5] The technique is also useful to provide surgical anesthesia for groin surgery,

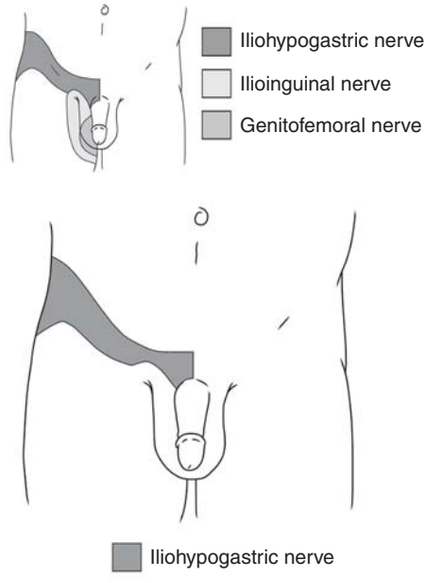


FIGURE 15–8
The sensory distribution of the iliohypogastric nerve.

including inguinal herniorrhaphy when combined with ilioinguinal and genitofemoral nerve block.[5] When a differential diagnosis needs to be made to delineate peripheral nerve entrapment versus lumbar radiculopathy, an illohypogastric nerve block with local anesthetic can be helpful. If destruction of the iliohypogastric nerve is being considered, this technique is useful as a prognostic indicator of the degree of motor and sensory impairment. Iliohypogastric nerve block with local anesthetic may be used to palliate acute pain emergencies, including postoperative pain relief while one is waiting for pharmacological methods to become effective. Iliohypogastric nerve block with local anesthetic and steroids is also useful in the treatment of persistent pain after inguinal surgery or groin trauma when the pain is thought to be secondary to inflammation or entrapment of the iliohypogastric nerve.[4]

Destruction of the iliohypogastric nerve is occasionally indicated for the palliation of persistent groin pain after blunt or open trauma to the groin or persistent pain mediated by the iliohypogastric nerve after groin or lower abdominal surgery. Iliohypogastric nerve block via a 25-gauge needle may be performed in the presence of coagulopathy or anticoagulation, albeit with an increased risk of ecchymosis and hematoma formation.

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represents a relatively strong contraindication to the performance of iliohypogastric nerve block. Some clinicians will consider performing iliohypogastric nerve block with a 25-gauge needle in the setting of patients with pain involving the iliohypogastric nerve in whom pain is uncontrolled by systemic analgesic. Local infection involving the area of the iliohypogastric nerve is also a contraindication to the performance of iliohypogastric nerve block.

EQUIPMENT

- Peripheral nerve block tray
- 22-gauge, 1-1/2-inch needle
- 25-gauge, 1-1/2-inch needle
- 18-gauge, 1-1/2-inch needle
- 12-ml sterile syringe

DRUGS

- 1% preservative-free lidocaine (for diagnostic or prognostic block)
- 0.25% preservative-free bupivacaine (for therapeutic block)
- Depot methylprednisolone (for therapeutic block)
- 6.5% aqueous phenol (for chemical neurolytic block)

PROCEDURE

The patient is placed in the supine position with a pillow under the knees if extending the legs increases the patient's pain because of traction on the nerve. The anterior superior iliac spine is identified by palpation. A point 1 inch medial and 1 inch inferior to the anterior superior iliac spine is then identified and prepared with antiseptic solution. A 25-gauge, 1-1/2-inch needle is then advanced at an oblique angle toward the pubic symphysis (Figure 15-9). Five to 7 ml of 1.0% preservative-free lidocaine is injected in a fanlike manner as the needle pierces the fascia of the external oblique muscle. A small amount of water-soluble contrast medium can be added to the local anesthetic solution, and the spread of contrast of local anesthetic can be observed under fluoroscopy. Care must be taken not to place the needle too deep and enter the peritoneal cavity and perforate the abdominal viscera.

If the pain has an inflammatory component, the local anesthetic is combined with 80 mg of methylprednisolone and is injected in incremental doses. Subsequent daily nerve blocks are performed similarly, substituting 40 mg of methylprednisolone for the initial 80-mg dose. Because of overlapping innervation of the ilioinguinal and iliohypogastric nerves, it is not unusual to block branches of each nerve when performing iliohypogastric nerve block. After injection of the solution, pressure is applied to the injection site to decrease the incidence of postblock ecchymosis and hematoma formation, which can be dramatic, especially in the patient on anticoagulants.

COMPLICATIONS

The main complication of iliohypogastric nerve block is postblock ecchymosis and hematoma formation. If needle placement is too deep and enters the peritoneal cavity,

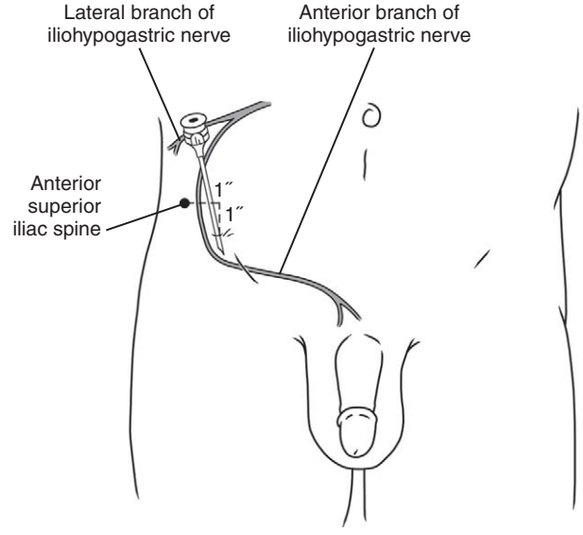


FIGURE 15–9
The relationship of the anterior superior iliac spine and the iliohypogastric nerve.

perforation of the colon may result in intra-abdominal abscess and fistula formation. Early detection of infection is crucial to avoid potentially life-threatening sequelae.

CLINICAL PEARLS

Iliohypogastric nerve block is a simple technique that can produce dramatic relief for patients suffering from the previously mentioned pain complaints. Neurolytic block with small quantities of phenol in glycerin has been done in the past. However, newer methods for neurolyis via cryotherapy or radiofrequency lesioning is now recommended. These methods have shown to provide long-term relief for patients suffering from chronic pain secondary to trauma to the ilioinguinal nerve in whom more conservative treatments have been ineffectual. As mentioned earlier, pressure should be maintained on the injection site post-block to avoid ecchymosis and hematoma formation.

If a patient presents with pain suggestive of iliohypogastric neuralgia and iliohypogastric nerve blocks are ineffectual, a diagnosis of lesions more proximal in the lumbar plexus or an L1 radiculopathy should be considered. Such patients often respond to epidural steroid blocks. Electromyography and magnetic resonance imaging of the lumbar plexus are indicated in this patient population to help rule out other causes of groin pain, including malignancy invading the lumbar plexus or epidural or vertebral metastatic disease at T12-L1.

ILIOINGUINAL NERVE BLOCK

ANATOMY

The ilioinguinal nerve is a branch of the L1 nerve root with a contribution from T12 in some patients.[6] The nerve follows a curvilinear course that takes it from its origin of the L1 and occasionally T12 somatic nerves to inside the concavity of the ilium. The ilioinguinal nerve continues anteriorly to perforate the transverse abdominis muscle at the level of the anterior superior iliac spine. The nerve may interconnect with the iliohypogastric nerve as it continues to pass along its course medially and inferiorly, where it accompanies the spermatic cord through the inguinal ring and into the inguinal canal. The distribution of the sensory innervation of the ilioinguinal nerves varies from patient to patient because there may be considerable overlap with the iliohypogastric nerve. In general, the ilioinguinal nerve provides sensory innervation to the upper portion of the skin of the inner thigh and the root of the penis and upper scrotum in men (Figure 15-10) or the mons pubis and lateral labia in women.

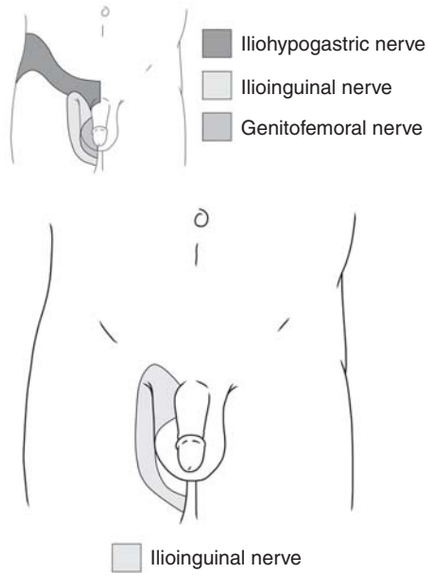


FIGURE 15–10
The relationship of the anterior superior iliac spine and the iliohypogastric nerve.

INDICATIONS

Ilioinguinal nerve block is useful in the evaluation and management of groin pain thought to be subserved by the ilioinguinal nerve, including the pain associated with ilioinguinal neuralgia.[6,7] The technique is also useful to provide surgical anesthesia for groin surgery, including inguinal herniorrhaphy when combined with ilioinguinal and genitofemoral nerve block.[5,6] Ilioinguinal nerve block with local anesthetic can be used diagnostically during differential neural blockade on an anatomical basis in the evaluation of groin pain when peripheral nerve entrapment versus lumbar radiculopathy is being evaluated. If destruction of the ilioinguinal nerve is being considered, this technique is useful as a prognostic indicator of the degree of motor and sensory impairment. Ilioinguinal nerve block with local anesthetic may be used to palliate acute pain emergencies, including postoperative pain relief while one is waiting for pharmacological methods to become effective. Ilioinguinal nerve block with local anesthetic and steroids is also useful in the treatment of persistent pain after inguinal surgery or groin trauma when the pain is thought to be secondary to inflammation or entrapment of the ilioinguinal nerve.[6]

Destruction of the ilioinguinal nerve is occasionally indicated for the palliation of persistent groin pain after blunt or open trauma to the groin or persistent pain mediated by the ilioinguinal nerve after groin or lower abdominal surgery. Ilioinguinal nerve block via a 25-gauge needle may be performed in the presence of coagulopathy or anticoagulation, albeit with an increased risk of ecchymosis and hematoma formation.

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represents a relatively strong contraindication to the performance ilioinguinal nerve block. Some clinicians

will consider performing ilioinguinal nerve block with a 25-gauge needle in the setting of patients with pain involving the ilioinguinal nerve in whom pain is uncontrolled by systemic analgesic. Local infection involving the area of the ilioinguinal nerve is also a contraindication to the performance of the ilioinguinal nerve block.

EQUIPMENT

- Peripheral nerve block tray
- 22-gauge, 1-1/2-inch needle
- 25-gauge, 1-1/2-inch needle
- 18-gauge, 1-1/2-inch needle
- 12-ml sterile syringe

DRUGS

- 1% preservative-free lidocaine (for diagnostic or prognostic block)
- 0.25% preservative-free bupivacaine (for therapeutic block)
- Depot methylprednisolone (for therapeutic block)
- 6.5% aqueous phenol (for chemical neurolytic block)

PROCEDURE

The patient is placed in the supine position with a pillow under the knees if extending the legs increases the patient's pain because of traction on the nerve. The anterior superior iliac spine is identified by palpation. A point 2 inches medial and 2 inches inferior to the anterior superior iliac spine is then identified and prepared with antiseptic solution. A 1-1/2-inch, 25-gauge needle is then advanced at an oblique angle toward the pubic symphysis (Figure 15-11). Five to 7 ml of 1.0% preservative-free lidocaine is injected in a fanlike manner as the needle pierces the fascia of the external oblique muscle. A small amount of water-soluble contrast medium can be added to the local anesthetic solution and the spread of contrast of local anesthetic can be observed under fluoroscopy. Care must be taken not to place the needle too deep and enter the peritoneal cavity and perforate the abdominal viscera.

If the pain has an inflammatory component, the local anesthetic is combined with 80 mg of methylprednisolone and is injected in incremental doses. Subsequent daily nerve blocks are performed similarly, substituting 40 mg of methylprednisolone for the initial 80-mg dose. Because of overlapping innervation of the ilioinguinal and iliohypogastric nerves, it is not unusual to block branches of each nerve when performing ilioinguinal nerve block. After the solution is injected, pressure is applied to the injection site to decrease the incidence of postblock ecchymosis and hematoma formation, which can be dramatic, especially when the patient is on anticoagulants.

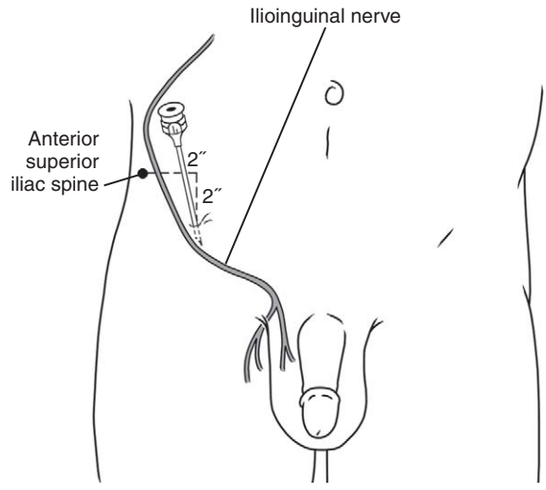


FIGURE 15–11
The relationship of the anterior superior iliac spine and the ilioinguinal nerve.

COMPLICATIONS

The main complication of ilioinguinal nerve block is postblock ecchymosis and hematoma formation. If needle placement is too deep and enters the peritoneal cavity, perforation of the colon may result in intraabdominal abscess and fistula formation. Early detection of infection is crucial to avoid potentially life-threatening sequelae.

CLINICAL PEARLS

Ilioinguinal nerve block is a simple technique that can produce dramatic relief for patients suffering from the previously mentioned pain complaints. Neurolytic block with small quantities of phenol in glycerin has been done in the past. However, newer methods for neurolysis via cryotherapy or radiofrequency lesioning is now recommended. These methods have been shown to provide long-term relief for patients suffering from chronic pain secondary to trauma to the ilioinguinal nerve in whom more conservative treatments have been ineffectual. As mentioned earlier, pressure should be maintained on the injection site postblock to avoid ecchymosis and hematoma formation.

If a patient presents with pain suggestive of ilioinguinal neuralgia and ilioinguinal nerve blocks are ineffectual, a diagnosis of lesions more proximal in the lumbar plexus or an L1 radiculopathy should be considered. Such patients often respond to epidural steroid blocks. Electromyography and magnetic resonance imaging of the lumbar plexus are indicated in this patient population to help rule out other causes of groin pain, including malignancy invading the lumbar plexus or epidural or vertebral metastatic disease at T12-L1 (Figure 15-12).

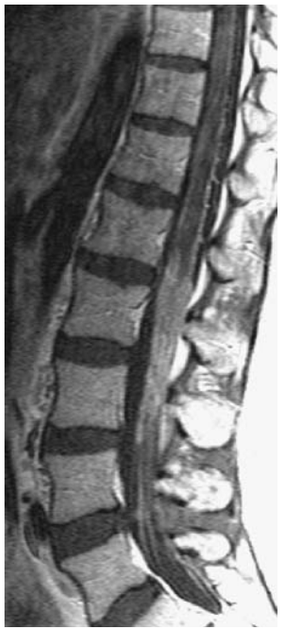


FIGURE 15–12

Leptomeningeal metastatic disease. A sagittal T1-weighted spin echo MR image obtained after intravenous gadolinium administration shows abnormal enhancement of the surface of the distal portion of the spinal cord, as well as of all the roots of the cauda equina. (From Resnick D: *Diagnosis of Bone and Joint Disorders*, 4th ed. Philadelphia, Saunders, 2002, p. 483, with permission.)

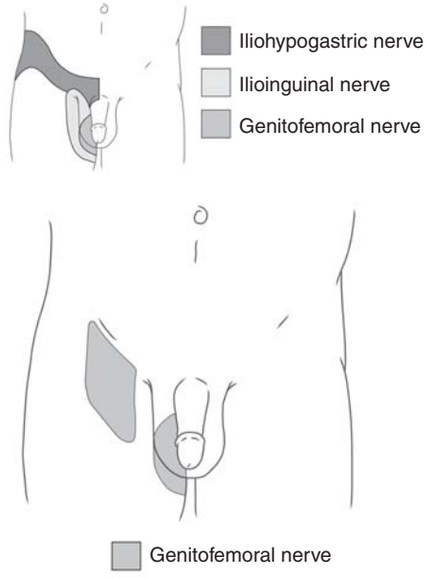


FIGURE 15–13

The relationship of the anterior superior iliac spine and the genitofemoral nerve.

GENITOFEMORAL NERVE BLOCK

ANATOMY

The genitofemoral nerve is a branch of the L1 nerve root with a contribution from T12 in some patients.[8] The nerve follows a curvilinear course that takes it from its origin of the L1 and occasionally T12 and L2 somatic nerves to inside the concavity of the ilium. The genitofemoral nerve descends obliquely in an anterior course through the psoas major muscle to emerge on the abdominal surface opposite L3 or L4. The nerve descends retroperitoneally behind the ureter and divides into a genital and femoral branch just above the inguinal ligament. In males, the genital branch travels through the inguinal canal passing inside the deep inguinal ring to innervate the cremaster muscle and skin of the scrotum. In females, the genital branch follows the course of the round ligament and provides innervation to the ipsilateral mons pubis and labia majora. In males and females, the femoral branch descends lateral to the external iliac artery to pass behind the inguinal ligament. The nerve enters the femoral sheath lateral to the femoral artery to innervate the skin of the anterior superior femoral triangle (Figure 15-13).

INDICATIONS

Genitofemoral nerve block is useful in the evaluation and management of groin pain thought to be subserved by the genitofemoral nerve, including the pain associated with genitofemoral neuralgia.[9] The technique is also useful to provide surgical anesthesia for groin surgery, including inguinal herniorrhaphy when combined with iliohypogastric and ilioinguinal nerve block. Genitofemoral nerve block with local anesthetics can be used diagnostically during differential neural blockade on an anatomical basis in the evaluation of groin pain when peripheral nerve entrapment versus lumbar radiculopathy is being evaluated. If destruction of the genitofemoral nerve is being considered, this technique is useful as a prognostic indicator of the degree of motor and sensory impairment. Genitofemoral nerve block with local anesthetic may be used to palliate acute pain emergencies, including postoperative pain relief while one is waiting for pharmacological methods to become effective. Genitofemoral nerve block with local anesthetic and steroids is also useful in the treatment of persistent pain after inguinal surgery or groin trauma when the pain is thought to be secondary to inflammation or entrapment of the genitofemoral nerve.

Destruction of the genitofemoral nerve is occasionally indicated for the palliation of persistent groin pain after blunt or open trauma to the groin or persistent pain mediated by the genitofemoral nerve after groin surgery. Genitofemoral nerve block via a 25-gauge needle may be performed in the presence of coagulopathy or anticoagulation, albeit with an increased risk of ecchymosis and hematoma formation.

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represents a relatively strong contraindication to the performance of genitofemoral nerve block. Some clinicians will

consider performing genitofemoral nerve block with a 25-gauge needle in the setting of patients with pain involving the genitofemoral nerve in whom pain is uncontrolled by systemic analgesic. Local infection involving the area of the genitofemoral nerve is also a contraindication to the performance of genitofemoral nerve block.

EQUIPMENT

- Peripheral nerve block tray
- 22-gauge, 1-1/2-inch needle
- 25-gauge, 1-1/2-inch needle
- 18-gauge, 1-1/2-inch needle
- 12-ml sterile syringe

DRUGS

- 1% preservative-free lidocaine (for diagnostic or prognostic block)
- 0.25% preservative-free bupivacaine (for therapeutic block)
- Depot methylprednisolone (for therapeutic block)
- 6.5% aqueous phenol (for chemical neurolytic block)

PROCEDURE

The patient is placed in the supine position with a pillow under the knees if extending the legs increases the patient's pain because of traction on the nerve. The anterior superior iliac spine, femoral artery, femoral crease, and the pubic tubercle are identified by palpation.

To block the genital branch of the genitofemoral nerve, the pubic tubercle and the inguinal ligament are identified. A point just lateral to the pubic tubercle just below the inguinal ligament is then identified and prepared with antiseptic solution. A 1-1/2-inch, 25-gauge needle is then advanced through the skin and subcutaneous tissues (Figure 15-14). Five milliliters of 1.0% preservative-free lidocaine are injected after careful aspiration. A small amount of water-soluble contrast medium can be added to the local anesthetic solution, and the spread of contrast of local anesthetic can be observed under fluoroscopy. Care must be taken not to place the needle too deep and enter the peritoneal cavity and perforate the abdominal viscera, or to inadvertently inject the local anesthetic into the femoral artery.

If the pain has an inflammatory component, the local anesthetic for the above blocks is combined with 80 mg of methylprednisolone and is injected in incremental doses. Subsequent daily nerve blocks are performed similarly, substituting 40 mg of methylprednisolone for the initial 80-mg dose. Because of overlapping innervation of the genitofemoral, ilioinguinal, and iliohypogastric nerves, it is not unusual to block branches of each nerve when performing genitofemoral nerve block. After the solution is injected, pressure is applied to the injection site to decrease the incidence of postblock ecchymosis and hematoma formation, which can be dramatic, especially when the patient is on anticoagulants.

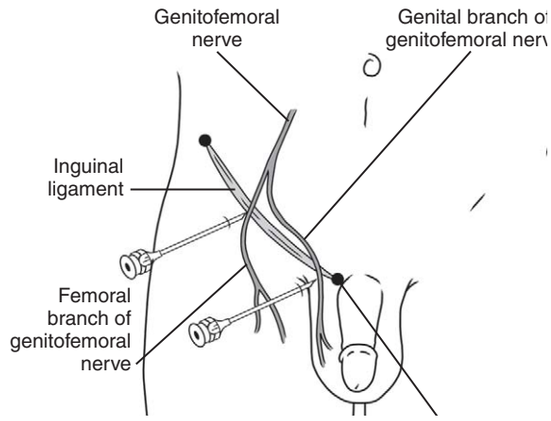


FIGURE 15–14
The relationship between the pubic tubercle and the genitofemoral nerve.

COMPLICATIONS

The major complication of genitofemoral nerve block is postblock ecchymosis and hematoma formation. If needle placement is too deep and enters the peritoneal cavity, perforation of the colon may result in intra-abdominal abscess and fistula formation. Early detection of infection is crucial to avoid potentially life-threatening sequelae.

CLINICAL PEARLS

Genitofemoral nerve block is a simple technique that can produce dramatic relief for patients suffering from the previously mentioned pain complaints. Neurolytic block with small quantities of phenol in glycerin has been done in the past. However, newer methods for neurolysis via cryotherapy or radiofrequency lesioning is now recommended. These methods have been shown to provide long-term relief for patients suffering from chronic pain secondary to trauma to the genitofemoral nerve in whom more conservative treatments have been ineffectual. As mentioned earlier, pressure should be maintained on the injection site postblock to avoid ecchymosis and hematoma formation.

If a patient presents with pain suggestive of genitofemoral neuralgia, and genitofemoral nerve blocks are ineffectual, a diagnosis of lesions more proximal in the lumbar plexus or an L1 radiculopathy should be considered. Such cases often respond to epidural steroid blocks. Electromyography and magnetic resonance imaging of the lumbar plexus are indicated in this patient population

to help rule out other causes of genitofemoral pain, including malignancy invading the lumbar plexus or epidural or vertebral metastatic disease at T12-L1.

LATERAL FEMORAL CUTANEOUS NERVE BLOCK

ANATOMY

The lateral femoral cutaneous nerve is formed from the posterior divisions of the L2 and L3 nerves.[10] The nerve leaves the psoas muscle and courses laterally and inferiorly to pass just beneath the ilioinguinal nerve at the level of the anterior superior iliac spine (Figure 15-15). The nerve passes under the inguinal ligament and then travels beneath the fascia lata, where it divides into an anterior and a posterior branch. The anterior branch provides limited cutaneous sensory innervation over the anterolateral thigh. The posterior branch provides cutaneous sensory innervation to the lateral thigh from just above the greater trochanter to the knee (Figure 15-16).

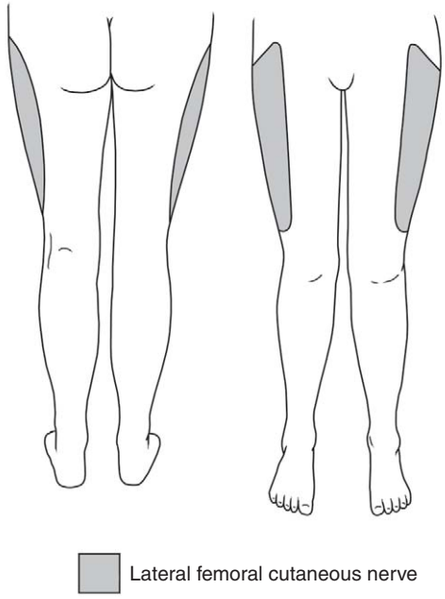


FIGURE 15–16
The sensory distribution of the lateral femoral cutaneous nerve.

INDICATIONS

Lateral femoral cutaneous nerve block is useful in the evaluation and management of lateral thigh pain thought to be subserved by the lateral femoral cutaneous nerve, including meralgia paresthetica.[11] The technique is also useful to provide surgical anesthesia for skin graft harvest procedures from the lateral thigh and to relieve tourniquet pain. Lateral femoral cutaneous nerve block with local anesthetic can be used as a diagnostic tool during differential neural blockade on an anatomical basis in the evaluation of lateral pain when peripheral nerve entrapment versus lumbar radiculopathy is being evaluated. If destruction of the lateral femoral cutaneous nerve is being considered, this technique is useful as a prognostic indicator of the degree of motor and sensory impairment that the patient may experience. Lateral femoral cutaneous nerve block with local anesthetic may be used to palliate acute pain emergencies, including postoperative pain relief while waiting for pharmacologic methods to become effective. Lateral femoral cutaneous nerve block with local anesthetic and steroid is also useful in the treatment of persistent pain after inguinal or bone harvest surgery from the iliac crest when the pain is thought to be secondary to inflammation or entrapment of the lateral femoral cutaneous nerve.

Destruction of the lateral femoral cutaneous nerve is occasionally indicated for the palliation of persistent groin pain after blunt or open trauma to the groin or persistent pain mediated by the lateral femoral cutaneous nerve after groin surgery. A 25-gauge needle may be used for lateral femoral cutaneous nerve block in the presence of coagulopathy or anticoagulation, albeit with an increased risk of ecchymosis and hematoma formation.

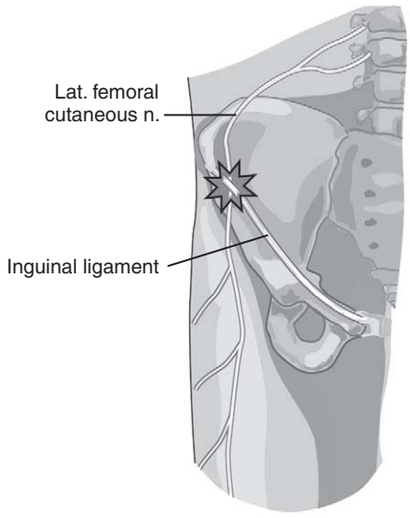


FIGURE 15–15
Meralgia paresthetica is caused by compression of the lateral cutaneous nerve by the inguinal ligament as it passes through or under the inguinal ligament. (From Waldman SD: *Physical Diagnosis of Pain: An Atlas of Signs and Symptoms*. Philadelphia, Saunders, 2005, p. 278, with permission.)

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represents a relatively strong contraindication to the performance of lateral femoral cutaneous nerve block. Some

clinicians will consider performing lateral femoral cutaneous nerve block with a 25-gauge needle in the setting of patients with pain involving the lateral femoral cutaneous nerve in whom pain is uncontrolled by systemic analgesic. Local infection involving the area of the lateral femoral cutaneous nerve block nerve is also a contraindication to the performance of lateral femoral cutaneous nerve block.

EQUIPMENT

- Peripheral nerve block tray
- 22-gauge, 1-1/2-inch needle
- 25-gauge, 1-1/2-inch needle
- 18-gauge, 11/2-inch needle
- 12-ml sterile syringe

DRUGS

- 1% preservative-free lidocaine (for diagnostic or prognostic block)
- 0.25% preservative-free bupivacaine (for therapeutic block)
- Depot methylprednisolone (for therapeutic block)
- 6.5% aqueous phenol (for chemical neurolytic block)

PROCEDURE

The patient is placed in the supine position with a pillow under the knees if the legs-extended position increases the patient's pain because of traction on the nerve. The anterior superior iliac spine is identified by palpation. A point 1 inch medial to the anterior superior iliac spine and just inferior to the inguinal ligament is then identified and prepared with antiseptic solution (Figure 15-17). A 25-gauge, 1-1/2-inch needle is then slowly advanced perpendicular to the skin until the needle is felt to pop through the fascia. A paresthesia is often elicited. After careful aspiration, 5–7 ml of 1.0% preservative-free lidocaine is injected in a fanlike manner as the needle pierces the fascia of the external oblique muscle. A small amount of water-soluble contrast medium can be added to the local anesthetic solution, and the spread of contrast of local anesthetic can be observed under fluoroscopy. Care must be taken not to place the needle too deep and enter the peritoneal cavity and perforate the abdominal viscera.

If the pain has an inflammatory component, the local anesthetic is combined with 80 mg of methylprednisolone and is injected in incremental doses. Subsequent daily nerve blocks are carried out in a similar manner, substituting 40 mg of methylprednisolone for the initial 80-mg dose. After injection of the solution, pressure is applied to the injection site to decrease the incidence of postblock ecchymosis and hematoma formation, which can be dramatic, especially in the patient receiving anticoagulants.

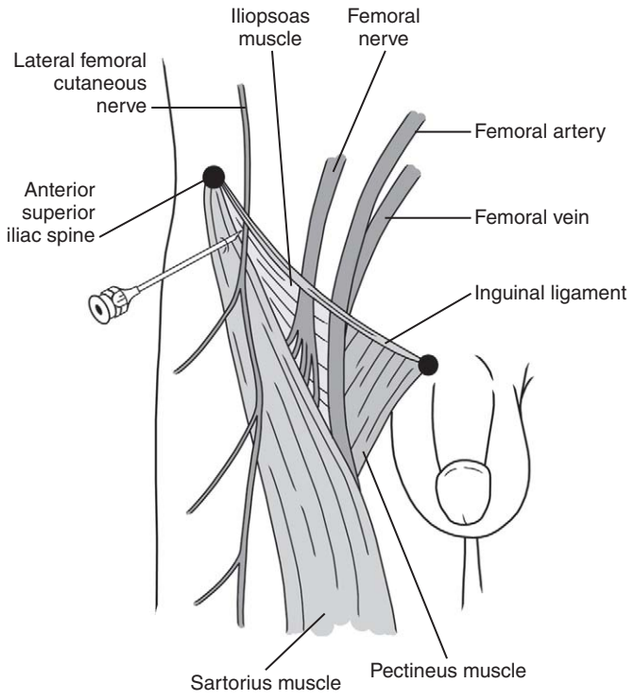


FIGURE 15–17
The relationship between the anterior superior iliac spine and the lateral femoral cutaneous nerve.

COMPLICATIONS

The main side effect of lateral femoral cutaneous nerve block is postblock ecchymosis and hematoma. If the needle is placed too deep and enters the peritoneal cavity, perforation of the colon may result in the formation of intra-abdominal abscess and fistula formation. Early detection of infection is crucial to avoid potentially life-threatening sequelae. If the needle is placed too medial, blockade of the femoral nerve may occur and make ambulation difficult.

CLINICAL PEARLS

Lateral femoral cutaneous nerve block is a simple technique that can produce dramatic relief for patients suffering from the mentioned pain complaints. Neurolytic block with small quantities of phenol in glycerin has been done in the past. However, newer methods for neurolysis via crotherapy or radiofrequency lesioning is now recommended. These methods have been shown to provide long-term relief for patients suffering from chronic pain secondary to trauma to the lateral femoral cutaneous nerve in whom more conservative treatments have been ineffectual. As mentioned earlier, pressure should be maintained on the injection site post block to avoid ecchymosis and hematoma formation.

Lateral femoral cutaneous neuralgia is often misdiagnosed as either trochanteric bursitis or lumbar radiculopathy. Also known as *meralgia paresthetica*, lateral femoral cutaneous neuralgia is characterized as dysesthetic pain and numbness in the lateral thigh. The pain is made worse when sitting or squatting for long periods. This painful condition may also occur secondary to compression on the nerve by wide belts or tool pouches. Blockade of the lateral femoral cutaneous nerve with local anesthetic should provide prompt relief of symptoms. Electromyography can help confirm the diagnosis. Therapeutic lateral femoral cutaneous nerve blocks with local anesthetic and steroid are extremely beneficial when treating meralgia paresthetica.

If a patient presents with pain suggestive of lateral femoral cutaneous neuralgia and lateral femoral cutaneous nerve blocks are ineffectual, a diagnosis of lesions more proximal in the lumbar plexus or L2-L3 radiculopathy should be considered. Such patients often respond to epidural steroid blocks. Electromyography and magnetic resonance imaging of the lumbar plexus are indicated in this patient population to help rule out other causes of lateral femoral cutaneous pain, including malignancy invading the lumbar plexus or epidural or vertebral metastatic disease at L2-L3.

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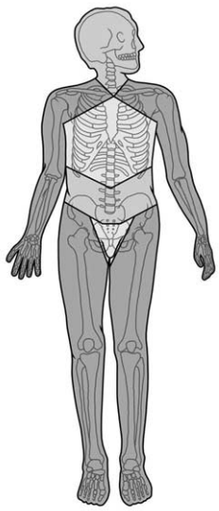

16

Lumbar Sympathetic Blocks

DAVID NIV AND MICHAEL GOFELD

CELIAC GANGLION BLOCK AND NEUROLYSIS

HISTORY

In 1914, Kappis[1] introduced the percutaneous splanchnic and celiac plexus block with local anesthetic via posterior approach and proposed it for a surgical anesthesia. He rapidly gained experience with this technique and reported a series of 200 patients in 1918.[2] At the same time, Wendling[3] described the anterior percutaneous approach, a method of blocking the celiac plexus utilizing a single needle placed anteriorly through the liver. Judged to be riskier than Kappis's posterior approach, it rapidly fell into disfavor. Seven decades later, an anterior approach was "rediscovered" using computerized tomography[4] or ultrasound[5] guidance. In 1996, a transgastric endoscopic technique was also developed.[6]

Over the ensuing 30 years since Kappis invented his technique, Labat, Farr, and others[7–9] introduced various modifications. It is important to note that the celiac plexus and splanchnic nerve block were initially used as a surgical anesthetic technique. However, because of the complexity and variable outcome of this technique, in due course neuroaxial anesthesia and segmental blockade of the somatic paravertebral nerves were needed to supplement it.[10]

As celiac plexus and splanchnic nerve blocks were falling into disuse for surgical anesthesia, the clinical utility of these techniques was becoming apparent in the new specialty of pain management. Jones first described alcohol neurolysis of the splanchnic nerves and celiac plexus for long-lasting relief of abdominal pain in 1957 via the classic retrocrural approach.[11] Bridenbaugh and colleagues[12] reported on the role of neurolytic celiac plexus block to treat the pain of upper abdominal malignancy. In 1978, Boas[13] set retrocrural technique apart from transcrural. Four years later, Ischia[14] described a transaortic one-needle injection. There is renewed interest in the anterior approach to celiac plexus block, using computed tomography (CT) or ultrasonography to allow more accurate needle placement.[15,16]

In spite of various modifications, Kappis's classic posterior approach to the celiac plexus and splanchnic nerves continues to serve as the basis for contemporary techniques. It is important to mention that none of the techniques developed show superiority in relation to safety and success aspects. In general, the celiac plexus and splanchnic neurolytic lesions yield 70–90% of long-lasting pain relief in abdominal and mainly in pancreatic malignancy.[17,18]

ANATOMY

Innervation of the abdominal viscera originates in the anterolateral horn of the spinal cord with the ventral spinal routes to join the white communicating rami and route to the sympathetic chain. In contradistinction to other preganglionic sympathetic nerves, these axons do not synapse in the sympathetic chain; rather, they pass through the chain, to synapse at distal sites, including the celiac, aortic renal, and superior mesenteric ganglia. Postganglionic nerves accompany blood vessels to their respective visceral structures.

Preganglionic nerves from T5-T9 and occasionally T4 and T10 travel caudally from the sympathetic chain along the lateral and anterolateral aspects of the vertebral bodies. At the level of T9 and T10, the axons coalesce to form the greater splanchnic nerve, course through the diaphragm, and end as numerous terminal endings in the celiac plexus. Most travel ipsilaterally, but a few cross and synapse with contralateral postganglionic cell bodies.

Sympathetic nerves from T10-T11 and, occasionally, T12, combine to form the lesser splanchnic nerve. Their course parallels the greater splanchnic nerve in a posterolateral position and ends in either the celiac

plexus or aorticorenal ganglion. The least splanchnic nerves arise from T12, parallel posteriorly the lesser splanchnic nerve, and synapse in the aorticorenal ganglion (Figure 16-1).

Afferent spinal fibers join sympathetic nerve fibers to transmit nociceptive signals from the abdominal viscera. The cell bodies of these afferent spinal nerves exist in the posterior roots. Next, the proximal axons of the afferent spinal nerve cell bodies synapse in the dorsal horn of the spinal cord.

The celiac plexus is about 3 cm in length and 4 cm in width, typically lies anteriorly and anterolaterally to the aorta at the level between the T12-L1 intervertebral disc and L2 vertebral body. It is located just anterior to the crus of the diaphragm, a fact that becomes important for selection of the approach (Figure 16-2). Fibers within the plexus arise from preganglionic splanchnic nerves, parasympathetic preganglionic nerves from the vagus, some sensory nerves from the phrenic and vagus nerves, and sympathetic postganglionic fibers. Afferent fibers concerned with nociception pass diffusely through the celiac plexus and represent the main target of celiac plexus blockade.

These fibers coalesce to form a dense, intertwining network of autonomic nerves. Three pairs of ganglia exist within the plexus: (1) celiac ganglia, (2) superior mesenteric ganglia, and (3) aortic renal ganglia. Postganglionic nerves from these ganglia innervate all of the abdominal viscera with the exception of part of the transverse colon, the left colon, the rectum, and the pelvic viscera.[19]

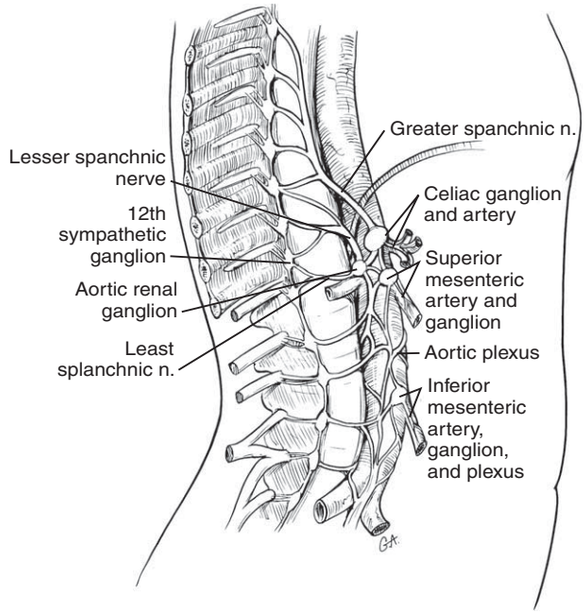

FIGURE 16–1

This line drawing shows the formation of the greater, lesser, and least splanchnic nerves arising from T5-T12. Note the final destination of these nerves into the celiac ganglion and the superior and inferior mesenteric ganglion.

INDICATIONS

Any pain originating from visceral structures and innervated by the celiac plexus can be effectively alleviated by block of the plexus. These structures include the pancreas, liver, gallbladder, omentum, mesentery, and alimentary tract from the stomach to the transverse portion of the large colon. It is commonly applied to treat pain of intra-abdominal malignant origin, particularly pain deriving from pancreatic cancer.

The vast majority of cancer patients receive opioids for the treatment of pain. An additional benefit in these patients may be the effect of celiac plexus block on gastric motility. Complete sympathetic denervation of the gastrointestinal tract allows unopposed parasympathetic activity and increases peristalsis. Whereas diarrhea has been reported in a few patients, concomitant decrease in the incidence of nausea and vomiting has also been

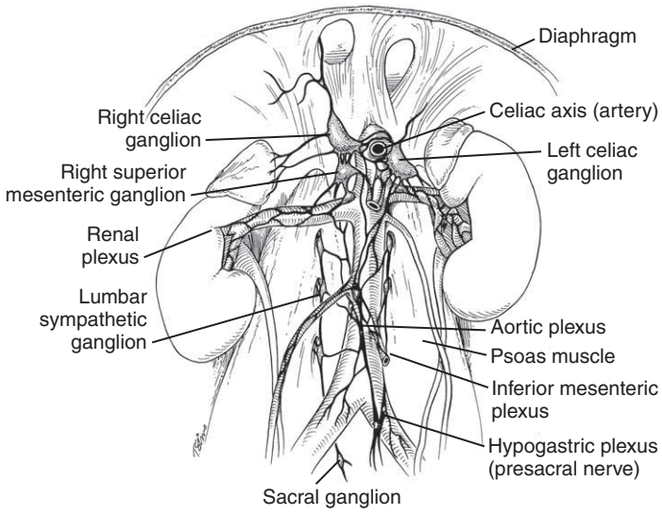

FIGURE 16–2

Anterior view of the celiac plexus. The relationship to nearby structures is shown. Note the dense, diffuse intertwining network of nerves that form the plexus.

reported. The presence of severe nausea and vomiting may be another important indication in patients with pancreatic cancer.

CONTRAINDICATIONS

Owing to the proximity to vascular structures, celiac plexus block is contraindicated in patients who are on anticoagulant therapy or suffer from coagulation abnormalities, antiblastic cancer therapies, or liver failure.[19,20] Local or intra-abdominal infection and sepsis represent absolute contraindications to celiac plexus block. Calcification, mural trombus, or aortic aneurysm at the level of celiac plexus makes transaortic approach excessively risky.

Blockade of the celiac plexus results in greater bowel motility; therefore, the technique should be avoided in patents with bowel obstruction.[19] Neurolytic celiac plexus block should probably be deferred in patients who suffer from chronic abdominal pain, who are chemically dependent or who exhibit drug-seeking behavior, until these relative contraindications have been adequately addressed.[20] The use of alcohol as a neurolytic agent should be avoided in patients on disulfiram therapy for alcohol abuse.

EQUIPMENT

- 25-gauge, skin-infiltration needle
- 22-gauge, 10-cm spinal needle for deep infiltration
- 20–21-gauge, 15-cm Chiba needle or 20-gauge blunt Coudé or straight block needle
- 16-gauge angiocath for facilitation of placement of blunt needle

DRUGS

- *Local anesthesia block*
- 0.2–0.5% ropivacaine, 0.25–0.5% bupivacaine, or 1–2% lidocaine, 20–40 ml
- Depo steroids (optional): methylprednisolone (DepoMedrol), 40 mg, triamcinolone, 40 mg or equivalent
- *Phenol neurolysis*
- 6–10% phenol in Omnipaque, 20–30 ml (total)
- *Alcohol*
- 50–100% alcohol, 20–30 ml (total)

PREPARATION OF PATIENT

Preoperative Tests

- PT, PTT, CBC
- Chest x-ray
- Abdominal CT or MRI (for recognizing the anatomical distortions due to cancer in area of block and detection of aortic pathologies)

Preprocedure Medication

Use the standard recommended protocol for conscious sedation by the American Society of Anesthesiologists.

PROCEDURE

Posterior (Classic, Retrocrural) Approach

The patient is placed in the prone position with a pillow beneath the abdomen to reverse the thoracolumbar lordosis. This position increases the distance between the costal margins and the iliac crests and between the transverse processes of adjacent vertebral bodies. Once the patient is positioned appropriately, a fluoroscopic view is taken in a PA position. Then the fluoroscope is turned obliquely for a "tunnel" view entry of the needle (Figure 16-3). For comfort, the patient's head is turned to the side, and the arms are permitted to hang freely off either side of the table. The operative field is prepared and draped in standard aseptic manner.

Some clinicians find it beneficial to delineate the pertinent landmarks on the skin with a sterile marker. The landmarks include the iliac crests, 12th ribs, dorsal midline, vertebral bodies (T12-T2), and lateral borders of the paraspinal (sacrospinalis) muscles. Moore[21] recommends that the intersection of the 12th rib and the lateral border of the paraspinal muscles on each side (which corresponds to L2) be marked and connected with lines to each other and to the cephalic portion of the L1 spine, forming an isosceles triangle, the sides of which serve as an additional guide to needle positioning (Figure 16-4).

The skin and underlying subcutaneous tissues and musculature are infiltrated with 1.0% lidocaine at the points of needle entry, 7.5 cm, approximately four fingerbreadths, lateral to the midline, just beneath the 12th ribs. Chiba needles, 20- or 21-gauge, 15-cm, are inserted

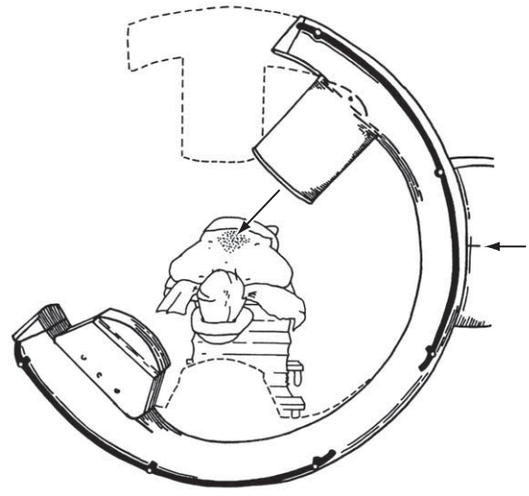

FIGURE 16–3

Patient is placed in prone position. The C-arm is placed in a PA position to locate the T12 vertebral body. The C-arm is then turned oblique to locate the print of entry of the tunnel view.

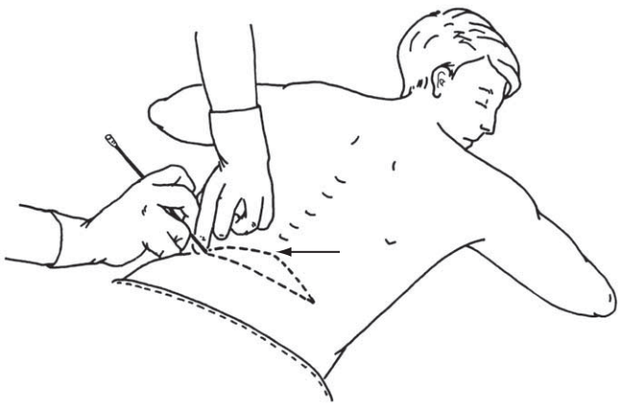

FIGURE 16–4

Moore's technique for identifying the landmarks to the celiac plexus. Note the dotted line shows the course of the 12th ribs from T12 vertebrae at a distance of 7-8cm. The lateral points of the 12th rib lines are closed with a transverse line. This forms an isosceles triangle.

bilaterally through the previously anesthetized areas. The needles are initially oriented 45 degrees toward the midline and about 15 degrees cephalad, to ensure contact with the L1 vertebral body (Figure16-5). Once contact with the vertebral body has been verified, the depth at which bone contact occurred is noted. Some clinicians find it useful to actually mark this measurement on the shaft of the needle using the Chiba needle silicone ring after the needle is withdrawn.

After bony contact is made and the depth is noted, the needles are withdrawn to the level of the subcutaneous tissue and redirected slightly lateral (about 60 degrees from the midline) in order to "walk off" the lateral surface of the L1 vertebral body. The needles are reinserted to the depth at which contact with the vertebral body was first noted. At this point, if no contact with bone is made, the left-sided needle is gradually advanced 1.5–2 cm or until the pulsations emanating from the aorta and transmitted to the advancing needle are felt (Figure 16-5).[21,22] The right-sided needle is then advanced slightly farther (i.e., 3–4 cm past contact with the vertebral body) (Figure 16-6). Ultimately, the tips of the needles should be just posterior to the aorta on the left and to the anterolateral aspect of the aorta on the right. It is essential that anteroposterior (Figure 16-3 and 16-7 and lateral images (Figure 16-8 and 16-9) are taken to confirm the correct position. The stylettes are removed once the needles are in correct position. The needle hubs are aspirated for blood, cerebrospinal fluid, thoracic duct fluid, and urine. At this point up to 5ml of nonionic radiographic contrast is injected to ensure correct spread of the solution (see Figure 16-10). For the temporary blockade, local anesthetic is injected in 3-5 ml intermittent boluses to a maximum of 20 ml for each side. If alcohol is injected for a neurolytic block, then a 50-100% concentration is used with a maximum volume of 20 ml per side

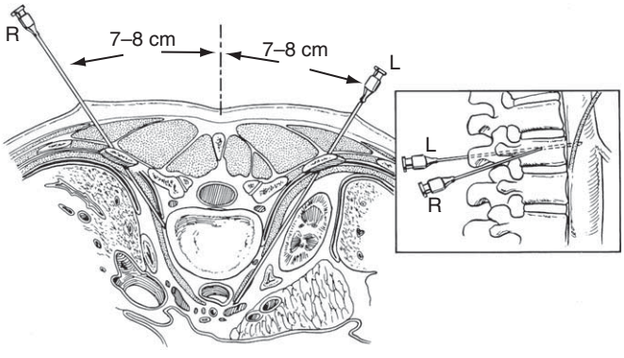

FIGURE 16–5

Retrocrural and transcrural needle placement for celiac plexus block. (Inset) The left needle (R) is retrocrural and results in solution to spread and block the splanchnic nerves. The left needle (L) is transcrural and is in close proximity to the celiac plexus.

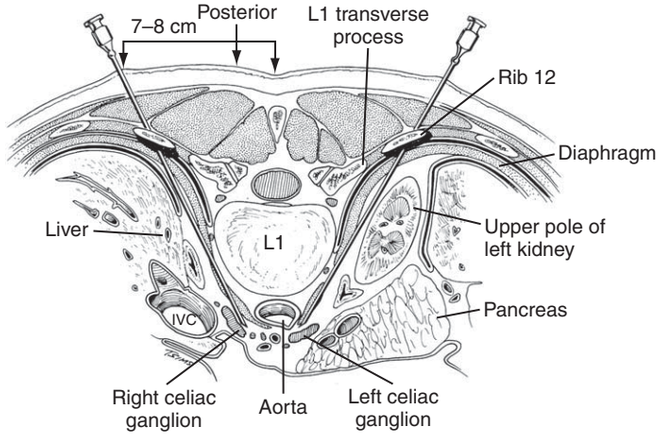

FIGURE 16–6

Cross-section of celiac plexus block. The proximity of renal parenchymal tissue necessitates placing needles no farther than 7–8 cm from midline.

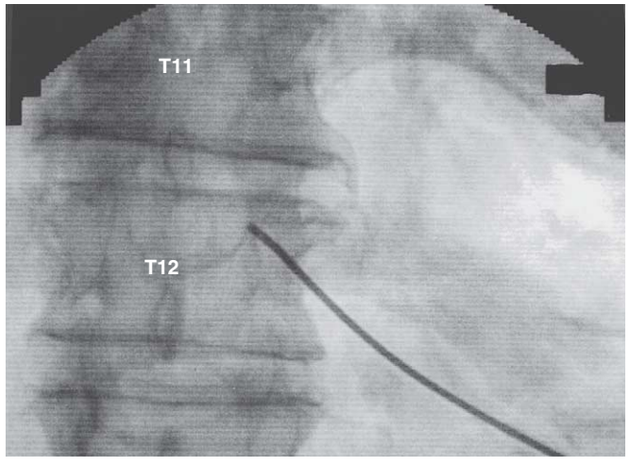

FIGURE 16–7

Anteroposterior (AP) view shows needle at the pedicular level of the T12 vertebrae ipsilaterally. This is the correct placement for the needel for the celiac plexus blocks in this view.

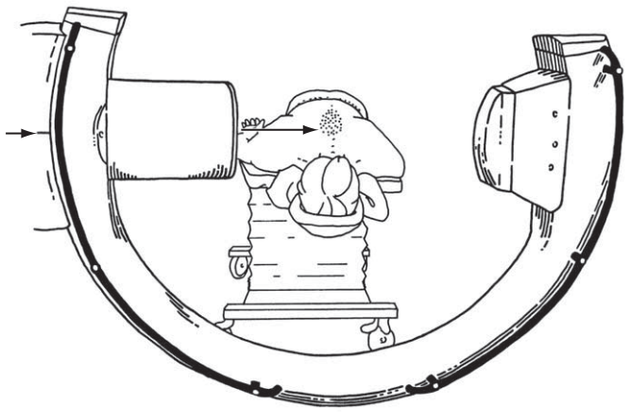

FIGURE 16–8
Patient with lateral view positioning of C-arm.

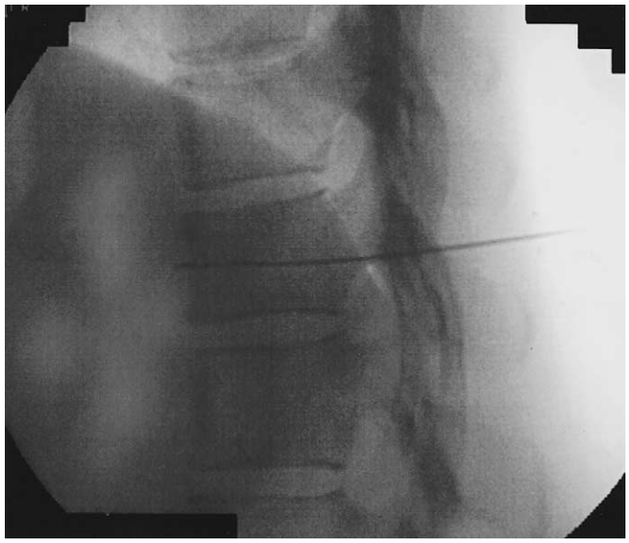

FIGURE 16–9
Celiac plexus anteroposterior view with contrast.

Transdiscal Approach

The technique may be considered when the risk of renal puncture is thought to be high, that is, due to hydronephrosis.

The procedure is performed under fluoroscopy. The patient is placed in the prone position with a pillow beneath the iliac crest to facilitate the opening of the interdiscal space as much as possible. The T12-L1 level is identified under flouroscopy. The fluoroscope is rotated obliquely at an angle of 15–20 degrees (Figure 16-11). It is important to align the inferior end-plates with a cephalocaudal projection (Figure 16-12). A one- or two-needle technique is utilized. Prophylactic use of systemic and/or intradiscal antibiotics is mandatory to prevent infectious discitis.

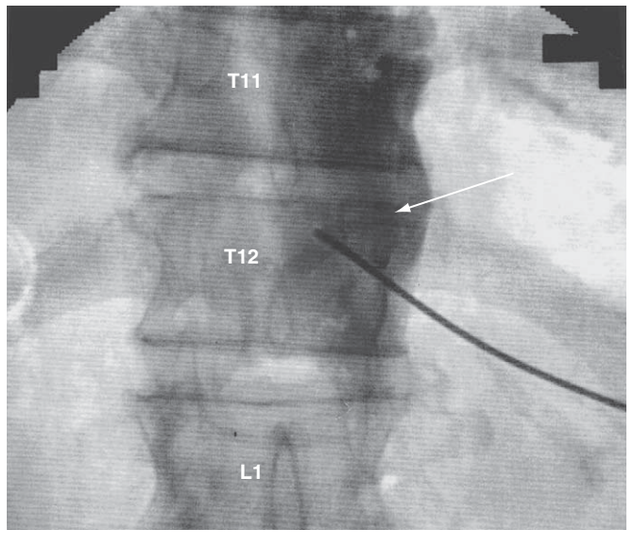

FIGURE 16–10
Radiographic image in the lateral position showing the needle top at T12 anterior border of vertebral body.

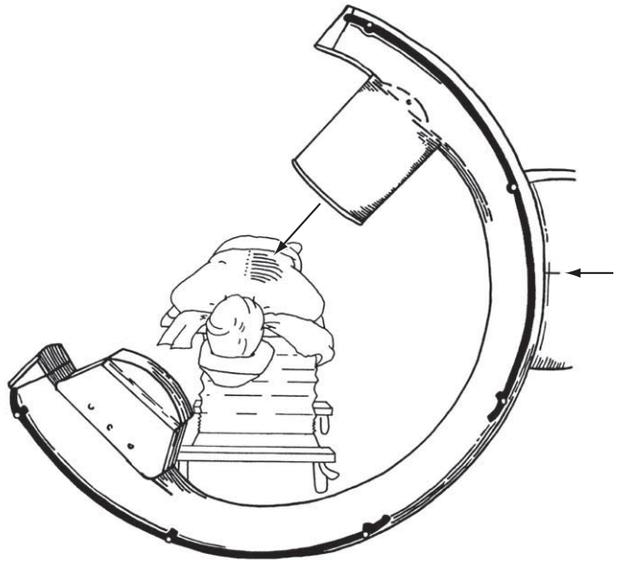

FIGURE 16–11
C-arm is rotated 15-20 degree in an oblique position for a paraspinous or intradiscal approach for celiac plexus blockade.

Paraspinous Posterior Approach

This approach is similar to the approach described for splanchnic nerve block (see Chaptet 12). The differences are that the needle is directed at L1 vertebra and should be anterior to the vertebral body.

Transaortic Approach

This technique was introduced in 1983 as a deliberate placement of the needle through the aorta in order to guarantee preaortic spread of the injected solution.[14] The

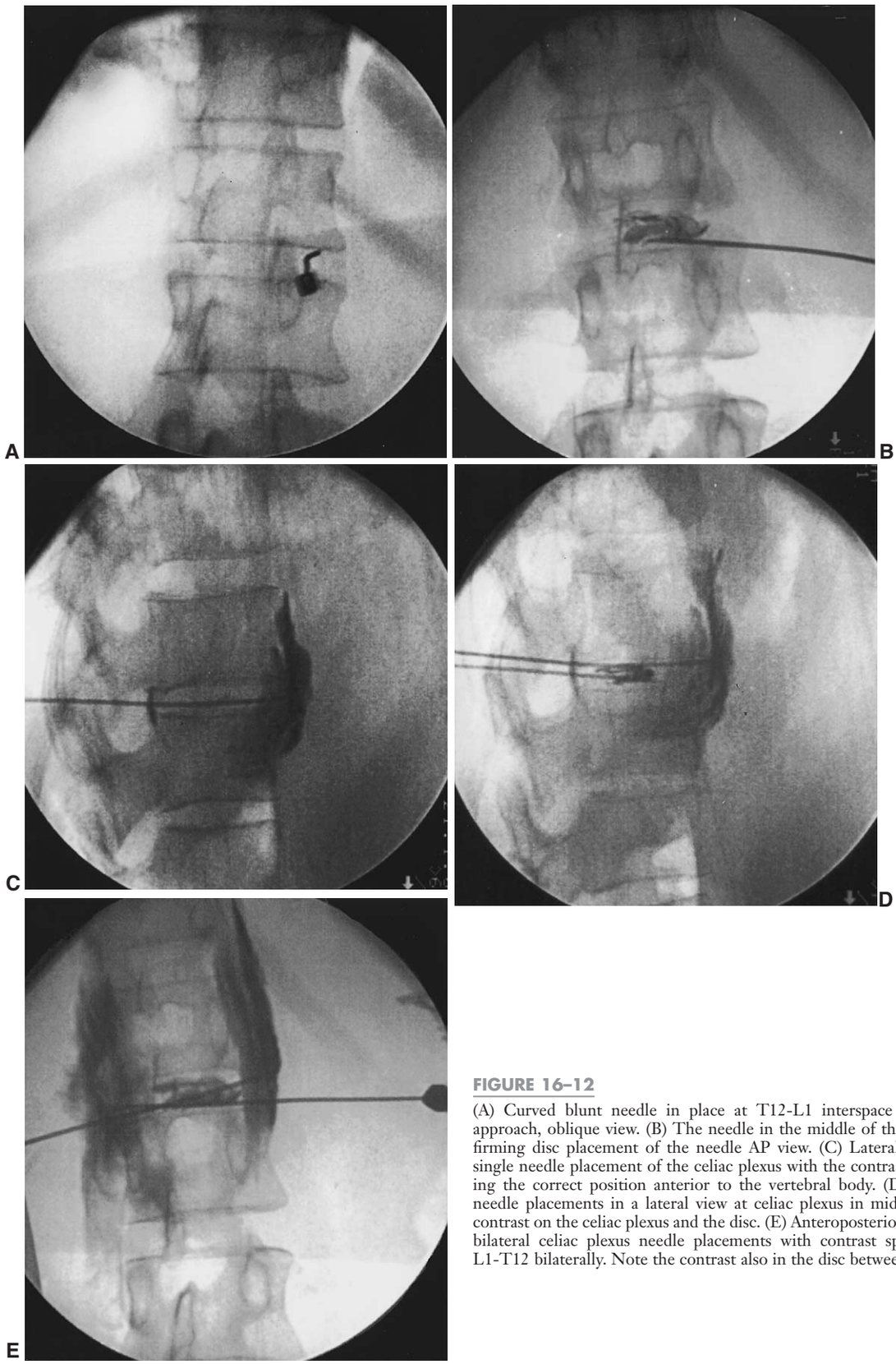

FIGURE 16–12

(A) Curved blunt needle in place at T12-L1 interspace intradiscal approach, oblique view. (B) The needle in the middle of the disc confirming disc placement of the needle AP view. (C) Lateral view of a single needle placement of the celiac plexus with the contrast confirming the correct position anterior to the vertebral body. (D) Bilateral needle placements in a lateral view at celiac plexus in middle of disc contrast on the celiac plexus and the disc. (E) Anteroposterior view with bilateral celiac plexus needle placements with contrast spread from L1-T12 bilaterally. Note the contrast also in the disc between T12-L1.

incidence of significant hemorrhage during translumbar aortography is as low as 0.1–0.5%.[24] Transaortic needle puncture seems to be safe owing to elasticity of aortic wall and tight surroundings of adjacent structures.

Basically the technique is a left-sided, fluoroscopic guided paravertebral approach. The needle is advanced until it penetrates the aortic wall, which is heralded by brisk arterial blood pulsation. Then, an increased resistance with subsequent loss of resistance and cessation of blood expulsion means that the anterior wall is pierced. The original technique has been modified. Continuous loss-of-resistance technique with saline seems to be more accurate and sensitive to the penetration of the anterior wall.[25] On the posterolateral view, contrast dye should remain midline with some tendency toward greater concentration around the lateral margins of the aorta. Lateral view should confirm predominantly preaortic T12-L2 spread and is often pulsating. The absence of a narrow longitudinal "line" is suggestive of aortic wall dissection. It is important to mention that in cases of excessive tumor involvement, the contrast might not spread in the expected pattern.

Choice of Drugs and Neurolytic Solutions

For diagnostic and prognostic block utilizing the retrocrural technique, 12–15 ml of 1.0% lidocaine or 3.0% 2-chloroprocaine is administered through each needle.[26] In order to prevent local anesthetic toxicity and recognize intravascular injection early, all local anesthetics should be administered in incremental doses.[27] For pain treatment of acute pancreatitis, an 80-mg dose of depot methylprednisolone is advocated for the initial celiac plexus block, and 40 mg for subsequent blocks.[28]

Most investigators suggest that 10–12 ml of 50% ethyl alcohol or 6.0% aqueous phenol be injected through each needle for retrocrural neurolytic block. Thomson and colleagues,[27] however, strongly recommend that 25 ml of 50% ethyl alcohol be injected via each needle.

After the neurolytic solution has been injected, each needle should be flushed with sterile saline solution. There have been anecdotal reports of neurolytic solution being tracked posteriorly along with the needles as they are withdrawn. Radiographic guidance, and in particular CT guidance, offers the pain specialist an added margin of safety when performing neurolytic celiac plexus block and thus should be utilized.

Catheter Placement

Patients with nonmalignant abdominal pain often fare poorly after neurolytic blockade of the celiac plexus, yet many derive temporary benefit from local anesthetic blockade. Because this pain is sympathetically mediated and reflexively perpetuated, continuous denervation of the plexus by local anesthetic infusion may provide prolonged analgesia.

The technique for placement is similar to that described previously.[29] Instead of 22-gauge needles, a 6- or 8-inch catheter system (e.g., Longdwel, Becton & Dickinson) placed bilaterally is recommended. Once they are placed, secure the catheters at the skin with either a 2-cm silk skin suture or benzoin and Steri-Strips. Place a sterile, clear dressing over the catheters, and start local anesthetic infusion of bupivacaine 0.1–0.25% or ropivacaine 0.2%, at 6–8 ml/hr. These catheters can be maintained for 4–7 days if placed sterilely and if the sites are checked daily.

Anterior Approaches to Celiac Plexus Block

A percutaneous anterior approach to the celiac plexus was advocated early in the 20th century, only to be abandoned because of the high incidence of complications.[3,17] The invention of fine needles, improvements in radiologic guidance technology, and the maturation of the specialty of interventional radiology and pain management have since led to renewed interest in the anterior approach to blockade of the celiac plexus.

Extensive experience with transabdominal fine-needle aspiration biopsy has confirmed the relative safety of this approach and provides the rationale and method for the modification of this radiologic technique for anterior celiac plexus block. The anterior approach to the celiac plexus necessarily involves the passage of a fine needle through the liver, stomach, intestine, vessels, and pancreas. Surprisingly, it is associated with very low rates of complications.[30–33]

Advantages of the anterior approach to blocking the celiac plexus include its relative ease, speed, and reduced periprocedural discomfort as compared with posterior techniques.[14,15] Perhaps the greatest advantage of the anterior approach is the fact that patients do not have to remain prone, which can be a significant problem for patients suffering from intra-abdominal pain. The supine position is also advantageous for patients with ileostomies and colostomies.

The anterior approach is probably associated with less discomfort because only one needle is used. Furthermore, the needle does not impinge on periosteum or nerve roots, and does not pass through the bulky paraspinous musculature. Because needle placement is precrural, there is less risk of accidental neurologic injury related to retrocrural spread of drug to somatic nerve roots or epidural and subarachnoid spaces.

Potential disadvantages of the anterior approach to celiac plexus block include the risks of infection, abscess, hemorrhage, and fistula formation.[30] Although preliminary findings indicate that these complications are exceedingly rare, further experience is needed to draw a definitive conclusion.

The anterior technique can be carried out under CT or ultrasound guidance. Patient preparation is similar to that for posterior approaches to celiac block. The patient is placed in the supine position on the CT or ultrasound table. The skin of the upper abdomen is prepared with antiseptic solution. The needle entry site is identified 1.5 cm below and 1.5 cm to the left of the xiphoid process (Figure 16-13).[31] At

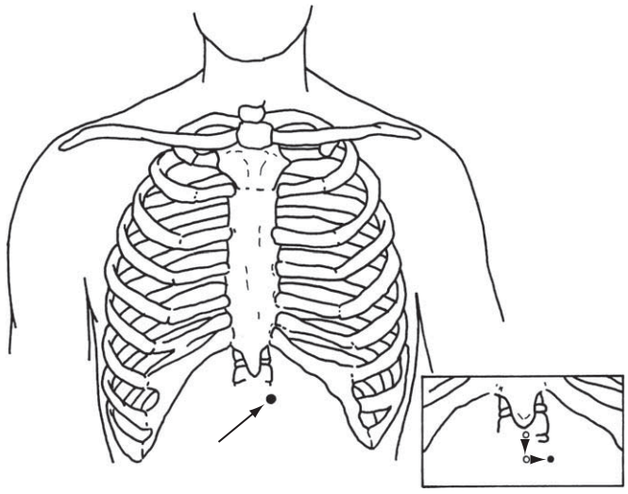

FIGURE 16–13
Line drawing of point of entry for anterior approach to the celiac plexus.

that point, the skin subcutaneous tissues and musculature are anesthetized with 1.0% lidocaine. A 22-gauge, 15-cm needle is introduced through the anesthetized area perpendicular to the skin and advanced to the depth of the anterior wall of the aorta, as calculated using CT or ultrasound guidance (Figure 16-14).

If CT guidance is being utilized, 4 ml of water-soluble contrast in solution with an equal volume of 1.0% lidocaine is injected to confirm needle placement (Figure 16-14). If ultrasound guidance is being used, 10–12 ml of sterile saline can be injected to help confirm needle position.[15] After satisfactory needle placement is confirmed, diagnostic and prognostic block is carried out using 15 ml of 1.5% lidocaine or 3.0% 2-chloroprocaine. Owing to the potential for local anesthetic toxicity, all local anesthetics should be administered in incremental doses.

Matamala and associates[15] recommended 35–40 ml of 50% ethyl alcohol for neurolytic blocks of the celiac plexus via the anterior approach. Other investigators have had equally good results utilizing 15–20 ml of absolute alcohol.

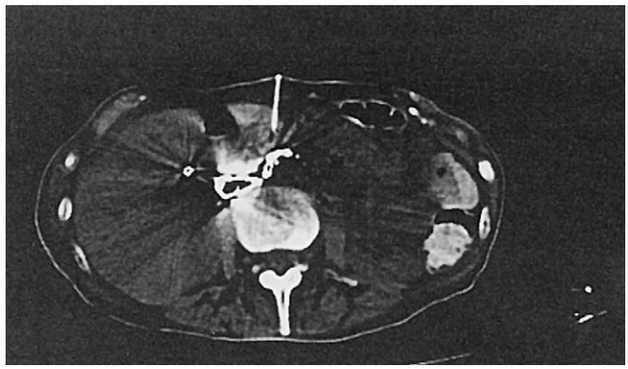

FIGURE 16–14
CT confirms proper needle placement for anterior celiac plexus block.

An alternative technique uses fluoroscopy to guide the passage of a single needle just to the right of the center of the L1 vertebral body, after which it is withdrawn 1–3 cm.[30] Important precautions for the anterior approach to celiac plexus block include the administration of prophylactic antibiotics and the use of needles no larger than 22 gauge, to minimize the risks of infection and trauma to the vasculature and viscera.

COMPLICATIONS

In the hands of the skilled clinician, serious complications should rarely occur from celiac plexus and splanchnic nerve block. Because of the proximity of other vital structures, however, coupled with the use of large volumes of neurolytic drugs, the following side effects and complications may be seen (in descending order of occurrence):

- Pain during and after procedure
- Hypotension/orthostatic hypotension
- Diarrhea
- Intravascular injection (venous or arterial)
- Renal injury
- Injury of lumbar somatic nerve
- Vascular trauma
- Perforation of viscus
- Pneumothorax
- Chylothorax
- Failure of ejaculation
- Vascular thrombosis or embolism
- Abscess
- Peritonitis
- Retroperitoneal hematoma
- Subarachnoid or epidural injection
- Urinary tract injury
- Discitis
- Paraplegia

The main side effect from the celiac plexus block is backache, which usually results from the passage of needles through the back muscles. This result can be minimized by gently positioning the needles, minimal repositioning, and adequate local infiltration. Although self-limiting, back pain can be a significant complaint and can require use of a nonsteroidal anti-inflammatory drug, muscle relaxant, or heating pad. Celiac catheter placement and subsequent maintenance can be distressing enough to require the ongoing treatments listed previously.

EFFICACY OF CELIAC AND SPLANCHNIC NERVE BLOCKS

Despite general agreement that celiac plexus block is indeed efficacious, significant controversy persists regarding (1) its efficacy relative to opioid therapy, (2) relative efficacy compared to other approaches and techniques, and (3) whether even a remote risk of paraplegia warrants a

commitment to neurolysis, especially when treatment with analgesics usually provides adequate relief. Regrettably, despite the legacy of extensively descriptive literature, these questions remain largely unresolved because of persisting scientific inadequacies.

A survey of the literature reviewed data from 24 studies on celiac neurolysis performed on 1145 patients, 63% of whom had pancreatic cancer pain and 37% of whom had pain caused by other intra-abdominal malignancies.[17] Good to excellent pain relief was achieved in 90% of available patients during the first 2 weeks after treatment, only 6% of whom required a repeated procedure for inadequate analgesia. Partial or complete pain relief was observed in 95% of patients alive at the time of last follow-up and 87% of patients at the time of death. In another review that addressed the treatment of pain due to intra-abdominal malignancy independent of the site of primary tumor, significant relief of pain and persistence of effect until death were reported in 62–100% and 35.5–100%, respectively, with most studies reporting favorable outcomes in the higher ranges.[34]

Another carefully conducted survey of the available literature draws similar conclusions. In this paper, Mercadante and Nicosia[35] conclude that favorable results are achieved in 85 and 73% of patients with pain caused by pancreatic and other malignancies, respectively, independent of the technique used. Such results include a low incidence of serious side effects, opioid dose reduction in most patients, and a half-life for pain relief in excess of 4 weeks, with the likelihood of pain relief receding with increased survival time.

In a small, prospective randomized controlled trial comparing celiac plexus neurolysis in 12 patients with medical management alone in 12 patients, all of whom suffered from pain caused by pancreatic cancer, neurolysis was associated with significant benefit, although this benefit was ultimately not as dramatic as the older literature would predict.[36] Patients treated with neural blockade had much greater initial pain relief and similar long-term results for pain but used reduced drug doses and differed significantly from untreated patients on the basis of drug-related adverse effects. Complications were limited to transient hypotension and diarrhea in treated patients, whereas control patients experienced more constipation (12 of 12 vs. 5 of 12), nausea and vomiting (4 of 12 vs. 1 of 120), and other events, including a gastric ulcer and a gluteal abscess.

Another control study addressed the relationship between tumor location and spread and efficacy of neurolysis. In this study, unilateral transcrural celiac plexus neurolysis has been shown to provide effective pain relief in 74% of patients with pancreatic cancer pain. Neurolysis was more effective in cases with tumor involving the head of the pancreas. In the cases with advanced tumor proliferation, regardless of the technique used, the analgesic effects of block were not satisfactory.[37]

Using a similar design in 20 patients with pancreatic cancer pain, Mercadante[38] also achieved similar pain scores in patients randomized to pharmacotherapy alone and celiac block with pharmacotherapy, but only as a consequence of a significantly greater opioid burden and attendant side effects. Factors influencing efficacy are uncertain but may include plexus invasion by tumor, which, in one study, was found in 70% of patients with pancreatic cancer and was independent of tumor size and histopathology.[39]

Time to maximal pain relief is variable. In most patients, relief is immediate and complete; in others, it will accrue over a few days.[33,34] In addition, pain relief is often re-established with repetition. If the interval of comfort is extremely short, repetition by an alternate route may be warranted.

Finally, a carefully conducted randomized, prospective evaluation of quality of life in patients with pancreatic cancer treated with celiac neurolysis versus pharmacotherapy reported on 10 and 11 patients, respectively.[40] Patients given neural blockade had less pain for the first 4 weeks after treatment and used less morphine through week 7, after which lower doses persisted but not at a statistically significant level. Whereas performance status improved only transiently after celiac block, the most striking observation was that of a profound deterioration of performance status noted in pharmacologically treated patients that appeared to have been prevented in patients treated with neural blockade.

LUMBAR SYMPATHETIC BLOCK AND NEUROLYSIS

HISTORY

Historically the first report of lumbar sympathetic block seems to be by Brunn and Mandl,[41] who in 1924 described Selheim's technique of injecting the lumbar sympathetic nerves as a component of his paravertebral approach to blocking the mixed spinal outflow in the lumbar region. Kappis[42] also described the technique of lumbar sympathetic block and surgical resection of the lumbar sympathetic nerves about this time. Others associated with expansion of the technique are von Gaza[43]; Mandl[44] and Lawen[45] in Germany; Jonnesco[46] and Leriche and Fountain[47] in France; and White[48] in the United States. During the 1950s, Bonica,[49] Moore,[50] and Arnulf[51] described in detail the importance of lumbar sympathetic blockade, particularly its relationship to the treatment of causalgia and post-traumatic reflex dystrophies in servicemen after World War II. Although the technique described by Mandl[44] in 1926 remains one of the most popular approaches to the lumbar sympathetic trunk, Reid and colleagues,[52] in a large series published in 1970, described a more lateral approach that avoids contact with the transverse process. Two techniques are described in this chapter: the "classic" technique first described by Kappis[42] and Mandl[44] and the lateral technique first described by Mandl[44] and redefined by Reid and colleagues.[52]

Techniques for neurolysis of the lumbar sympathetic chain appeared after 1924, when Royle[53] in Australia attempted to modify skeletal muscle tone in patients with spastic paralysis. Physiological effects such as increased skin blood flow and dryness were observed and utilized for the treatment of Raynaud's disease in 1929.[54] In the latter half of the 20th century, arterial reconstructive surgery largely supplanted the use of sympathectomy for peripheral vascular disease with the possible exception of vascular ulcers. Lumbar sympathetic block continues to be advocated for hyperhidrosis with some justification. Percutaneous and endoscopic techniques have become the methods of choice, as with the cervicothoracic chain.

ANATOMY

Peripherally, the sympathetic nervous system consists of preganglionic and postganglionic efferent fibers that innervate deep somatic structures, skin, and viscera. The two paravertebral sympathetic trunks are connected segmentally by preganglionic neurons, whose cell bodies are situated in the lateral horn, intermediate nucleus, and paracentral nuclei of the thoracolumbar spinal cord. The cell bodies responsible for vasoconstriction in the lower limbs are in the lower three thoracic and first three lumbar segments. The preganglionic fibers pass by way of their corresponding nerves as white rami communicantes, which communicates with considerable convergence in the paravertebral ganglia with postganglionic efferents and in the prevertebral ganglia by postganglionic efferents to the pelvic viscera (Figure 16-15A). A small percentage of postganglionic fibers pass directly to ganglia in the aortic plexus and the superior and inferior hypogastric plexuses (Figure 16-15B). The postganglionic fibers leave the sympathetic trunk as gray rami communicantes, some passing to the L1 nerve to contribute to the iliohypogastric and genitofemoral nerve territories, some to the L2-L5 nerves, and some to the upper three sacral nerves, where they pass on to their respective destinations in the lumbosacral plexus.

Intermediate ganglia found in the psoas and iliacus muscles also communicate with postganglionic fibers that pass through the segmental lumbar and sacral nerves. The S1 and S2 nerves contain the largest numbers of postganglionic fibers. Most of these represent gray rami communicantes that subserves vasomotor, pilomotor, and sudomotor functions. It has been determined that although each root of the lumbosacral plexus receives one group of gray rami communicantes, the S1-S3 nerves contain several (i.e., a large convergence), because they innervate the blood vessels in the lower extremity.[54]

Each lumbar sympathetic chain enters the retroperitoneal space under the right and left crura, continuing inferiorly in the interval between the anterolateral aspect of the vertebral bodies and the origin of the psoas muscle to enter the pelvis and the L5-S1 disc. Posteriorly, the periosteum overlies the vertebral bodies and the fibroaponeurotic origin of the psoas muscles and their fascial coverings. Anteriorly is the parietal reflection of the peritoneum, the aorta lying anteromedial to the left trunk and the vena cava anterior to the right trunk. It should be noted that the white and gray rami communicantes pass to their respective ganglia beneath the fibrous arcades of the psoas attachments to each vertebral body. Also, they tend to pass alongside the middle of the vertebral body.

The sympathetic ganglia of the lumbar sympathetic chain are variable in both numbers and position. Rarely are five ganglia found on each side in the same individual,[55] in most cases, only four are found. There tends to be fusion of L1 and L2 ganglia in most patients, and ganglia are aggregated at the L2-L3 and L4-L5 discs. Also, there is considerable variability in the size of the ganglia, some being fusiform and as long as 10–15 mm, others being round and approximately 5 mm long.[56] Because of this aggregation and the fact that the right crus extends to L3 and the left to L2, lumbar sympathetic blockade is more efficacious.

INDICATIONS

The indications for lumbar sympathetic block may be divided into four broad categories:

1. *Circulatory insufficiency in the leg*: Includes arteriosclerotic vascular disease, diabetic gangrene, Buerger's disease, Raynaud's phenomenon and disease, reconstructive vascular surgery after arterial embolic occlusion, and frostbite.
2. *Sympathetically maintained pain*: CRPS types I and II.
3. *Other conditions*: Hyperhidrosis, phlegmasia, Alba dolens, erythromelalgia, amputation stump pain and phantom pain, acrocyanosis, intractable urogenital pain, and trench foot, among others.
4. *Discogenic pain* with pseudo-sciatic radiation has been recently suggested as an indication for segmental sympathetic neurolysis.

The rationale for sympathetic blocks, particularly in treatment of pain, is based on the observation that pain under certain conditions is potentiated or mediated by sympathetic hyperactivity. Laboratory evidence has demonstrated that the sympathetic postganglionic neuron may not only act at the effector terminal, but also on the primary afferent (PA) in certain pathologic conditions; it may communicate with the PA neuron at other sites (direct and indirect coupling).[57] Although the mechanism remains unclear, blocks of the sympathetic nervous system may have two actions: (1) interruption of preganglionic and postganglionic sympathetic efferents may influence function of the primary afferent (PA) neuron[58,59] or (2) visceral afferents

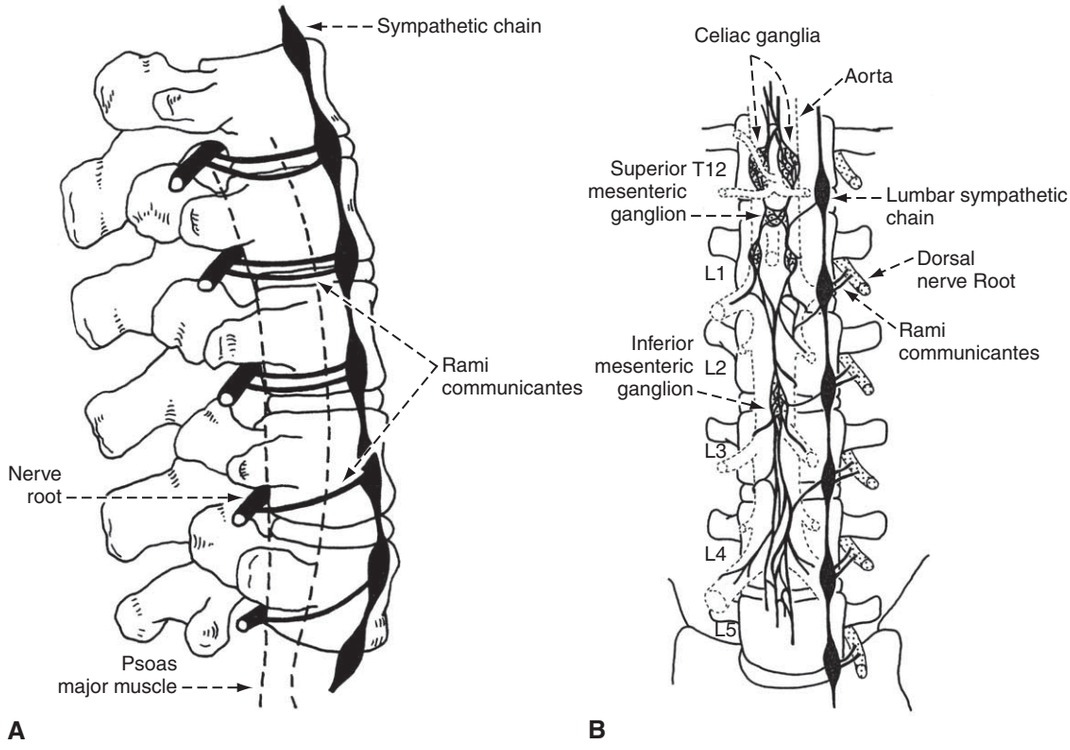

FIGURE 16–15

(A) This line drawing shows the sympathetic chain as it courses the lordotic curve of the lumbar vertebrae. (B) This line drawing shows the course of the sympathetic chain and its connection to celiac and aortic plexus.

from deep visceral structures in the leg that travel with the sympathetic nerves may be blocked.[1,18,19]

As a diagnostic and prognostic procedure, sympathetic blocks are helpful in determining the nature of the pain (i.e., whether it is sympathetically maintained (SMP) or whether it is independent of sympathetic function (SIP). Such procedures are always used to test the effects of destructive (neurolytic, or surgical) sympathectomy.

CONTRAINDICATIONS

Contraindications to sympathetic blocks are a bleeding diathesis, local infection, and certain anatomical anomalies, which may be considered relative contraindications if they are likely to render the procedure difficult or hazardous.

EQUIPMENT

- 25-gauge, skin-infiltration needle
- 21-gauge, 1-1/2-inch needle for deep infiltration
- 20-gauge Chiba or 6-inch Coudé needle
- 16-gauge angiocath
- 10- or 15-cm (with a 10- or 15-mm active tip) curved, blunt radiofrequency (RF) electrode
- RF generator with cables and probes

DRUGS

- *For diagnostic and therapeutic block*
- 0.5% ropivacaine, 0.5% bupivacaine, or 2% lidocaine = 40 ml
- *For neurolytic block*
- Phenol neurolysis (preferred)
- 6–10% aqueous phenol in Omnipaque (5–10 ml)
- *Alcohol*
- 50–100% alcohol (5–10 ml)

PROCEDURE

The prone position is most convenient for lumbar sympathetic blockade, but pain or anatomic deformity may make it necessary to place the patient in the left or right lateral decubitus position (Figure 16-16).

Paramedian (Classic or Mandl) Approach

Skin wheals are made 5–8 cm lateral to the spinous processes of L2-L4. With the spinal needle held perpendicular to the skin, a track of local anesthetic is infiltrated down to the transverse process at each level. The sympathectomy needle is then directed to the same point, and the depth is noted, after which the needle is withdrawn

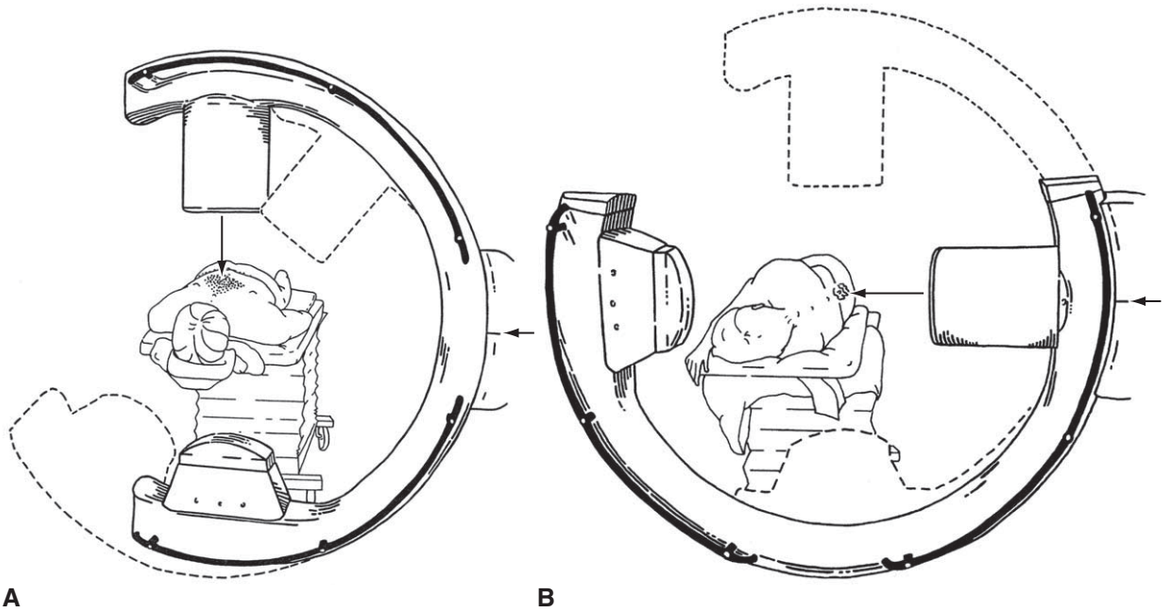

FIGURE 16–16

(A) The C-arm is first positioned AP to locate the appropriate vertebral body and then turned obliquely for needle placement through the skin in a tunneled view.

to the subcutaneous tissue. The needle is then redirected so as to pass below and slightly medial to the inferior border of the transverse process, at an angle of about 10 degrees to the sagittal plane (Figure 16-17). It is advanced about 2 cm deep to the transverse process, where it should contact the side of the vertebral body. A slight decrease in the angle is made so as to allow the needle to slip past at a tangent to the lateral aspect of the vertebral body. Fluoroscopy at this point will confirm both the needle position and the distance to the anterolateral surface of the vertebral body. A small (1–2 ml) amount of Omnipaque is injected through each needle. The contrast will hug the contour of each vertebral body if the needle tips are in the correct tissue plane.

For diagnostic or prognostic blocks, a local anesthetic is used, whereas for neurolytic blocks, one can use a mixture of Omnipaque and phenol or RF (Figure 16-18)

Lateral (Reid's) Approach

With the patient in a prone position, a skin wheal is made 10–14 cm lateral to the superior border of the spinous processes of L1 and L4. Usually, L3 is chosen, with the intent of having a spread of contrast over the L2-L4 sympathetic ganglions. The spinal needle inserted at an angle of 60 degrees to the sagittal plane, toward the body of L3 of vertebra, is used to track the direction and for local anesthetic infiltration. The sympathectomy needle is then introduced and advanced until it contacts the vertebral body. Fluoroscopy in two planes will confirm its position and the angle to assume for redirection of the needle to its final position at the anterolateral aspect of the vertebral body (Figure 16-19). With the fluoroscopy positioned laterally, any final adjustments can be made to ensure that the needle tip lies exactly at the anterolateral edge of the vertebral body. A small amount of contrast material will identify the correct tissue plane. The dye should spread to form a line conforming to the anterolateral margin of the vertebral bodies (L2-L4) (Figure 16-18). If the spread of contrast dye is restricted to one ganglion, the procedure is then repeated with the second needle at an adjacent level, that is, the L2 or L4 vertebral body.

Injection of Test Solution

A long-acting agent such as bupivacaine or ropivacaine is advantageous for both therapy and prognosis, because it enables the patient adequate time to evaluate the effects of sympatholysis and any effect this might have on the pain. A concentration of 0.5% bupivacaine or 0.5% ropivacaine gives optimal duration without the need for an added vasoconstrictor. However, a short-acting local anesthetic is commonly used first in order to obtain quick skin response (temperature elevation, plethymography curve heightening, galvanic skin response), and then a long-acting agent is injected.

NEUROLYTIC BLOCK

Neurolysis of the lumbar sympathetic chain is easily performed and is one of the most useful procedures.[60] It can be indicated for recalcitrant CRPS I or II peripheral vascular disease, pelvic malignancies, and deafferentation pain syndromes. Neurolysis should be considered only

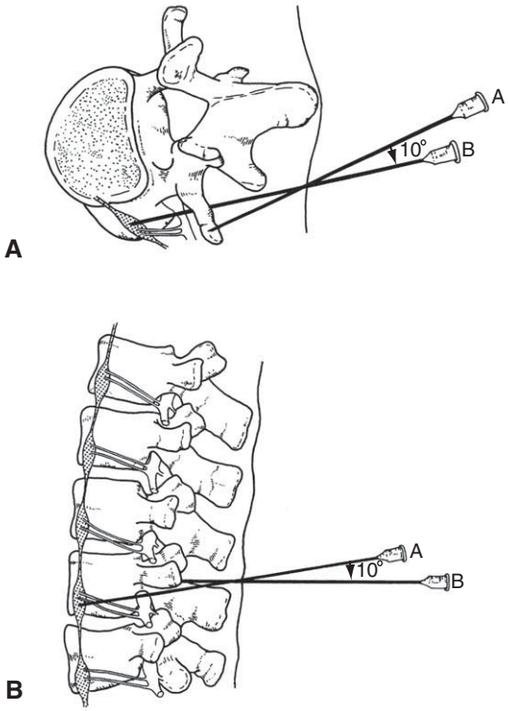

FIGURE 16–17

(A) Coronal view. "Classical" technique with the needle "A" touching the transverse process and slipping off to contact the vertebral body (needle "B") (B) Lateral view. Needle "A" and "B" doing the same as previously described.

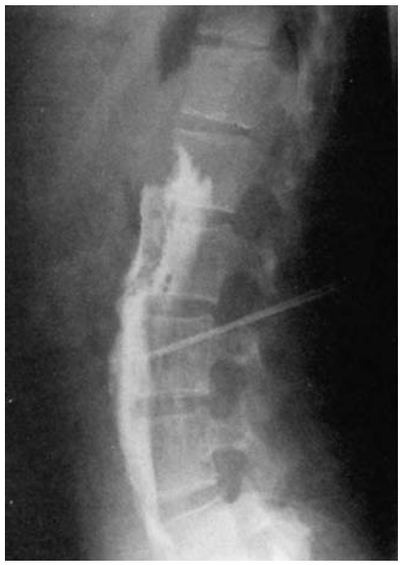

FIGURE 16–18

The radiographic image of lumbar area showing the needle in position and the flow of contrast solution at L2-L4 in a lateral view during a lumbar sympathetic block.

after local anesthetic blocks of the lumbar sympathetic chain have documented efficacy but have failed to produce long-lasting relief.

Needle placement for neurolysis does not differ from that for a local anesthetic lumbar sympathetic block. Image intensification, and fluoroscopy in particular, greatly facilitates placement, allows real-time visualization of drug diffusion, and helps prevent possible complications by ill-placed needles or neurolytic solution. When a single-needle technique is used, fluoroscopy can document adequate cephalad spread to the upper limits of L2 and caudal diffusion of drug to L4.

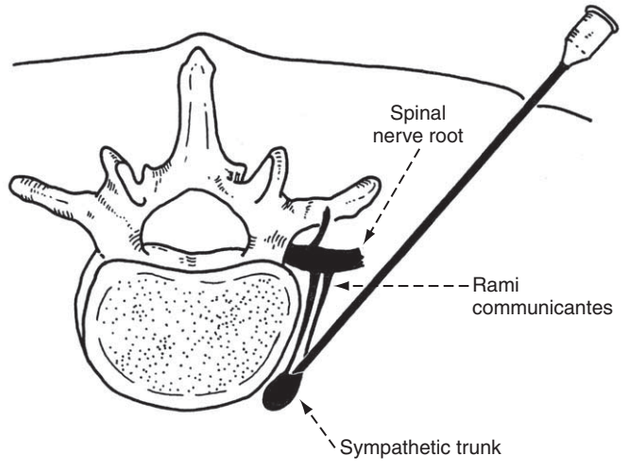

FIGURE 16–19

Line drawing of the needle being inserted for a lumbar sympathetic block in the lateral technques (Reid technique).

Significant longitudinal spread of drug along the sympathetic chain is required for adequate neurolysis. Because spread cannot be reliably achieved with a single needle, a two- or three-needle approach is needed. One needle is positioned at the inferior aspect of L2, and the second needle is positioned at the superior aspect of L3. The third needle can be placed at the L4 vertebral body. No comparative studies have reported any difference in efficacy with the use of one, two, or three needles. Check needle placement before injecting contrast agent in both the anteroposterior and the lateral views. C-arm fluoroscopy is ideal and allows real-time visualization.

Monitor distal skin temperatures during neurolysis for further documentation of block. Inject a local anesthetic solution before neurolysis and evaluate the efficacy by a temperature rise and relief of symptoms.

The spread of contrast is characteristic and reproducible. The dye confines itself to the anterolateral border of the vertebral body in a tight, linear fashion (see Figure 16-18). Movement of contrast is cephalad and caudal, with no lateral diffusion of drug to the vertebral bodies. Contrast dye that diffuses laterally is usually deposited either in the psoas muscle or on the fascia; the effect appears either as a roundish, poorly circumscribed picture or band-like with muscular striations visibly present. In neither situation should neurolytic agents be injected.

Phenol is the agent of choice for neurolysis. It produces a lower incidence of neuralgias than that with equivalent injections with alcohol.[61] Although volumes as small as 2 ml through each of three needles have been

used, larger volumes (15 ml) through a single needle have been equally efficacious.[62] Concentrations of 6% phenol have been replaced with 10% and 12% solutions, due to evidence from animal studies that higher concentrations provide prolonged nerve destruction.[63] After completion of the neurolytic agent injection, the needle should be flushed with 1 ml of saline solution to prevent tracking of neurolytic agent during withdrawal.

RADIOFREQUENCY OF LUMBAR SYMPATHETIC CHAIN

The position of the patient is similar to that described for other procedures in prone positions. A curved, blunt RF electrode is used for this technique. The lesion should be at the inferior one-third of L2, upper one-third of L3, and middle of L4 vertebral bodies (Figure 16-20).

TECHNIQUE

The C-arm is obluqued to 15-20 degrees until the vertebral body "covers" the transverse process (Figure 16-21). Mark the point of entry of the needle below the transverse process and in line with the lateral edge of the vertebral body (Figure 16-22A). A 16-gauge angiocath is introduced at the appropriate levels at L2, L3, or L4 using a tunnel vision technique (Figure 16-22B). A curved blunt electrode is then introduced through the angiocath with the tip directed laterally (Figure 16-22C). The C-arm then should be rotated laterally. The electrode

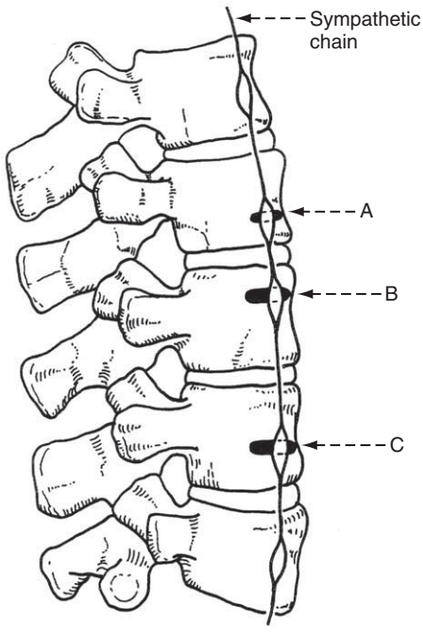

FIGURE 16–20

Line drawing showing anatomical location of lumbar sympathetic chain and sites of lesion (arrows A, B, and C) with radiofrequency.

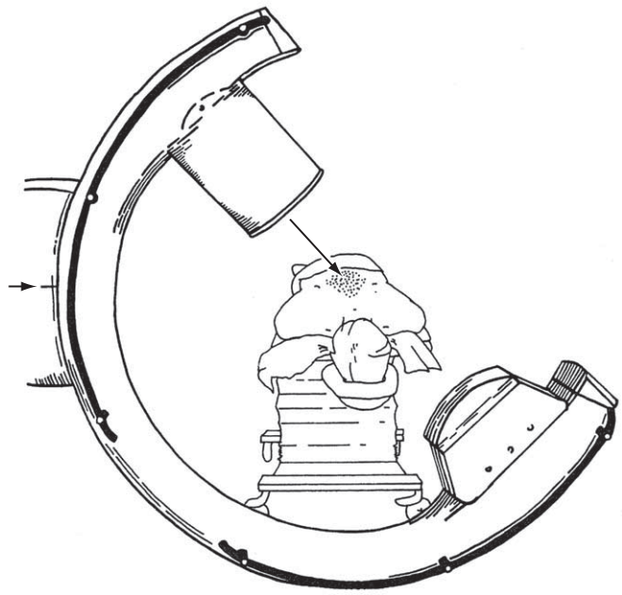

FIGURE 16–21

Line drawing of patient in prone position and C-arm in oblique position prior to performing lumbar sympathetic radiofrequency.

should be advanced to the anterior edge of the lumbar body (Figure 16-22D and E).

At the lateral position, the electrode's tip should stay at the anterior edge of the vertebral body. At the PA view, the electrode's tip should be positioned at the pedicle level (Figure 16-22F). The correct electrode position will show that the tip is behind the facetal line while in bone contact with the vertebral body.

Verify the position with 1–2 ml of contrast. If it spread into the psoas, the tip is too posterior and should be repositioned (more anteriorly). There should be no resistance on injecting the contrast.

STIMULATION PARAMETERS

Once the needle tip is in the correct position, the following should be done for RF neurolysis.

1. Sensory stimulation at 50 Hz. Vague discomfort may be felt in the back with 0.2–0.5 V; stimulate up to 1 V. If paresthesia elicited in the groin at L2 and L3 level, the electrode must be repositioned (due to its proximity to the genitofemoral nerve).
2. Motor stimulation at 2 Hz. There should not be any motor response up to 2V in the lower extremity.
3. Next, inject 1-ml of lidocaine (2%) or equivalent before the lesion; wait for 45 seconds.
4. Then insert the probe and complete lesion at 80°C, for 90 seconds when electrode's tip is rotated cephalad. The lesion should be done with 10-mm avtive needle tip on L2, L3, and L4, and sometimes at L5. Make the second lesion after the tip is rotated caudally.

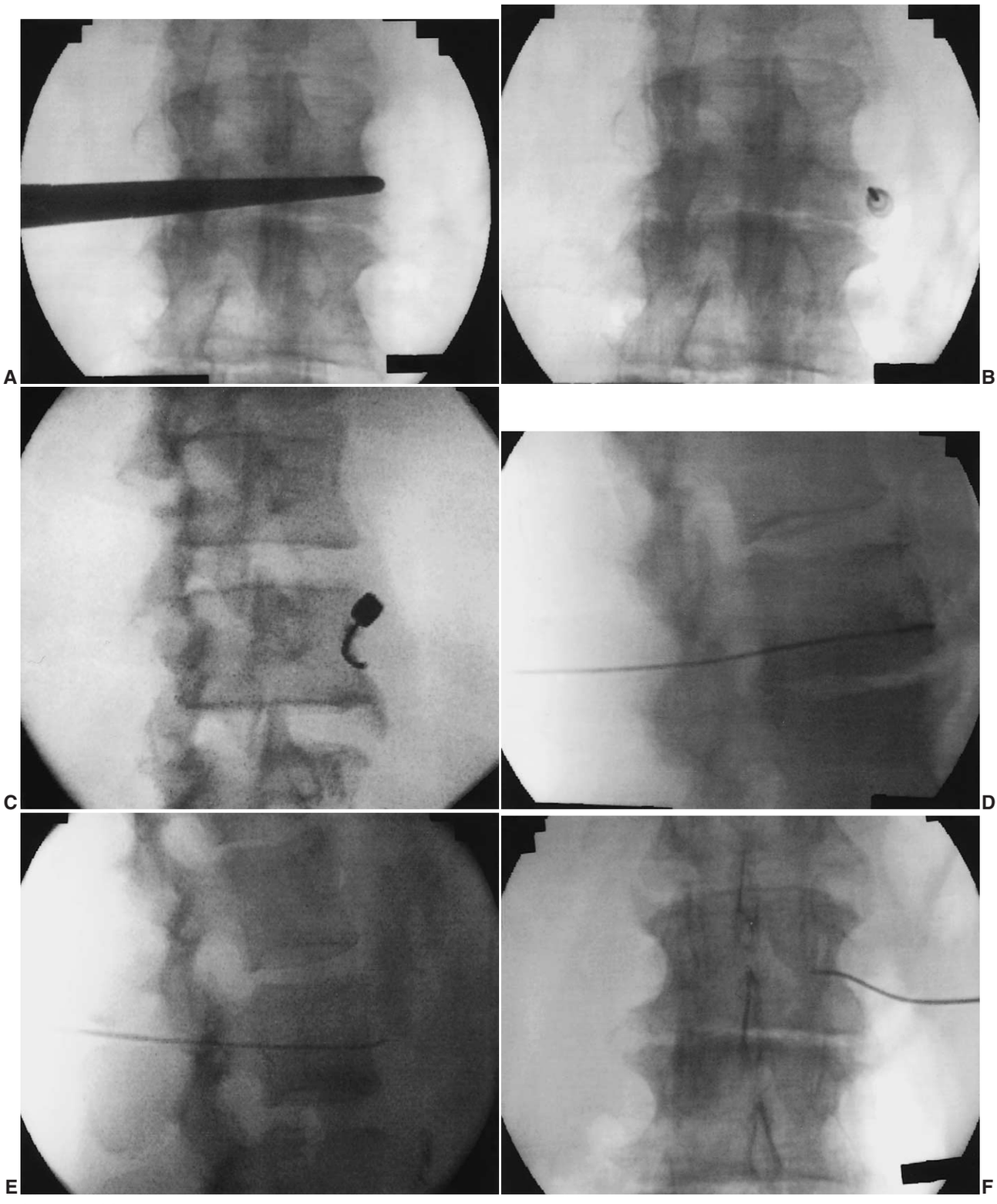

FIGURE 16–22

(A) Radiographic image showing the place of entry of the needle at L2 vertebra for lumbar sympathetic block. (B) The 16-gauge angiocatheter insertion at that side after local anesthetic infiltration at that site (note the tunnel view). (C) Insertion of 10-cm, curved, blunt radiofrequency (RF) needle through the angiocatheter with the tunnel view. The curved needle should be inserted with the tip directed laterally. (D) The lateral view of the RF needle tip at the anterior border of the L2 vertebral body in correct position. (E) The RF needle at L4 vertebral body level slightly in front of the anterior border of the vertebral body. This position avoids the lesion of the lumbar plexus in the psoas muscle. (F) The RF needle position of L4 (AP view) correctly placed at the waist and pedicle.

POSTPROCEDURE MONITORING

After the lesion, the patient should be monitored for 30–45 minutes. After a normal neurological checkup, the patient can be discharged.

COMPLICATIONS

- Postoperative discomfort for approximately 5 days.
- Intravascular or subarachnoid injection.
- Retrograde ejaculation (rare). (Do not perform bilateral sympathectomy.)
- Neuralgia may occur owing to spread of the neurolytic material onto a somatic nerve root. The most susceptible nerve to this complication is the genitofemoral nerve.[64]

The list of possible complications is significant but these complications can be largely avoided if meticulous attention is paid to the approach track of the needle/cannula, the final position of the tip and the spread of contrast agent.[65] The post procedural discomfort felt in the groin, particularly after chemical sympathectomy, is thought to represent a chemical irritation of the genitofemoral nerve. It is suggested that the more discrete lesion of RF reduce the incidence of neuralgic pain. An alternative explanation might lie in convergence phenomena, as sympathetic afferents pass to the central nervous system through the nerve roots L1-L2.[66] The tracking of neurolytic agents to components of the lumbar plexus or contents of the spinal canal can lead to significant neurological deficit. Although this can be largely avoided by careful technique, such rare occurrences are inherently more likely with the uncontrolled spread of neurolytic solutions.

CLINICAL PEARLS

Lesion L2 if there is lumbar pain, L3 if pain is axial in spine, L4 if pain is in lower limb, and L4 and L5 if pain is in the ankle or foot. Several lesions may be performed at different levels.

It is advisable to perform a previous diagnostic blockade with lidocaine 1% (or bupivacaine 0.25%) to avoid false positives (impregnation of somatic nerves). The blockade should be performed in L2-L3-L4, and if the foot is involved, block L3, L4, L5. No sensory or motor blockade should be produced.

The most common side effect after lumbar sympathetic block is backache, which results from the placement of the needles through the paravertebral muscles of the back. This possibility should be carefully explained to the patient before blockade, and the use of a heating pad and ice packs, along with rest and occasional muscle relaxants, may be necessary.

Intravascular injections of larger volumes of local anesthetics can produce serious, systemic, toxic reactions. These are best avoided by the use of test doses, repeated aspiration, and epinephrine-containing solutions in combination with electrocardiographic monitoring.

Inadvertent subarachnoid injections occur rarely if the needle is mistakenly positioned into a dural sleeve. The length of the needle and its small diameter hinder free flow of cerebral spinal fluid (CSF). The high pressure generated during aspiration of the small, 22-gauge needle often sucks the arachnoid against the bevel, resulting in no flow of CSF. An initial, small injection of local anesthetic followed by testing for spinal effect avoids the subsequent total spinal block seen if 15 ml of local anesthetic is injected into the subarachnoid space.

Not uncommonly, the needle passes through the intervertebral disk. The sensation of passing through "Swiss cheese" is easily noted, necessitating removal of the needle and repositioning. Medication cannot be easily injected into a disk. No sequelae have been reported from this occurrence, and any extrusion of disk material would be lateral, away from the spinal canal, and not of any clinical significance. Purposeful transdiscal technique has been published. With this technique, the risk of genitofemoral neuritis, the most common complication after neurolytic lumbar sympathetic block, was reduced because the needle does not penetrate the psoas muscle.[67]

Renal trauma or puncture of a ureter can occur if proper technique is not followed. Most important, the needle should not be inserted more than 7–8 cm from the midline. Sequelae are minimal unless neurolytic agent is injected, resulting in possible urethral stricture or extravasation of urine.

Blockade of the genitofemoral nerve or lumbar plexus within the psoas muscle can occur if the needle is placed too far laterally or posteriorly. If a local anesthetic solution is used, a resulting numbness or weakness can occur in the groin, anterior thigh, or quadriceps. To avoid the 18–24 hour weakness seen with bupivacaine, a short-duration agent (2-chloroprocaine) can be injected initially and the strength of the quadriceps tested.

Lateral spread of neurolytic solution from the lumbar sympathetic chain can result in genitofemoral neuralgia and, less often, lumbar plexus involvement.[61,68,69] Boas[70] reported a 6% incidence of genitofemoral neuralgia in one study. Cousins and associates[61] reported on 35 patients receiving 100% alcohol using a technique without image intensification. Mild neuralgia (one or less per week) occurred in 14%, and severe neuralgia (more than one per week) occurred in 26%. Use of a similar technique with phenol resulted in a respective incidence of 6 and 16%. Sensory loss was reported in 5% of patients, and motor weakness occurred in 6%.[61]

The genitofemoral nerve is most susceptible at the L4-L5 vertebral level, after it has emerged from the psoas major muscle, and lies anterior to the fascia in close proximity to the sympathetic chain. Most mild neuralgias can be treated with nonopioid analgesics and reassurance that this complication is transient. For severe cases, Boas[70] reported

success using IV lidocaine 1–2 mg/kg over 2–3 minutes, sufficient to produce light toxicity symptoms. The pain will disappear, and normal cutaneous sensation returns. In refractory cases, transcutaneous electrical nerve stimulation (TENS), tricyclic antidepressants, and antiepileptic agents may be necessary. Similarly, intravenous lidocaine may be used in a dose of 1–2 ml/kg.

INTERPRETATION OF AND RESPONSES TO LUMBAR SYMPATHETIC BLOCK

It is important to understand the patient's personality when interpreting the subsequent effects of sympatholysis. Although evidence of sympatholysis, that is, vasodilation, increased temperature, and reduction of edema, is important, the qualitative effect on the preexisting symptoms, manifested by continuous pain, hyperalgesia, or touch-evoked pain such as allodynia, requires careful assessment after sympatholysis. It should be remembered that technical failure might be the cause of therapeutic failure, even on repeated occasions. A placebo response is possible and may merely be the response of a grateful patient to the fact that something fundamental has been done to unravel a particular medical condition. It should also be noted that the amount of local anesthetic used for sympatholysis might have an effect on multisynaptic pathways in the central nervous system, producing central inhibition of nociception, an effect that may erroneously be attributed to sympatholysis.[71]

Some have questioned the efficacy and reproducibility of sympathetic block, particularly in relation to pain relief, as a response. Nevertheless, carefully performed, sympathetic block is a useful and important therapeutic diagnostic procedure.[72]

EVALUATION OF COMPLETENESS OF BLOCKADE

Whenever possible, monitor the effectiveness of a sympathetic block. Many tests have been reported to monitor sympathetic activity. Unfortunately, a number of scientific tests lack applicability for the practicing clinician secondary to their intricate apparatus involved, cost, and time for setup. The test described here can be performed at the bedside, and one or two should be used to monitor all blocks.

SURFACE TEMPERATURE MONITORING

Skin temperature recording represents the easiest and fastest way to test sympathetic blockade. Advanced temperature monitors have two or three channels combined with very sensitive sensors and easily read digital displays. Lower extremity temperature should be best measured at the anterior thigh, the medial aspect of the leg, the dorsum of the foot, and the great toe. It is recommended to measure skin temperatures 15–20 minutes before the block to allow for equilibration with the ambient surrounding. Wrapping extremities eliminates environmental influences. It is important to monitor both affected and unaffected sides.

Thermography has been advocated for documentation of sympathetic blockade. It records skin temperature either by an infrared technique or by liquid crystals. Both methods effectively demonstrate changes in skin temperature.

Look for a minimum positive change of 2°C after sympathetic block. At times, this may not occur despite appropriate blockade. A fixed proximal blockage and a larger artery may prevent enhanced distal flow. Some CRPS patients also present with a very warm extremity that does not become warmer with sympathetic blockade.

If a skin temperature change cannot be evoked or further documentation is deemed necessary, record the sympathogalvanic reflex. Initially described by Lewis in 1955,[73] this reflex is also known as skin conductance response, galvanic skin reaction, electrodermal reaction, and psychogalvanic reflex.

In order to measure the sympathogalvanic reflex, one should place standard electrodes on the dorsal and plantar surfaces of the distal extremity (i.e., foot or hand), with a third grounding electrode remotely located. The skin should be free of epithelial cells before electrode placement. The patient is allowed to rest in silence for several minutes to permit the tracing to return to baseline. A short deep breath, loud noise, or pinch of the skin usually suffices to elicit the response, which is recorded as a deflection on the electrocardiography paper or monitor screen. The deflection lasts 4–5 seconds, and changes of 1–3 mV are normal. It will be wise to measure both blocked and unblocked extremities. The blocked side should have an absent sympathogalvanic reflex 20–30 minutes after the procedure.

The presence of the sympathogalvanic reflex varies among patients, with younger patients having much greater deflections and unstable baseline patterns. Not all patients have an obtainable sympathogalvanic reflex, particularly older, diabetic, or significantly depressed individuals. Furthermore, patients receiving drugs such as opiates, barbiturates, atropine, or other centrally acting agents will exhibit minimized or absent sympathogalvanic reflex. Marked habituation can also occur, with smaller deflections occurring with succeeding stimuli.

SWEAT TEST

Three methods of sweat testing have been used clinically to test sympathetic blockade.

1. The *ninhydrin test* relies on the protein in sweat to change the color to yellow.[74] The blocked extremity cannot sweat and shows no color change. The test is considered accurate but time consuming and cannot produce immediate results for clinical use.

2. The *cobalt blue test* involves filter papers that are saturated with cobalt blue, and then dried and stored in a desiccator. When needed, the papers are removed from the desiccator and placed on a clean dry skin of the blocked and unblocked extremity. The presence of sweat changes the paper from blue to pink. An extremity that has been sympathetically denervated shows no color change.
3. The *starch-iodine test* also relies on color change. Its major drawback concerns the length cleanup involved after the starch-iodine application.

PAIN ASSESSMENT

The assessment of pre-block and postblock pain also provides some indication of sympathetic blockade. Pain relief can be reported almost immediately after block or can be delayed for several hours in some patients.[75] The long-lasting neurolytic effect of phenol is often delayed, but its local anesthetic action is usually immediate. If opioids or sedative drugs have been employed, pain assessment scores are rendered meaningless in the immediate postblock period. Patients should be instructed to keep a pain diary after block to aid in assessment of effectiveness.

EFFICACY

There are no extensive data of efficacy in the literature. Gleim and colleagues[76] found that lumbar neurolytic block provides immediate and long-lasting improvement of painless walking distance and muscle metabolism in patients with severe peripheral vascular disease. Median painless walking distance increased from 95 m (10–200) before to 355 m (25–1003) after neurolytic block. Dabrowski et al.[77] demonstrated significant improvement of muscle and popliteal blood flow after sympathectomy. Limited data suggest efficacy of RF sympathectomy among carefully selected patients with sympathetically maintained pain.[78]

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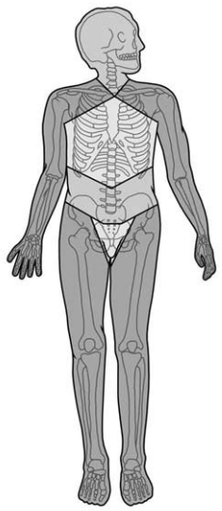
C H A P T E R



17

Lumbar Spinal Neuroaxial Procedures

MILTON H. LANDERS, RODNEY L. JONES, RICHARD M. ROSENTHAL, AND RICHARD DERBY

LUMBAR TRANSFORAMINAL EPIDURAL STEROID INJECTION

Lumbar radicular pain (in the past referred to as sciatica) and lumbar radiculopathy are problems frequently encountered by the interventional pain physician. These entities result from inflammation and irritation of the spinal nerves and the dorsal root ganglion (1,2). The most common etiology for these symptoms is a herniated nucleus pulposus or foraminal stenosis secondary to spondylosis.

Regarding radicular pain, it is well known that compression alone cannot account for these symptoms, in that mechanical manipulation of nonpathologic nerves during surgical procedures evokes numbness and paresthesia rather than pain (3,4). Substances known to be highly inflammatory, such as phospholipase A2, metalloproteases, and nitric oxide, have been shown to be present within disc material and are present at high levels around the segmental neural structures in cases of disc disruption. In experimental models, these same substances can produce pain and inflammatory changes (5–9). This chemical inflammatory response has been indicated as the primary cause of most radicular-type of pain (10–12). A corticosteroid, methylprednisolone, applied to the neural structures has been shown to reduce some of the experimentally induced inflammatory changes (13).

Injecting corticosteroids into the epidural space has been the mainstay for the conservative treatment of sciatica (14). Although the original technique described injection via the S1 posterior foramen, the interlaminar and caudal approaches to the epidural space historically became predominant in the United States. Questions about the efficacy of introducing corticosteroids into the dorsal epidural space, and the anatomically far-removed caudal canal, have been presented in reviews of the subject (15–18). In addition, with fluoroscopically guided spinal injections becoming more common, if not the norm, precise access to the intervertebral foramen, as well as the pain generators, is possible through a safe and reliable technique. Injection of corticosteroids into the intervertebral foramen, and segmental nerve canal, allows for a concentrated application of steroid around the inflamed neural structures. Anecdotal and good controlled trials have shown long-term clinical outcomes using a transforaminal approach for intraspinal steroid therapy, this technique seems to provide better relief than blind interlaminar technique. (19–24) This is not surprising in that placement of injectate into the dorsal epidural space by the interlaminar or transflaval approach will often fail to reach the target structures, that is, the segmental nerve and dorsal root ganglion, which lie ventral and lateral. This is especially true when pathology, such as scarring, further impedes the flow of medications. In addition, the interlaminar approach may not be appropriate post surgery, due to removal of the ligamentum flavum and adherence of the dura to the dorsal structures.

However, advantages of transforaminal corticosteroid injection come with increased complexity, requiring technical expertise, and bringing with it a significantly increased risk of morbidity. As with all interventional pain procedures, extensive clinical experience and good medical judgment is required to assess each patient's clinical condition and to offer an algorithm leading to the proper diagnosis and treatment.

INDICATIONS

The primary indication for transforaminal epidural steroid injection is radicular pain resulting from irritation and inflammation of the dorsal root ganglion and other neural structures in its vicinity.

CONTRAINDICATIONS

As with all spinal injections, contraindications include patients with significant bleeding diathesis, systemic or local infections at the procedure site, mental state making

communication difficult, including heavy sedation, and patients who are uncooperative. Surgical posterior fusion, with or without hardware, severe degenerative changes, large lateral disc displacement, and severe facet hypertrophic changes result in altered anatomy increasing the difficulty and risks. Substandard radiographic equipment, imaging tables, supplies and equipment, facility layout, and support staff untrained in interventional pain procedures will degrade the quality of care and can significantly increase risks.

PREPROCEDURE STUDIES

If the patient reports a history of prolonged bleeding or easy bruising, or is currently taking medications that interfere with blood coagulation, then appropriate coagulation studies are obtained prior to the procedure to assess elevated bleeding risk. Care and consideration should be taken when stopping these medications as to the reasons they were initially prescribed. Medications known to interfere with clotting are stopped at an appropriate time prior to the procedure (25). Stopping anticoagulation medications should be coordinated with the prescribing physician.

INFORMED CONSENT

Transforaminal injections carry risks of nerve damage from direct needle trauma, perforation of the dura, infection, and bleeding. Unrecognized, unintentional arterial injection with particulate corticosteroids can have catastrophic sequelae and is the possible mechanism for ischemia of the spinal cord with paraplegia, which has been much discussed. Informed consent should be obtained, explaining these risks.

ANATOMY

The dimensions of the intervertebral foramen are defined by the pedicles above and below, the dorsal aspect of the vertebral bodies, above and below, the posterior disc annulus, the zygapophyseal joint, and the ligamentum flavum. The contents of the foramen include the nerve root, arteries, veins, connective tissue, and fat. There are no "empty spaces" in the normal anatomy of the spine. Every point in and around the foramen where a needle can be placed is occupied by some tissue. The goal of transforaminal injections is to place the needle tip outside the segmental nerve and dorsal root ganglion and avoid the vascular structures. Degenerative processes and spinal roto-scoliosis can greatly alter the anatomy. The space is highly vascular, and increased venous engorgement can occur in response to space-occupying lesions.

In a true anteroposterior (AP) view, a so-called "safe triangle" has been described, although in fact it is a "safer" triangle (Figures 17-1 and 17-2) (26,27). The upper border is a transverse line running lateral from a point under the pedicle at the "6:00 o'clock" position, when the pedicle is

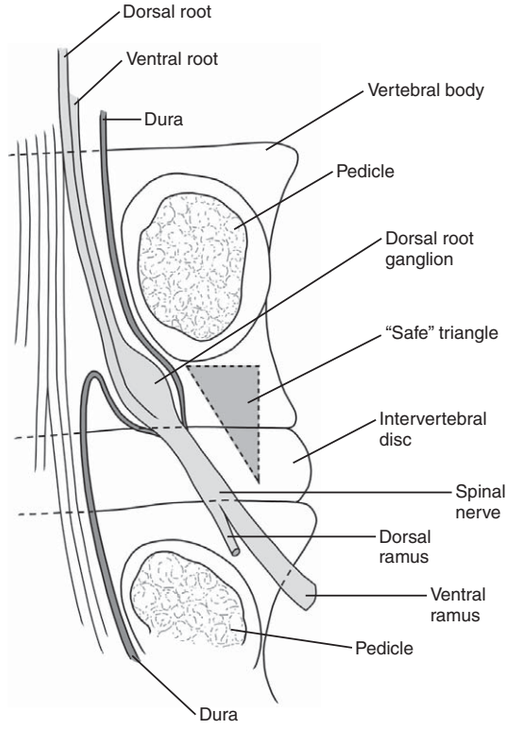


FIGURE 17–1

Illustration of transforaminal injection "safe zone" and contiguous structures.

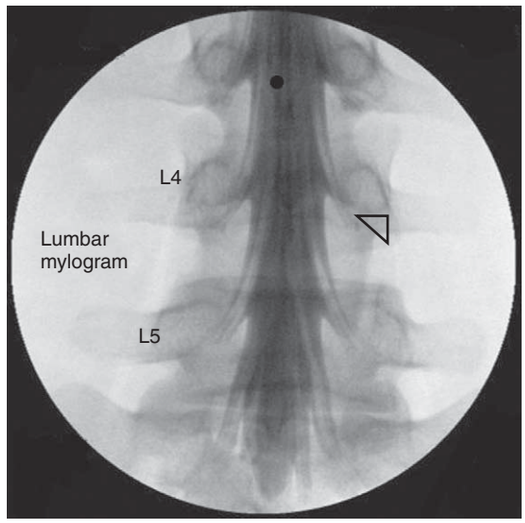


FIGURE 17–2
Lumbar transforaminal "safe zone" overlying AP view of lumbar myelogram. Note extent of dural root sleeves.

seen as a clock face, to the lateral pedicular line. The lateral boundary is a sagittal line extending caudad from the lateral aspect of the pedicle to the segmental nerve, and the hypotenuse connects the two lines and runs parallel to the lateral border of the nerve. Although in the vast majority of individuals, the dural investment of the segmental nerve root ends medial to the "6 o'clock" position, dural ectasia may be present with dural cysts within the lateral foramen. However, for the most part, a needle placed anywhere within this imaginary triangle would not be expected to risk dural, neural, or arterial impingement unless the anatomy of the region has been significantly changed due to surgical or degenerative changes.

The "magnus ramus radicularis anterior", i.e. great radicular, or medulary artery, was first described by Adamkiewicz and is often referred to by his name, that is, the artery of Adamkiewicz (28). The microanatomy has been elegantly reported as to origin and course (29). This vessel is the major arterial supply of the anterior spinal artery of the thoracolumbar spinal cord. This artery is found on the left approximately 80% of the time, and the origin is known to be from T9-L2 in the great majority of specimens. The artery courses medially through the rostral or mid portion of a foramen, and lies in close proximity to the dorsal root ganglion-ventral root complex. Injection into, or damage to, this artery including emboli derived from particulate corticosteroid injectate has been proposed as the probable etiology of paraplegia and other neurological sequelae associated with transforaminal injection procedures (30,31). Branches of the artery of Adamkiewicz lie in and around each foramen and supply various structures, including the spinal nerve, and dorsal and ventral roots, and go on to anastamose with vessels arising from the conus medullaris.

EQUIPMENT

Although historically lumbar transforaminal, or paravertebral somatic nerve blocks, were performed by blind, nonfluoroscopic-guided techniques, this is no longer an acceptable medical practice in any community (32). Fluoroscopy is required when performing lumbar transforaminal injections, and a fluoroscope capable of excellent image quality is a necessity. A C-arm fluoroscope, which allows the x-ray beam to be directed from any angle, has advantages and is the instrument of choice utilized by the vast majority of spinal injectionists. The ability to save the last image is a must. Low-dose and pulsed x-ray modes are of great benefit to minimize overexposure to the radiation inherent in any fluoroscopically guided procedure. A sterile cover over the image intensifier allows optimum positioning of the fluoroscope and fine adjustments during the procedure. Although not a standard of care at the present, digital subtraction angiography (DSA) is being widely used to ensure adequate visualization of the contrast pattern and prevent possible unintentional arterial injection.

Although advocated by a small group of practitioners, computed tomography (CT)–guided transforaminal injections are not appropriate and offer no benefit and increased risk. With CT, although the skin insertion point is exactly visualized, needle advancement toward the target endpoint is essentially performed with no x-ray guidance. This results in additional discomfort to the patient and an increased chance of needle misadventures. In addition, CT use necessitates the injection of contrast without active, real-time, x-ray visualization. Vascular injection would in all likelihood be missed due to the rapid clearing of the contrast in an artery or vein. No literature exists as to any benefit of using CT, and the increased exposure to radiation by

the patient is a real concern. In guidelines as published by the International Spine Intervention Society (ISIS) and PASSOR (Physiatric Association of Spine, Sports and Occupational Rehabilitation), CT imaging is specifically not mentioned (33,34). It can therefore be inferred that it is not recommended. There is at least one case in the literature where a transforaminal L2 injection was performed with CT guidance, with resultant spinal cord infarction and severe permanent neurological complications (35). The lesion was presumed to be secondary to injection into the artery of Adamkiewicz, which as noted earlier, is the dominant arterial supply of the anterior spinal artery of the thoraco-lumbar spinal cord. Contrast was not utilized in this mishap, although it is questionable whether its use would have prevented this disastrous outcome since real-time imaging during injection of contrast cannot be utilized. CT is simply not the appropriate imaging modality for transforaminal injections at any level.

A radiolucent procedure table compatible with the fluoroscope is utilized. Intravenous access is advocated by some. Monitoring equipment, including pulse oximetry, noninvasive blood pressure, and EKG should at the least be immediately available. Oxygen, airway supplies, emergency drugs, suction and other resuscitation equipment and supplies should be in the procedure room and checked on a regular basis. Well-trained personnel who are able to assist the physician, monitor the patient, and operate the fluoroscope are essential to lessen complications and improve safety. Appropriate radiation protection and radiation exposure monitors should be provided for all personnel in the room.

Procedure needles typically 22- or 25-gauge of 3.5–5.0 inches in length are utilized. When a two-needle technique is required, a shorter, slightly larger-gauge introducer needle is also used. Sharp-tipped needles, Quinke or Chiba, are employed by the vast majority of injectionists when performing lumbar transforaminal injections. A small bend placed at the tip opposite the bevel, in the direction of the point, to aid in needle control during insertion, is desirable (36). This bend allows change of direction of the needle by rotation during insertion. Occasionally, when the two-needle technique is required, a large bow in the needle is utilized to negotiate around obstacles such as intertransverse fusion masses (Figures 17-3, 17-25, and 17-26). Although there is no explicit evidence against their continued use, some eminent authorities have stated that the use of sharp needles should be curtailed and blunt needles utilized to reduce the complication rate of unintentional vascular injection. This argument has been addressed in a recent publication (36). Because a point is lacking in blunt needles, being a truncated cylinder, such needle cannot pierce the skin without an introducer, and advancement will be difficult through any dense tissue. Although blunt needles may have a bend on the tip, they are harder to control and require larger gauges. In addition, their supposed advantage of lowering the frequency of intervascular injections has been shown to be false. In a recent small, thus far unpublished study, more intravenous injections were noted with blunt

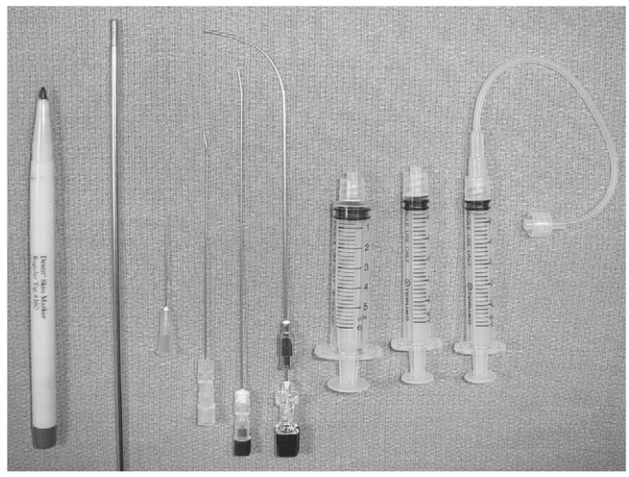


FIGURE 17–3

Equipment used in lumbar transforaminal injections. From left to right: Skin marker, pointer, 25-gauge 1-1/2-inch needle for skin anesthesia, 25-gauge 3-1/2-inch procedure needle with mild curve at distal tip; 22-gauge, 5-inch procedure needle with mild curve at distal tip; 22-gauge, 9-inch procedure needle with significant curve through 18-gauge 3.5-inch introducer needle; 5–6-cc syringe for skin local anesthetic; 3-cc syringe for injectate (corticosteroid with or without local anesthetic); 3-cc syringe with extension tubing for contrast.

than sharp needles (Figure 17-4) (37). In short, blunt needles are not a substitute for excellent technique, precise needle placement, and vigilance in interpretation of the contrast test dose, that is, a trained, skilled operator.

Skin preparation with an iodine-based solution (e.g., Povidone-iodine), or chlorhexidine, with or without alcohol, followed by draping with sterile towels or a fenestrated drape is advocated. Sterile gloves, a metal pointer to allow determination of the skin entry point while using fluoroscopy, and a sterile skin marker should be provided. Syringes

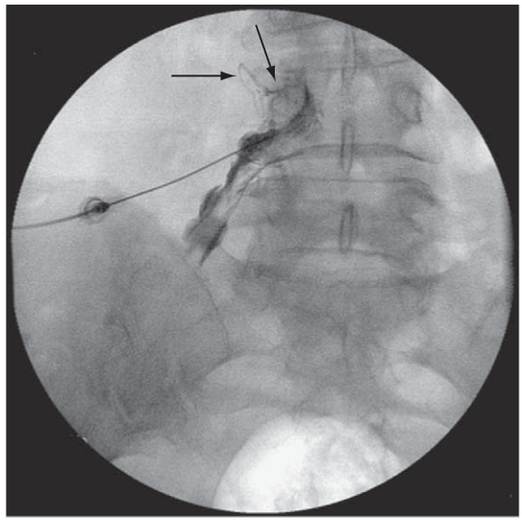


FIGURE 17–4

Left L5 transforaminal injection using a blunt needle and intra-vascular injection. (Courtesy of Kevin Pauza, MD.)

of 3 and 5 cc are utilized. A small-bore, low-volume extension tubing allows contrast to be injected under active fluoroscopy to confirm nonvascular needle placement without irradiation of the hands. In addition, extension tubing minimizes the movement of the needle while syringes are being changed.

Sedation, although not required in the vast majority of cases, is advocated by some physicians. To a large extent, regional bias and patient expectation, rather than medical necessity, appear to dictate this practice, since the discomfort experienced during a transforaminal injection by a competent practitioner with small-gauge needles is minimal. If the physician chooses to sedate his patients, intravenous access and monitoring are mandatory. Midazolam in a dose of 1–2 mg, should be adequate to provide sedation. It is unacceptable to render the patient unconscious during any spinal injection procedure. If a patient demands a level of sedation in excess of that which the physician feels reasonable, a psychological overlay should be considered, the risk–benefit ratio explored, and the procedure possibly canceled. Although small doses of analgesics (fentanyl 50 mcg, meperidine 50 mg, or morphine 5 mg) may lessen the discomfort of the injection, if any diagnostic trend is to be forthcoming, these opioids may render any response by the patient questionable.

The use of a water-soluble, nonionic contrast medium, iohexol (Omnipaque) or iopamidol (Isovue), must be utilized in all fluoroscopically guided spinal injections to ensure that the injectate is covering the proposed target—the spinal nerve and dorsal root ganglion in the case of transforaminal injections—and that no arterial, or marked venous, uptake is noted. The contrast solution concentrations between 180 and 240 are adequate for this purpose.

The primary purpose of a lumbar transforaminal injection is placement of an anti-inflammatory agent, corticosteroids, in the vicinity of, and bathing the possibly inflamed structures generating the radicular type pain. As noted previously, many of the catastrophic problems associated with this procedure appear to be due to spinal cord ischemic infarction, associated with injection of particulate corticosteroids into the radicular artery. Therefore, common sense dictates that a less particulate agent may offer some margin of safety. Methylprednisolone, due to its large particulate formulation, might not be considered the best choice for this application. Rather, triamcinolone 40–80 mg, betamethasone 6–18 mg, or dexamethasone 4–12 mg might be a better alternative. Derby et al.[38] have recently compared red blood cell size to the particle and aggregate size of the frequently used corticosteroid solutions and concluded that dexamethasone "will not cause arterial or capillary obstruction if inadvertently injected into a vertebral or radicular artery." (38)

Although the main purpose of a transforaminal injection is delivery of corticosteroids, local anesthetics are often utilized. The amide group of local anesthetics, without preservative, is preferred due to the allergenic profiles. Lidocaine is an extremely safe, versatile, and inexpensive medication. It is often used for skin infiltration in the 1% (10 mg/cc) concentration. For the transforaminal injectate, 2-4% (20-40mg/cc) is preferred. Bupivacaine 0.5–0.75% (5-7.5 mg/cc), a longer-duration, amide-type local anesthetic, can be substituted for lidocaine. Less than 1 cc of local anesthetic, total volume 1.5-2cc, needs be used for transforaminal injections.

The local anesthetic response can validate the procedure, in that if local anesthetic is utilized with the corticosteroid, and the pain is decreased markedly in the postprocedure period, by inference the pain generator has been addressed.

Karasek and Bogduk[39] reported a case of temporary neurological deficit while performing a transforaminal injection, following injection of a small aliquot of local anesthetic (0.8 cc of 2% lidocaine). A transforaminal injection was confirmed by prior injection of contrast, and although some venous uptake was noted, no arterial pattern was appreciated. Although this occurred during a cervical rather than lumbar, transforaminal injection, the result of a lumbar injection into the medulary artery would be expected to be analogous. In response to this and other cases, some have maintained that a "test dose" of local anesthetic, followed by a 1- to 2-minute period where the patient is observed and examined for neurological deficits, might prevent unintentional injection of corticosteroids into a radicular artery, with possible devastating sequelae (30).

Warning: Spinal injections in the pain patient, whether for diagnosis or therapy, should be performed only by physicians who have the extensive training required to evaluate such patients, interpret imaging studies, perform the procedures in a safe manner, and analyze in real time the radiographic information obtained during the procedure.

NEEDLE PLACEMENT TECHNIQUE

Although various techniques for the accessing of the lumbar inter vertebral foramen have been proposed, the majority target the subpedicular area, that is, the rostral and ventral portion, of the intervertebral foramen (27,40). Recently, a "retroneural" approach has been described which results in the needle tip being placed subpedicular, but in the mid-foramen slightly dorsal to the segmental nerve than seen in the more classic position (1).

The purported advantage to this retroneural approach is that it attempts to address the problem of unintentional injection into the artery of Adamkiewicz, which as noted earlier, courses medially through the mid or rostral portion of the foramen, enters the dura, and supplies the anterior spinal artery, occlusion of which has been proposed to be associated with paraplegia and other neurological sequelae. However, the above is supposition based on anatomical dissections, and no true evidence exists indicating that the retroneural approach is clinically safer. Additionally, all

studies used to validate the efficacy of transforaminal deposition of steroids utilized the classic, more ventral needle tip position (19–22). Clinically, the difference between the retroneural and the more ventral needle placement is often merely a matter of needle insertion depth, with little actual difference in skin entry or needle insertion targets.

When a C-arm fluoroscope is utilized for lumbar transforaminal injections, the patient is placed in prone position. Often a pillow under the upper abdomen will decrease the physiologic lumbar lordotic curve and allow for optimum visualization. Depending on target level, the lower thoracic, lumbar, and/or sacral regions are prepared and draped in a sterile manner.

Accurate target identification requires that an examination of the lumbar spine by fluoroscopy precede any needle placement. Verification that five lumbar, non–rib-bearing vertebral bodies are present must be ensured. Approximately 10% of the population will be noted to have either a nonsacralized S1, or sacralized L5 vertebra, which can lead to misidentification of the level being treated and any diagnostic inferences derived. Using an AP image, the pedicle corresponding to the targeted foramen is identified. To ensure a true AP image, using cephalad-caudad tilt of the image intensifier, the inferior end-plate just caudad to the pedicle identified is "squared," that is, the x-ray beam passes tangentially. The inferior end-plate should be seen as a line rather than an oval. For example, if the target is the L4 foramen, dorsal root ganglion (DRG) and segmental nerve, the L4 pedicle is identified and the fluoroscope is maneuvered until the beam is parallel to the inferior end-plate of L4, which is seen as a single line.

The final needle-tip target is within the foramen, subpedicular, approximately halfway between the ventral and dorsal extent of the pedicle when imaged in a true lateral view. This location will place the needle tip rostral and lateral to the DRG and segmental nerve, and dorsal to the anterior radicular artery.

In most spinal injections, a down-the-beam, so-called "tunnel vision" is best utilized. This involves aligning the skin entry point with the anatomical target. This technique obviates the need to guess at the correct angle of needle insertion, and if the anatomy lying between the skin and target structure is well known to the injectionist, offers the safest approach. After the target level is identified and the end-plate "squared," as detailed earlier (Figures 17-5 and 17-15) to ensure a true AP view, the C-arm is then rotated until an ipsilateral oblique view projects the superior articular process (SAP) of the infrasegmental level so that it appears to lie under the 6 o'clock position of the target pedicle (Figures 17-6 and 17-16). The skin is marked over the target, caudal to the pedicle. If a needle larger than 25 gauge is used, a skin wheal is made, through which the procedure needle is introduced. The needle is slowly and carefully advanced through the tissues toward the target (Figures 17-6, 17-7, and 17-17). Intermittent, spot fluoroscopic images are used throughout the needle insertion while the needle is advanced in small increments. If the needle is noted to stray from the desired course, it is slightly withdrawn, rotated, and utilizing the bent tip, advanced along the corrected direction. The needle should not be allowed to stray medial to the superior articular process (SAP), 6 o'clock position, or lateral to the lateral-pedicular line. Although not required, touching the caudal aspect of the pedicle shadow, which is the caudad aspect of the

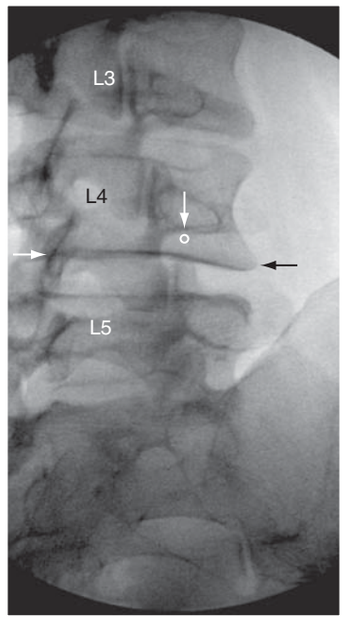


FIGURE 17–5

Right oblique view of lumbar spine with target for a right L4 transforaminal injection indicated. Note that the inferior end plate of L4 is parallel to x-ray beam and SAP of L5 is positioned under the "6:00" position of the L4 pedicle.

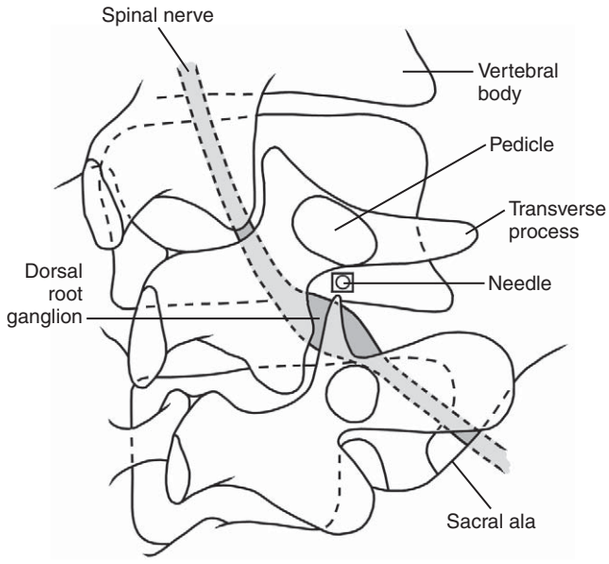


FIGURE 17–6

Illustration of needle placement for an idealized right L4 transforaminal injection. Note position of needle lateral and rostral to the segmental nerve.

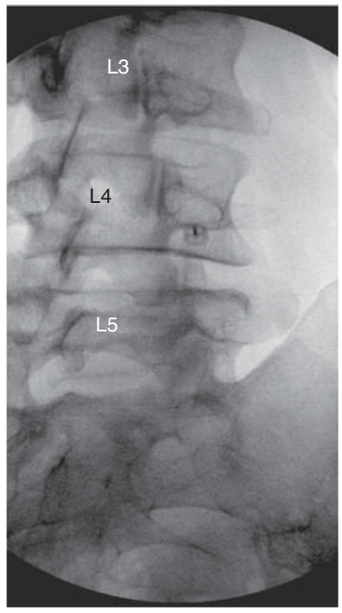


FIGURE 17–7
Right oblique view of the lumbar spine. Needle in place for L4 transforaminal injection.

transverse process or lateral lamina, ensures verification of depth prior to entering the foramen. The needle can then be slightly withdrawn so that the tip is not restricted by bone, and using the slight bend at the tip, rotated and advanced so as to "slide off" into the rostral aspect of the foramen. Needle insertion continues until either resistance to further advancement is noted or the patient experiences a dysethetic radicular-type pain.

If resistance is met during needle insertion, a lateral fluoroscopic view should be obtained. If a posterior element of the spine, transverse process, lamina, or SAP is preventing passage, the bend in the needle tip can be utilized to pass around the structure. Occasionally, withdrawal of the needle up to 5 mm may be required to bypass the impeding structure. If on lateral view the needle is noted to have contacted the dorsal-lateral aspect of the vertebral body, withdrawal 2–3 mm is advised. This lessens the chance of the radicular artery having been "trapped" between bone and needle and accidentally cannulated.

If radicular pain is noted by the patient at any point during needle insertion, the spinal nerve or DRG may have been touched, and the needle should be immediately withdrawn a small amount. If marked pain continues after withdrawal of the needle, termination of the procedure should be considered after documenting the needle position with AP, oblique and lateral fluoroscopic spot films. If the pain is noted to abate, a lateral view should be obtained. If the needle tip is noted to lie within the foramen, and on AP view the tip is seen to be within the "safe triangle," the procedure should proceed without further needle advancement.

Verification of final needle position by fluoroscopy in AP and lateral views is mandatory at this stage of the procedure. In the AP view (Figures 17-8, 17-9, 17-18, and 17-27), the needle tip should be positioned just caudal to the pedicle shadow under the 6 o'clock position, while a lateral image (Figures 17-10, 17-11, 17-19, and 17-28) will find the

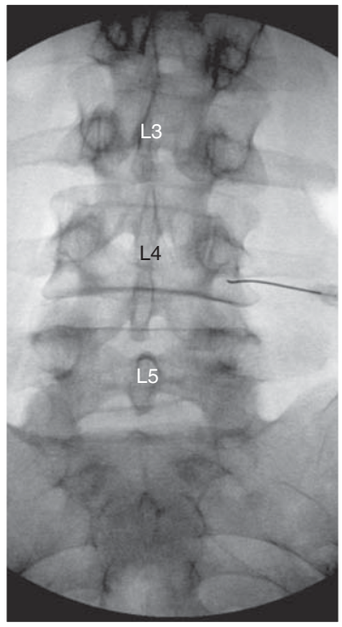


FIGURE 17–8
Anterior posterior view of lumbar spine. Needle in final position for L4 transforaminal injection. Note needle tip is in excellent position just lateral to the "6:00" position under the L4 pedicle.

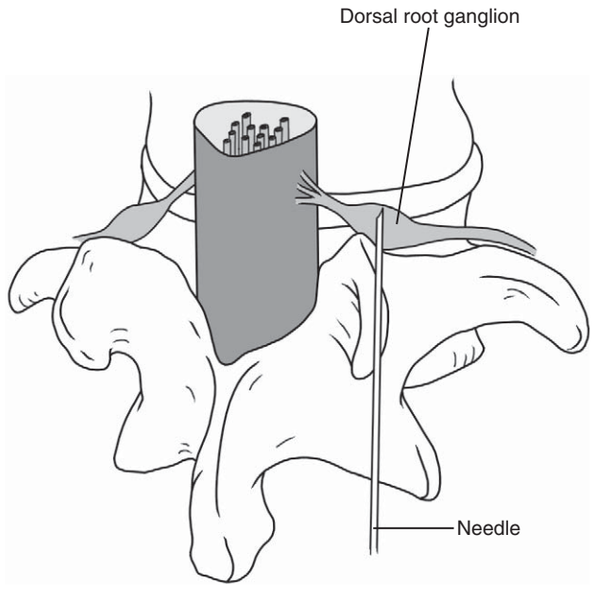


FIGURE 17–9
Illustration with a slight right oblique orientation of lumbar spine. Final needle position for a lumbar transforaminal. Note needle tip is lateral and rostral to the DRG and segmental nerve in the mid-portion of the foramen.

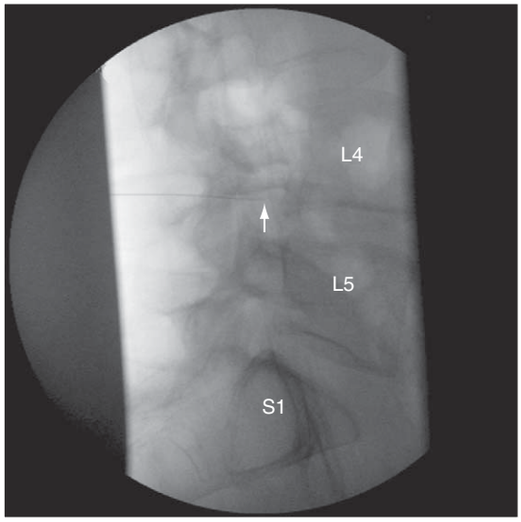


FIGURE 17–10

Lateral view with needle in excellent position within the L4 foramen, subpedicular and in the middle aspect of the foramen.

tip in the mid or ventral aspect of the foramen and subpedicular. The needle tip will lie lateral and rostral to the segmental nerve and DRG (Figures 17-9 and 17-11). If required, further adjustment of the needle into the correct position can be entertained.

Once needle tip position is noted to be within the target zone, a contrast-containing syringe with low-volume extension is attached to the procedure needle. Using AP, real-time imaging, with digital substraction angiography (DSA) if available, a small volume of contrast medium (0.25–0.5 cc) is injected. If no vascular uptake is noted, an additional 0.5–1.0 cc is injected to verify that the injectate will cover the desired target structure, that is, the pain generator and site of suspected inflammation. Total contrast volumes of 1.0–1.5 cc are usually adequate. Contrast should be seen to flow medially from the needle tip, through the foramen, and rostral around the pedicle, covering the segmental nerve and DRG (Figures 17-12a–e, 17-13, 17-20, 17-21 and 17-26). Volume of contrast required to cover the target structure should be noted. If an aberrant contrast pattern is observed, the injection should be stopped, needle repositioned, and further contrast injected. If a vascular pattern is noted, determination of whether it is arterial or venous must be made. As noted earlier, an injection into a radicular artery can have disastrous results. Any vascular structure noted to flow medially and seen to end at the midline in AP view must be considered arterial until proved otherwise (Figures 17-23 and 17-24). If a radicular arterial pattern is evidenced, the needle should be withdrawn and the procedure terminated and rescheduled at a later time.

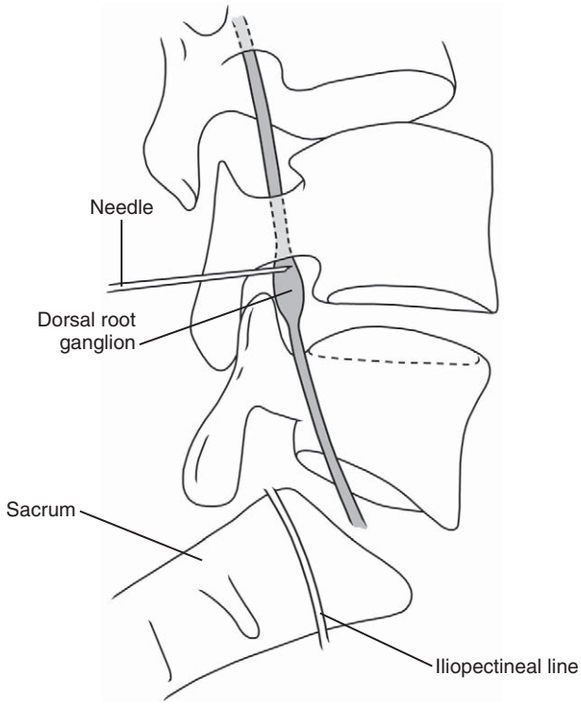


FIGURE 17–11

Illustration of a L4 transforaminal injection (lateral view). Note needle tip in the middle aspect of the foramen lateral to the DRG.

Venous contrast patterns may be noted to flow laterally or will cross to the contralateral side in AP view. If the contrast pattern evidences a venous pattern the needle position should be readjusted and contrast re-injected. If the repositioning affords a pattern covering the target, without vascular uptake, the procedure can continue. If a significant venous injection is noted with multiple repositionings, the procedure should be terminated.

Spread of contrast laterally along the ventral ramus, rather than medially, warrants repositioning of the needle since the target structure, that is, the DRG, is not being addressed. A lateral view (Figures 17-14 and 17-22) should confirm contrast in the foramen and ventral epidural space.

Occasionally, if an obstacle, such as an intertransverse fusion mass, is present, a two-needle technique is required in order to gain access to the intervertebral foramen. This entails the use of a larger gauge introducer needle, which is inserted laterally and slightly ventral to the obstruction. A second, smaller-gauge procedure needle, with varying degrees of curve at the distal end, is then advanced through the introducer needle (Figures 17-3, 17-25, and 17-26). As the procedure needle emerges from the introducer needle, the curve is reinstituted and maneuvered into the target area. Once the procedure needle passes from the distal end of the introducer needle, the latter may be withdrawn slightly as the former needle is advanced. Due to the myriad variations that may be encountered, a more detailed discussion is not possible in this venue. Care must be taken at all times due to significant loss of needle control due to the needles extreme curved profile. Confirmation of needle position with contrast is described in Figures 17-25 and 17-26.

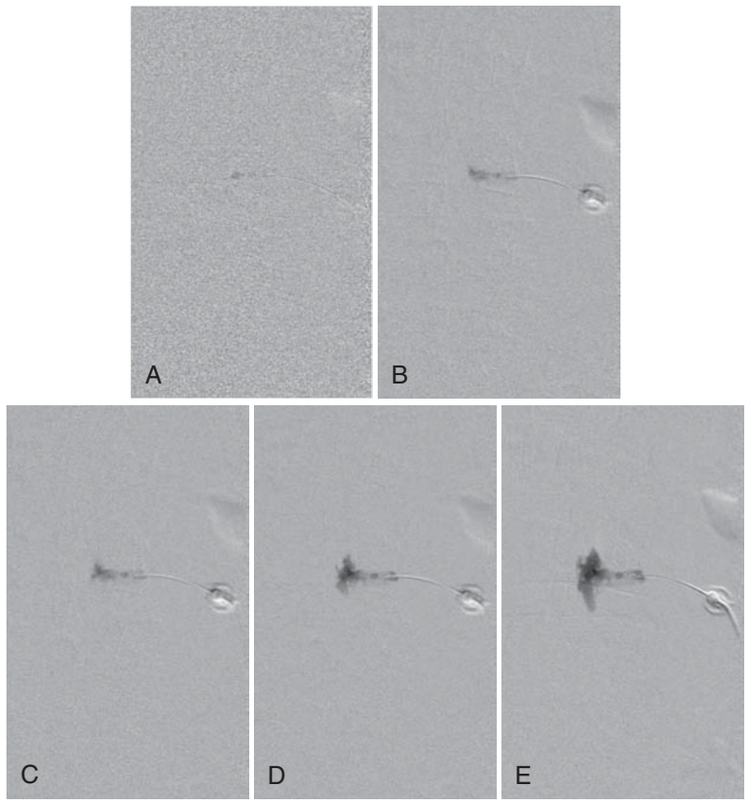


FIGURE 17–12

(A to E) Sequential digital subtraction angiography, digital subtraction angiography, images of a L4 transforaminal injection. Note flow through foramen, around pedicle within the nerve canal and into the epidural space without vascular uptake.

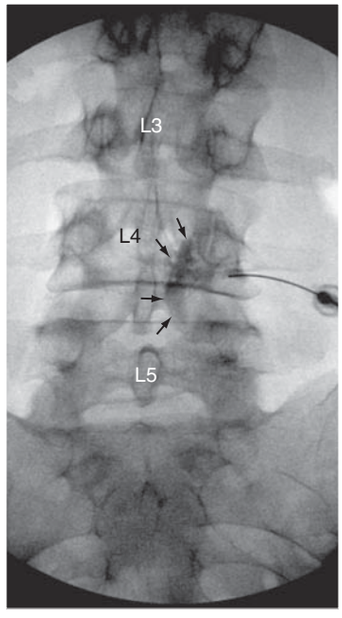


FIGURE 17–13

Plain film anteroposterior of L4 transforaminal injection with contrast. Note flow through foramen into the lateral epidural space.

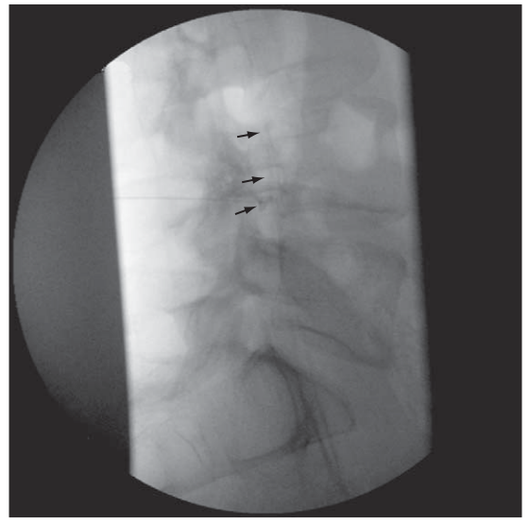


FIGURE 17–14

Lateral view of L4 transforaminal injection. Note contrast in foramen and ventral epidural space.

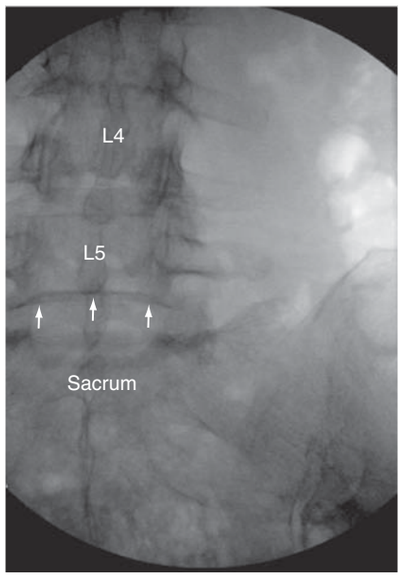


FIGURE 17–15

Scout AP image for right L5 transforaminal injection. Note inferior end plate of L5 is parallel to x-ray beam.

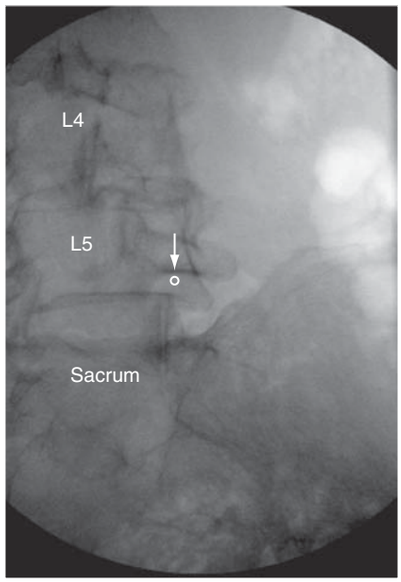


FIGURE 17–16

Right oblique initial view of lumbar spine for L5 transforaminal injection. Open circle indicates target. Note SAP of S1 appears to be under the "6:00" position of the L5 pedicle.

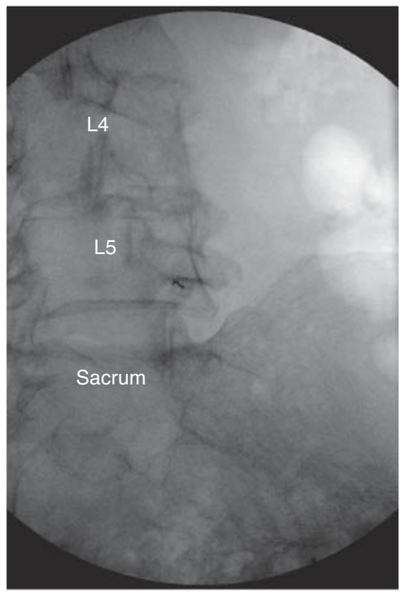


FIGURE 17–17

Ipsilateral oblique view with 25-gauge needle in place for a right L5 transforaminal injection.

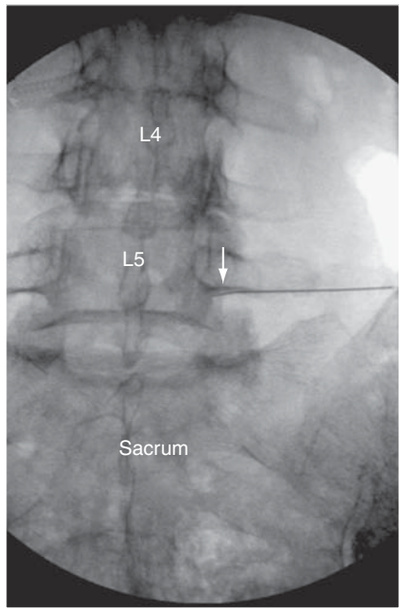


FIGURE 17–18

AP view of needle in final position for L5 transforaminal injection. Note needle tip is seen as lying under the "6:00" position of the L5 pedicle.

On rare occasions, although the needle tip is seen to lie in proper position by the bony landmarks, some radiolucent structure might, in fact, have been penetrated. Figures 17-27 to 17-31 demonstrate needle entry into a far lateral intervertebral disc extrusion with cephalad migration, producing an unintentional discogram with injection of contrast.

Needle placement and injection between the dura and arachnoid layers, a subdural injection, is a rare occurrence (Figures 17-32 and 17-33) and must be differentiated from an intrathecal placement. Subdural contrast will be noted to be maintained in a cyst-like structure and does not layer out in the ventral thecal sac as will be seen with an intrathecal injection. In addition, the contrast will not be

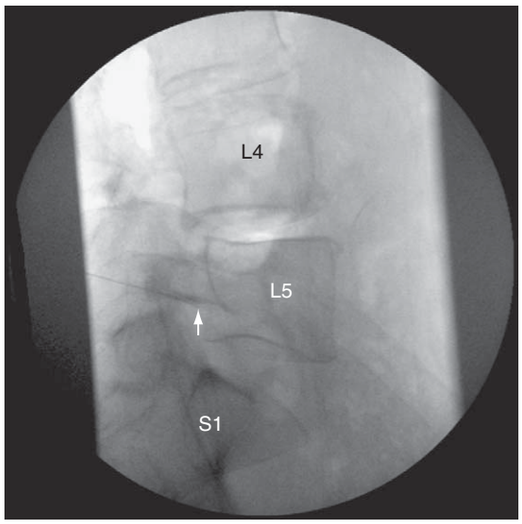


FIGURE 17–19

Lateral view with needle in excellent position within the L5 foramen, subpedicular and in the middle aspect of the foramen.

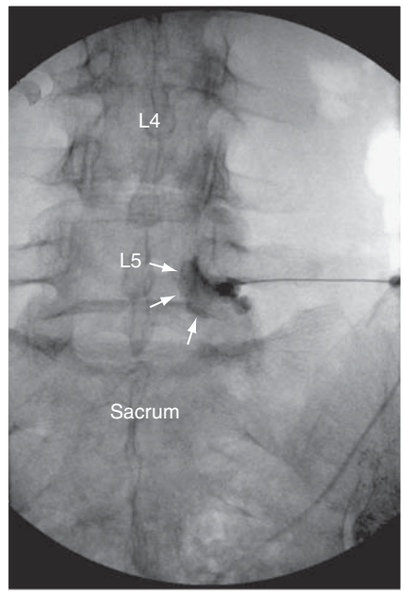


FIGURE 17–21

Excellent contrast pattern for an L5 transforaminal injection.

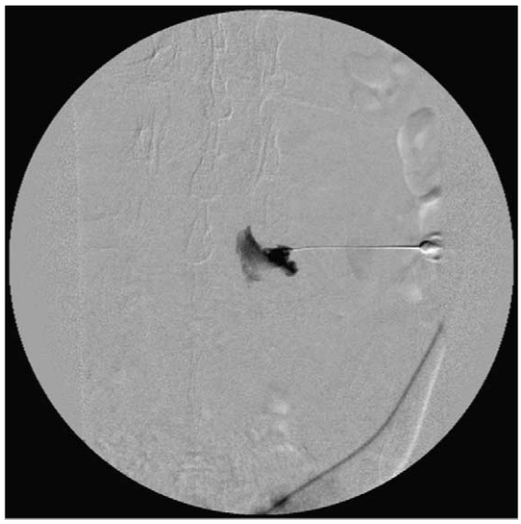


FIGURE 17–20

Digital subtraction angiography image of a L5 transforaminal injection. Note flow of contrast around the pedicle into the epidural space without intravascular pattern evident.

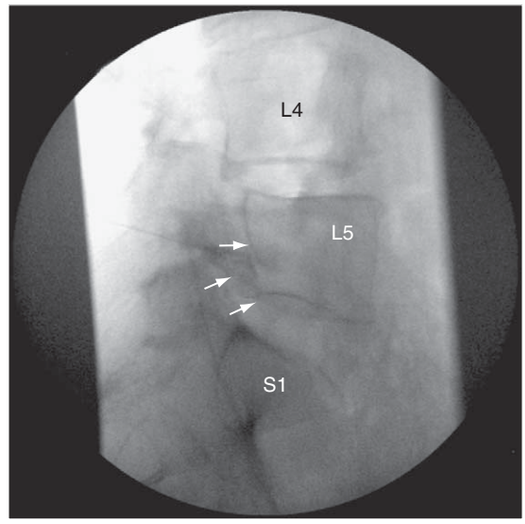


FIGURE 17–22

Lateral view of the L5 foramen with contrast within the foramen and ventral epidural space.

diluted by the cerebral spinal fluid (CSF). Often aspiration and injection of the contrast can be seen to produce rapid changes in the volume of the contrast body. If a subdural injection is noted, the needle can be slightly withdrawn and repositioned, followed by confirmation of good placement with reinjection of contrast. Unlike subdural placement, if an intrathecal pattern is observed, the procedure should be terminated.

The capsule of the zygapophyseal (i.e., facet) joint covers the inferior and superior articular processes and is known to be loose at the superior pole. The capsule "balloons upwards toward the base of the next transverse process." (2) While excellent position may be noted in AP and lateral views while performing a transforaminal injection, there is a small probability of its being within the superior capsule of the zygapophyseal joint. This is

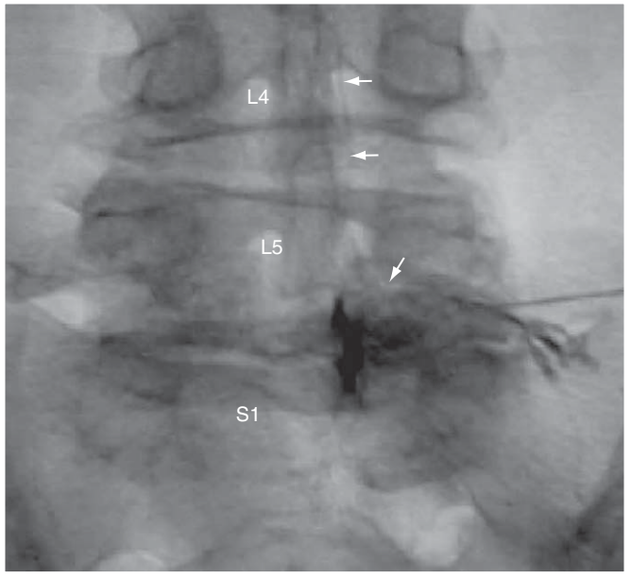


FIGURE 17–23

Right L5 transforaminal injection. Arrows indicate contrast within the radicular artery, artery of Adamkiewicz, and anterior spinal artery. (Courtesy of Way Yin, MD.)

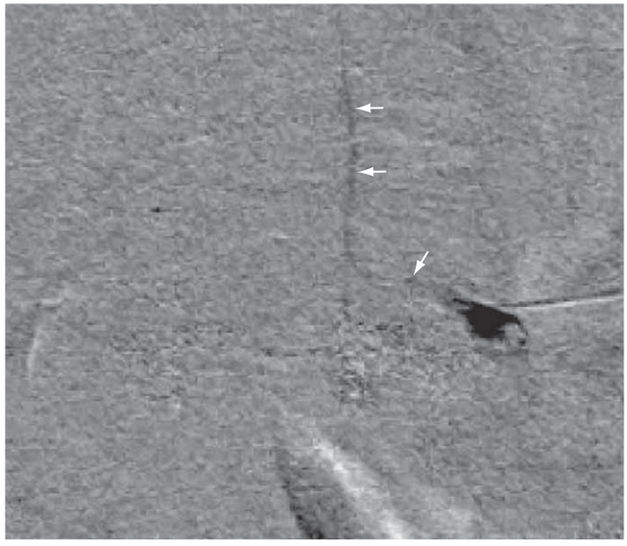


FIGURE 17–24

DSA image of the right L5 transforaminal injection seen in Figure 17-23. Note relative ease in visualization of the radicular and anterior spinal artery as compared with the previous figure. (Courtesy of Way Yin, MD.)

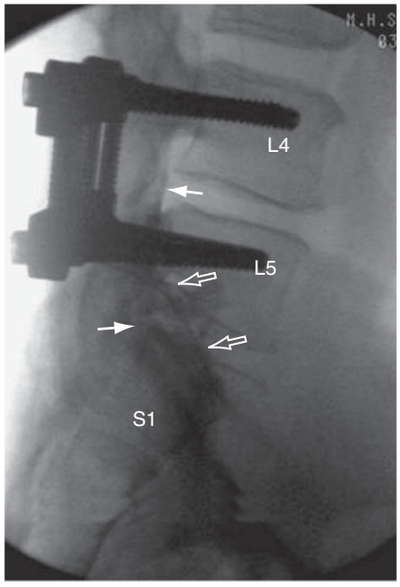


FIGURE 17–25

Lateral view of dual needle technique. Open arrow indicates tip of needle within the L5 foramen. Solid arrows note contrast within the foramen, epidural space and following segmental nerve.

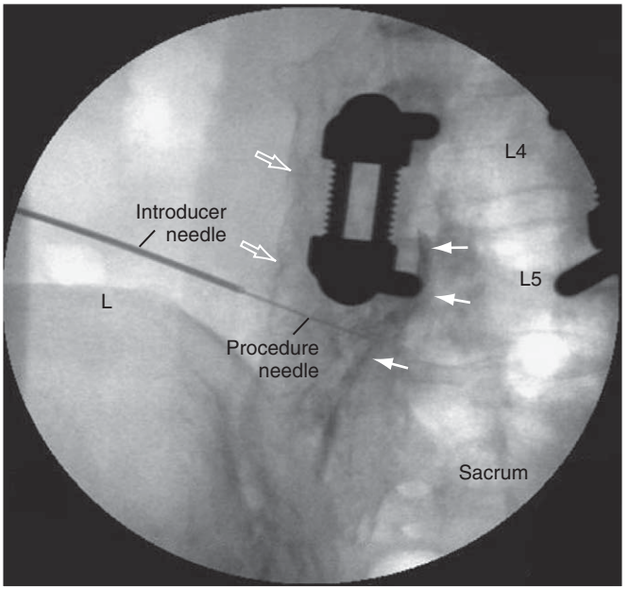


FIGURE 17–26

Anteroposterior view of intertransverse fusion mass with L5 transforaminal injection utilizing a dual needle technique. Open arrows indicate lateral aspect of fusion mass. Note 18-gauge introducer needle lateral to fusion mass with 22-gauge curved procedure needle proceeding ventral to mass into foramen. Solid arrows indicate contrast within epidural space.

seen most often during an L5 transforaminal injection (Figure 17-34). If a "Z" joint arthrogram is noted, the needle must be repositioned and additional contrast injected. Often advancing the needle as little as 1–2 mm is enough so that the needle tip is ventral to the capsule and a good transforaminal contrast pattern noted.

Although extremely rare, an intraneural injection is possible, especially if heavy sedation is utilized. If not overly sedated, excruciating radicular dysethetic pain would be evidenced by the patient. Injection into the epineurium might occur without marked discomfort; however, the contrast pattern would evidence

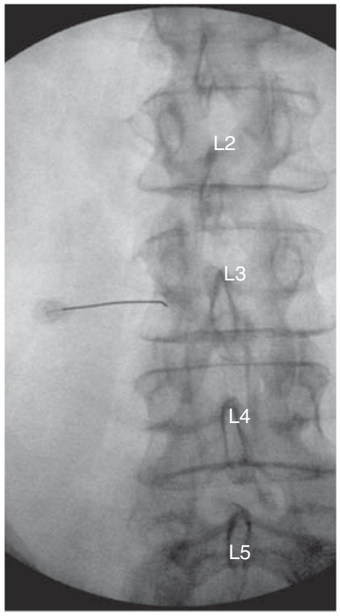


FIGURE 17–27

Anteroposterior view of needle in excellent position for a left L3 transforaminal injection.

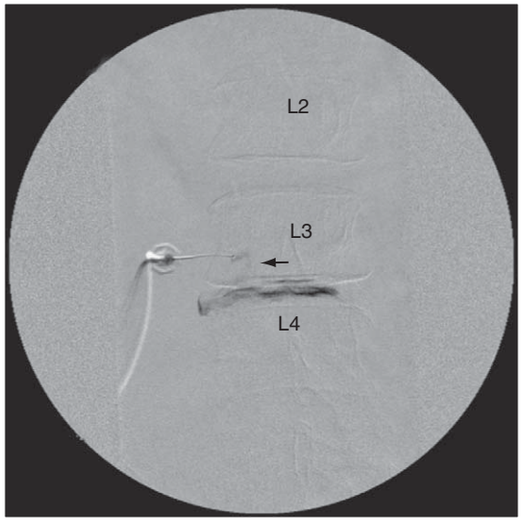


FIGURE 17–29

Digital subtraction angiography anteroposterior image during injection from Figures 17-27 and 17-28. Note contrast flow into intervertebral disc through disc extrusion within foramen.

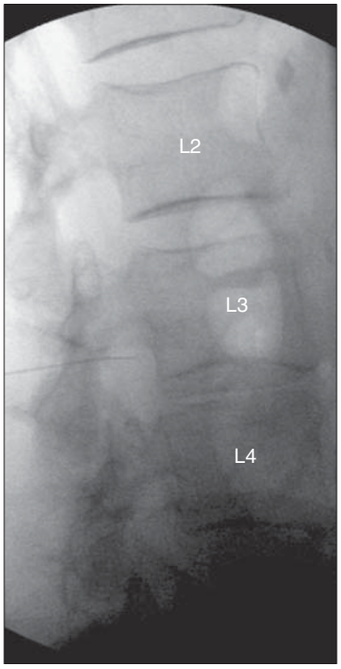


FIGURE 17–28

Lateral view of needle in excellent position within the L3 foramen.

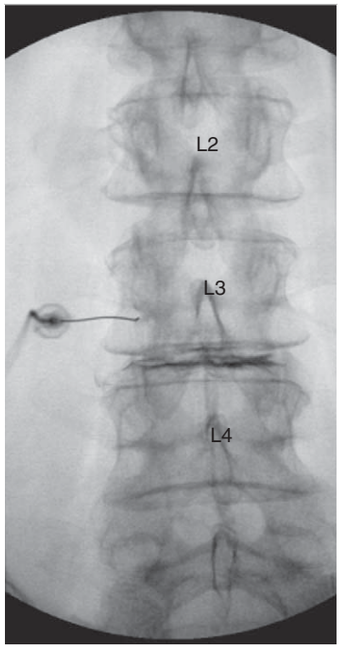


FIGURE 17–30

Anteroposterior view of disc injection via extruded disc material.

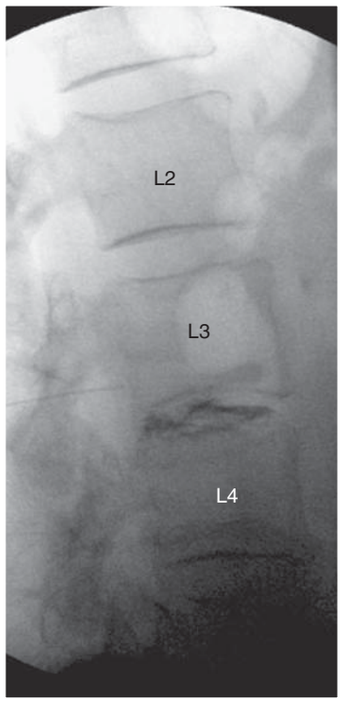


FIGURE 17–31
Lateral view following injection into disc extrusion with unintentional discogram.

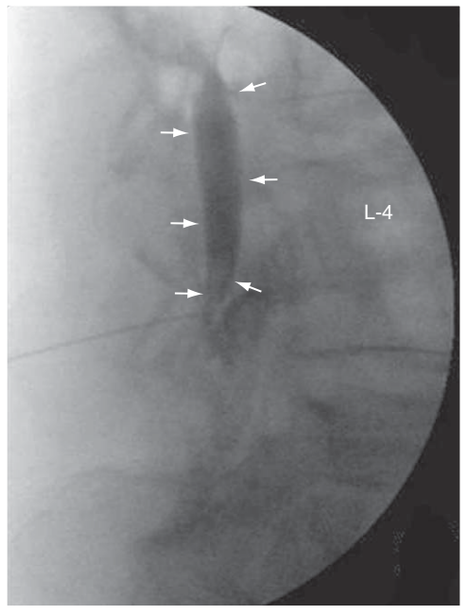


FIGURE 17–33
Lateral view of subdural injection during L4 transforaminal injection

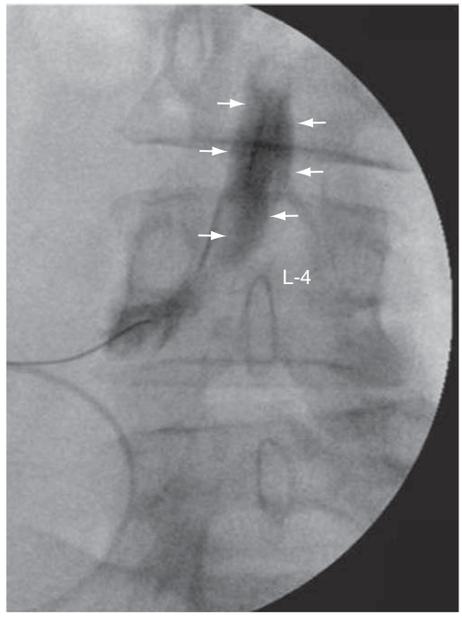


FIGURE 17–32
Anteroposterior image of left L4 transforaminal injection with subdural pattern.

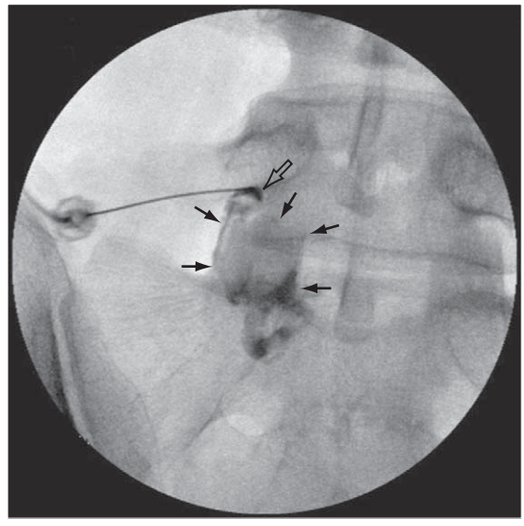


FIGURE 17–34
Unintentional intra-articular zygapophyseal, facet, joint injection during L5 transforaminal injection. Open arrow indicates needle tip in superior capsule of the joint.

outline of the nerve within the intrathecal space (Figure 17-35).

Final permanent documentation following a lumbar transforaminal injection should include AP and lateral images with and without contrast, and DSA if utilized.

INJECTION OF THERAPEUTIC AGENT

Following confirmation of needle placement and evidence of a good contrast pattern, the syringe containing the contrast is disconnected from the extension tubing and replaced by one containing the therapeutic agent(s). Care is taken to remove all air from the syringe. A mixture of local anesthetic and corticosteroid can be utilized. As discussed earlier, some evidence suggests that injecting the local anesthetic may provide an additional margin of safety. The minimum volume of injectate is dictated by the volume of contrast required to adequately cover the target structure, usually between 1.5–2cc.

During injection the patient might be aware of a pressure paresthesia, that is, paresthesia or dysesthesia into the lower extremity. If this is not severe, injection can proceed, and the patient queried on whether the paresthesia is in the same distribution as their usual pain, that is, concordant with, and notation of this made on the procedure note. If extreme pain is noted, slight repositioning of the needle may alleviate or lessen the discomfort and the procedure continued. If severe pain on injection continues after needle reposition, the procedure should be terminated.

POSTPROCEDURE CARE

Once the therapeutic agent has been injected, the needle is removed, skin cleaned of any blood and antiseptic preparation, and a sterile adhesive dressing applied. The patient is then taken to a recovery room where he or she is observed by trained personnel with physiologic monitoring utilized. Any complications must be diagnosed and managed in a timely and appropriate manner. Unless a problem is noted, a recovery period of 30 minutes is adequate in most instances. Assistance with initial standing and walking is prudent given the possibility of motor blockade secondary to local anesthetic.

Prior to discharge, and during the period between the time of onset and duration of the local anesthetic, the patient is evaluated as to any change in the preprocedure pain. Assessment must include provocative movements that elicited pain prior to the procedure. A neurologic examination to document neurological changes, such as numbness in the L5 dermatome or weakness in extensor hallucis longus, validates the procedure. If a local anesthetic was utilized and no reduction of pain realized, either a technical problem exists or the diagnosis must be reconsidered. This assessment must be included in the procedure note.

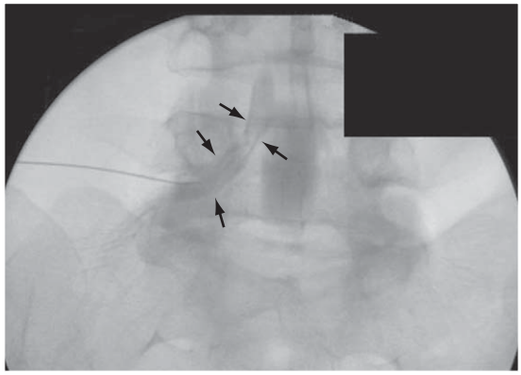


FIGURE 17–35

Intraneural injection, probably into epineurium, during L5 transforaminal injection with intrathecal and possible subdural spread of contrast. (Courtesy of Michael Hammer, MD.)

CLINICAL APPLICATION

The sole indication for lumbar transforaminal injection of corticosteroids is treatment of radicular pain. Patient response dictates whether repetition of the injection is justified. If no relief is noted in the immediate postprocedure period and local anesthetic of an appropriate concentration was utilized, the diagnosis must be questioned and possibly a different transforaminal level targeted at a future session or further evaluation considered. When greater than 70% pain relief is noted in the immediate postprocedure period, it can be assumed that this is in response to the local anesthetic effect on the pain generator. A positive corticosteroid response might then be considered if the patient were seen to benefit from the injection for days to weeks. Transforaminal injections should be repeated no more often than at 7-day intervals and limited to a maximum of 3 within a 6-month period. Patients responding to transforaminal corticosteroid injections require an average of approximately two injections (19–22).

SUMMARY

The literature indicates that nonfluoroscopically guided interlaminar, transflaval, epidural injection of corticosteroids is of little or no value in the treatment of "sciatica," that is, lumbar radicular pain (23,24). On the other hand, several papers have left little doubt that transforaminal injections provide long-term benefit in the same patient population (19–22). As with all spinal injections, there can be significant risks involved with transforaminal epidural injections. However, these risks are well managed if meticulous technique and due diligence are prac-

ticed by well-trained physicians. Today, transforaminal injections should always be considered the treatment of choice for lumbosacral radicular pain when conservative measures have failed and prior to surgical intervention. This procedure should no longer be thought of as "special" or exotic; rather, it must be considered as fundamental and within the armamentarium of all physicians who claim to practice standard of care for interventional pain management.

LUMBAR SPINAL NERVE BLOCK

HISTORY

Diagnostic lumbar spinal nerve blocks are used to evaluate the cause of sciatica in patients reporting lower extremity pain.[41–49] In these patients, the precise mechanism is not always clear and the MRI may not reveal the etiology since it provides only anatomic information.[42,48] Alternatively, nerve root damage seen on an MRI may not be the cause of the pain. To further elucidate the pain generator, diagnostic lumbar spinal nerve root block has been advocated.[41–49] The procedure involves anesthetizing the affected nerve root with a small amount of local anesthetic in order to determine the patient's response. If the pain is relieved, this supports the hypothesis that the suspected nerve is causative. If the pain persists despite successfully anesthetizing the targeted nerve, then the hypothesis is refuted.[50] MacNab et al.[45] first reported on the technique of selective nerve root injection in 1971. Since that time it has been used extensively as a physiological means of evaluating the etiology of sciatica.

Prior to the pervasive use of fluoroscopy, the procedure was performed by contacting the ventral ramus of the spinal nerve outside of the intervertebral foramen. Performance of this procedure involved contacting the nerve and resulting in a radicular dysthetic sensory complaint by the patient. Refinements in fluoroscopic techniques led to modification of the target to contact the spinal nerve where it lies within the intervertebral foramen.[50] The advantage of this approach is that it lessens the risk of needle trauma to the nerve root.

ANATOMY

There are five paired lumbar nerves that exit their respective foramina from the L1-L2 to the L5-S1 levels.[51] Rootlets come off the dorsal and ventral surface of the spinal cord to form the dorsal and ventral nerve roots.[51,52] These join to form the spinal nerve in the region of the intervertebral foramen. The spinal nerve is relatively short and immediately divides into anterior and posterior primary divisions.[51,52] Just as the orientation of the lumbar zygapophyseal joints differ from L1-2 to L5-S1, the lumbar nerves exit their respective foramina at different angles from L1 through L5.[53] At L1 the nerves exit downwards and forward at an acute angle, whereas at L5 the nerves exit somewhat horizontally and at a more obtuse angle (Figures 17-36).

Located in the upper aspect of the foramen is a quadrant known as the safe triangle.[50] A needle placed in this location will allow infiltration of the nerve without risk of injury to other structures including the exiting nerve root. The safe triangle has an imaginary base tangential to the pedicle, a side in line with the outer margin of the intervertebral foramen, and a hypotenuse formed by the spinal nerve in an AP view (Figures 17-1 and 17-2).

The anatomy of the arterial system is important when performing spinal injections because some of the vessels that supply the spinal nerve roots also anastomase with the anterior spinal artery (Figure 17-37).[52] Injection into the medulary artery has been discussed previously. Injection of particulate corticosteroid preparations appears to be the cause of the severe neurological complications seen.[49,54,55] However, corticosteroid use is not indicated in this purely diagnostic procedure. In a single case report, injection into a cervical medulary artery has been postulated as the cause of temporary paralysis secondary to local anesthetic effect on this cord.[56]

INDICATIONS

Conservative treatment options should be tried and have failed to produce a benefit before considering this procedure, although no conservative treatment has been proven to provide definitive benefit.[47,50] Patients should not be considered for this procedure until 6 weeks have passed

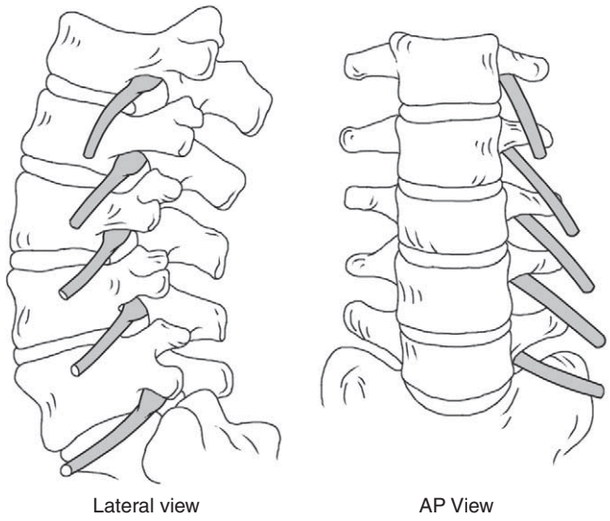


FIGURE 17–36
Direction of the lumbar nerve roots as they exit the foramen.

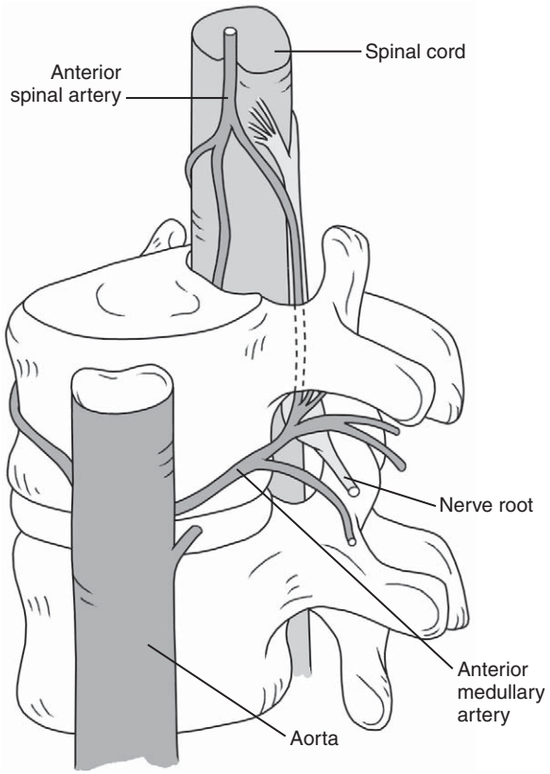


FIGURE 17–37
Anterior medullary artery.

since the onset of symptoms. The majority of patients will have healed during this time. Those who have not may require a more invasive approach.

The indication for diagnostic lumbar or sacral spinal nerve block is to investigate the cause of radicular symptoms in the following patients:

Imaging studies implicate more than one nerve as a possible cause of the symptoms.
Imaging studies are difficult to interpret due to previous surgery.
Clinical features do not suggest a specific spinal segment.
Clinical symptoms suggest radicular involvement, but the MRI appears "normal."[41,42,48,50,52]

Contraindications, equipment, and pre-procedure testing have been discussed previously.

DRUGS

- Water-soluble, nonionic contrast such as Isovue or Omnipaque 200 or 240
- Preservative-free local anesthetic such as 2 or 4% lidocaine or 0.5 or 0.75% bupivacaine
- 1% lidocaine to anesthetize a skin wheal

PREPARATION OF PATIENT

Informed consent should be obtained prior to the procedure.

History

The patient will commonly complain of pain, numbness, tingling, or paresthesia confined to one or two dermatomes. Weakness may also be noted. The pain is most often felt in the lower extremity if the lower lumbar or upper sacral nerve roots are involved. The pain travels in a narrow band and is burning, shooting, or lancinating in nature. The pain is more often below the knee and above it, and it is felt both deep and superficially in the involved extremity.[41]

Physical Exam

The following exam findings may be seen alone or in combination:

- Dural tension signs (positive straight leg raise, femoral stretch, etc.)
- Weakness in the involved muscle groups
- Numbness or hypoesthesia to touch or noxious stimuli
- Decreased reflexes

Imaging and Neurodiagnostic Studies

- MRI or CT should be done in all cases of suspected radicular pain or radiculopathy.
- EMG can be helpful to differentiate radicular from peripheral neuropathy.

Preprocedure Medication

As discussed previously, sedation is rarely medically indicated or required. In that selective spinal nerve blocks are utillized to obtain diagnostic information by reduction of pain immediately post-procedure, no analgesics should be administered pre-, peri-, or post-procedure.

PROCEDURE

Patient Positioning

See previous discussion.

Technique

The target for a lumbar selective nerve block is above the nerve with the needle tip located at the six o'clock position relative to the pedicle when seen in an AP view.[50] The procedure is performed by first squaring the inferior vertebral end plate and then rotating the image intensifier into an oblique position toward the affected side until the target point is not obstructed by the superior articular process, lamina, or transverse process (Figure 17-38). This

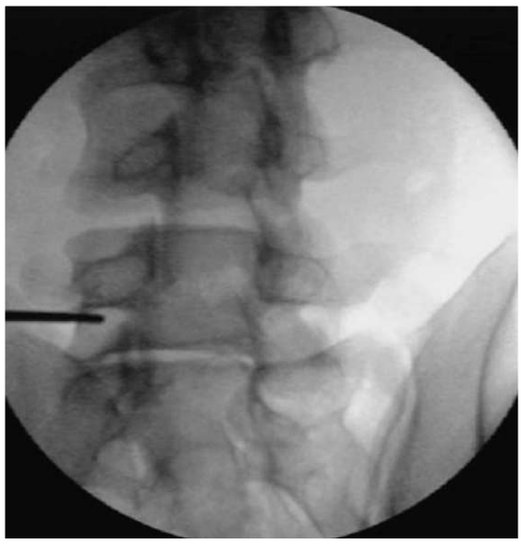


FIGURE 17–38

This image shows the starting position for the needle on the skin. Note the location of the pedicle just inside the border of the vertebral body. Also note the location of the superior articular process approximately one third of the way across the end plate of the vertebral body. This serves as a visual guide for the correct amount of obliquity.

usually requires that the C-arm is obliqued until the pedicle is seen at or just inside the shadow of the vertebral body (about 20–25 degrees) (Figure 17-39).

The skin is anesthetized, and the needle is advanced down the x-ray beam toward the superior aspect of the foramen. As the needle is advanced, rotating into an AP projection and visualizing the extent of medial placement helps to determine needle depth. If further insertion is required, rotate back to an oblique view 6 o'clock and advance. Continue to advance and check depth until the needle tip is located at the 6 o'clock was previously used position relative to the pedicle (Figure 17-40). A lateral view is then checked (Figure 17-41). The needle tip should appear in the superior aspect of the foramen. Ideally, the tip should appear in the middle of the foramen in the anterior posterior or parasagittal plane. This way, the tip is located slightly dorsal to the location of the anterior medullary artery as it enters the foramen.

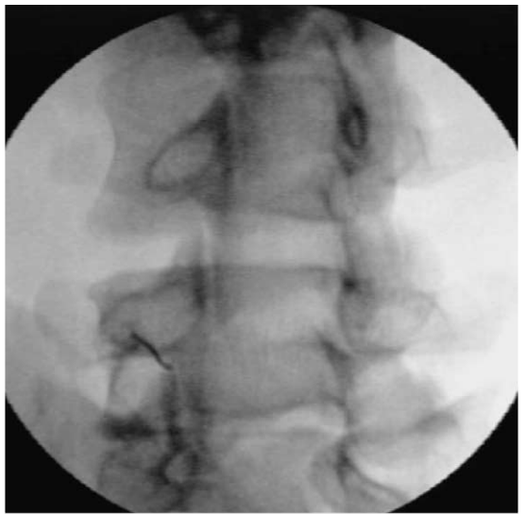


FIGURE 17–39

This shows the final needle position in the safe triangle.

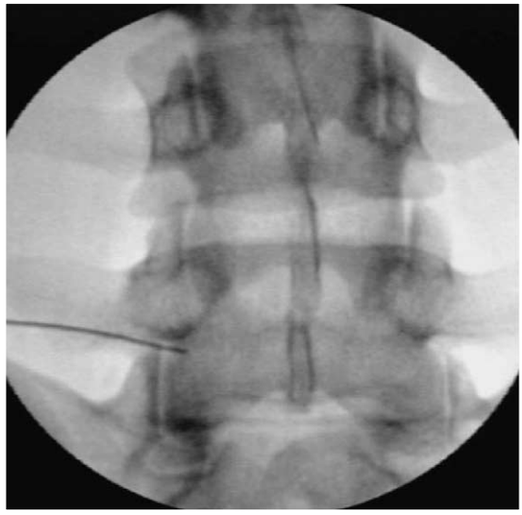


FIGURE 17–40

This is an anteroposterior view with the needle tip located at the 6 o'clock position.

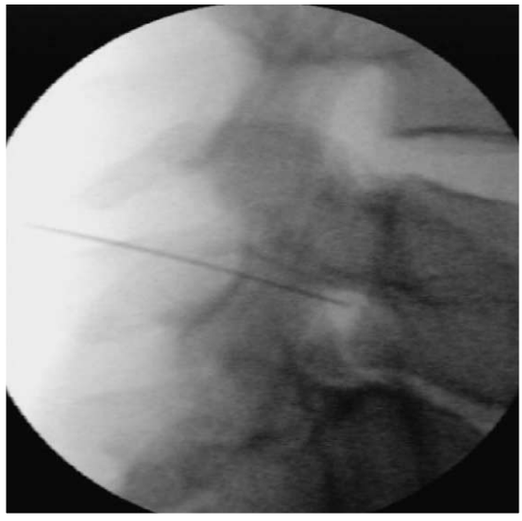


FIGURE 17–41

This is a lateral view showing the needle tip slightly posterior to the vertebral body. This avoids contact with the anterior medullary artery as it enters the foramen.

Confirm needle placement by injection of a small amount (<0.5 ml) of contrast medium under live fluoroscopy. The usual volume should not exceed 0.5 ml or the injection may lose specificity for a single nerve. A short IV extension can be attached to the needle hub for this purpose. The injectionist should be watching for a wisp of dye spreading into the central canal (indicating uptake by an anterior medullary artery) while the x-ray is on (Figure 17-42). The dye pattern should outline the spinal nerve root (Figure 17-43). The dye should not spread distal to the edge of the vertebral body or more proximal than the superior aspect of the pedicle (Figure 17-44). This pattern ensures that the injection is specific to the involved spinal nerve root (Figures 17-45 to 17-50). The injection should be terminated if dye is seen spreading into the central canal or into the intrathecal space to prevent serious complications. The needle should be repositioned if venous uptake is seen, since this finding indicates that the injectate instillation is intravascular rather than into the desired foraminal location and would interfere with the diagnostic utility.[57–59]

The volume of local anesthetic injected should correspond to the volume of dye used. For example, if 0.3 ml was seen outlining the nerve, then no more than 0.3 ml of local anesthetic should be injected. The most commonly used anesthetics are 0.5 or 0.75% bupivacaine or 2% or 4% lidocaine. Assuming that the injected local anesthetic contacts the targeted nerve, it should relieve symptoms if the nerve is responsible for production of the patient's symptoms. This can be ensured by observing that contrast dye outlines the targeted nerve. To ensure that the anesthetic does not spread to adjacent structures, a low volume (less than 0.5 ml) should be used. In a study using CT scanning to analyze the percent of patients showing spread of dye into the lumbar plexus, the group who received the lowest volume of dye (0.5 ml) had the fewest number of patients who exhibited spread to the lumbar plexus compared to with those who received more (1–2 ml).[49]

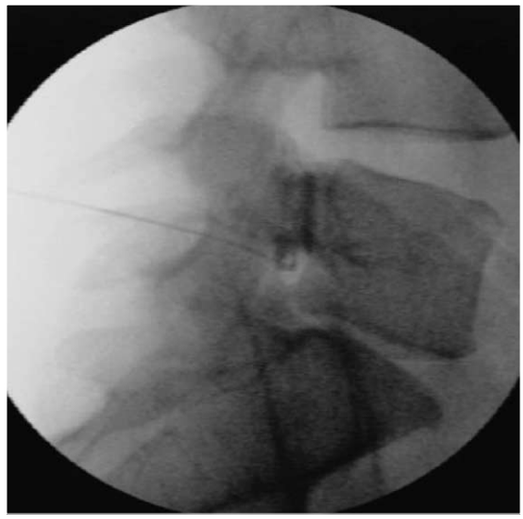


FIGURE 17–43
This is the lateral view after dye injection showing dye spreading within the epidural space but not extending to the foramen above.

COMPLICATIONS

Bleeding and infection are commonly listed but rare complications. Mechanical nerve root damage can occur if the needle inadvertently contacts the nerve. Starting in

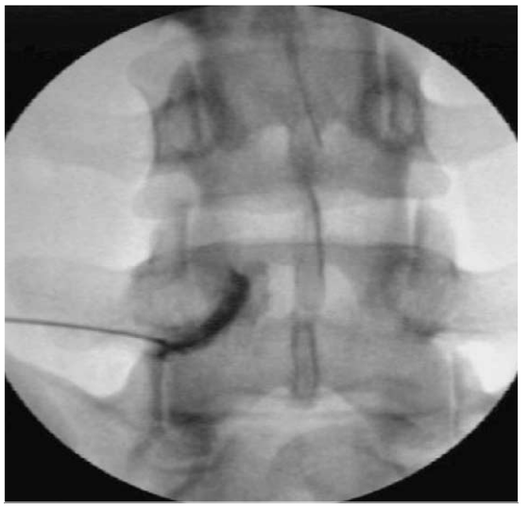


FIGURE 17–42
This shows dye outlining the nerve, but not extending laterally beyond the pedicle or superiorly beyond the border of the vertebral body.

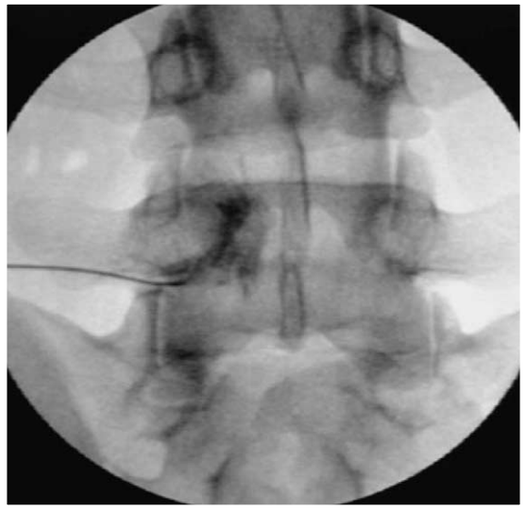


FIGURE 17–44
This shows postinjection dye spread. Again, note that the dye does not extend beyond the image of the pedicle superiorly.

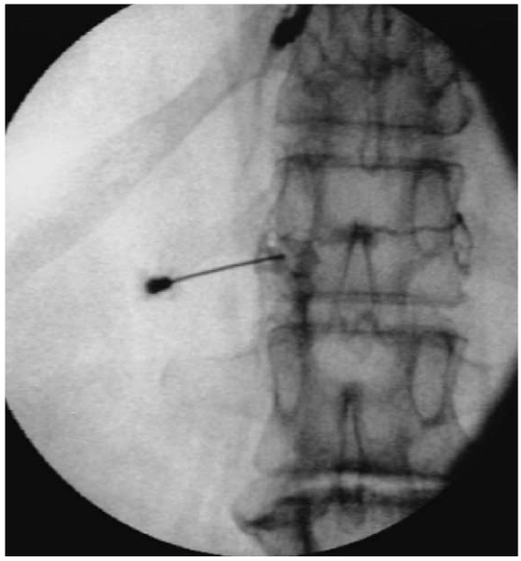


FIGURE 17–45
This shows dye filling veins within the epidural space. Injection here could not be considered diagnostic..

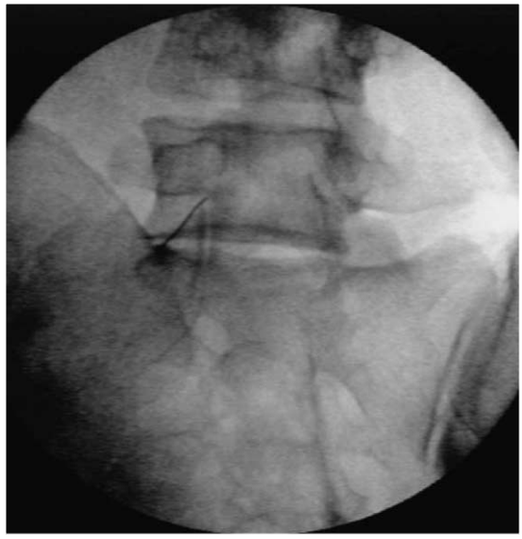


FIGURE 17–47
This shows a needle being advanced down to the superior articular process that is rather large and is obstructing passage of the needle.

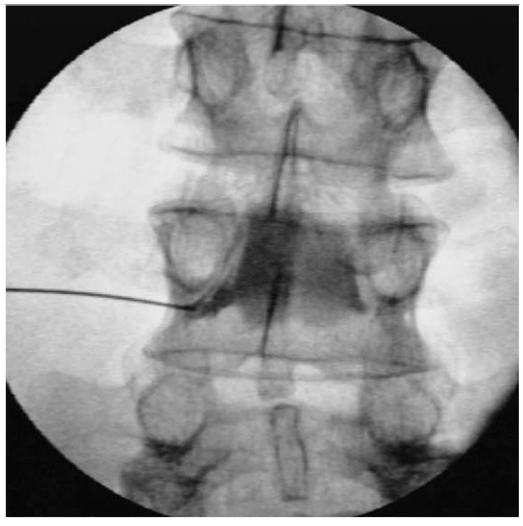


FIGURE 17–46
This shows dye filling the dural root sleeve and extending into the intrathecal space. Note the smooth appearance of the dye and the fact that it appears on both sides of the spine.

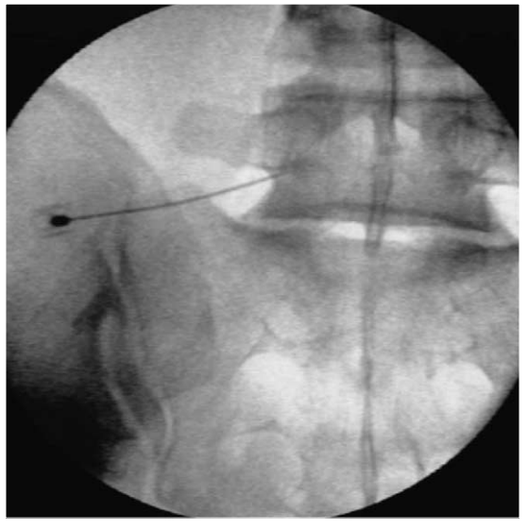


FIGURE 17–48
This shows an anteroposterior view prior to dye injection.

too oblique a position can result in intrathecal placement of local anesthetic, resulting in temporary lower extremity paralysis. Intrathecal placement may also occur if the needle is advanced beyond the 6 o'clock position in a patient with long dural root sleeves. Another uncommonly encountered complication is placement of the needle tip into the facet joint in those patients with a large superior articular process. Minor complications include facial flushing, nonpositional headache, leg pain, vasovagal reaction, back pain, and intraoperative hypertension.[49] Complications arising from injection of the medulary artery have been discussed previously, but have never been noted with injection of local anesthesia alone.

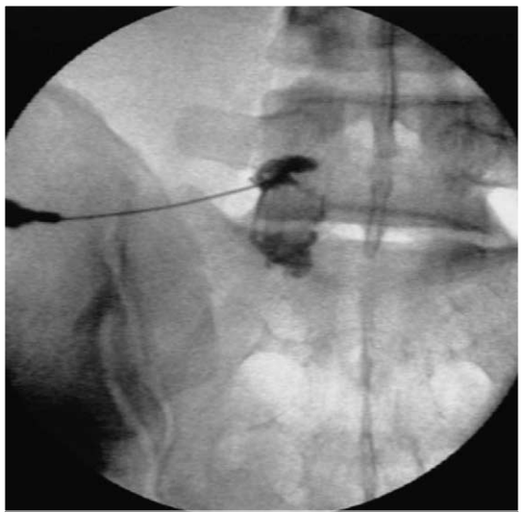


FIGURE 17–49
This shows an anteroposterior view of the dye outlining the facet joint.

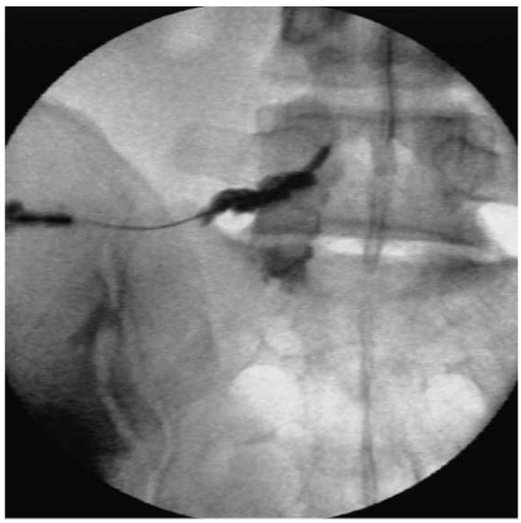


FIGURE 17–50
This is an anteroposterior view of the dye injection after slightly advancing the needle outlining the facet joint and nerve root. This can be avoided by angling the C-arm more steeply (to square the inferior end plate). This would throw the superior articular process inferiorly enough to allow passage of the needle.

EFFICACY

In a study examining the positive predictive value of the procedure, 62 patients who had undergone lumbar spinal nerve root block were explored at surgery.[42] Eighty-five percent of patients whose pain was relieved by the procedure showed pathology at surgery. The ISIS guidelines state that this implies that the false-positive rate of lumbar nerve blocks is low and, therefore, the specificity is high. In those patients with negative response to lumbar nerve block who underwent surgery, pathology that was found involved multiple levels or anomalous nerve roots. The ISIS guidelines report on another study measuring sensitivity by performing spinal nerve blocks in 46 patients with clinical and radiological evidence of nerve root compression subsequently confirmed at surgery.[50] The sensitivity was reported at 100%, with 95% confidence intervals of 88–100%. The same study estimated specificity by performing blocks in 23 patients at asymptomatic nerve levels. No false-positive responses were noted, and the authors concluded that the specificity was approximately 90%.

LUMBAR DISCOGRAPHY

There is no doubt that the lumbar intervertebral disc can hurt and has the necessary innervation to be a clinically significant source of pain.[60,61] Discitis provokes excruciatingly intense pain, and probing disc protrusions during awake surgery is painful.[62] What is controversial is not whether the disc is a source of pain, but whether disc pain can be reliably diagnosed.

Regardless, discography is the only available means for diagnosing lumbar discogenic pain. Because discography is a provocational test requiring reproduction of the patient's pain by stressing the disc with an injection of contrast medium, the response is dependent on the intensity of the provocation stimulus. In addition, the response is subjective and therefore there may be confounding factors other than the intensity of the stimulus. Since its introduction in 1948,[63] discography has been mostly evaluated without stipulating or requiring how strongly the disc is stimulated and often without requirements of the intensity of required provoked pain. Taxonomically unsound, emerging standards require unambiguous operational criteria that establish a threshold intensity for both pain response and stimulation intensity. Both require a precise method to apply the stimulus and strict criteria for interpretation.

PATIENT SELECTION

Discography was first used to diagnose protrusions in preparation for surgical interventions in patients with radicular pain.[63,64] Prior to the introduction of CT in the 1980s, and later MRI, plain film x-ray and myelography were the only imaging studies available to assess pathology in the spine. Since myelography could only evaluate the thecal sac, dural root sleeves, and structures within the dural sac, lateral protrusions could not be visualized. Since Lindblom first advocated the use of discography to diagnose disc protrusions, modern imaging techniques have made this indication obsolete and interesting only from a historical perspective.

Discography is not an initial screening examination. Disc stimulation follows failed conservative treatment modalities and is only used when other less invasive diagnostic tests are inconclusive. Discography is highly invasive, and irreversible surgical procedures may be chosen based on the results.

Indications

The primary purpose of discography is to examine the intervertebral disc by mechanical stimulation to help determine whether or not the disc is painful and to evaluate the extent of internal annular or end-plate disruption. Inclusion criteria include the following:

- Failed conservative treatment for low back pain of probable spinal origin.
- Pain has been ongoing for more than 3 months.
- Other pain generators have been ruled out.
- Symptoms are clinically consistent with disc pain.
- Symptoms are severe enough to consider surgery or percutaneous interventions.
- Surgery is planned, and the surgeon desires an assessment of the adjacent disc levels.
- The patient is capable of understanding the nature of the technique and can participate in the subjective interpretation.
- The patient needs to know of the source of his or her pain.

Contraindications

Contraindications are summarized below.

- Patient is unable or unwilling to consent to the procedure.
- Inability to assess patient response during the procedure.
- Inability of patient to cooperate.
- Known localized or systemic infection.
- Pregnancy.
- Anticoagulants or bleeding diathesis.

Relative contraindications to discography follow:

- Allergy to contrast medium, antibiotics, or local anesthetics.
- Significant psychological overlay.
- Any other condition, medical, anatomical or psychological, that would increase the risk of the performance of the examination to unsafe levels.

PROCEDURE CONSIDERATIONS

A medical history is taken and a physical examination is performed to ensure the discographer that there are no contraindications and the patient is an appropriate candidate for the procedure. If intravenous sedation is to be utilized, NPO (no oral intake) status is verified according to institutional guidelines. In females of childbearing age, pregnancy must be ruled out.

If the patient has a history of allergies to nonionic water-soluble contrast media iohexol or iopamidol, or other drugs, the risks versus benefits of the procedure must be weighed and discussed with the patient. In the case of iodine allergies, one can pretreat patients with corticosteroids and H1 and H2 blockers prior to the procedure. If the risk of allergic reaction to contrast is significant, saline instead of contrast can be used, or add a very small amount of gadolinium to the saline and obtain an MRI directly after the procedure.[65,66]

Informed consent should include discussion of the purpose of the procedure, risks, complications, and alternative diagnostic tests. Patients should be told that the procedure is potentially painful, and during the stimulation of the disc, a description of this discomfort will be required in regards to concordance and intensity as compared with their ongoing complaint.

Intravenous access is standard. Because disc space infection is the most common complication, prophylactic antibiotic (cefazolin 1 g, gentamicin 80 mg, clindamycin 900 mg, or ciprofloxacin 400 mg) is administered intravenously within 30 minutes of needle insertion. Aminoglycosides are not needed for postprocedural prophylaxis.[67] In sheep studies, Fraser et al.[68] noted antibiotic levels in the annulus 30 minutes following intravenous administration, but none were demonstrated at 60 minutes. In addition to intravenous antibiotics, many discographers mix between 1 and 6 mg per milliliter of cefazolin or an equivalent dose of another antibiotic with the contrast injected into the disc.[69–72] Klessig et al.[72] note that cefazolin and gentamicin 1 mg/cc, and clindamycin 7.5 mg/cc, exceed the minimum inhibitory concentrations (MICs) for the three most common organisms implicated in discitis, *Escherichia coli*, *Staphylococcus aureus*, and *Staphylococcus epidermidis.*

Intravenous sedation will increase patient compliancy during the procedure. Medications are titrated according to the patient's response to avoid oversedation during the testing phase. Intravenous midazolam provides effective sedation during discography in doses between 2.0 and 5.0 mg, but often causes amnesia, which may or may not be a desired consequence. The ultra–short-acting hypnotic, propofol, is used by many injectionists who have an anesthesia background. Propofol produces rapid sedation and amnesia during the needle insertion, but due to the short half-life, the patient can be awake when the discs are stimulated. Patients should be fully monitored and personnel competent in airway management and resuscitation should be present during the procedure. General, epidural, or spinal anesthesia is inappropriate.

How much and which drug to use for preoperative sedation varies depending on the discographer's convictions. Some discographers feel that opioids[73–75] should not be utilized prior to or during discography,

unless the patient is taking chronic long-acting medications. Their reasoning maintains that since discography is a provocational test, pain intensity needs to be compared and quantified in relation to the patient's usual pain intensity, and opioid analgesics could attenuate a pain response and cause a higher rate of false negatives. On the other hand, others[76] argue that giving a small dose of analgesics (meperidine 50 mg, fentanyl 50 mcg, or morphine 5 mg) prior to the procedure will help decrease the rate of false positives in patients with clinically insignificant discogenic pain. Most discographers do, however, agree that patients who are taking narcotics regularly and have been NPO 6 hours prior to the procedure will have an exaggerated pain response if they are undergoing early narcotic withdrawal.

Discography can be performed in any procedure room appropriate for aseptic procedures. Safety concerns require imaging equipment that provides good visualization of the relevant spinal anatomy. One must be able to view the spine in AP, lateral, and oblique projections. Although bi-plane fluoroscopy can be utilized, most discographers use C-arm fluoroscopic units that allow the discographer to obtain fluoroscopic views without repositioning the patient. Most also use a radiolucent procedure table that can be raised and lowered as needed. Monitoring equipment should include pulse oximetry, noninvasive blood pressure, and EKG. Oxygen, airway supplies, emergency drugs, suction, and other resuscitation equipment and supplies should be immediately available. There should be adequate personnel to monitor the patient and operate the fluoroscope.

Sterile technique requires preparation of the skin and draping analogous to that used for surgery. Povidone-iodine 10% (Betadine solution), and/or DuraPrep (iodophor 0.7% and isopropyl alcohol 74%) is the preparation of choice. If the patient indicates allergies to the above, chlorhexidine and alcohol can be substituted safely. Standard draping is utilized to provide a sterile field and may include the use of sterile towels and fenestrated drapes as per the injectionist's preference. The procedure room staff should be dressed in clean clothes (scrub suits). Masks and surgical caps are mandated by anyone coming in close proximity to the sterile field. Many injectionists scrub, gown, and glove as for an open surgical procedure. The C-arm image intensifier should also be draped.

Although the history and physical examination can be used to select levels, most discographers select levels based on the appearance of the MRI T2-weighted images. Most will include any disc that has a decreased signal intensity on the T2-weighted image and will often include adjacent less degenerated disc as a control. Rarely is it necessary to inject more than four segments. When injecting, the patient should be blind as to the onset and level of stimulation.

TECHNIQUE

Prior to the late 1960s, disc puncture was performed using a posterior interpedicular, or transdural, approach. This technique is seldom utilized today because it requires puncture of the dura. A lateral, or extrapedicular, approach is now used,[77,78] except in rare situations where anatomical variation or postsurgical changes prevent disc access using the lateral approach.

Although some physicians perform discography with the patient in a lateral position, most position the patient in a prone position with a bolster placed under the upper abdomen to slightly flex the spine and decrease the normal lumbar lordotic curve. Monitoring and light sedation are initiated. The lower thoracic, lumbar and upper sacral and gluteus regions are prepped and draped as discussed earlier.

The target disc is identified using an AP view (Figure 17-51). The image intensifier of the C-arm is then tilted in a cephalad-caudad direction until the subchondral end plate of the vertebral body, caudad to the target disc, is parallel to the x-ray beam. The subchondral plate will be seen as a line rather than an oval. To ensure against the patient mistaking the discomfort from needle placement for provoked pain secondary to disc stimulation, the disc is preferentially approached from the opposite side of the patient's usual pain. When the patient's pain is central, bilaterally equal, or there are anatomical variations that prevent disc puncture from

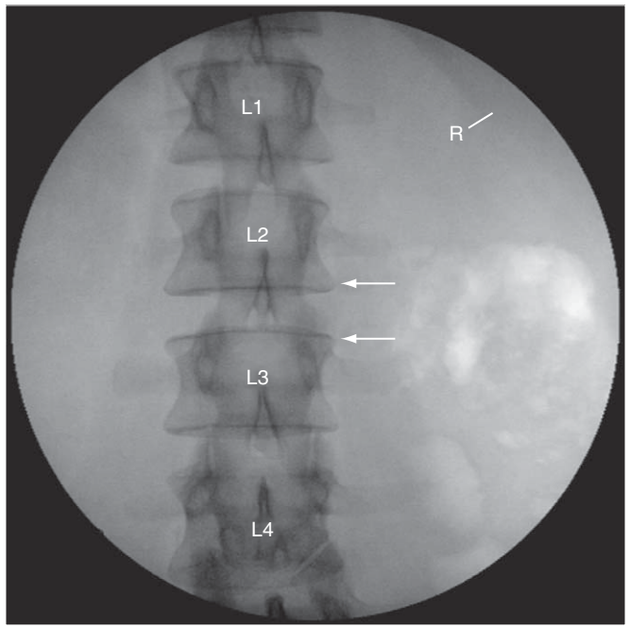


FIGURE 17–51

Anteroposterior view of lumbar spine. Arrows indicate end plates parallel to x-ray beam. R, 12th rib.

the contralateral side of the pain, needle insertion from either side is appropriate.

After squaring the end plate, the C-arm is rotated to an oblique view until the tip of the superior articular process (SAP) of the level below appears to lie under the approximate midpoint of the subchondral plate of the inferior end plate of the disc above (Figures 17-52 and 17-54). This positioning of the fluoroscope allows needles to be passed using "tunnel vision" (i.e., parallel to the beam when the skin puncture site is aligned with the target structure) just lateral to the SAP (Figures 17-53 and 17-59). The needle will travel under the segmental nerve, which courses medial to lateral, and dorsal to ventral, and will puncture the annulus fibrosis of the disc at the midpoint of the disc when seen in lateral and AP views (Figures 17-56 and 17-57).

Once the oblique view as described earlier is obtained, the skin is marked overlying the target (see Figure 17-52 and Figure 17-54). A skin wheal is made using a 25-gauge, 1.5-inch needle with lidocaine 1% (~1 cc). A 25- or 22-gauge, 3.5-inch needle is then advanced, using "tunnel vision," that is, parallel to the x-ray beam, to the level of the SAP, and lidocaine (~4–5 cc) is injected while withdrawing the needle, creating an anesthetized track (see Figure 17-55). One should be careful not to anesthetize the dorsal root ganglion within the foramen. Besides obscuring nerve root impalement, the sinu vertebral and ramus communicans nerves will partially anesthetize the disc.

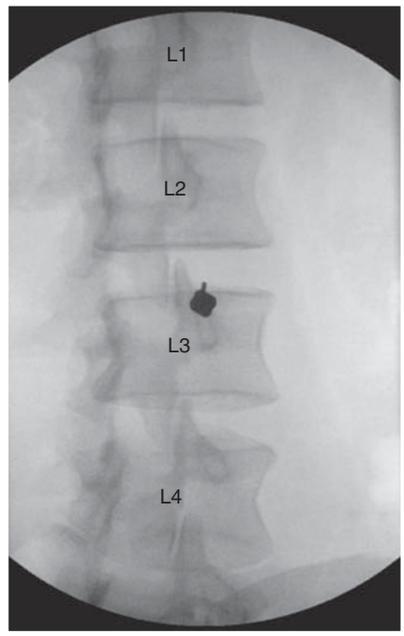


FIGURE 17–53
Right oblique view with introducer needle in place at L2-L3.

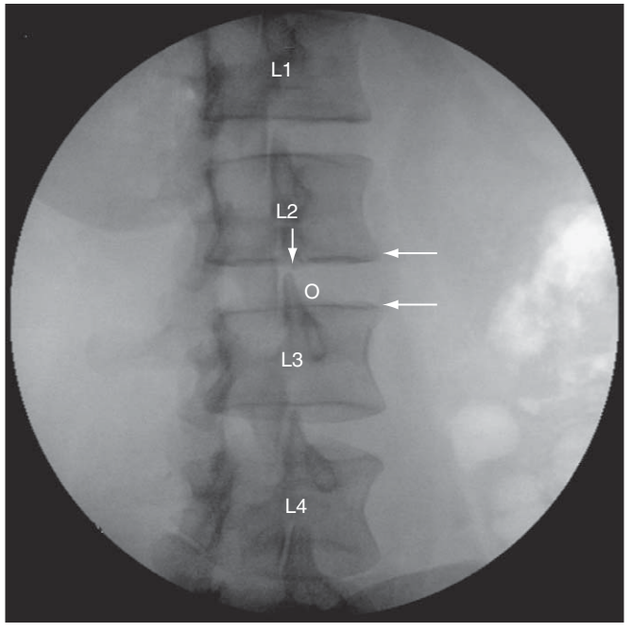


FIGURE 17–52
Right oblique view. Tip of the superior articular process (SAP) of L3 appears to lie under the approximate midpoint of the inferior end-plate of the L2 vertebral body (black arrow). Open circle represents target.

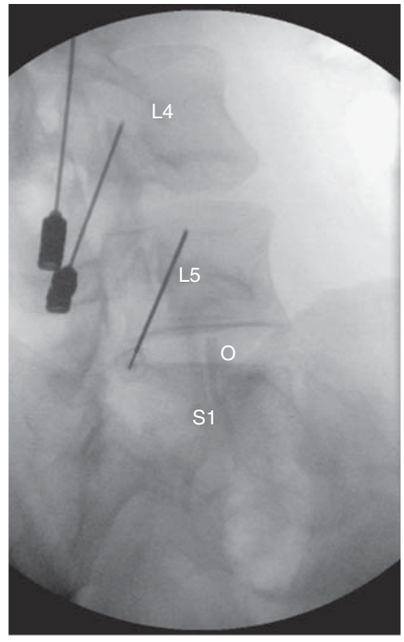


FIGURE 17–54
Right oblique view with end plates of L5-S1 parallel to beam. Superior articular process of S1 positioned as closely as possible to the midpoint of the inferior end plate of L5. Open circle indicates target. Introducer needles in place at L2-3, L3-4, and L4-5. Note differing angles needed to access each disc.

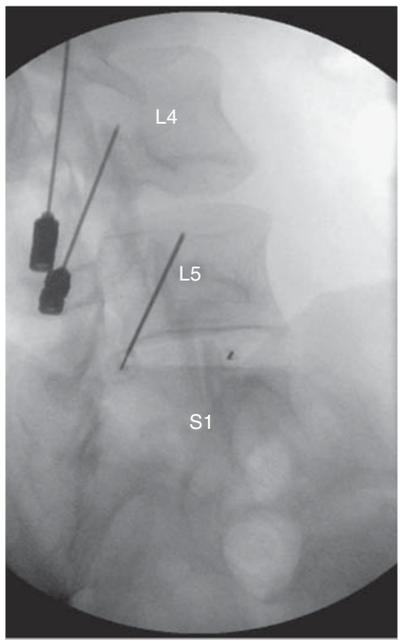


FIGURE 17–55

Right oblique view with end plates of L5-S1 parallel to beam. 25-gauge 3-1/2-inch spinal needle in place, parallel to beam, to anesthetize a track of tissue down to superior articular process level.

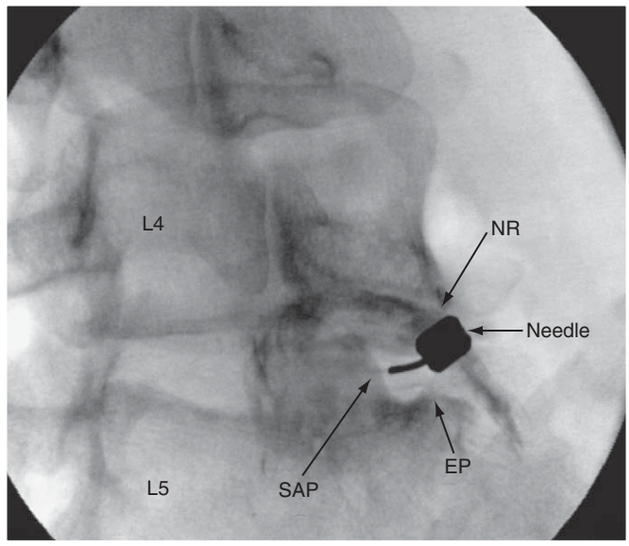


FIGURE 17–57

Needle in place at L4-L5 intervertebral disc following transforaminal injection of contrast. Note needle medial and caudal to segmental nerve. EP, superior end plate of L5; NR-L4, ventral ramus; SAP, superior articular process.

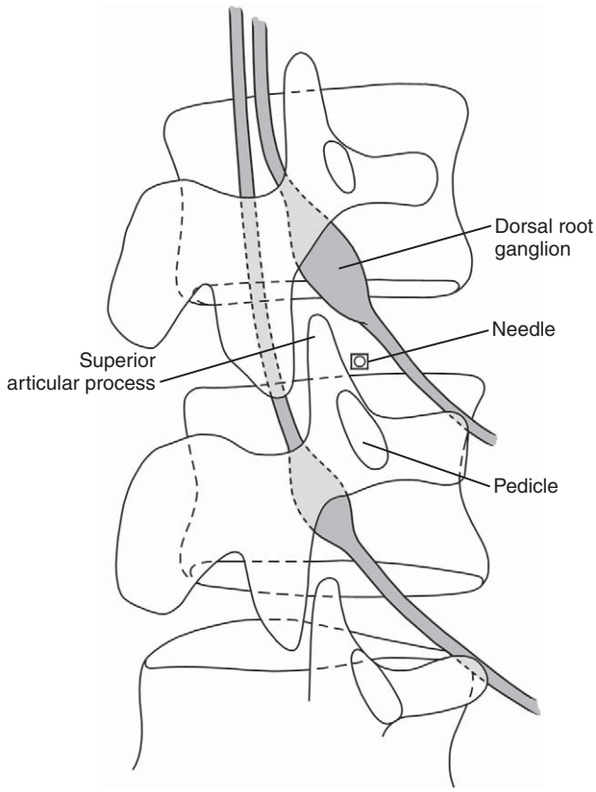


FIGURE 17–56

Course of segmental nerve running medial to lateral in close proximity to target. o, needle in position.

A one- or two-needle technique may be used. Prior to the routine use of prophylactic antibiotics, Fraser et al.[79] reported a rate of discitis with single nonstyletted needles of 2.7% versus 0.7% when a double-needle technique with stylettes was employed. Using a single styletted needle technique, Aprill[70] has reported one case of discitis in approximately 2000 patients (~0.05% per patient); however, both the North American Spine Society[80] and the International Spinal Injection Society[76] recommend a two-needle approach.

The two-needle technique utilizes a shorter, larger-gauge introducer needle through which a longer, smaller-gauge needle is advanced past the tip of the introducer needle and into the targeted intervertebral disc. The introducer needles are 18- or 22-gauge, 3-1/2 or 5 inches, while the complementary disc puncture needles are 22- or 25-gauge and 6 or 8 inches. The body habitus of the patient often dictates the combination of needles used at each level. Both the introducer and disc puncture needles should be styletted to prevent skin from being picked up and introduced into the disc. Many advocate a slight bend, opposite the bevel, placed at the tip of the disc puncture needle to enable the operator to control the course of (i.e., "steer") the needle during advancement.[81–84] At times, a larger curve on the distal third of the disc puncture needle must be utilized to compensate for less than ideal anatomy or postsurgical change (Figure 17-58). The introducer needle is passed through the skin wheal at the skin puncture point, using a down-the-beam, "tunnel-vision" technique toward the disc entry site (see Figures 17-53 and 17-69). Forward advancement is stopped at the

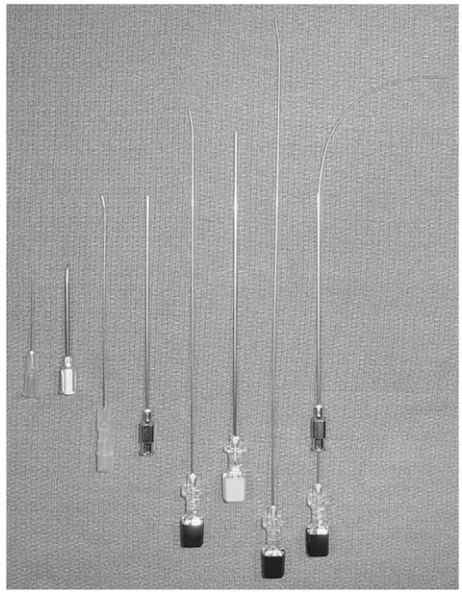


FIGURE 17–58

Needles utilized for discography. From left to right: 25-gauge 1-1/2-inch needle for skin anesthesia; 15-gauge, 1-1/2-inch needle for skin puncture; 25-gauge 3-1/2-inch for deeper anesthesia; 18-gauge 3-1/2-inch introducer; 22-gauge, 6-inch disc puncture with bend at tip; 18-gauge, 5-inch introducer; 22-gauge, 8-inch disc puncture with bend at tip; 22-gauge, 8-inch, curved disc puncture needle with marked curve through 3-1/2-inch introducer needle.

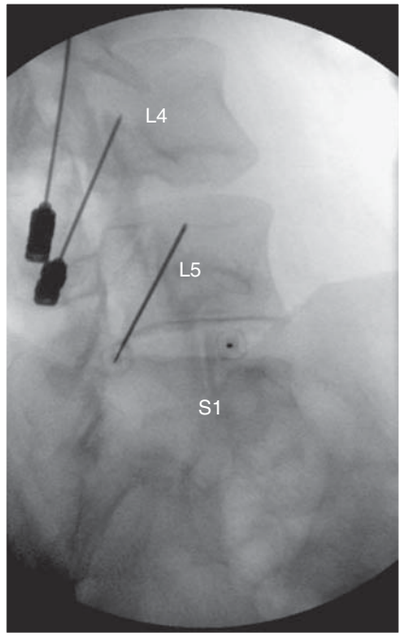


FIGURE 17–59

Right oblique view. Introducer needles in place at L5-S1, L4-L5, L3-L4, and L2-L3. Note that correct placement of each introducer needle requires a different angle of entry.

approximate level of the SAP, although placement within the foramen is acceptable. A lateral view with the fluoroscope is used to check needle depth (Figure 17-60). An AP view will indicate the needle tip as lying at the lateral extent of the intervertebral disc (Figure 17-61). The stylette is removed from the introducer, and the longer, smaller-gauge disc puncture needle is advanced slowly

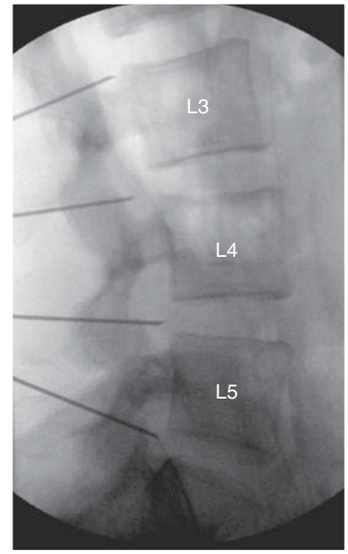


FIGURE 17–60

Lateral view. All introducer needles in place, at or just ventral to the posterior elements.

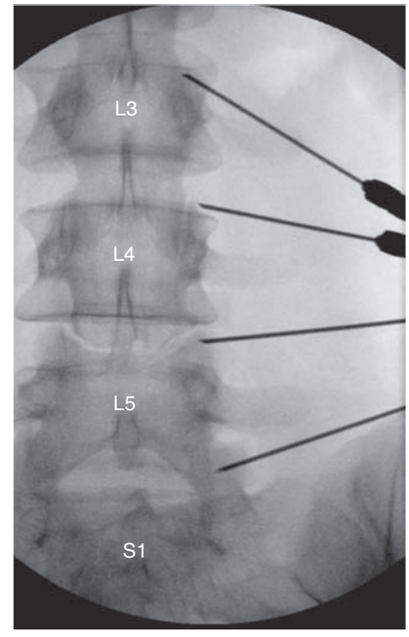


FIGURE 17–61

Anteroposterior view. All introducer needles in place in close proximity to the lateral aspect of each intervertebral disc.

under active lateral fluoroscopy. The needle will be seen to transverse the intervertebral foramen, and firm resistance will be noted as the needle touches and enters the annulus fibrosis.

Because the ventral ramus crosses the posterior-lateral aspect of the disc in close proximity to the disc entry site, if at any point during advancement of the needles radicular type dysesthesia is noted by the patient, insertion of the needle is stopped, the needle is partially withdrawn, and its course is altered and redirected toward the disc. A slight bend of the tip on the disc puncture needle facilitates this change of direction (see Figure 17-58). If more aggressive direction changes are required, the introducer needle can be withdrawn and redirected as well. Often redirection of the needle in a more caudal medial direction will allow insertion of the needle under the segmental nerve.

After the annulus is contacted, using active lateral fluoroscopy, the needle should be advanced into the center of the disc, that is, into the nucleus pulposus. As the outer third of the annulus is abundantly supplied with nerve endings, some axial discomfort, with referral into the thigh or buttock, is often felt by the patient. AP and lateral projections are used to ensure good needle placement, and spot films are saved for documentation prior to injection of contrast (Figures 17-62 and 17-63).

Although the above technique can be utilized for disc puncture in more than 95% of lumbar disc levels, occasionally, due to anatomical variations (i.e., overriding iliac crest, osteophytes), or postsurgical changes (i.e., posterior intertransverse fusion mass or fusion hardware), variations in the procedure must be utilized.

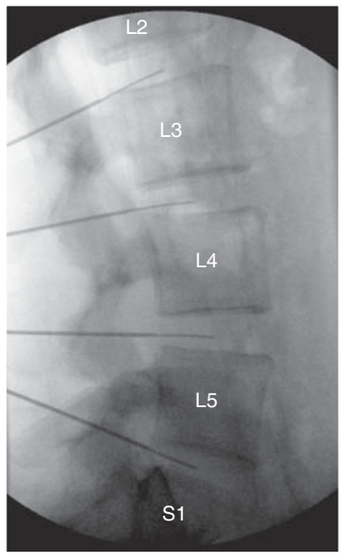


FIGURE 17–63

Lateral view. Disc puncture needles have been advanced under active lateral fluoroscopy into the center, nucleus pulposus, of each intervertebral disc.

A detailed description of the myriad modifications with which a discographer might be faced is beyond the scope of this forum; however, most involve either a more lateral or more medial needle insertion with the disc-puncture needle bent or curved to varying degrees (Figures 17-64, 17-65, and 17-66).

Rarely, the posterior interpedicular, transdural approach must be utilized to gain access to the disc

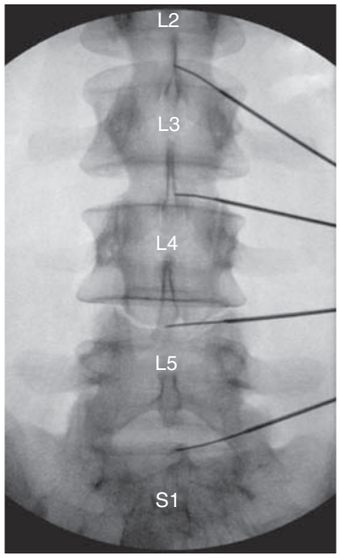


FIGURE 17–62

Anteroposterior view. Disc puncture needles verified as lying in the center, nucleus pulposus, of each intervertebral disc.

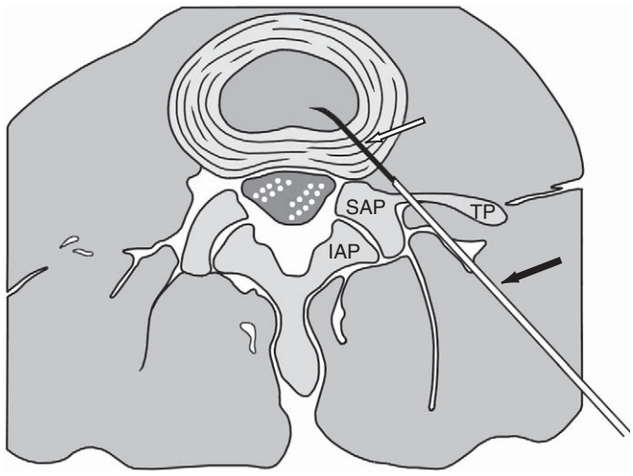


FIGURE 17–64

Straight approach to disc access. Small bend in distal 1 cm of disc puncture needle to aid in needle control. closed arrow, introducer needle; inferior articular process; open arrow, disc puncture needle with bent tip; SAP, superior articular process; TP, transverse process.

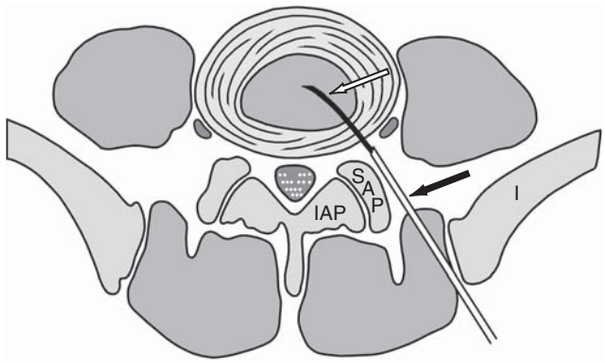


FIGURE 17–65

Moderate bend in needle utilized to gain access to disc nucleus. Closed arrow, introducer needle; I, ilium; IAP, inferior articular process; open arrow, slightly curved disc puncture needle; SAP, superior articular process.

(Figure 17-67). This approach increases the chance for morbidity since the dura is punctured twice. Risks and benefits of this technique must be weighed. At levels above the L3-4 intervertebral disc, the posterior approach should not be utilized since the chance of impaling the spinal cord is high.

Once all needles are positioned within the nuclei pulposi of the discs to be stimulated, injection can proceed. The patient should be blinded as to disc level and the initiation of the injection. At this point, the patient must be conversant enough to describe any sensations produced by the disc stimulation.

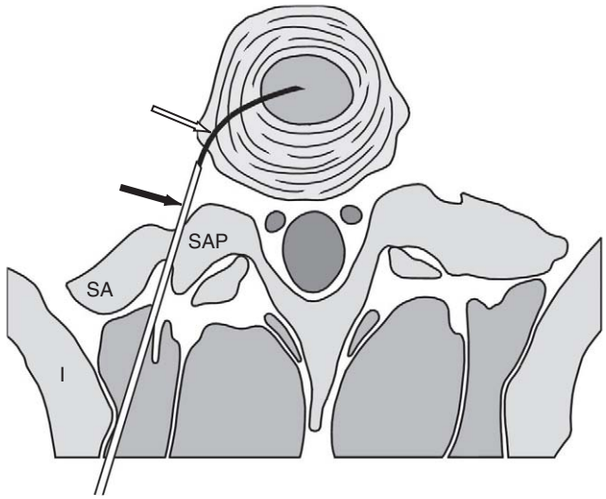


FIGURE 17–66

Significant curve on needle required to gain access to nucleus. Closed arrow, introducer needle; I, ilium; open arrow, curved disc puncture needle; SA, sacral ala; SAP, superior articular process.

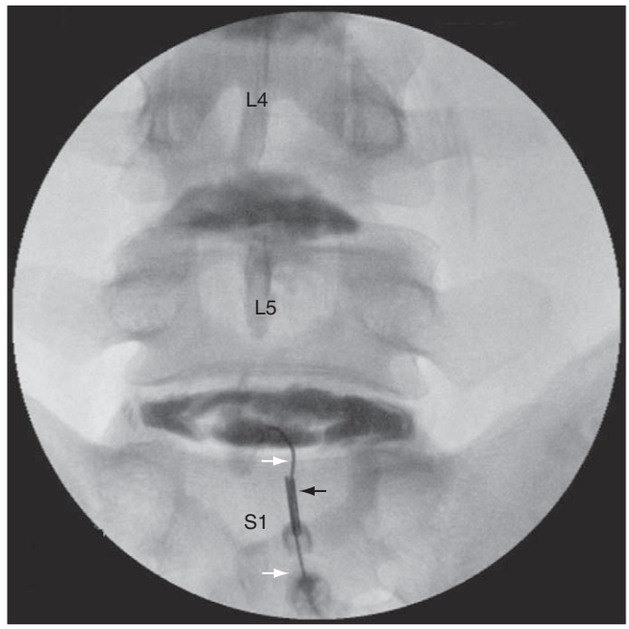


FIGURE 17–67

Anteroposterior view. Posterior, that is, interpedicular or transdural approach to the intervertebral disc. Closed arrow, introducer needle, 18-gauge, 1-1/2-inch; open arrow, 22-gauge, disc puncture needle.

Only nonionic myelographic contrast agents (isohexol or iopamidol) with added antibiotic[13] should be utilized. Using active fluoroscopy, the injectate is slowly injected into the disc. A manometer-syringe is preferred, but a 3-cc syringe is a much less-than-ideal substitute. Once the intrinsic or opening pressure of the disc is exceeded, contrast will be seen flowing into the disc nucleus. As the nucleus is filled, the disc space height is known to increase rather than axial cross-sectional area.[85] Pressure is applied slowly, in 0.5-ml aliquots, until one of the following four endpoints is noted: 3.5-ml volume is reached, significant pain is noted by the patient, epidural or vascular pattern is evident, or a maximum pressure of 90psi, or 50–70psi above opening (psi a.o.) has been reached.[86,87]

During pressurization of the disc, parameters of the injection are recorded on a standardized form by procedure room personnel. The opening volume and pressure are recorded. At predetermined increments, personnel should record the volume injected, static and dynamic pressures, pain description (none, nonconcordant, concordant), vocal or physical patient pain response, pain intensity, and the observed contrast pattern as visualized in the AP and lateral fluoroscopic projections.

Although a 3-cc syringe and manual thumb pressure are still utilized by some, the emerging standard is to use a manometer to accurately quantify the opening pressure and the pressures generated during disc injection. When utilizing a 3-cc syringe, it is difficult to maintain digital (thumb) pressure of over 60–75 psi.[70] Therefore, with the 3-cc syringe technique (i.e., nonmanometric), pressures

can be described as low or high with a variable degree of accuracy. Although the exact quantification of pressure by manometry during provocation discography should be considered as the most appropriate technique, nonmanometric studies should not be automatically assumed to be invalid, but rather suboptimal and highly operator-dependent.

Anteroposterior and lateral images of all discs injected must be saved for a permanent record of the study. These images should include AP and lateral both pre- and post-contrast (Figures 17-68 to 17-72).

STRUCTURAL INTERPRETATION

In 1986, Adams et al.[88] described the contrast patterns seen on the lateral x-ray view of 139 cadaver spines after injecting contrast into the lumbar discs. In order of progressing disc degeneration, patterns were classified as cotton ball, lobular, irregular, fissured, and ruptured. They found that when contrast media is injected into the disc nucleus, contrast media first pushes the disc matrix aside and creates pools of fluid. Fluid then slowly mixes with the matrix caused by the swelling pressure of the hydrophilic proteoglycans and diffusion. Because mixing and diffusion are slow, the location of the pools depends on the degree of fibrosis of the nucleus and any fissures present in the annulus. In other words, the terminology describes successive degrees of degeneration visualized by the pooling of contrast.

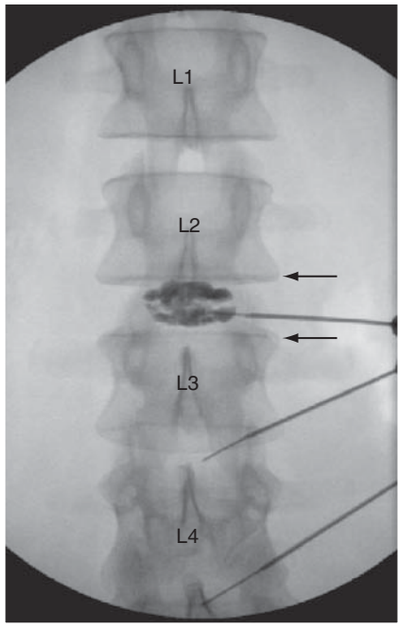


FIGURE 17–68

Anteroposterior view. Injection into L2-L3 intervertebral disc. Note that end plates are parallel to x-ray beam. Normal pattern of contrast.

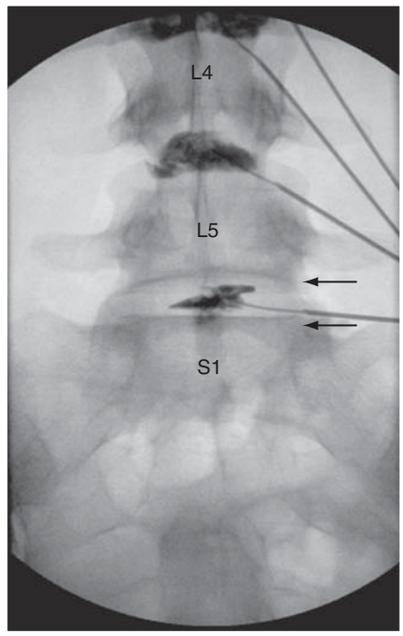


FIGURE 17–69

Anteroposterior view. Injection into L5-S1 intervertebral disc. Note that end plates are parallel to x-ray beam. Pattern not grossly abnormal.

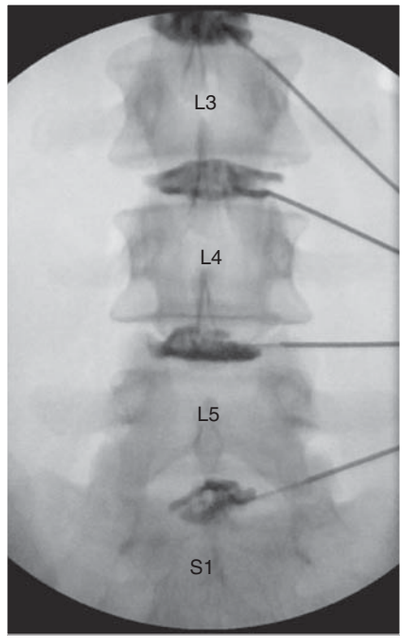


FIGURE 17–70

Anteroposterior view following injection into intervertebral discs. No gross pathology noted.

Although this descriptive classification is used to describe the contrast pattern seen on the fluoroscopy images during the discogram, a CT scan performed following contrast media injection provides the most detailed view of the internal disc architecture.[89] The extent of degeneration is described by dividing the disc as seen

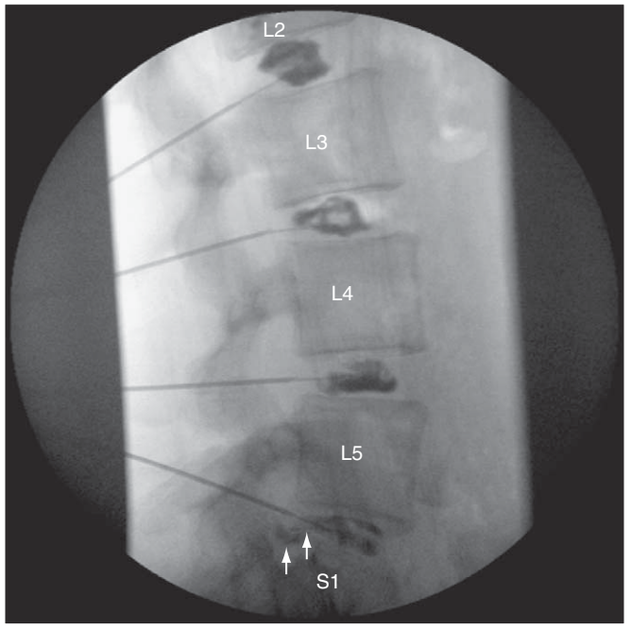


FIGURE 17–71

Lateral view following injection into intervertebral discs. Note posterior annular disruption in the L5-S1 intervertebral disc.

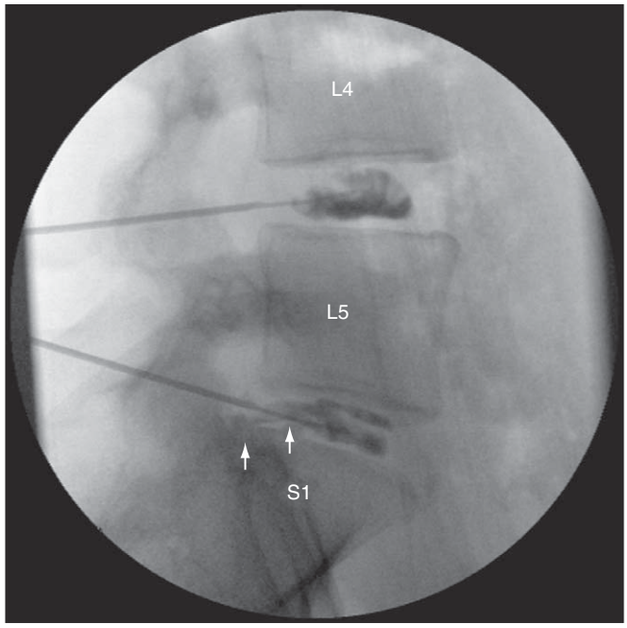


FIGURE 17–72

Lateral view with magnification. Note significant annular disruption at L5-S1 with associated protrusion. During stimulation of this intervertebral disc, marked concordant pain was noted by the patient at low pressure.

in an axial image into four quadrants.[90] If contrast is contained within the nucleus, then no quadrants are disrupted, but when disrupted the extent is described by indicating the location, such as single-quadrant, left posterior lateral; or two-quadrant disruption including the right posterior lateral and right lateral quadrants. In addition to describing the extent, a grading scale is typically used to describe the degree of radial and annular disruption.[90,91] As visualized on the axial CT images following discography, annular tears are graded based on how far radial annular fissures extend into the outer annulus, the degree of circumferential disruption, and whether there was rupture through the outer annulus (Figure 17-73). A grade 0 nuclear pattern indicates no annular disruption (Figures 17-74 to 17-77); grade 1 fissures are into the inner annulus only (Figures 17-78 and 17-79); grade 2 into the middle and outer annulus (Figures 17-80 and 17-81); grade 3 into the periphery, or outer third, of the annulus (Figures 17-82 and 17-83); grade 4 annular tear is a grade 3 annular tear with spread of contrast medium circumferentially within the substance of the annulus fibrosus, subtending a greater than 30-degree arc at the disc center (Figures 17-84 and 17-85); grade 5 annular tear represents spread of contrast through the outer annulus, and thus could involve either a grade 3 or grade 4 annular disruption (Figures 17-86 to 17-89).

When extensive disruption of the normal intervertebral disc architecture is present, no discrete annular tear(s) may be noted (Figure 17-90). Pain with disc stimulation may or may not be elicited.

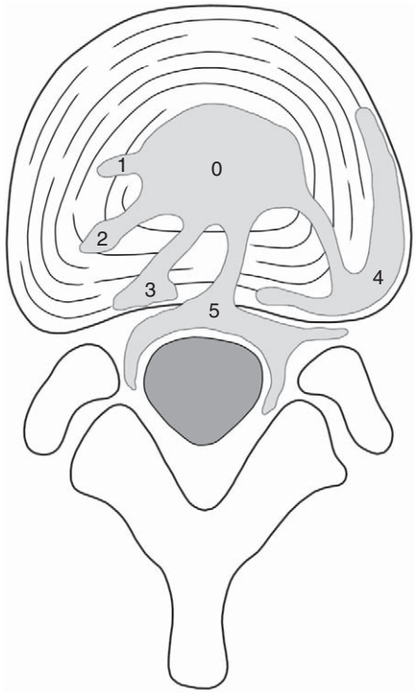


FIGURE 17–73

Modified Dallas Discogram Scale. Grade 0—no annular disruption; grade 1—radial disruption into the inner third of the annulus; grade 2—contrast spread into the middle third of the annulus; grade 3—contrast into the innervated outer third of the annulus; grade 4—grade 3 with >30-degree circumferential tear; grade 5—spread of contrast into epidural space.

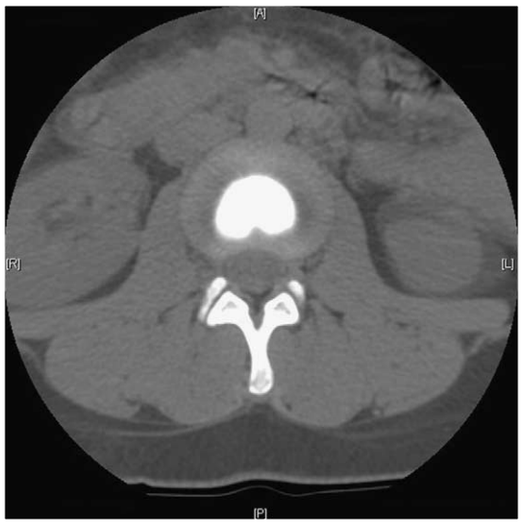


FIGURE 17–74
L2-L3, grade 0 with no disruption of the annulus. From postdiscogram study of Figures 17-68, 17-70 and 17-71.

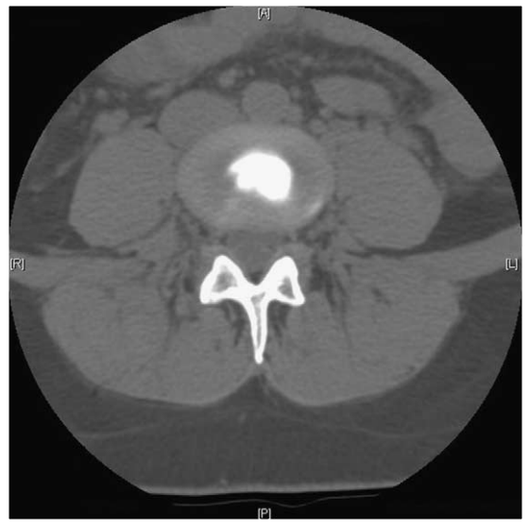


FIGURE 17–76
L4-L5, grade 0 with no annular disruption. From postdiscogram CT of Figures 17-69, 170-70, 17-71, and 17-72.

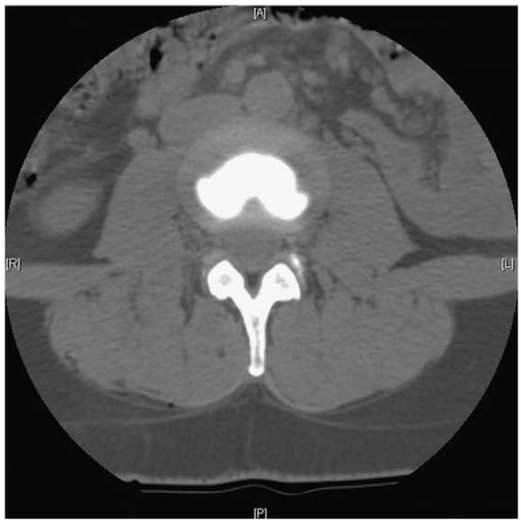


FIGURE 17–75
L3-L4, grade 0 with no annular disruption. From postdiscogram CT of Figures 17-69, 17-70, and 17-71.

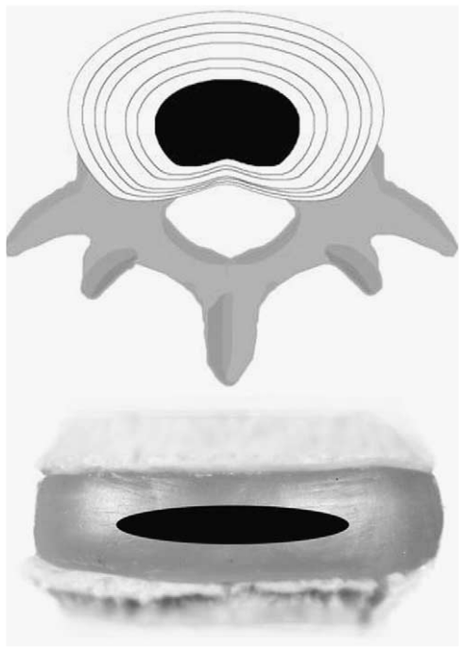


FIGURE 17–77
Axial and lateral illustrations of a grade 0 nuclear pattern.

The degree of annular disruption is important primarily as it relates to pain provocation during stimulation and therefore its possible likelihood of identifying a symptomatic disc. The relation between disc morphology and clinically significant discogenic pain is, however, controversial. The frequency of morphologic abnormalities revealed by discography in the back pain population is high and increases with age,[92,93] putatively from painless degenerative changes.[93] Discrepancies between morphologic appearance and pain provocation have also been described.[94] Milette and Melanson[95] retrospectively reported that concordant pain was provoked by injection in only 37% of patients with a morphologic abnormality documented by discography. Antti-Poika et al.[96] reported only a 52.8% concordant pain provocation rate in discs with discographically abnormal morphology.

While degenerative morphologic changes are not necessarily associated with a symptomatic disc, annular tears are associated with pain provocation during discography.[86,97] Vanharanta et al.[93] found that pain reproduction during

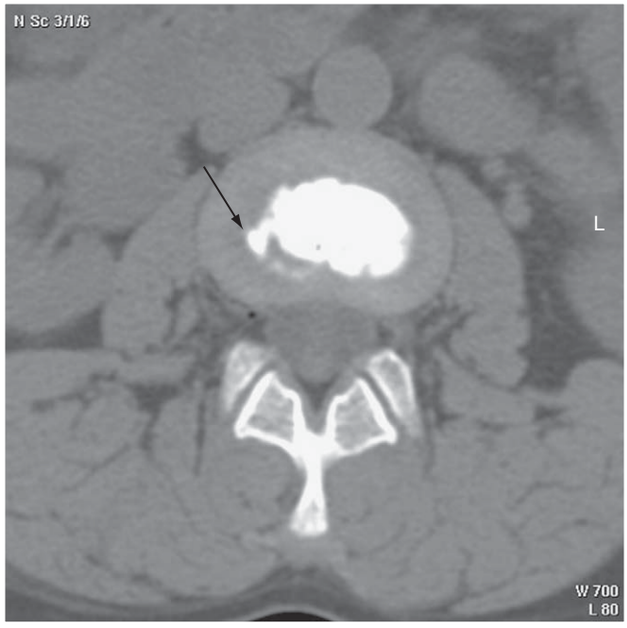


FIGURE 17–78
Grade 1 annular disruption. Contrast into medial third of the annulus.

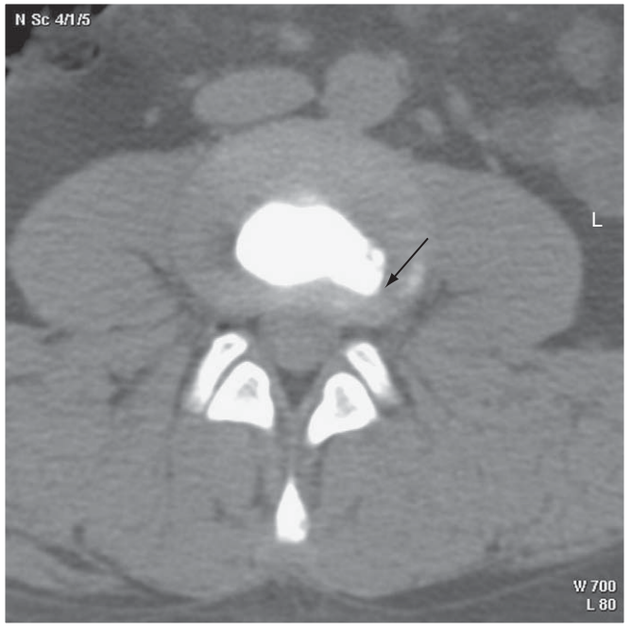


FIGURE 17–80
Grade 2 annular disruption. Contrast into middle third of the annulus.

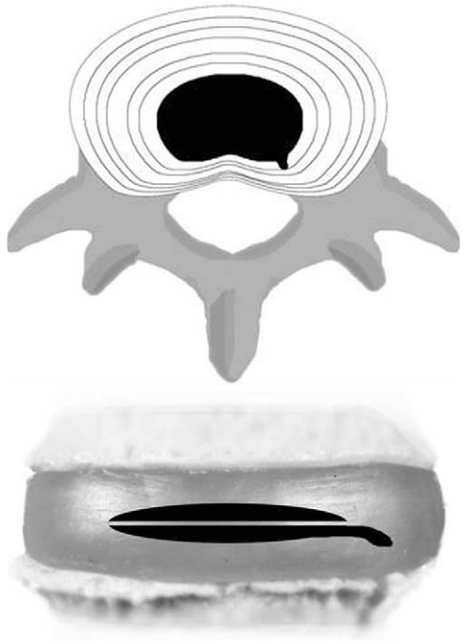


FIGURE 17–79
Axial and lateral illustrations of a grade 1 nuclear pattern.

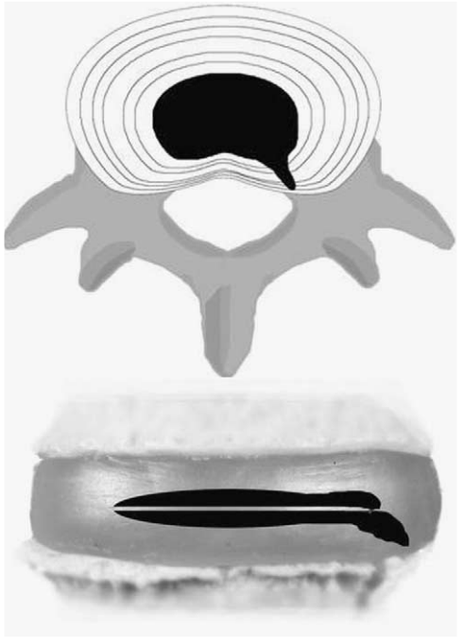


FIGURE 17–81
Axial and lateral illustrations of a grade 2 nuclear pattern.

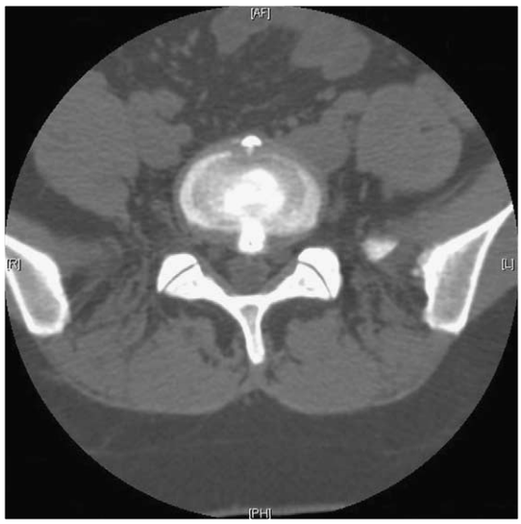


FIGURE 17–82
L5-S1, grade 3 annular disruption with associated protrusion. From postdiscogram CT of Figures 17-71 through 17-74.

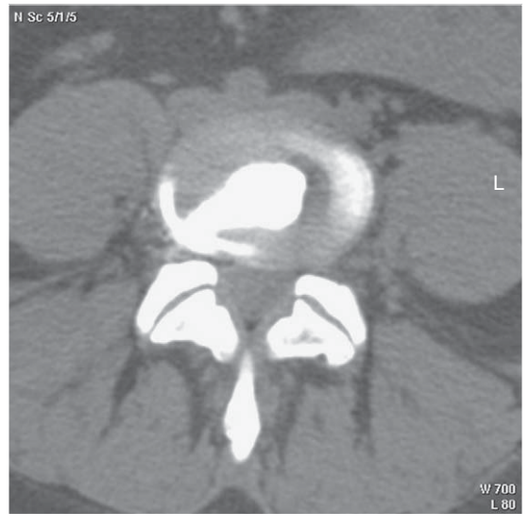


FIGURE 17–84
Grade 4 disruption into the outer third of the annulus with >30 degrees circumferential tear.

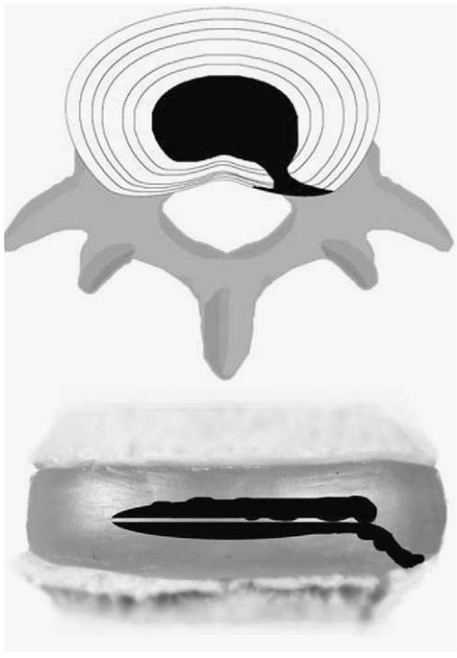


FIGURE 17–83
Axial and lateral illustrations of a grade 3 nuclear pattern.

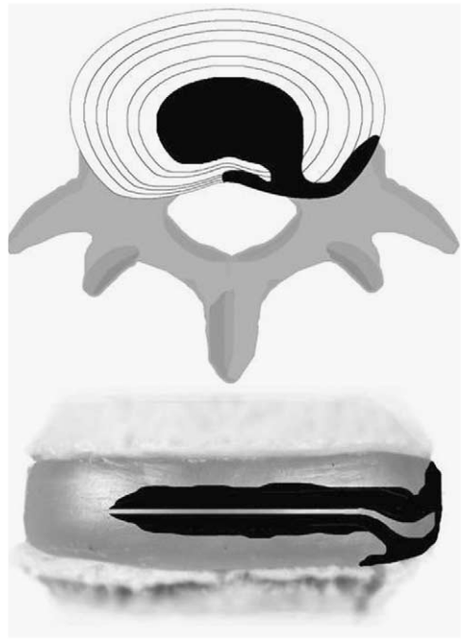


FIGURE 17–85
Axial and lateral illustrations of a Grade 4 nuclear pattern.

discography correlated with the extent of annular disruption. Grades 0 and 1 disruptions are rarely painful, but 75% of grade 3 disruptions were associated with exact or similar pain reproduction. Conversely, 77% of discs with exact or similar pain reproduction exhibited grade 3 annular disruptions. Grade 2 disruptions were less regularly associated with pain reproduction. Using strict criteria and pressure controlled discography, Derby et al.[98] similarly showed a relatively high rate of symptomatic disc (94.6%, 88/93 symptomatic discs). Like the former study, they found that symptomatic disc rates in grades 1 and 2 discs were extremely low (2/93 and 3/93), respectively. Higher pain intensities were observed in grade 3–5 discs relative to grade 0–2 discs at the same pressure, and thus supporting the importance of annular disruption reaching the outer annulus for pain generation. Although not statistically

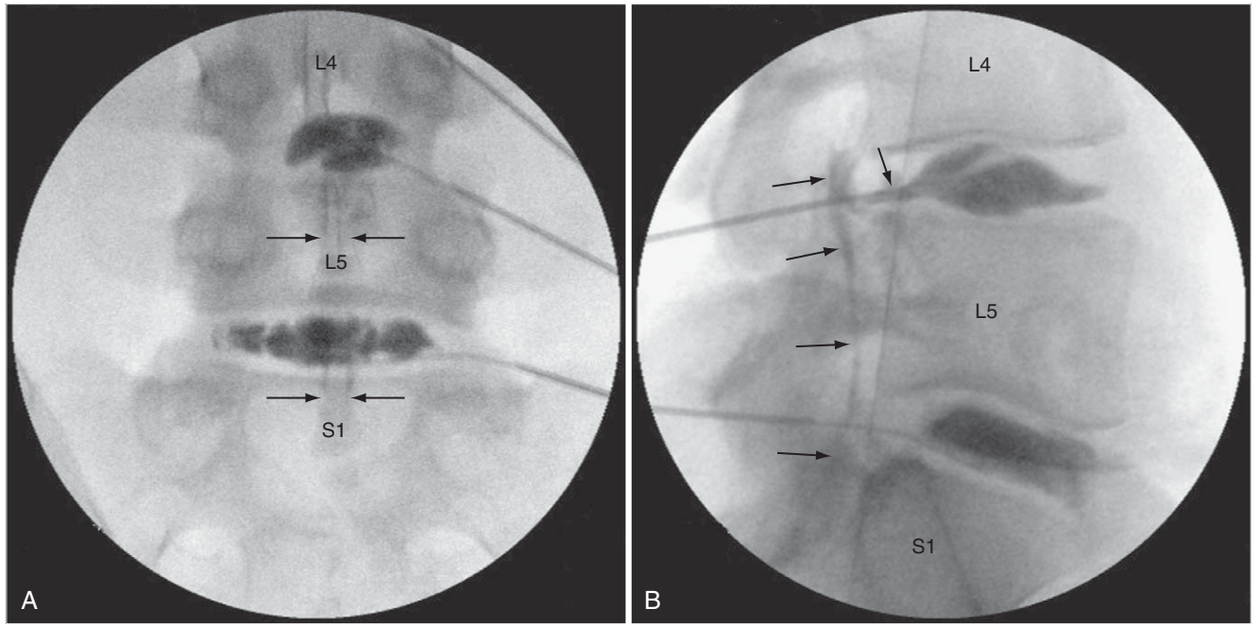


FIGURE 17–86
Grade 5 annular disruption L4-L5 with epidural spread of contrast.

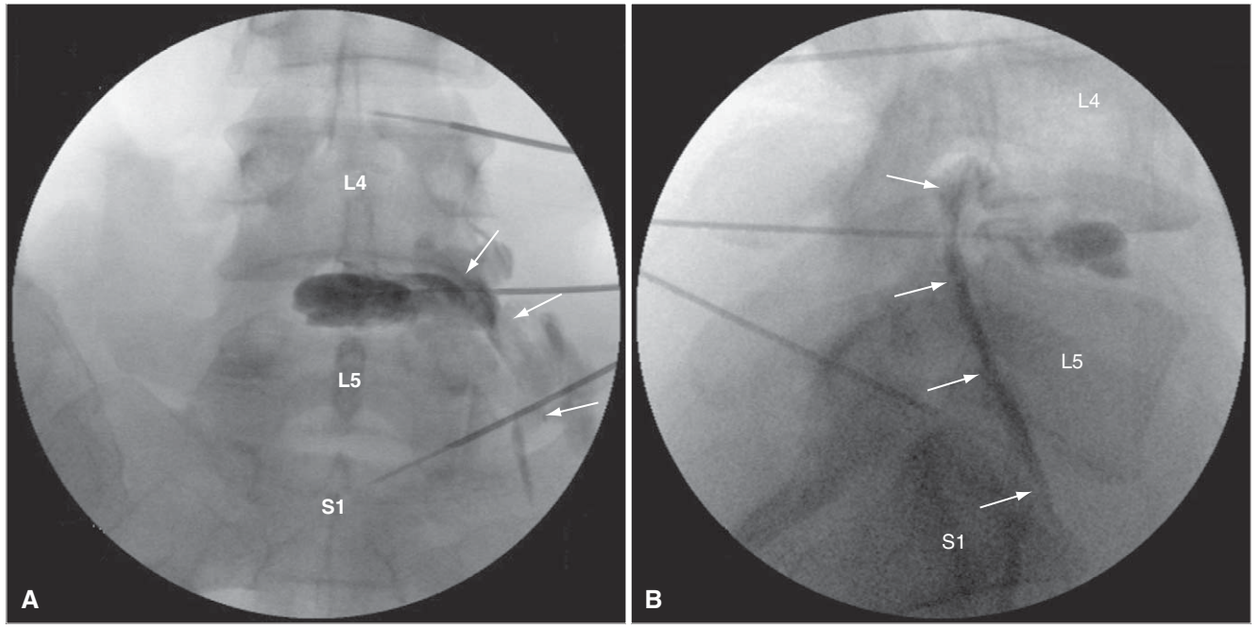


FIGURE 17–87
Injection into the L4-L5 intervertebral disc with foraminal spread of contrast, and an obvious radicular pattern. Might the inflammatory chemical milieu of the nucleus pulposus be causing the radicular pain noted in some patients without a comprehensive lesion?

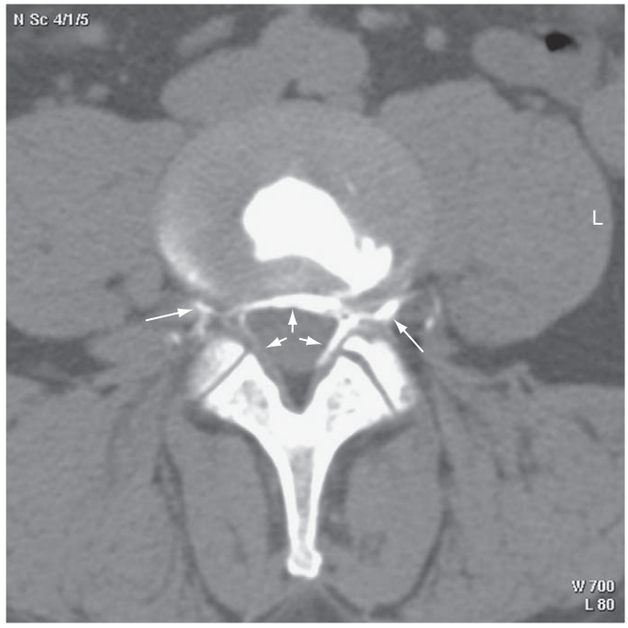


FIGURE 17–88
Grade 5 annular disruption. Contrast seen to spread into the epidural space and foramen bilaterally secondary to full-thickness disruption.

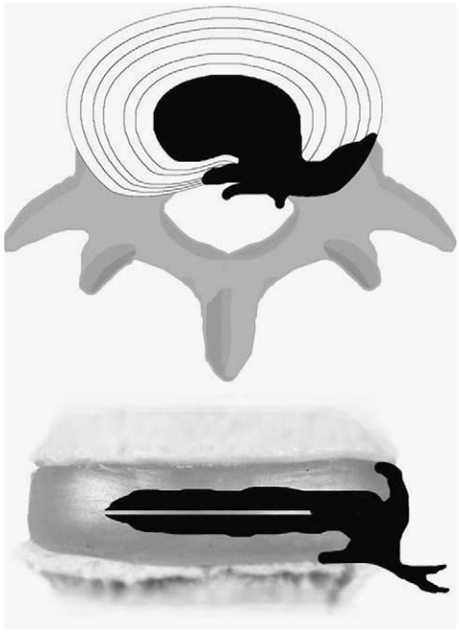


FIGURE 17–89
Axial and lateral illustrations of a grade 5 nuclear pattern.

significant, there was more severe pain intensity with increasing pressure in discs with circumferential extension of tearing (grade 4) and contrast media leakage through the outer annulus (grade 5) than in discs with only radial tearing to the outer annulus (grade 3). Theoretically, more nociceptive structures would be exposed in grade 4 and 5 discs than in grade 3 discs and thus might account for the increased intensity of pain provocation. Furthermore, leakage of contrast through the outer annulus could stimulate innervated structures outside the disc and should be taken into consideration when interpreting the results. In addition, discs classified as low-pressure sensitive (6 or greater concordant pain provocation at ≤15 psi a.o. pressure) showed no significant differences at each annular disruption grade; however, there was a decreasing rate of low-pressure sensitive discs with increasing annular disruption from grade 3 to grade 5 (62.5% at grade 3, 39.4% at grade 4, and 34.2% at grade 5).

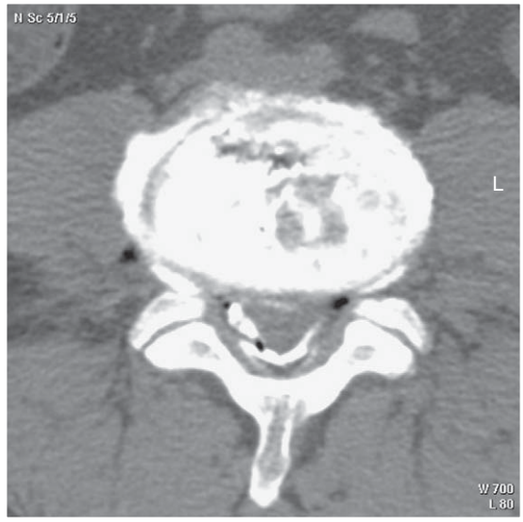


FIGURE 17–90
No discrete annular tear noted, but extensive disruption of normal internal disc architecture is present.

Despite the strong correlation between annular tears and disc disruption in symptomatic patients, in asymptomatic volunteers undergoing discography, Derby et al.[112] found no correlation between pain and the extent of annular disruption. Although nearly all discs that were painful had a grade 3 annular tear, an equal number of such discs were not painful.

PROVOCATION STIMULUS

As defined by Bogduk, provocative discography is conceptually an extension of the physical examination, tantamount to palpating for tenderness. The stimulus is typically created by the injection of nonionic contrast medium that provides a distending force on a fissured annulus and end plates. How closely this stimulus mimics a physiologic load on the disc is speculative. Normally compressive loads are placed on the disc. A healthy, well-hydrated nucleus buffers these loads by tensing the surrounding inner annulus, but in a

"degenerated" disc the dehydrated and fragmented nucleus becomes ineffective and the compressive loads are transferred to the middle and innervated outer annulus and longitudinal ligaments.

Lee et al.[99] studied the pressure effects on the nucleus and outer annulus in pig cadaver discs. Using two pressure transducers, they measured the differences in pressure patterns between the nucleus pulposus and the outer third of the annulus fibrosis during intradiscal injection both in the intact annulus and after making a grade 2 to 3 annular tear. When the annulus was intact, the intact annulus buffered the distending pressure of the injected contrast media, and even with consistent pressures above 150 psi, the outer annular pressures remained at a relatively lower pressure. In comparison, when the annulus was torn, the periannular pressure continuously increased proportionally to the intradiscal pressure. The differences were approximately 0 until 45 psi, when a small pressure difference between 20 to 25 psi was observed. Although not reported, the study also showed that the pressures directly measured within the nucleus were almost exactly the same as the pressures recorded on the external pressure-monitoring device attached to the syringe.

The expanding tensional loads placed on the annulus while injecting contrast media may therefore differ from the compressive loads of activities of daily living that stress both the nucleus and annulus. In the situation where the inner annulus is intact and the patient's pain is caused by an outer rim tear, distention with contrast may under-load the outer annulus, which could result in a false-negative response. Discs with an intact annulus may therefore need to be evaluated using different techniques. In fact, the studies of asymptomatic volunteers in the Walsh et al.,[86] Carragee and colleagues,[100] and Derby and associates[98] studies all showed negative responses (no false positives) when the inner annulus was intact. On the other hand, the measured intranuclear pressure as measured on the injecting syringe during manometric discography accurately reflects the increase in outer annular pressure and will permit the evaluation of pain caused by a graded increase in outer annular tension created by increasing volumes of contrast medium.

As a provocational test, discography is characterized by the liability inherent in all provocation tests; the response may be dependent on the intensity of provocation, and therefore one could therefore argue that since measuring and recording injected pressures should provide better inter- and intra-observer consistency compared to estimating manual injection pressures or ignoring them altogether. Furthermore, because one is attempting to mimic a physiologic load, typical loads experienced by discs with various grades of internal disruption during activities of daily living should be appreciated.

In an unloaded position, the intrinsic pressure of the disc nucleus is created by the osmotic swelling pressure of proteoglycans resisting the tensional compressive force of the anterior and posterior longitudinal ligaments. This opening pressure can be indirectly measured using the pressure at which contrast medium is first seen entering the disc while injecting through a 22–25-gauge needle. In the early 1990s, Derby[101] showed that the direct nuclear pressures could be measured with a hand-held manometer during discography with patients in prone, side, and sitting positions and that the measured opening pressures were the same as those measured by other authors that were specifically evaluating the changes in pressures caused by various disc loading positions. The typical opening pressure in the various positions in a healthy hydrated disc in psi (multiply ~6.9 = neutrons) are as follows: prone = ~15, side = ~25, standing = ~50, and sitting = ~90. He proposed a classification system based on concordant pain provocation created at various pressure values and ranged from the most sensitive discs, which he labeled "chemically" sensitive in which concordant 6/10 or greater pain was provoked at 15 psi or greater above opening pressure; "mechanically" sensitive discs that were painful at 16 to 50 psi a.o. pressure; and indeterminately sensitive discs, in which concordant pain was provoked at pressures greater than 50 psi a.o.. More recently, O'Neill and Kurgansky, [102] also using pressure manometry, classified discs as contact sensitive in which pain was provoked at 0 psi and mechanically sensitive in which pain was provoked during measured pressurizations. No subject experienced pain of intensity 6 with an injection pressure below 50 psi. If attention is paid to pressure of injection and intensity of response, operational criteria can be defined that provide lumbar discography with a potential false-positive rate of zero.

These studies and the majority of prior studies have been based on readings of plateau static pressure recorded postinjection. Previous research[104,105] anecdotally reported two pressures—dynamic and static, but dynamic pressures have not typically been clinically utilized. Although some physicians have used dynamic pressure,[98,104] the parameters have only been recently evaluated. The difference between dynamic and static pressures caused by the speed of contrast injection could be a potential confounding factor. In asymptomatic subjects undergoing discography, Derby et al.[98,103] showed that pain intensity corresponded with the peak manometric dynamic pressure, rather than static pressure. More recently, Seo et al.[125] showed that when injecting contrast medium at 0.08 ml/sec, the mean peak pressure difference between the dynamic and static pressure was minimal, but at faster rates there was an abrupt increase in mean pressure differences. Since it is the peak pressure that most likely provokes the initial pain response, it is important to record and limit the dynamic pressures to reduce the possibility of false-positive results and so that different studies may more easily be compared.

PROVOCATIONAL INTERPRETATION

Evaluating the significance of the pain response is the most difficult aspect of discography and requires the most experience. Interpretation of results will be improved if one closely examines the data of prior studies and should include those that support and those that refute discography's reliability in diagnosing disc pain and its ability to predict outcome.

All provocational tests, and in particular discography, are reliable only if a predefined set of parameters defining the intensity and concordance of a provoked pain response during stimulation of an asymptomatic structure are considerably different from that provoked in a symptomatic structure under the same stimulus conditions. In other words, if the disc is not symptomatic, the patient should either report no pain, discordant pain, or significantly less pain at any given level of pressure-volume of injected contrast medium compared to a disc that is symptomatic. The caveat is that because there is no gold standard to identify discogenic pain, one cannot directly test this hypothesis nor can one directly predefine the level of confidence that a particular disc is symptomatic based on the level of response.

Although one cannot prove that a disc is symptomatic, one can, however, assume that if a subject is not experiencing pain at the time of disc injection, the discs are not painful at that particular time and level of activity. The injection of discs of asymptomatic recruited subjects has historically[86,106] been used to both refute and support[107] test reliability, and in particular, the potential incidence of false-positive response. As one might expect, controversy is fueled by differing results and differing interpretations of results by authors with a particular bias.

TABLE 17-1 PROBABILITY OF EXPERIENCING PAIN AT INTENSITY AND PRESSURE INDICATED

Pressure (psi a.o.)	Group	Pain Score 0–10						
		0	1	2	3	4	5	6
100	No LBP	0.25	0.75	0.44	0.13	0.00	0.00	0.00
	Occ LBP	0.46	0.54	0.38	0.23	0.15	0.08	0.00
90	No LBP	0.25	0.75	0.44	0.13	0.00	0.00	0.00
	Occ LBP	0.46	0.54	0.38	0.22	0.15	0.08	0.00
80	No LBP	0.31	0.69	0.38	0.06	0.00	0.00	0.00
	Occ LBP	0.53	0.47	0.33	0.20	0.13	0.07	0.00
70	No LBP	0.42	0.58	0.32	0.05	0.00	0.00	0.00
	Occ LBP	0.59	0.41	0.29	0.18	0.12	0.06	0.00
60	No LBP	0.64	0.36	0.27	0.05	0.00	0.00	0.00
	Occ LBP	0.64	0.35	0.29	0.18	0.12	0.06	0.00
50	No LBP	0.70	0.30	0.22	0.04	0.00	0.00	0.00
	Occ LBP	0.68	0.32	0.26	0.18	0.11	0.05	0.00
40	No LBP	0.78	0.22	0.17	0.04	0.00	0.00	0.00
	Occ LBP	0.68	0.25	0.26	0.18	0.11	0.05	0.00
30	No LBP	0.83	0.17	0.13	0.00	0.00	0.00	0.00
	Occ LBP	0.75	0.25	0.20	0.10	0.05	0.00	0.00
20	No LBP	1.00	0.09	0.09	0.00	0.00	0.00	0.00
	Occ LBP	0.90	0.10	0.10	0.00	0.00	0.00	0.00
10	No LBP	1.00	0.00	0.00	0.00	0.00	0.00	0.00
	Occ LBP	1.00	0.00	0.00	0.00	0.00	0.00	0.00

LBP, lower back pain; Occ, occasional; psi a.o., pounds per square inch above opening.
Source: Derby R, Kim BJ, Lee SH, et al: Comparison of discographic findings in asymptomatic subject discs and the negative discs of chronic LBP patients: can discography distinguish asymptomatic discs among morphologically abnormal discs? *Spine J* 5:389–394, 2005, with permission.

DISCOGRAPHY IN VOLUNTEERS WITHOUT CHRONIC PAIN

Performed in asymptomatic volunteers without a history of chronic pain, discography has a low rate of false-positive responses.[86,98,100] In fact, the studies by Walsh et al.[86] and Derby and associates[86,98,100] showed a zero-percent false-positive rate when a positive response is defined as 6 or greater pain response at a pressure equal to or less than 50 psi a.o. pressure (see Table 17-1, Figure 17-91). If one includes only the asymptomatic, the Carragee volunteers' pain provocation at various static pressures were relatively mild and similar to the responses of Derby and associates.[98,99] The differing results are probably due to the more precise and slow stimulating pressures used by Derby et al.[98,99] and Walsh.[86] Since the dynamic pressure is transferred directly to the outer annulus when a grade 3 annular fissure is present,[99] the dynamic pressure should be and was used when evaluating the results. During manual injection, there is likely to be a pressure difference of 20 psi or greater between static and dynamic values and would further explain the difference between the Carragee and associates' results, which recorded static pressures, and Derby and colleagues' results in which the slow injection speed gave closer values between static and dynamic pressures.

Lumbar discs can be made to hurt in asymptomatic volunteers, and in 13 volunteers of the Derby et al. study,[103] 50% of discs were painful during pressurization. However, the response was variable and depended on the segmental level stimulated, the nature of the disc, and the intensity of stimulation. When painful, the 13 subjects on average rated their pain as 2 to 3 on a 10-point scale, no subjects had a 6 or greater pain response, and only 1 subject had pain that reached pain at 5. Using the pressure versus pain intensity scores, a receiver-operator curve was constructed (see Figure 17-91, Table 17-1) to show that false-positive responses occurred only above certain pressures and pain scores. This table can be used to estimate the likelihood of a false-positive response during discography in a particular disc and, depending on one's willingness, to accept a certain percentage of false-positive responses can be used to determine whether the disc is in fact symptomatic. In practice, however, many discographers use more conservative boundaries. In particular, most discographers require a 6 or greater concordant pain response at equal to or less than 50 psi a.o. and may

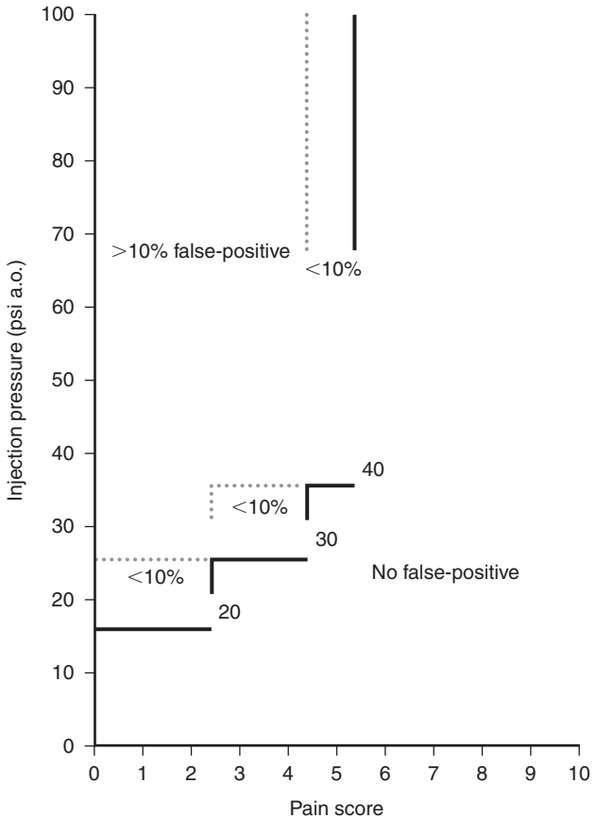


FIGURE 17–91

A receiver-operator curve showing the combined pressures and pain scores, below which no false-positive or less than 10% false-positive response might be expected during discography in symptomatic patients, and above which false-positive response has a likelihood greater than 10%. psi a.o., pounds per square inch above opening.

require more stringent requirements, such as 7 or greater pain at ~20 psi above opening in patients with perceived low pain tolerance or increased pain sensitization caused by chronic unremitting pain.

DISCOGRAPHY IN ASYMPTOMATIC CHRONIC PAIN VOLUNTEERS

Although the results of the studies by Lee et al.[99] and, to a lesser extent, Walsh et al[92] and Carragee and associates[100,105,108–111] (Figure 17-92) show that discography in an asymptomatic disc is usually mild and generally occurs only at higher pressures, patients undergoing discography are typically not asymptomatic and often have a long history of chronic pain. Chronic pain may cause patients to overreact because of compromised pain tolerances and abnormal psychology. In Carragee and associates'[100] original study cohort of asymptomatic volunteers, there were 10 subjects with chronic cervical pain due to a failed surgical fusion surgery, who claimed they had no remembered history of prior low back pain. Defining the false-positive rate as 3/5 pain, Figure 17-93 shows the false-positive rate at three pressure values and the highest unrestricted value. As can be seen by the results, the potential false-positive value per patient could be a standard deviation above or below 30%. Because discogenic pain is ~40% or higher in the population being tested, the probability of finding at least one painful disc may be high if the patient has multiple discs with grade 3 annular tears. On the other hand, most physicians use or should use discography to determine whether a particular disc is painful. In other words, one should not perform discography in a patient for the purpose of hopefully finding at least one painful level to justify a surgical intervention. Using a response of 6 or greater in patients without a history of low back pain, Carragee and associates' data show a

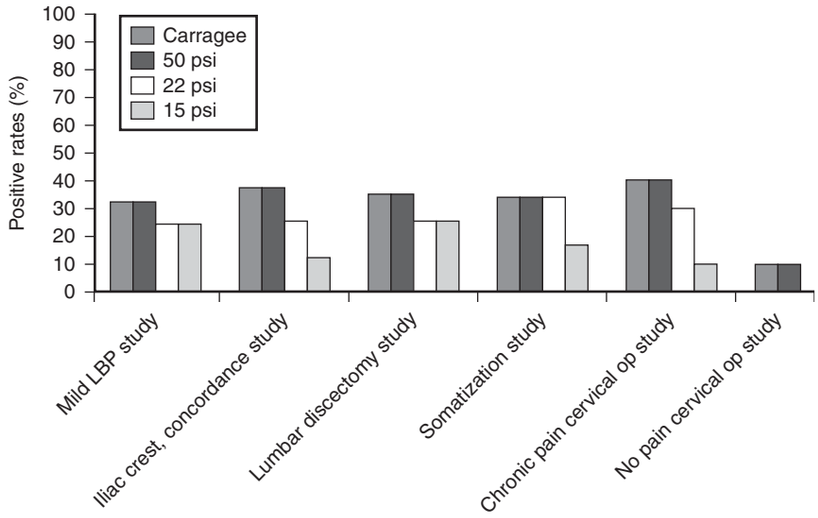


FIGURE 17–92

Positive rates per patients in all of Carragee and associates' studies (with criteria of grade 3 torn discs).

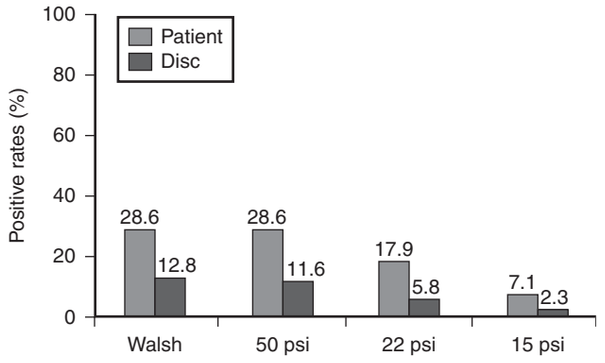


FIGURE 17–93

Positive rates according to four different diagnostic criteria in three studies by Carragee and associates (chronic pain and iliac crest studies) with no lower back pain (with criteria of grade 3 torn discs). These included the 10 patients with a prior successful cervical spine surgery, the 10 chronic pain patients without low back pain symptoms after cervical spine surgery, and 8 patients who had a posterior iliac crest bone graft harvesting for nonthoracolumbar procedures evaluated with lumbar radiography.

false-positive rate on a disc-by-disc basis of ~12% at 50 psi a.o. and ~6% at 15 psi above opening (Figure 17-93). However, analyzing the results of discograms performed on patients and all of Carragee and colleagues' volunteers, including those with "benign" chronic low back pain, O'Neill and Kurgansky[102] determined the estimated probability of obtaining a false-positive response in a patient undergoing discography at 50 psi is 100%, at 25 psi is 50%, at 19 psi is 25%, and at 14 psi is 10%.

COMPARISON BETWEEN ASYMPTOMATIC VOLUNTEERS AND PATIENTS

How do the responses of patients with chronic pain differ from those of asymptomatic volunteers with similar degrees of annular disruption? Since discography is a test that depends on a patient's subjective evaluation of pain provocation, many assume that a patient with chronic pain will over-report pain because of abnormal psychology and low pain tolerances. Whether or not this assumption is true is partially answered by the Derby et al.[103] study comparing the discogram results of patients and asymptomatic volunteers. The data show that many patients undergoing discography have either normal or minimally elevated (DRAM) scores and most have normal or high pain tolerances. Contrary to the preconceived assumptions of many, the study found no significant relationship between abnormal DRAM scores and the intensity of Numeric Rating Scale (NRS) pain intensities reported by patients at pressures of 50, 30, and 15 psi a.o. pressure. There was a weak relationship between pain tolerance and abnormal DRAM scores, but there was only a trend and not a statistical difference between pain tolerance and reported pain intensity scores at the various pressure levels. Nevertheless, 65% of the patient discs with normal pain tolerances had discs that did not meet the criteria of a positive response compared with 52% in patients with a low pain tolerance. This 13% difference might potentially be due to false-positive responses secondary to over-reporting of pain. A patient with a low pain tolerance and abnormal psychological profile is not an ideal candidate for discography, nor do we expect that he or she is an ideal candidate for any invasive surgical procedure.

CONCORDANCE DEBATE

Many discographers feel that reproduction of pain during disc provocation is all that is important. While the absence of pain provocation or the provocation of discordant pain could be defended, one may have a harder time defending the validity of concordant pain provocation in patients with chronic pain due to a variety of structures innervated by overlapping segmental levels. Carragee and associates[105] studied a cohort of eight asymptomatic volunteers whom he just asked to compare the pain on disc injection with the pain experienced after bone graft harvesting. In four discs of 24 (16.7%) in eight patients, Carragee and colleagues were able to provoke pain during his discography that was reported to be in a similar location as their iliac crest bone pain. Even though these four cases of remembered pain will probably not convince most discographers that concordant pain provocation is unreliable, other sources of pain should be ruled out prior to discography.

CLINICAL SIGNIFICANCE DEBATE

Discography cannot and should not determine the clinical significance of the provoked pain. In Carragee and associates'[109] cohort of volunteer subjects with a history of "benign" low back pain, a significant number of the discs provoked pain during provocative testing (see Figure 17-91). Similarly, the study by Derby et al.[103] had three asymptomatic volunteers who had a history of frequent flares in low back pain. Even though none of their discs (eight with grade 3 annular tears) provoked pain greater than 5/10 at 50 psi a.o., a higher percentage of their discs were painful compared with volunteers with only occasional or no history of prior back pain. It is quite probable that opening these fissures during contrast pressurization created a stress similar to the cause of their intermittent flare ups, and in fact one might expect that the forceful opening of a healed but recently asymptomatic fissure should provoke pain.

Discography tests for the presence of nociceptors. If significant concordant pain is provoked at low volumes and pressures, it is likely that the nociceptors stimulated by the

injected contrast are a source of pain. Whether the disc is the majority of the patient's pain or whether the pain is enhanced by functional or central reorganization is beyond the scope of this test. Contrast may stimulate nociceptors not only within the disc itself but also within adjacent structures due to high tensional loads on the outer annulus or direct stimulation by contrast leaking through annular or end-plate ruptures. Responses that are only marginally positive or indeterminate may be a warning that pain is caused by other sources or that this disc is either not symptomatic or only marginally symptomatic at the time of discography. Most complainers have a low level of background low back pain not dissimilar to Carragee's group with "benign" pain and most have intermittent pain flares provoked by activities required at their jobs.

PREDICTIVE VALUE

The ability of manometric discography to predict surgical outcome has been investigated in several studies. In a retrospective review, Derby et al.[112] found that patients who had one or more discs that were painful at a pressure of 15 psi or less above opening ("chemically sensitized discs") had a poor outcome when the disc was not removed and fused and a significantly better outcome when either the anterior column was fused or a combined procedure was performed. Discs that were painful at lower pressures and volumes were chosen because the authors felt these discs were probably more likely to be symptomatic. It does not, however, mean that discs that were less positive are not also symptomatic or whether the presence of a "chemically sensitized disc" will predict better surgical outcome. Combined reconstructive procedures in which manometric controlled discography was used as one of the diagnostic criteria performed will provide patients with degenerative spine pain on average of 30% improvement in overall bodily pain.[113,114]

NEGATIVE DISCOGRAPHY

Although the diagnostic reliability of a positive discogram may never be resolved to the satisfaction of all, the value of a negative response is seldom discussed. Probably the most common use for discography is not to decide which level to fuse but to evaluate adjacent levels. If the disc is not the primary source of pain, some surgeons may want the option to leave the disc alone or use more flexible means of stabilization. In many cases one or two segments are going to be reconstructed for reasons unrelated to whether or not the disc can be proved painful. It is the mildly degenerate adjacent level(s) that is in question.

Comparing the discographic findings in asymptomatic subject discs and the negative discs of chronic low back pain patients using the same pressure controlled techniques,[103] there may be no significant NRS pain score differences between asymptomatic volunteers and the approximately 60% of patient discs that did not meet the criteria of a positive response at 15, 30, and 50 psi a.o. pressure. In contrast, the pressure and pain intensities for negative patient discs and positive patient discs differed significantly (Figure 17-94). The study concluded that advanced discography techniques and strict criteria may distinguish negative asymptomatic discs among morphologically abnormal discs in patients with suspected chronic discogenic low back pain.

DISCOGRAPHY STANDARDS

Since its introduction in 1948, lumbar discography has been mostly practiced without strict standards for pressure, volume, speed of injection, or limits of injection. These practices are no longer supportable.

The authors recommend using the following criteria for a positive response when using pressure-controlled manometric discography: *numeric rating scale of pain above 6/10*, *less than 50 psi intradiscal pressure above opening pressure*, *less than 3.5 ml total volume*, *and at least one negative control disc*. If the provocation at the tested level does not meet these requirements (especially in a patient with a low pain tolerance), the provocation response is no more than

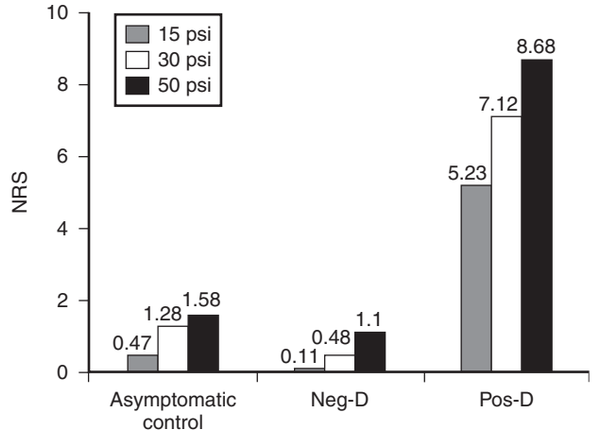


FIGURE 17–94

Mean self-reported NRS scores for discs in each group at three pressure levels: Pain intensity in grade 3 annular torn discs at each pressure. Pressures indicate the pressure above opening pressure. The Neg-D group scores did not differ significantly from asymptomatic control subject scores. The Pos-D group differed significantly from the control and Neg-D groups ($p<0.001$). NRS, numeric rating scale (0-10); Neg-D, discs meeting negative response criteria among grade 3 annular tear discs in patient; Pos-D, discs not meeting negative response criteria among grade 3 annular tear discs in patient. (Adapted from Derby R, Kim BJ, Lee SH, et al: Comparison of discographic findings in asymptomatic subject discs and the negative discs of chronic LBP patients: can discography distinguish asymptomatic discs among morphologically abnormal discs? *Spine J* 5: 389–394, 2005, with permission.)

that which could be reproduced in an asymptomatic "normal" subject and therefore there is no confirmatory evidence that the tested disc is a source of pain. Even if the requirements for a positive response are met, the degree of confidence that the tested disc is a significant source of pain will depend on sound medical judgment.

Remember to keep the rate of injection slow enough to avoid high dynamic pressures. In patients with grade 3 annular tears an injection speed of 0.05–0.1 ml/sec (one revolution of Merit syringe over 5–10 seconds) both the externally measured dynamic and static pressures are an accurate reflection of the pressures transferred to the outer annulus[99] (see Figure 17-93). Record both the dynamic and static pressures, but it is the dynamic pressure that is used to determine a positive response. In addition, transient pain provocation may occur when a fissure is opened or when a thin membrane sealing the outer annulus is ruptured; however, the provocation response should not be accepted as a positive unless it can be confirmed by a repeat pressurization. Pain that does not decrease more than 50% over 30 seconds, increased pain in the postoperative recovery, and a significant flare in symptoms over the next 3–7 days may be used as additional but not the sole criteria indicating a positive response.

CAVEATS

Disc degeneration will directly and indirectly cause pain originating from multiple sources. Once one source of pain is eliminated, other sources tend to become worse. Because a disc is painful does not mean that other sources of pain are absent or even that the disc is the primary source of pain. In most cases, other sources of pain should be investigated with appropriate analgesic diagnostic interventions.

A subjectively interpreted diagnostic test like discography is more than just collecting unfiltered responses. The following are examples of methods used by many experienced discographers when trying to decide if the disc is a source of pain and hopefully decrease both false-positive and false-negative interpretations.

1. If one is unskilled in placing needles into intervertebral discs, the patient will be so traumatized that any further stimulus will be difficult to interpret.
2. The patient's pain tolerance must be evaluated and his or her sensitivity and scoring of pain provocation must be taken into account. A stoic response to skin and subcutaneous infiltration of 1% unbuffered xylocaine is an average score of 2 or less and 5 or less in patients with a normal pain tolerance. Patients must be educated to properly score pain intensity and concordance. Record the patient's responses fairly without coaching, but depending on the pain tolerance, the criteria for a positive or negative response can be adjusted. Remember that the Bogduk table of probabilities is based on asymptomatic volunteers with normal to stoic pain tolerances.
3. One can further refine the pain response and criteria for a positive response by closely observing facial expressions and vocalization. In fact, the Walsh criteria includes the requirements that both vocalization and grimacing need to be observed before the test is positive.
4. Needles should be inserted from the asymptomatic or least symptomatic side.
5. Be very skeptical of pain provocation that occurs on the same side as the needles. Leg and hip pain is usually caused by the discogram needle pushing on and displacing the dorsal root ganglion. The needle will falsely stimulate even back pain. Before accepting ipsilateral pain, one should gently jiggle the needle and make sure that the same pain is *not* provoked.
6. The first report of pain as contrast flows into a grade 3 or greater annular fissure (or end-plate defect or disc protrusion) should be recorded, but the pain intensity must be validated. Record the response but also record the persistence of pain at 30–60 seconds postprovocation. Pain that quickly subsides within 10 seconds should be ignored. The provoked pain could be nothing more than that which would occur if one tore off a quickly opened and otherwise asymptomatic partially healed skin wound.
7. All positive responses must be validated with a confirmatory pressurization. The subsequent pressurization should provoke pain at the same or greater intensity. The pain intensity at the highest pressure equal to or less than 50 psi a.o. is the intensity determining whether a positive response was achieved.
8. Annular tears often heal with a fibrous cap of tissue. The disc may be asymptomatic, but rupture of this membrane during contrast pressurization may cause transient or even prolonged pain. For example, in the study by Derby's group, a fibrous membrane in the outer lateral annulus was ruptured in the L3-L4 disc of one of the asymptomatic volunteers. There was transient provocation of 4/10 groin pain, but one might imagine that if the disc had been more enthusiastically pressurized or the patient's pain reporting was less constrained, this would have been labeled a false-positive response.
9. If a patient complains of pain in a disc without a grade 3 annular tear, look for other causes. Is this just a sign that everything hurts? If so, no

response can be considered positive. If an adjacent disc is painful at a low pressure and volume, inject 1 ml of 4% xylocaine into the painful adjacent disc and retest the normal appearing disc in 10 minutes. One will often find that the disc no longer is painful. Even if the disc remains painful, the results are indeterminate. The disc could have a symptomatic concentric annular or rim annular tear, but this diagnosis is conjecture.

10. Limit the injected contrast volume to 3.5 ml or less. A severely degenerated but asymptomatic disc (at the time of discography) can be made to hurt if enough volume is injected. The actual volume limitation will vary; if there is a leak, the volume restrictions may not apply.
11. A characteristic of "false" pain provocation in an asymptomatic person is a quick resolution of the pain postprocedure in contrast to patients with painful disc that will typically have prolonged pain aggravation. The exception is when an annular fissure that is asymptomatic at the day of the discogram is performed, but is reopened during contrast pressurization. Pain is usually provoked, and the person will usually experience a flare resembling usual episodes that occur with re-injury. Derby and associates saw this response in several asymptomatic volunteers with a history of recurrent back pain.
12. A disc with a leak either through the end plates, outer annular vessels, or into the surrounding structures is more difficult to evaluate. In addition, one may not be able to pressurize the disc. In this case, a more forceful injection may be the only way to get the pressure above 50 psi a.o. In addition, be aware that provocation of proximal and distal pain or even back pain may be due to stimulation of structures adjacent to the disc.
13. Patients with chronic pain often take copious quantities of opioids. Most centers tell patients not to eat or drink after midnight. By the time the discogram is performed the next day, the patient is having early opioid withdrawal symptoms. Everything will hurt. Unless one's intent is to create false-positive responses, these patients must be given a reasonable dose of narcotic before doing the procedure.
14. The false-positive response is probably higher at the level of a previous discectomy. Unless the disc is painful at low volumes and pressures, the results should be called indeterminate. Unless the patient has severe pain at pressures no higher than 20 psi a.o., one should look for other sources of pain.

POSTPROCEDURE CARE

After completion of the discogram, sterile self-adhesive dressings are applied to the puncture wounds and the patient is taken to a recovery room with nurses trained to care for post–spinal injection patients. Periodic evaluation of the patient, including vital signs, level of comfort, level of consciousness, and visualization of the injection sites are recommended. Analgesic medications (oral, IM, or IV) are provided as needed. Patients are observed and discharged as per institutional criteria. Once the patient is stable, he or she may be discharged for a postdiscogram CT scan to provide axial images of the injected discs, if painful levels were noted. The patient is discharged into the care of a responsible adult with discharge instructions to include no driving the day of the procedure. The patient is told to expect some increase in discomfort for a few days postprocedure, and a limited prescription for oral analgesics is provided. Patients are encouraged to call if they feel any unusual or severe pain not relieved by the oral analgesics.

COMPLICATIONS

A myriad of complications following discography have been well documented.[68,93,76] Complications can be inherent to disc penetration, the medications utilized, or unintentional misadventures involving needle placement, and range in severity from minor inconveniences (i.e., increase in low back pain, nausea, and headache) to seizure and death.[115]

Discitis is the most common complication of discography with a rate of less than 0.08% per disc injected.[80] Fraser et al.,[68] have provided evidence that all discitis is due to an infectious process with the most common organisms being *Staphylococcus aureus*, *Staphylococcus epidermidis*, and *Escherichia coli* from the skin. The intervertebral disc is an excellent growth medium for bacteria since it is an essentially avascular structure. However, with the use of preprocedure screening for chronic infections, strict aseptic preparation of the skin, styletted needles, meticulous technique, and intravenous and intradiscal antibiotics, discitis is an exceedingly rare occurrence today.[69,74]

Whether seen in the postdiscogram or postsurgical patient, discitis presents in a similar fashion.[79,117] The patient with discitis usually will present with severe, intractable, debilitating pain of the cervical, thoracic, or lumbar spine days to weeks following the procedure; however, mild self-limiting cases have been described.[74] Discitis needs to be ruled out in any postdiscogram patient who notes a change in severity and/or quality of their pain postprocedure. Workup consists of obtaining laboratory and imaging studies. The C-reactive protein will increase within days of the onset while the sedimentation rate may remain in the normal range for over a month. Blood cultures and CBC will be negative until

the end plates are breached and often remain normal. MRI is the imaging study of choice[118–120] with hyperemia of the end plates and marrow space changes in T2 sequence weighted images 3–4 days after onset of symptoms. Radionuclide bone scanning has been shown to be inferior to MRI in specificity and sensitivity.[121] If an adequate sample of tissue can be obtained, disc aspiration and/or biopsy will be positive in the acute phase of discitis, but once the end plates are violated, a sterile environment is soon noted in response to the patient's immune system.[74]

Treatment of infections within the disc and sepsis often require antibiotic therapy. Although rare, abscess or empyema[122–124] may necessitate surgical intervention. Boswell and Wolfe[115] described a case in which a woman developed intractable seizures, coma, and death following discography. Their conclusion was that an unintentional int rathecal administration of cefazolin (12.5 mg/cc), which had been included in the contrast agent for prophylaxis of infection, precipitated this catastrophic event.

SUMMARY

The importance of determining whether an intervertebral disc is a source of pain is critical. There is, however, an ongoing debate of whether discography can confirm or refute the hypothesis that a particular disc is a source of pain. Any diagnostic test that interprets results based on pain provocation is liable to false-positive and false-negative errors. In addition, if strict control is not applied to the prevocational stimulus, the test can be easily abused to suit one's bias. The reliability of the provoked response will vary from patient to patient and level to level, depending on how intense the stimulus needed to provoke a response, the skill of the discographer, and the sensitivity of the patient. The degree of sensitivity between symptomatic and asymptomatic discs is, however, usually enough for patients to differentiate between the true and false provocation of pain. If a patient has a normal pain tolerance, the provocation of concordant pain at a low pressure and volume will in most cases reliably detect the presence of nociceptors within the disc or adjacent tissue. Even in patients whose pain tolerance is compromised, a positive response has a higher chance of being a true positive then false positive, but in these patients one should insist an adjacent disc with a grade 3 annular tear that is relatively painless at similar or higher pressures and volumes. If such a control can be found, a spurious result secondary to generalized pain overreaction cannot be supported. Furthermore, in many cases, a negative response to disc stimulation provides more important and perhaps more reliable information.

Interpreting discogram results is an art of clinical judgment. Never printed but recognized by the wise, discography is an informative presurgical challenge regardless of the results. Performed by an expert, the test is not particularly painful. A patient that kicks and screams during needle insertion, who bitterly complains that the procedure was the worst thing that ever happened to him or her, and has been forever worse since the procedure may not be the patient you want to live with when his or her surgery fails. Expertly performed and interpreted, discography will help identify asymptomatic discs and to a greater or lesser degree identify a painful disc or segment. Its best future use may be to help limit the number or prevent altogether the number of levels subjected to interventional disc procedures.

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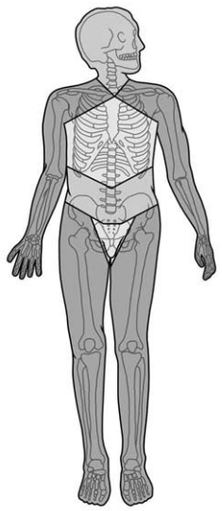
C H A P T E R



18 Lumbar Facet Joint Blocks and Neurotomy

AARON K. CALODNEY

The zygapophyseal joints have been recognized as a source of chronic low back pain for nearly a century.[1] The term "facet syndrome," used by Ghormley[2] in 1933, defined these joints as a pain generator even before Mixter and Barr reported painful lumbar disc herniation.[3,4]

Credit for advancing the concept of zygapophyseal joint denervation as a treatment for low back pain goes to the Australian physician W.E.S. Rees,[5] who in 1971 proposed a surgical approach to severing the nerves that innervate these joints. Currently, zygapophyseal joint denervation is carried out via a percutaneous approach using a small Teflon-coated electrode or cannula. Radiofrequency current directed along the uninsulated electrode tip produces heat that is used to lesion the nerves supplying the symptomatic joint. This was first reported by Shealy[6,7] in 1975 and advanced by others[8–11] and is currently a commonly used treatment for patients with facet joint pain.

Anatomical, technical, and diagnostic inaccuracies in some early reports have largely been corrected. Anatomical studies[12] have defined anatomically correct target points. In the lumbar region, the course of the medial branch nerves is consistent and predictable. This allows for reliable denervation when proper technique is followed. Technique continues to evolve in an effort to maximize the length of nerve coagulated. The effectiveness and duration of pain relief is proportional to the length of the lesion.[13] Laboratory studies demonstrate that the radiofrequency lesion does not extend beyond the tip of the electrode, but rather spreads radially around the long axis of the electrode[14] (Figure 18-1). This suggests that an electrode placed perpendicular to a nerve would be unlikely to coagulate the nerve. The electrode must lie parallel to the nerve in order to obtain an adequate lesion[15] (Figure 18-2). Accurate anatomic targets and proper technique do not overcome an incorrect diagnosis. Dual diagnostic medial branch blocks should be used with small volumes of local anesthetic. The use of a single medial branch block to make the diagnosis of zygapophyseal joint pain carries a false-positive rate of 38%.[16] The use of dual blocks improves the specificity of this diagnostic procedure to 75–85%.[17]

ANATOMY

The lumbar zygapophyseal joints are formed by the articulation of the inferior articular processes of one lumbar vertebra with the superior articular processes of the next vertebra. These joints may be flat or curved in the transverse plane.[18] A concave orientation of the superior facet accommodates the convex orientation of the inferior facet.[19] The L5-S1 joint is typically flat, while the L2-3 and L3-4 joints are usually curved (Figure 18-2A). The extent to which the superior articulating facet faces posteriorly prevents anterior displacement of the intervertebral joint restricting excessive flexion. Resistance to excessive rotation of the joint depends on the degree to which the superior articulating facet faces medially.[18,20] These joints are typical synovial joints. The facets of the inferior and superior articulating processes are covered with articular cartilage.[18] Every lumbar zygapophyseal joint is enclosed by a fibrous capsule. This capsule is made up of collagen fibers passing from one articular surface to the other along the posterior, inferior, and superior joint margins. The anterior margin of the joint effaces the ligamentum flavum, which replaces the fibrous joint capsule anteriorly. Posteriorly, the capsule attaches about 2 mm from the edge of the articular cartilage. At the superior and inferior poles this attachment is even from bone creating subcapsular pockets superiorly and inferiorly.[18] Targeting these subcapsular pockets at the upper or lower pole can provide the injectionist access to the joint space when needle entry along the joint line itself is not possible.

The synovium of the lumbar zygapophyseal joints attaches along the margin of the cartilage on one facet and crosses the joint to the opposite articular cartilage, lining the surface of the fibrous capsule posteriorly, superiorly, and inferiorly, and lining the ligamentum flavum anteriorly.[18]

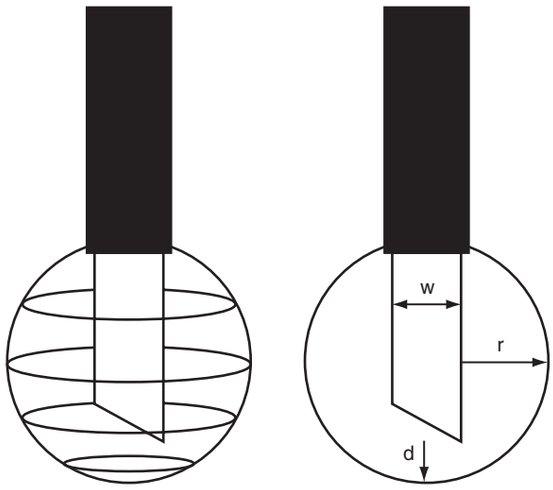


FIGURE 18-1

The radiofrequency lesion spreads radially along the long axis of the electrode. The lesion does not extend far beyond the tip itself. The radial extent (r) of the lesion beyond the surface of the electrode and its distal extent (d) beyond the tip of the electrode are functions of the width (w) of the electrode. A larger electrode produces a larger lesion.

Synovial reflections filled with fat at the superior and inferior poles of the joint have been termed menisci and have been implicated erroneously as the cause of "acute locked back" due to meniscus entrapment.[21]

NERVE SUPPLY

The nerve supply to the lumbar zygapophyseal joints is via the medial branches of the dorsal rami of L1-L5.[18,22,23] Each medial branch supplies the joints above and below its course except for L5, which sends an ascending articular branch only to the L5-S1 joint.[18] Conversely, each zygapophyseal joint is supplied by the two medial branches that course over the transverse processes at the levels comprising the joint. The name of the joint blocked is numerically the same as the names of the transverse processes targeted for injection, but the names of the nerves are one segment higher. The L3-L4 joint is supplied by the medial branches that cross the L3 and L4 transverse processes, which are the medial branches of the dorsal rami of L2 and L3. Similarly, to denervate the L5-S1 joint the L4 medial branch and L5 dorsal ramus must be blocked, which cross the transverse process of L5 and the sacral ala, respectively.[23–25] The lumbar dorsal rami of L1-L4 are short nerves arising from lumbar spinal nerves. These nerves are directed inferiorly and posteriorly toward the top of the transverse process of the vertebral body below. The L1-L4 dorsal rami divide into a medial and lateral branch as they near the transverse process. A variable intermediate branch often arises from the lateral branch rather than the dorsal ramus proper. The L5 dorsal ramus has a longer course and travels inferiorly and posteriorly over the top of the sacral ala below.[18,24] The L5 dorsal ramus divides into only two branches, a medial and an intermediate branch (Figure 18-3).

The medial branches of the L1-L4 dorsal rami run a constant and predictable course. The nerve exits from the intervertebral foramen, piercing the intertransverse ligament and traveling across the neck of the superior articular process below. To do so it crosses over the top of the transverse process at its junction with the superior articular process. This junction is often referred to as a "groove"[22] and represents a readily identifiable landmark where the medial branch lies on bone in a consistent and dependable manner.[26] The medial branch nerve runs in the groove along the lateral aspect of the neck of the superior articular process traveling caudally and posteriorly. Occasionally, the superior articular process will be elongated and the nerve may be found slightly higher, up the wall of the superior articular process. The nerve passes under the mamillo-accessory ligament and medially around the base of the superior articular process. Here the articular branches emerge to supply the zygapophyseal joints above and below (Figure 18-4). Other branches then supply the multifidus and interspinous muscles and interspinous ligament.[18,25]

The L5 medial branch arises from the longer L5 dorsal ramus. It is the dorsal ramus itself, which pierces the intertransverse ligament and runs caudally and posteriorly along the groove formed by the junction of the superior articular process and sacral ala. At the base of the L5-S1 joint, the L5 medial branch then curves medially, sending an articular branch to this joint and then supplying the multifidus muscle.[18,25] There is no mamillo-accessory ligament at this level; however, fibrous tissue that represents an analog of the ligament does fix the position of the nerve at the base of the superior articular process.

The medial branches of the lumbar dorsal rami innervate the zygapophyseal joints, multifidus muscle and interspinous ligament and muscle. The lateral branches innervate the iliocostalis lumborum muscle and often become cutaneous at L1, L2, and L3 innervating the skin over the buttock. The intermediate branches innervate the longissimus muscle.

ZYGAPOPHYSEAL JOINT PAIN

Lumbar zygapophyseal joints have been implicated as the cause of pain in 15–45% of patients with chronic lower back pain.[27–31] Lumbar zygapophyseal joint pain is more common in an older population.[30] Degeneration, inflammation and injury can lead to pain with joint motion. The concept of the three joint complex consisting of two opposing zygapophyseal joints and one intervertebral disc suggests that degeneration of a disc would lead to degeneration of the associated facet joints.[32,33] Prevalence data, however, suggest clinically that facet pain and disc pain are distinct in the lumbar spine, occurring together only 8% of the time.[29] The concept of

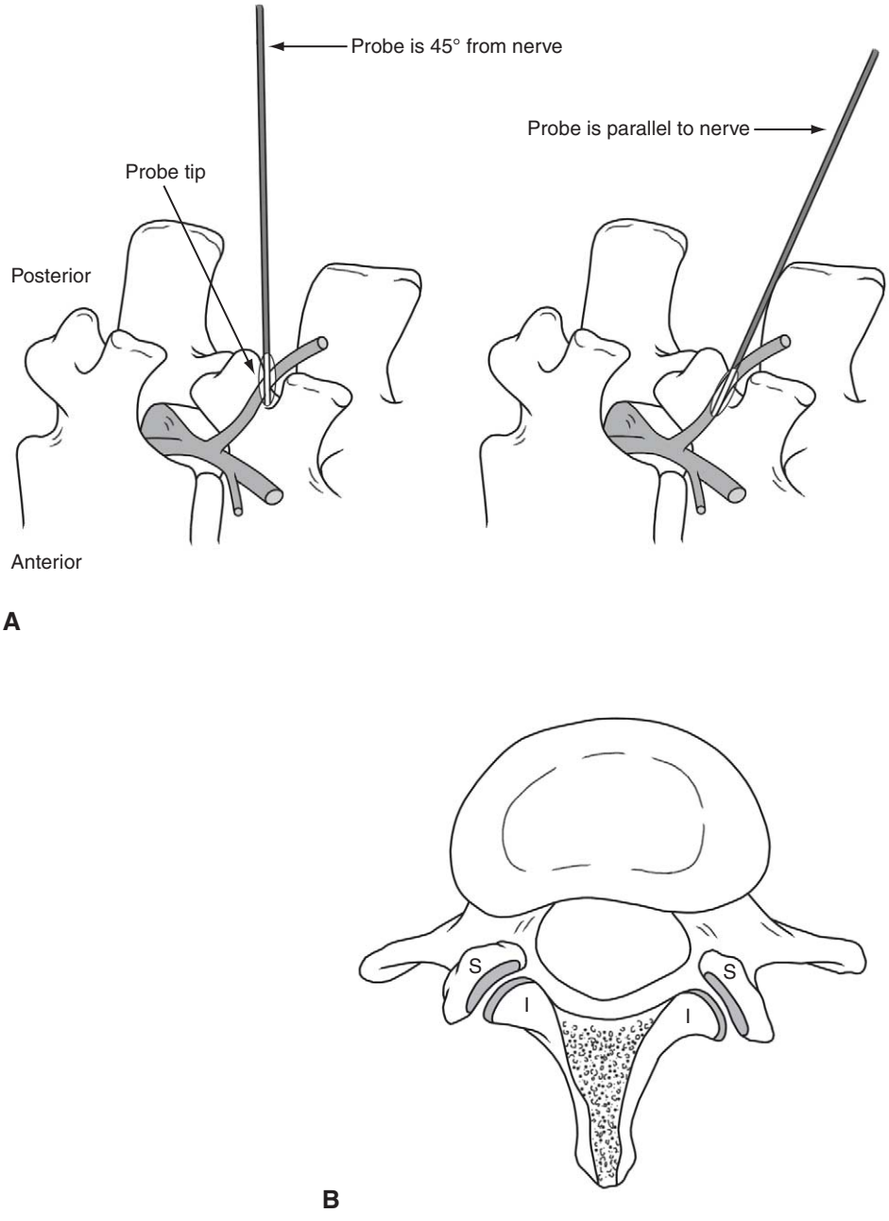


FIGURE 18–2

(A) The electrode should lie parallel to the nerve. When the electrode is placed perpendicular to the nerve, the lesion does not incorporate an adequate length of the target nerve. (B) Sagittal view of an L3-L4 joint. Note the curve of the articular facets and joint space. A backward facing sap resists forward displacement, while a medially facing sap resists excessive rotation. I, inferior articular process L3; S, superior articular facet L4.

the three-joint complex with degeneration occurring in both disc and joint may be anatomically correct but has no clinical correlate. Disc-related pain and zygapophyseal joint pain seem to be discreet entities.[29] Findings of osteoarthritis on radiographs are equally common in symptomatic and asymptomatic individuals. There is no dependable correlation between imaging including CT, MRI, or plain films and zygapophyseal joint pain.[33–35] Multiple sources of zygapophyseal joint pain have been described including small fractures, capsular tears, splits in the articular cartilage, hemorrhage, osteoarthritis, joint subluxation, and capsular and synovial inflammation, among others.[33,36]

PATIENT SELECTION

Patients with predominately axial lumbar pain who have failed at least 3 months of conservative care may be appropriate candidates to be screened for the presence of zygapophyseal joint pain. Other serious causes of lumbar pain must be excluded including infection, tumor, vascular disease, and metabolic disease.[37] Lumbar zygapophyseal joint pain cannot be diagnosed clinically. Clinical signs and symptoms have not been shown to be sensitive and specific for the diagnosis of lumbar zygapophyseal joint pain.[25,30] The correlation of clinical examination findings with results of diagnostic blocks is poor.[38,39] An exception to this

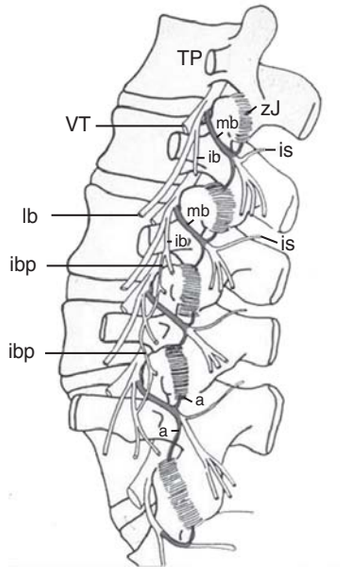


FIGURE 18–3

Nerve supply of the lumbar zygapophyseal joints. The medial branches are in red. Note the very long dorsal ramus at L5. (From Bogduk N, Macintosh J, Marsland A: Technical limitations to the efficacy of radiofrequency neurotomy for spinal pain. *Neurosurgery* 20:529–535, 1987, with permission.)

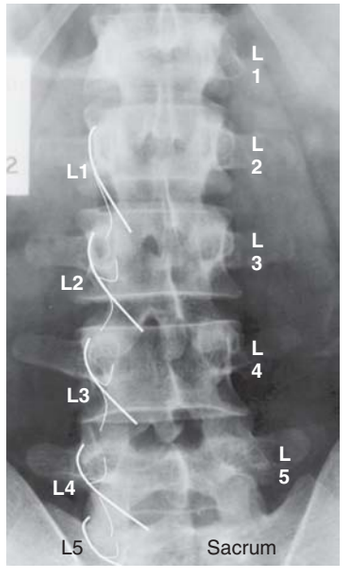


FIGURE 18–4

Medial branch anatomy. Medial branches of the lumbar dorsal rami (labeled on left) curve around the lateral aspect of the neck of the superior articular process. Articular branches pass to the zygapophyseal joints at and below the level of the nerve.

is the screening criteria of Revel et al.,[40] which include relief of pain when recumbent, as well as four of the following six characteristics: age over 65, back pain without exacerbation by flexion, rising from flexion, hyperextension, extension-rotation, or coughing. These criteria were studied using a single intra-articular block to establish the diagnosis, which limits their reliability.[41]

PREVALENCE

Chronic axial lumbar pain is found to be due to the zygapophyseal joints in 15–45% of patients and due to the lumbar intervertebral disc in 26–39% of patients based on prevalence studies.[27–30,38,42] This suggests that investigation of the disc along with the spinal synovial joints should provide a diagnosis in the majority of patients. If the discs appear normal on MRI, the likelihood of disc-related pain is low, and therefore the likelihood of zygapophyseal joint pain is higher. A disc that appears normal on MRI can be painful, although this is uncommon.[37,43] In a patient with a normal MRI, the zygapophyseal joints should be investigated first. If the MRI demonstrates an abnormal lumbar disc, the discs should be tested first. An exception is in the older patient where the prevalence of zygapophyseal joint pain is 40% or higher.[30,31] In this group, investigating the joints first is logical even if the MRI is abnormal.[37] The presence of referred pain radiating into the lower extremity even as far as the foot has been relieved by blocking the lumbar zygapophyseal joints and should not be seen as a contraindication.[37,44]

LUMBAR MEDIAL BRANCH BLOCK

INDICATIONS

Lumbar medial branch blocks are used to determine whether specific lumbar zygapophyseal joints are the cause of a patient's lower back pain. The lumbar zygapophyseal joints have been known to be a source of lumbar pain for nearly a century, and treatment of lower back pain by denervation of these joints has been utilized for over 30 years.[6,45]

The only validated treatment of lumbar zygapophyseal joint pain is radiofrequency neurotomy of the medial branch nerves.[37,46,47] It follows that blocking these nerves with local anesthetic would be the diagnostic test of choice to select patients for this procedure. Kaplan and associates,[48] as well as Kaplan and colleagues,[49] have shown that medial branch blocks are a specific and valid test for lumbar synovial joint pain. Manchikanti et al.[17] demonstrated that the specificity of dual-controlled comparative local anesthetic blockade of the lumbar medial branch is 75–85%.

The false-positive rate of making the diagnosis of lumbar zygapophyseal joint pain using a single medial branch block is 38%.[16] The use of dual comparative blocks with a short- and long-acting local anesthetic is recommended. The most stringent application of our present knowledge would suggest the use of controlled, dual medial branch blocks with at least 80% pain relief on two occasions.[46,50]

CONTRAINDICATIONS

- Systemic or local infection at proposed injection site
- Bleeding diathesis, either primary or due to anticoagulant use
- Pregnancy

EQUIPMENT

- C-arm fluoroscopy with the capability of hard copy or digital image storage: mandatory
- 22- or 25-gauge 3-1/2-inch needle
- Low-volume extension tubing
- Two 3-cc syringes
- Skin preparation solution (povidone-iodine, chlorhexidine, or equivalent)

DRUGS

- 0.5% bupivacaine
- 2% lidocaine
- Nonionic contrast, myelogram compatible

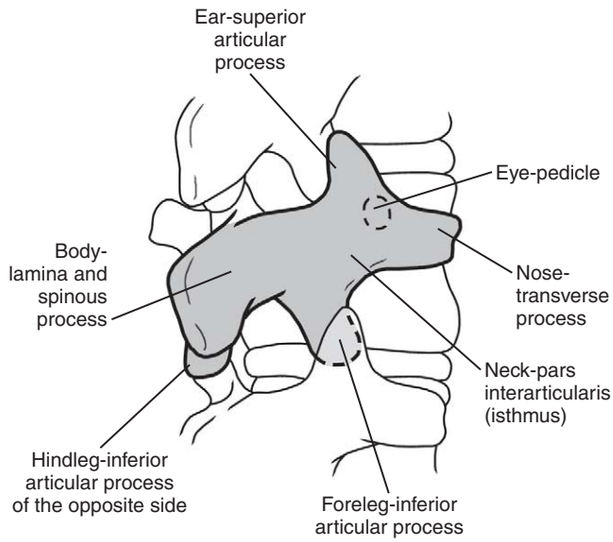


FIGURE 18–5
Oblique "Scottie dog" view.

PREPARATION OF PATIENT

The diagnosis of lumbar zygapophyseal joint pain on clinical grounds is challenging. Clinical features, physical examination, and imaging have not been shown to reliably predict which patients will respond positively to diagnostic block.[25,30,33–35,38,39] Generally, the patient's clinical picture is absent of any neurological deficit, and if a deficit is detected, other causes of back pain and abnormal neurological examination have to be ruled out. History and physical examination, imaging, and laboratory tests can rule out more serious causes of back pain such as tumor, infection, vascular disease, and metabolic disease.[37] Baseline pain scores should be documented and informed consent obtained.

PREOPERATIVE MEDICATION

Standard recommendations by the American Society of Anesthesiologists for conscious sedation and monitoring should be utilized. Sedation, if used, should be minimal to avoid difficulty in interpreting the results of this diagnostic block.

PROCEDURE

L1-L4 Medial Branch

The patient is positioned prone on the fluoroscopy table. The lower back is prepared in a sterile fashion and draped. An oblique approach is utilized. The level chosen is identified in the posteroanterior view. The C-arm is then rotated toward the side to be injected approximately 20 degrees until the "Scottie-dog" view is obtained (Figure 18-5).

The skin entry point should overly the target so that a "down-the-beam" approach can be used. A subtle curve at the tip of the needle can aid in steering the needle to the target point. The needle is passed through skin and superficial tissues directly to the target. Multiple fluoroscopic images are obtained to make sure that the path of the needle remains directly toward the target point. The target is a point along the superior articular process-transverse process junction halfway between the superior border of the transverse process and the mamillo-accessory notch. This point has also been described as just behind the "eye" of the Scottie-dog (Figure 18-6).

The needle is advanced until bony contact is made. Placement is confirmed with a posteroanterior view demonstrating the needle tip at the lateral margin of the superior articular process or even slightly medial to this margin (Figure 18-7). A thick transverse process can deflect the needle to a position where the tip is seen lateral to the lateral superior articular process margin and requires repositioning of the needle. Once confirmation of needle position is made in both the posteroanterior and oblique views, a small amount of contrast is injected to ensure the absence of intravascular uptake. This is followed by injection of less than 0.5 ml of local anesthetic. Directing the bevel caudally helps to avoid spread of injectate toward the intervertebral foramen.[49]

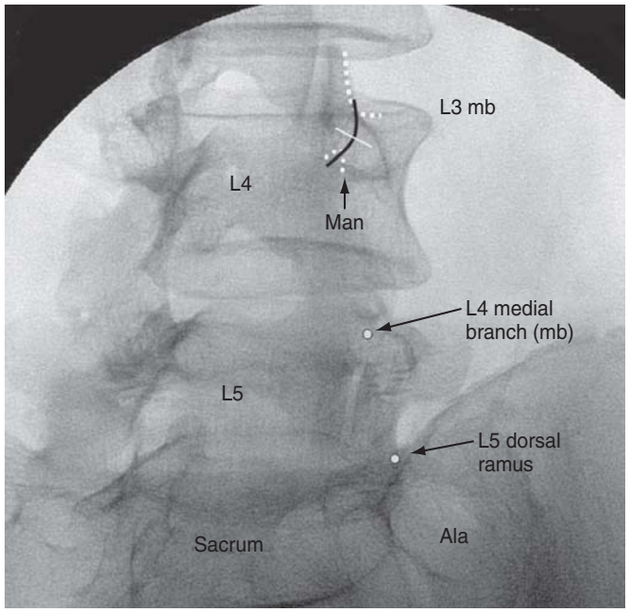


FIGURE 18–6

Oblique view of the lumbar spine. Blue dots show targets for medial branch block. The target is midway between the mammalo-accessory notch (man) and the superior border of the transverse process–superior articular process junction.

L5 DORSAL RAMUS

For the L5 level, recall that the dorsal ramus itself is the target nerve. A "Scottie-dog" oblique view may place the iliac crest in the path of the needle. Using a slightly less oblique (0–10 degrees) approach provides access to the target point, which is along the superior articular process–sacral ala junction below the upper margin of the sacral ala. The needle is advanced until contact is made with bone. Posteroanterior and oblique views are used to confirm proper needle placement (Figure 18-8).

With the bevel pointed medially, a small amount of contrast is injected to rule out venous uptake followed by injection of less than 0.5 ml of local anesthetic.

LUMBAR INTRA-ARTICULAR ZYGAPOPHYSEAL JOINT INJECTION

INDICATIONS

Lumbar intra-articular zygapophyseal joint injection is largely a therapeutic procedure. It has not been rigorously studied as a diagnostic test to identify patients with lumbar synovial joint pain or predict the outcome of lumbar medial branch neurotomy. Zygapophyseal joint injection, although not a validated diagnostic tool, offers the patient an opportunity to respond to the corticosteroid and engage in physical therapy.[51] Joint injection using corticosteroid without local anesthetic can be carried out at the same time as a diagnostic medial branch block, providing validated diagnostic information, as well as intra-articular steroid effect.

CONTRAINDICATIONS

- Systemic or local infection at proposed injection site
- Bleeding diathesis, either primary or due to anticoagulant use
- Pregnancy

EQUIPMENT

- C-arm fluoroscopy with the capability of hardcopy or digital image storage: mandatory
- 22- or 25-gauge 3-1/2-inch needle
- Low- volume extension tubing
- Two 3-cc syringes
- Skin preparation solution (povidone-iodine, chlorhexidine, or equivalent)

DRUGS

- 0.5% bupivacaine
- 2% lidocaine

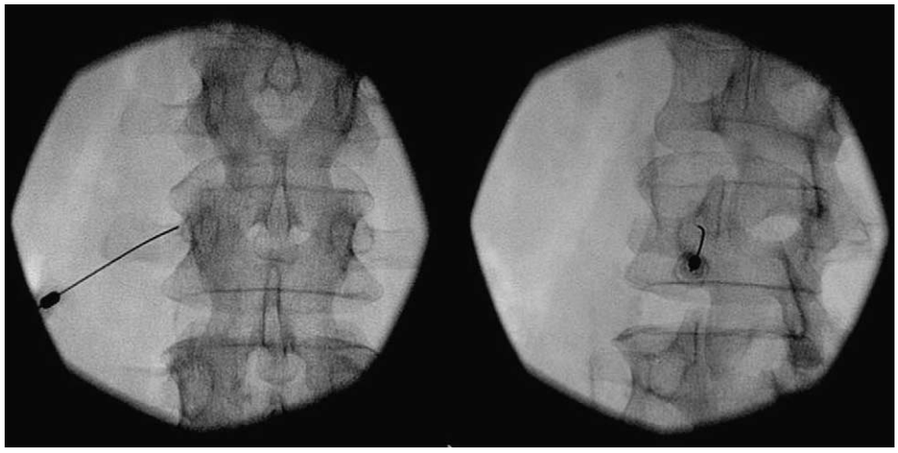


FIGURE 18–7

Oblique and posteroanterior views of medial branch block.

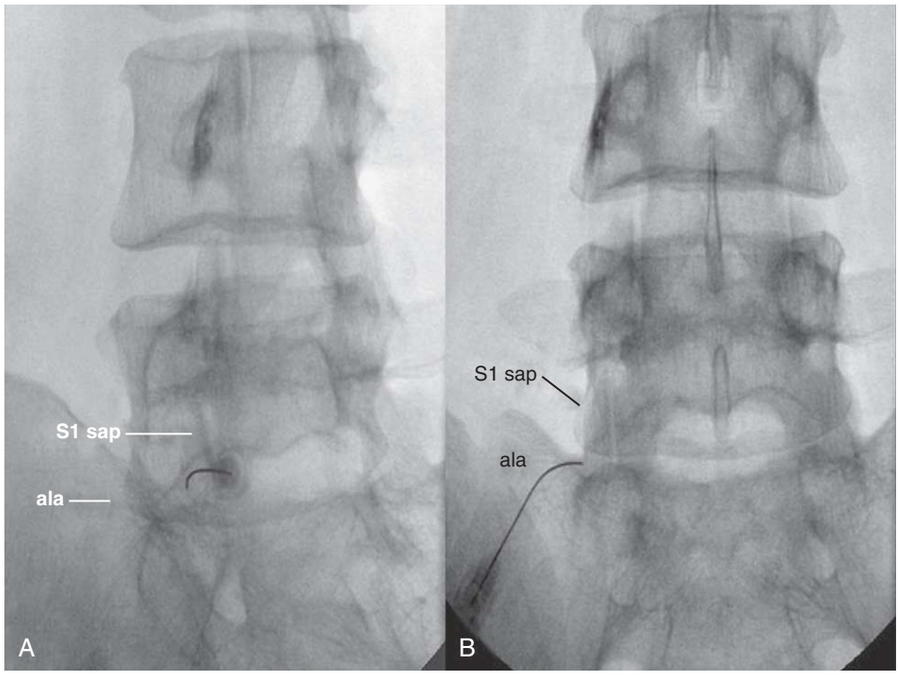


FIGURE 18–8

Proper needle placement for L5 dorsal ramus block. (From International Spinal Injection Society: International Spinal Injection Society Practice Guidelines and Protocols. *ISIS Newsletter*, 2005, with permission.)

- Corticosteroid solution (methylprednisolone, triamcinolone, betamethasone, dexamethasone)
- Nonionic contrast, myelogram compatible

PREPARATION OF PATIENT

See medial branch block above.

PREOPERATIVE MEDICATION

Standard recommendations by the American Society of Anesthesiologists for conscious sedation and monitoring should be utilized. Sedation, if used, should be minimal to avoid difficulty in interpreting the results of this diagnostic block.

PROCEDURE

The patient is positioned prone on the fluoroscopy table. The lower back is prepared in a sterile fashion and draped. An oblique approach is utilized. The level chosen is identified in the posteroanterior view. The C-arm is then rotated toward the side to be injected approximately 20 degrees until the "Scottie-dog" view is obtained. As the C-arm is rotated obliquely, the first visualizationof the joint space is the standard target view. Further rotation of the C-arm may produce a clearer silhouette, but remember that the joint is curved, and the posterior aspect of the joint (our point of entry) will appear open first. The target is anywhere along the joint line (Figure 18-9). Alternatively, the needle can be placed into the superior or inferior capsular recesses above and below the joint. Redundant capsule in these areas provides for intra-articular spread of injectate when osteophytes prevent entry along the joint line itself. The skin entry point should overlie the target so that a "down-the-beam" approach can be used. A subtle curve at the tip of the needle can aid in steering the needle to the target point. The needle is passed through skin and superficial tissues directly to the target. Multiple fluoroscopic images are obtained to make sure that the path of the needle remains directly toward the target point. The needle is advanced until bony contact is made. Placement is confirmed with a posteroanterior, oblique, and lateral view. A small amount of contrast is injected to demonstrate intracapsular spread with filling of the superior and inferior recess. Contrast injection should be observed closely to ensure the absence of intravascular uptake or intraspinal spread. This is followed by placement of 0.5–2 ml of injectate including corticosteroid with or without local anesthetic as discussed above.

RADIOFREQUENCY NEUROTOMY

INDICATIONS

Percutaneous radiofrequency neurotomy of the medial branch nerves is the only validated treatment of lumbar zygapophyseal joint pain.[37,52,53] Successful outcome requires accurate diagnosis. Patients with lumbar facet joint pain cannot be selected by history, physical examination,

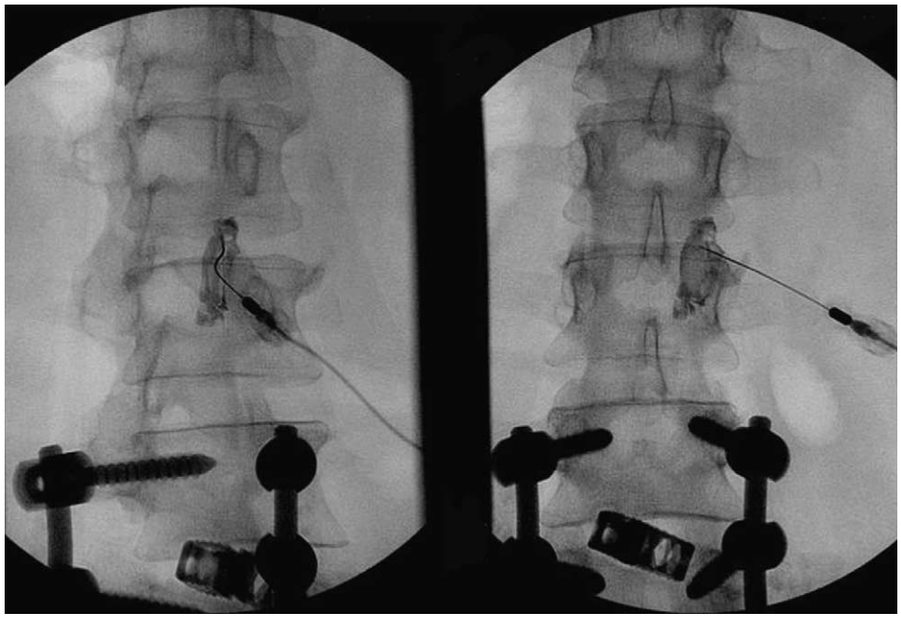


FIGURE 18–9
Intra-articular facet joint injection at L3-L4. Note contrast in the superior and inferior recess.

or imaging studies.[25,30,33,34,35,38,39] It is imperative that diagnostic blocks be carried out using small volumes of local anesthetic under fluoroscopic guidance.

CONTRAINDICATIONS

Absolute
Systemic or local infection at proposed injection site
Bleeding diathesis
Pregnancy
Patients with negative or indeterminate response to diagnostic blocks
Relative
Anticoagulated patients
Pacemaker or defibrillator
Surgical or congenital changes in anatomy (including posterior instrumentation) that render the procedure less reliable

EQUIPMENT

- C-arm fluoroscopy with the capability of hardcopy or digital image storage: mandatory
- Radiofrequency generator
- Radiofrequency cannula and matching thermocouple, typically a 10–15-cm cannula with a 10-mm exposed tip
- Grounding pad
- Syringes for local anesthetic injection
- Needles for anesthetizing the skin entry and cannula path
- Low-volume extension tubing
- Skin preparation solution (povidone-iodine, chlorhexidine, or equivalent)

DRUGS

- 0.5% bupivacaine
- 2% lidocaine

PREPARATION OF PATIENT

The patient must have responded to diagnostic medial branch blocks as outlined above. Patients should have realistic expectations of the outcome and duration of relief from the procedure. Patients who have had previous lumbar surgery remain candidates for the procedure as long as the targets remain accessible. Pain return following previously successful neurotomy does not preclude repeating the procedure. Repeat neurotomy has been shown to be effective as long-term management of lumbar zygapophyseal pain in a series of patients by Schofferman and Kine.[54] Each radiofrequency neurotomy had a mean duration of relief of 10.5 months, and was successful more than 85% of the time. The patient should be educated regarding postoperative activities and restrictions including work and medications. Informed consent should be obtained.

PREOPERATIVE MEDICATION

Standard recommendations by the American Society of Anesthesiologists for conscious sedation and monitoring should be utilized.

PROCEDURE

Lumbar radiofrequency neurotomy is carried out with the patient positioned prone on a fluoroscopy table. Intravenous access is required, and monitoring should include

EKG, pulse oximetry, and blood pressure. Intravenous sedation, if provided, must allow the patient to remain conversive and able to verbalize response to nerve stimulation and identify painful paresthesia. Resuscitative equipment should be available. A high-quality C-arm is needed for cannula placement and to provide hardcopy documentation of the procedure.

After an informed consent is obtained, the patient, procedure, and site are verified. The lower back is prepped and draped using standard solutions for skin preparation and sterile drapes. Sterile gloves are used, and a gown is recommended as the cables, electrodes, connections, and cannulae can be cumbersome and tend to contact an uncovered forearm.

The target point for radiofrequency neurotomy at the L1-L4 medial branch levels is the groove formed by the junction of the transverse process and superior articular process.[25] The needle tip should pass snugly along this groove, until it reaches a point at or immediately below the junction of the superior articular process and transverse process. This can be seen as the "leading edge" of the transverse process–superior articular process junction on the oblique "Scottie-dog" view. The cannula should lie along the lateral aspect of the superior articular process just above its junction with the transverse process (Figure 18-10).

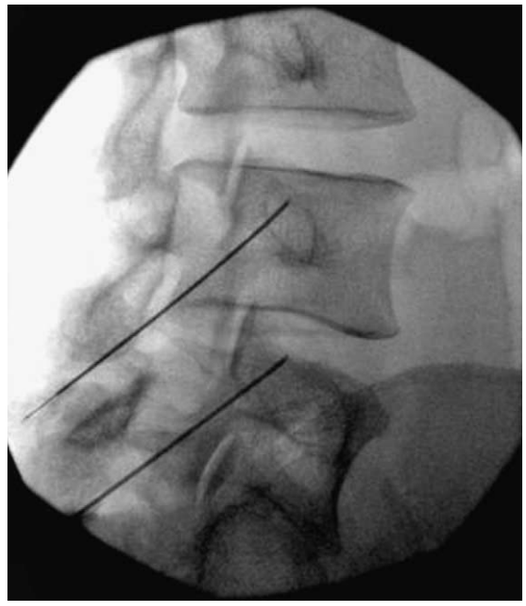


FIGURE 18–10

Oblique view showing needle tips at the superior aspect of the transverse process–superior articular process junction.

Confirmatory posteroanterior views should show the cannula tip above the transverse process directly adjacent to the superior articular process; in fact, the cannula tip may lie just medial to the lateral shadow of the superior articular process (Figure 18-11).

Lateral views should show the tip along the middle of the neck of the superior articular process. There should always be bone in front of the needle tip between the tip of the cannula and the edge of the foramen (Figure 18-12A).

Oblique views should demonstrate that the radiofrequency cannula lies parallel to the expected course of the nerve with the tip at or immediately below the "leading edge" of the transverse process–superior articular process junction (see Figure 18-10).

The procedure can be carried out with an assortment of disposable probes ranging from 18 to 22 gauge in diameter. Remember that the size of the lesion is directly proportional to the diameter of the probe. Using a larger-gauge needle allows for fewer lesions and a higher probability of incorporating the target nerve into one of the lesions. Probe lengths of 5–15 cm with blunt or sharp tips and curved or straight shafts are available with a variety of exposed, active tips. We prefer the 20-gauge, curved, 10-mm active tip, Racz-Finch needles. A 10- or 15-cm length is adequate for lumbar procedures. The curved tip facilitates needle placement, hugs the bony target, and can be turned 180 degrees to make a second burn, enlarging the size of the lesion.

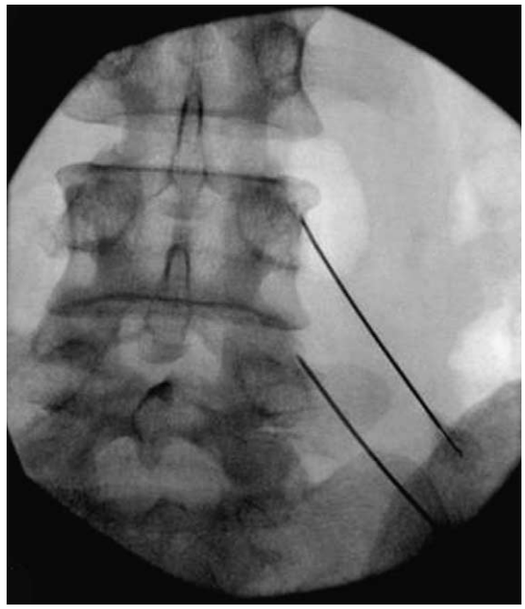


FIGURE 18–11

Posteroanterior view shows needle tips snug against the superior articular process and above the transverse process.

The target point at L5 is the dorsal ramus proper.[22] The medial branch itself is not fully accessible. This is because the dorsal ramus at L5 follows a slightly different anatomical course. It is longer than the other lumbar dorsal rami, running across the top of the sacral ala and along the groove formed by the superior articular process and sacral ala before giving off a medial and intermediate branch. At the L5 level, the needle initially contacts bone at the caudal aspect of the superior articular process near its base. The needle slides snugly along the groove until reaching the notch formed by the

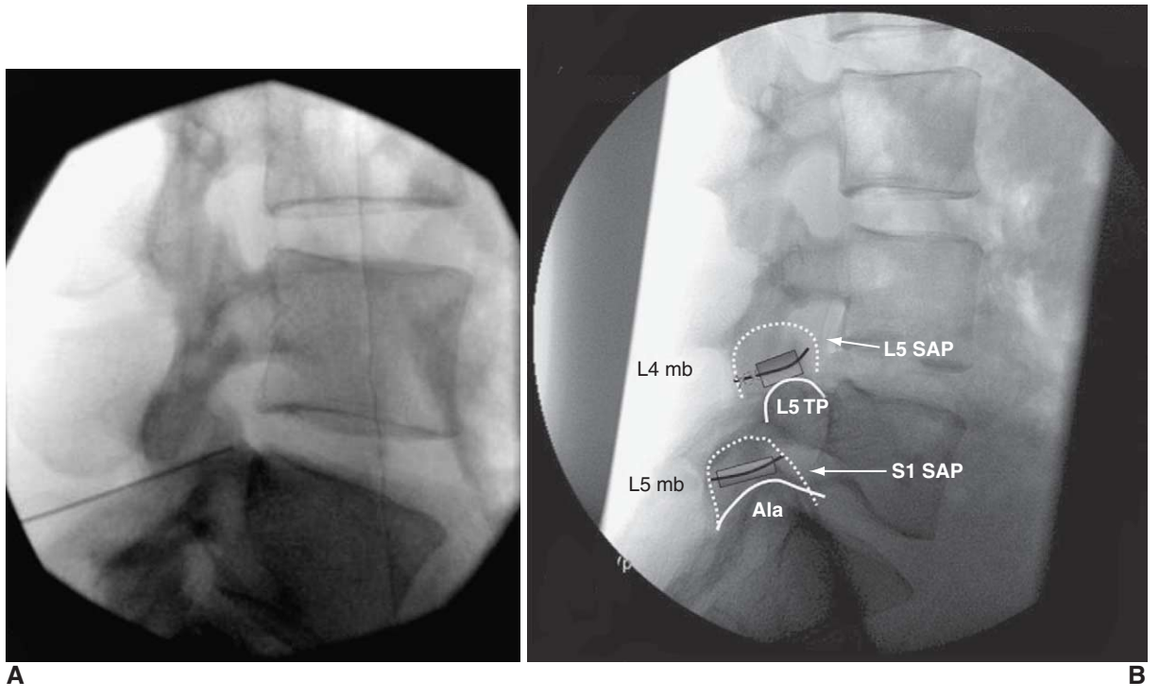


FIGURE 18-12

(A) On the lateral view the active tip should lie across the middle two fourths of the neck of the SAP for L1-L4 and across the posterior three fourths of the SAP for the L5 dorsal ramus, where there is no mammalo-accessory ligament that shields the nerve from the radiofrequency lesion. (B) In the lateral view, the needle should lie along the neck of the SAP. Bone should be visible between the needle tip and the neuroforamen. SAP, superior articular process.

superior articular process–sacral ala junction. Once again, posteroanterior views show the shaft and tip directly against the superior articular process, above sacral ala, and often with the tip seen medial to the lateral shadow of superior articular process. The lateral views should show the tip opposite the middle of the neck of the superior articular process and posterior to the neural foramen. The oblique view shows the cannula parallel to the expected course of the target nerve with the tip at or just below the "leading edge" of the superior articular process-sacral ala groove (Figure 18-13).

The procedure is expedited by starting with the C-arm rotated 10–15 degrees laterally and caudally 15–25 degrees. The degree of caudal tilt varies from level to level on the basis of the degree of lordosis present. The steepest tilt (20–25 degrees) is used at the sacral ala. This view has been called the "pillar view" and approximates the angle of the "groove" between transverse process and superior articular process as seen from below. This allows for initial needle contact with bone to be accomplished using essentially a tunnel-vision technique (Figure 18-14).

Once bony contact is made, the C-arm can be placed in a more conventional posteroanterior or oblique view with the beam parallel to the end plates at the target level. The needle is advanced along the groove until reaching its destination at the superior edge of the groove just above the transverse process or sacral ala. This corresponds to a point just at or below the "leading edge" of the transverse process–superior articular process or sacral ala–superior articular process junction in the oblique view and provides for a long lesion of the nerve along the middle two fourths of its path coursing the lateral aspect of the neck of the superior articular process. Remember the posteroanterior view must show needle tip snugly against the superior articular process and above the transverse process. The oblique view must show the needle immediately against the superior articular process, but not beyond its "leading edge" into the intertransverse space. The lateral view must show the needle along the lateral aspect of the neck of the superior articular process with bone between needle tip and foraminal border (Figure 18-15).

Once all views show proper position of the needle, the stylette may be removed and replaced with the radiofrequency electrode. Impedance should be noted, and if high, can be lowered by injection of a small volume of local anesthetic.[55] Injection of local anesthetic prior to lesioning makes the procedure more comfortable but precludes later the use of stimulation at that level. Sensory and motor stimulation can be utilized to optimize needle placement adjacent to the target medial branch nerve and to ensure a safe distance between the probe and the ventral ramus. Sensory testing is 50 Hz and up to 1 volt. Motor testing is 2 Hz up to 2 volts. These are standard methods for thermal radiofrequency lesioning.

Recently published guidelines suggest that electrical stimulation is unnecessary and superfluous.[56] Dreyfuss et al.[46]

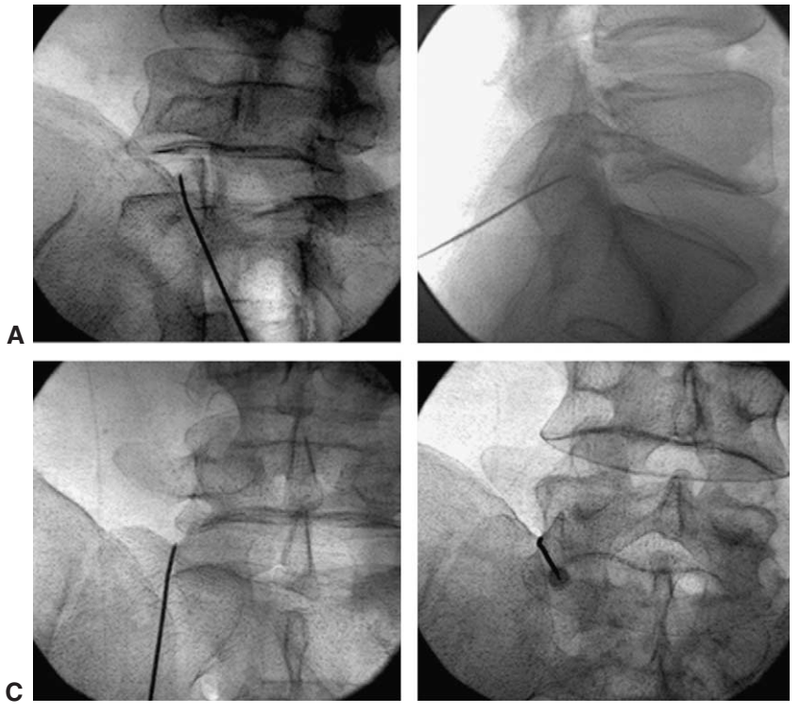


FIGURE 18–13

A-D, Posteroanterior, lateral, oblique, and pillar views of proper needle position for L5 dorsal ramus lesioning. (From International Spinal Injection Society: International Spinal Injection Society Practice Guidelines and Protocols. *ISIS Newsletter*, 2005, with permission.)

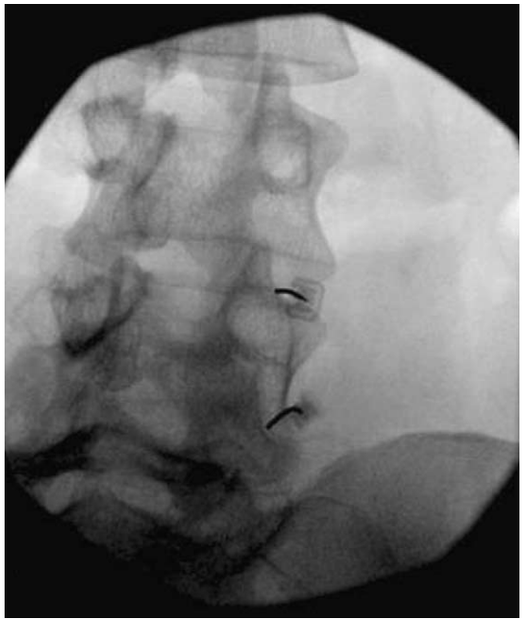


FIGURE 18 –14

Pillar view for initial needle placement using tunnel vision. This view uses 15-degree oblique tilt and a 15–25-degree caudal tilt. Note needles in position for L3 and L4 medial branch lesioning.

found no correlation between impedance values or minimum voltage for multifidus twitch pre-procedure and the presence or absence of multifidus denervation postprocedure. The use of electrical stimulation for verification of needle placement appears to be unnecessary. Anatomically accurate positioning of the electrode may be judged radiographically and by feel of the needle in the target groove.[25]

After position is verified and stimulation completed, 1 cc of local anesthetic is injected to anesthetize the target area. Radiofrequency thermal lesioning is carried out at 80 to 90°C for 90 seconds. A second lesion can be carried out after carefully pulling the needle back along the same trajectory 3–4 mm, enlarging the coagulated area and increasing the likelihood of incorporating the medial branch nerve into the lesion. An additional lesion can be made as a parallel pass, slightly higher up the wall of the superior articular process, particularly if the superior articular process appears elongated.

This process is carried out at each of the levels to be lesioned. A minimum of two levels must be addressed to denervate a single joint. In general, if more than two nerves are to be targeted, lesion only one side at a time, starting with the most highly symptomatic side, and bringing the patient back a few weeks later to treat the other side. This decreases postprocedure discomfort, and often demonstrates that lesioning of the opposite side is not needed as the patient may report significant relief after treatment of the more painful side.

After the procedure, the patients are instructed to ice the procedure site and are excused from work for 1–2 days. Most patients will appreciate a 2–3 day prescription of hydrocodone or equivalent following the procedure.

OUTCOME DATA

Manchikanti et al.[34,57] published a systematic review of medial branch neurotomy and more recently a set of evidence-based guidelines for the treatment of spinal pain. These

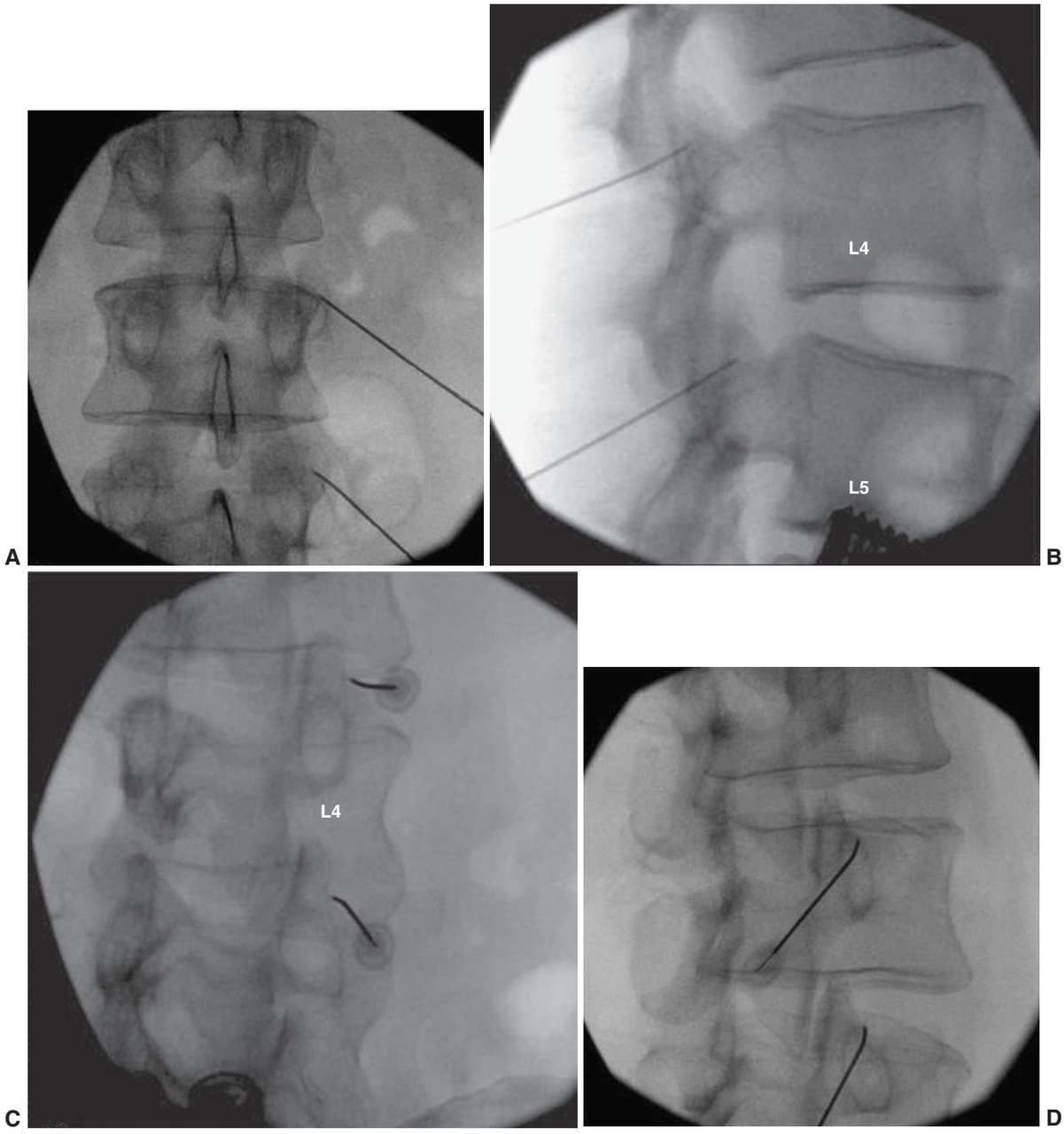


FIGURE 18–15 (A–D)
Posteroanterior, lateral, oblique, and pillar views of needle position for L3 and L4 medial branch neurotomy.

publications reviewed the published literature on lumbar medial branch neurotomy using criteria as described by the Agency for Healthcare Research and Quality (AHRQ). This review concluded that radiofrequency neurotomy of medial branches provided strong evidence of short-term relief and moderate evidence of long-term relief of chronic spinal pain of zygapophyseal joint origin.

Several randomized controlled trials (RCTs) of medial branch neurotomy have been published. Lord and colleagues,[50] in a double-blinded, placebo-controlled trial, evaluated radiofrequency neurotomy for treatment of chronic cervical zygapophyseal joint pain. Strict inclusion criteria were used, including complete pain relief with anesthetic blocks and no relief with placebo block. The median time before return of pain to 50% of pretreatment levels was 263 days in the treatment group and 8 days in the sham procedure group.

Van Kleef and colleagues,[47] in a prospective, randomized, double-blinded trial, evaluated radiofrequency lumbar zygapophyseal denervation for chronic low back pain. At 3, 6, and 12 months, the number of successes was significantly greater in the radiofrequency group compared with the

sham treatment group. A single medial branch block was utilized to screen patients for study inclusion. Two other RCTs by Gallagher and Leclaire also both utilize a single diagnostic block rather than comparative blocks.[9,61] This suggests that a large and unknown portion of the study subjects did not have zygapophyseal joint pain.[41] Patients with other causes of low back pain would not be expected to improve after medial branch neurotomy, perhaps explaining the less successful outcomes in the RCTs when compared with the open prospective trial by Dreyfuss et al.[46]

Dreyfuss and colleagues[46] published a nonrandomized, prospective trial of radiofrequency neurotomy for lumbar zygapophyseal joint pain. This study is remarkable for several reasons.[25,34] First, it used dual diagnostic medial branch blocks to select patients for neurotomy. Second, this study used an anatomically accurate operative technique insuring placement of the radiofrequency needle parallel to the medial branch nerve. Third, objective evidence of target nerve coagulation was provided by postneurotomy multifidi EMG. Fifteen patients underwent lumbar medial branch neurotomy. Sixty percent of patients obtained at least 90% relief at 12 months. Eighty-seven percent of the patients obtained at least 60% relief. Most patients remained stable for the entire 12-month period, but a few reported gradual return of symptoms suggesting regeneration of the medial branch nerves.[46]

CONCLUSION

Kuslich et al.[58] demonstrated that multiple structures are capable of transmitting pain in the low back. These included ligaments, fascia, muscles, intervertebral discs, zygapophyseal joints, and nerve root dura. Prevalence studies have shown that the zygapophyseal joints are a common cause of axial lumbar pain, occurring in 15–45% of patients with chronic low back pain.[27–30,38,42] Clinical history, physical examination, and radiological and electrophysiological testing do not provide an accurate diagnosis in the vast majority of patients with chronic low back complaints.[59,60] Medial branch blocks are a valid, sensitive, and specific test for the diagnosis of zygapophyseal joint pain.[17,48,49] The anatomy of the lumbar medial branch nerves is very consistent, providing clear anatomical target points for lesioning.[12] The use of radiofrequency neurotomy in the treatment of lumbar zygapophyseal joint pain has been validated,[37,46,47] and remains a useful treatment option for carefully selected patients.

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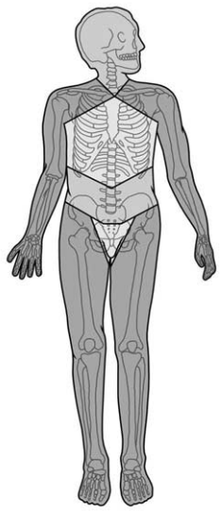
C H A P T E R



19 Lumbar Myofascial Blocks

LEE ANN PARADISE, P. PRITHVI RAJ, AND JASON ROBERT FRYER

HISTORY

Defined by the presence of trigger points, myofascial pain syndrome (MPS) was conceptualized by Janet Travell in the 1950s.[1] Credited with identifying the psoas and quadratus lumborum muscles as the cause of low back pain, Travell and Simons[2,3] were the first to suggest trigger point injections as a way to obtain pain relief. According to Simons and Travell,[3,4] these trigger points are characterized by taut bands of muscle fibers that are "ropy" and tender to the touch, which upon palpitation create a local twitch response. Patients often have trigger points in more than one location.

ANATOMY

MYOFASCIAL TRIGGER POINTS

Myofascial trigger points (TPs) are clinically defined by their motor and sensory characteristics, and a diagnosis can be made if only a few of the characteristics are present.[5] Trigger points are small (2–5 mm in diameter) nodules of hypersensitivity located in taut, "rope-like" bands of skeletal muscle that are detectable through palpatory examination.[1,6–8] Palpation of myofascial TPs will produce or increase a referred, radiating pain pattern recognizable by the patient.[1,3,6–8] Pain will be referred to distal or proximal locations.[8] In the "zone of reference," which is the specific region of referred pain, the patient will experience deep pain that ranges in intensity from a dull ache to severe and incapacitating.[2,6,7,9] Additionally, "snapping" palpation or needling of a myofascial TP may provoke an involuntary twitch in the muscle and/or skin.[6] More commonly, the patient will flinch away from the palpation in a reaction known as the "jump sign."[1,4,7,8] Verbalization may accompany the jump sign.[1] Finally, myofascial TP activation may evoke an autonomic phenomenon, including dermal flushing, lacrimation, sweating, vasoconstriction (blanching), and temperature changes.[8] An itemized list of criteria for identifying myofascial TPs can be found in Table 19-1.[6,8,10,11]

TPs can be classified as either active or latent. Active myofascial TPs remain symptomatic and painful, even at rest.[12] They can be found at the "spot of maximum tenderness" along the taut band of muscle.[1] Palpation of active TPs produces local or referred pain (or both) in a predictable pattern specific to the involved muscle or muscle group.[1] Pain will be described as "spreading" or "radiating," reproduces the patient's pain complaint.[8] Jump sign and local twitch response will also be present.[1,8,12,13] Latent myofascial TPs are not associated with spontaneous pain, but are tender to palpation.[8] They can be inactive or years, precipitated by previous muscular injury.[8] Muscle shortening and weakness, stiffness, and restricted range of motion are present with latent TPs.[8] Unlike active TPs, latent TPs usually do not require treatment unless activated by mechanical overload, stress, and prolonged muscle shortening.[8]

As reported by Travell and Simons,[3] the origin of low back pain in the lumbar region is commonly due to muscular factors, such as those involving the iliopsoas and quadratus lumborum muscles. Pain patterns involving these muscles are referred to as superficial (lateral) or deep (medial), depending on the location of the TP.

ILIOPSOAS MUSCLE

Origins of the psoas major muscle start at the T12 vertebra and along the side of all the lumbar vertebrae. It attaches to the lesser trochanter of the femur. The iliacus muscle originates from the upper two thirds of the iliac fossa and joins the psoas major tendon to attach directly to the femur near the lesser trochanter. The psoas muscle is active while sitting, standing, and maintaining posture. Flexion of the hip at the thigh is the primary function of the iliacus

TABLE 19-1 Clinical Characteristics of Myofascial Trigger Points

Taut band palpable (if muscle is accessible)
Focal exquisite spot tenderness of a nodule in a taut band
Painful restriction to range of motion
Patient's recognition of current pain complaint by pressure on the tender nodule (identifies as a trigger point)
Referred pain to a regional site upon trigger point activation
Reproducibility of pain complaints
Presence of taut band
Visual or tactile identification of local twitch response on trigger point activation
Pain or altered sensation on compression of tender nodule
Muscle weakness without muscle atrophy
Jump sign
Symptoms of autonomic dysfunction (e.g., sweating and localized vasoconstriction)

Sources: Simons DG, Travell JG, Simons LS: *Myofascial Pain and Dysfunction: The Trigger Point Manual*, vol. 1, 2nd ed. Baltimore, Williams & Wilkins, 1999; Raj PP, Paradise LA: Myofascial pain syndrome and its treatment in low back pain. *Semin Pain Med* 2:167–174, 2004; Simons D: Muscular pain syndromes. In Fricton JR, Awad E, editors: *Advances in Pain Research and Therapy*, vol. 17. New York, Raven Press, 1990, pp. 1–41; Gerwin RD, Dommerholt J: Treatment of myofascial pain syndromes. In Weiner RS, editor: *Pain Management: A Practical Guide for Clinicians*, 5th ed. Boca Raton, FL, St. Lucie Press, 1998, pp. 217–229.

and psoas major muscles. It is also possible that these muscles assist some slight lateral rotation (Figure 19-1).

QUADRATUS LUMBORUM MUSCLE

The quadratus lumborum muscle attaches to three different structures: the ilium, 12th rib, and transverse processes of the upper four lumbar vertebrae. The muscle functions as a lateral flexor and stabilizer of the lumbar spine.

INDICATIONS

Psoas Major Muscle

Pain from the psoas major muscle is often referred from the ipsilateral spine in the thoracic region of the sacroiliac area. Occasionally, this pain extends to the upper buttocks. When patients with unilateral iliopsoas TPs are asked to describe their low back pain, they tend to run their hands vertically up and down their spine rather than horizontally.[3] When bilateral iliopsoas muscles have active TPs, patients tend to describe the pain as running across the low back, which is also the case with quadratus lumborum TPs.[3] Patients often have difficulty getting up from a deep-seated chair and describe the pain as worse when they stand up as compared with when they are sitting down.[3]

Quadratus Lumborum Muscle

Symptoms of quadratus lumborum spasm are low back pain, pain with weight-bearing posture, and discomfort turning over in bed. Relief can be provided by positions or maneuvers that unload the lumbar spine of the upper body's weight. Simple coughing or sneezing can exacerbate the pain. In some cases, the pain can be so severe that the patient finds it impossible to bear any weight in an upright position.[3] Some patients have described the referred pain

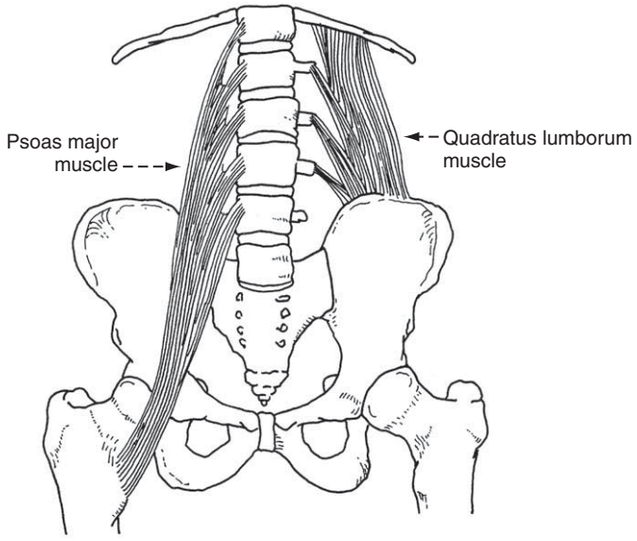


FIGURE 19–1
Origin and insertion of the psoas and quadratus lumborum muscles.

from deep TPs in the quadratus lumborum muscle as being like a lightning bolt to the front of the thigh that extends from the anterior superior iliac spine to the lateral side of the upper part of the patella.[3] History of irritation of pain is associated with falling, major body trauma, or any activity where one is simultaneously bending and reaching to one side to pull or lift something. Other factors that can cause persistence of this pain are length discrepancies, small hemipelvis, and/or short upper arms.

CONTRAINDICATIONS

Absolute
Local infection
Abnormal bleeding disorders (coagulopathy)

Relative
No radiographic imaging equipment

EQUIPMENT

- 25-gauge, 1-1/2-inch skin infiltration needle
- 22-gauge, 5-inch needle for local anesthetic injection
- 3-ml syringe
- 10-ml three-ring syringe for contrast injection
- 20-ml syringe for local anesthetic injection
- Intravenous T-piece extension
- Metal clamp for radiographic identification of entry site

DRUGS

- Radiographic contrast solution: iohexol (Omnipaque)

Local Anesthetics

- Lidocaine
- Bupivacaine
- Ropivacaine

Steroids

- Methylprednisolone acetate (Depo-Medrol)
- Methylprednisolone sodium succinate (Solu-Medrol)
- Dexamethasone (Decadron)
- Triamcinolone acetonide (Aristocort)

Neurolytics

- Sarapin
- 3% phenol
- 50% alcohol

Prolonged Action

- Botulinum toxin A
- Botulinum toxin B

TREATING LOW BACK PAIN ASSOCIATED WITH MYOFASCIAL PAIN SYNDROME

The use of medications in association with other modalities is indicated for any myofascial pain syndrome treatment regimen.[14] Four classifications of medication are typically used in the treatment of low back pain associated with myofascial pain syndrome: antidepressants, nonsteroidal anti-inflammatory drugs, muscle relaxants, and antiepileptic drugs.[15,16] The alleviation or suppression of perpetuating factors through the administration of medications will increase the efficacy of other modalities and speed the recovery process.

INJECTION TECHNIQUE FOR LUMBAR MUSCLES

The lumbar back muscles are classified as superficial, intermediate, and deep layers. For example, sacrospinalis is considered a superficial lumbar muscle, multifidus is considered an intermediate layer of back muscle, and psoas and quadratus lumborum are considered deep layers of the back. The trigger point injection techniques for all the muscles are well described by Travell and Simons.[3] The superficial and intermediate layers of back muscle do not require special imaging techniques and hence will not be described in this chapter. The psoas and quadratus lumbar muscle injections are described below.

PREPARATION OF PATIENT FOR PSOAS AND QUADRATUS LUMBORUM INJECTIONS

Physical Examination

PSOAS MAJOR MUSCLE

Clinical examination involves tests that restrict extension of the thigh at the hip. There is increased pain with an active straight-leg raise, which is decreased with passive lifting. Extension of the leg at the hip in the lateral decubitus position often increases the pain. Pressure at the insertion site deep in the lateral border of the femoral triangle over the trochanter elicits tenderness of the iliacus and psoas muscles. The uppermost iliacus muscle fibers can be palpated at the ilium behind the anterior superior iliac spine. Psoas muscle tenderness is found by palpating through the abdominis muscle and compressing the psoas muscle medially against the lumbar spine (Figure 19-2). Reference zones of the psoas muscle are shown in Figure 19-2.

QUADRATUS LUMBORUM MUSCLE

Physical examination shows muscular guarding and truncal rigidity with rolling over or rising into an upright posture. TP examination requires that the patient be in a prone position or any position where there is a palpable muscle area between the 12th rib and the iliac crest. There are four TP locations consisting of two superficial areas and two deep areas with a cephalad and caudal component for each pair. Palpation of the muscle just below the 12th rib and approximately 5–6 cm lateral to the spinous process of L1 elicits pain to the iliac crest and sometimes to the ipsilateral lower abdominal quadrant. The cephalad superficial TP is found at the L4 level about 1–2 cm above the posterior iliac crest and can refer pain to the greater trochanter. Deep triggers of the quadratus lumborum muscle can be palpated at the transverse process of L3 and 2 cm above the posterior superior iliac spine, with referred pain to the sacroiliac joint and lower buttocks, respectively. Figure 19-3 shows the course of the quadratus lumborum muscle from its origin to insertion, as well as the reference zones of the quadratus lumborum muscle.

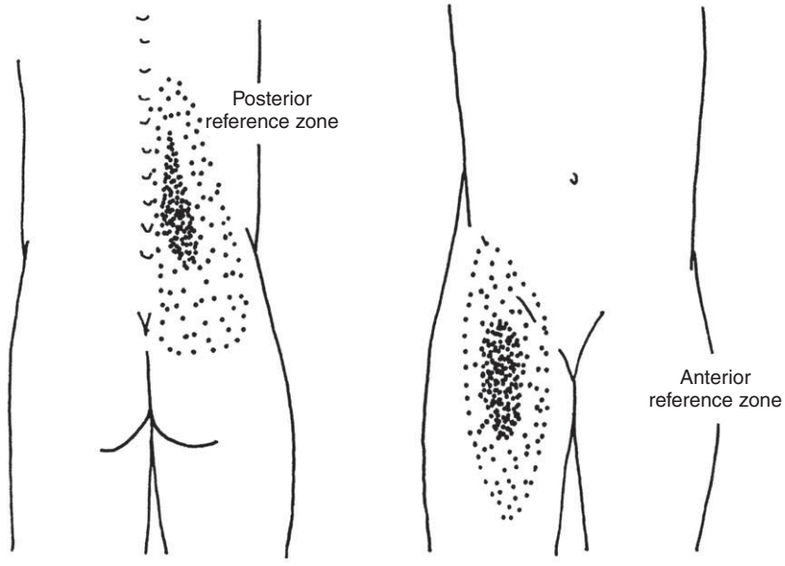


FIGURE 19–2
Reference zones of the psoas muscle.

PREOPERATIVE MEDICATION

Standard recommendations by the American Society of-Anesthesiologists sedation may be provided when necessary.

Monitoring

- Electrocardiogram
- Blood pressure
- Pulse oximeter
- Intravenous access
- Nasal cannula for O_2 if necessary

PROCEDURE

Patient Positioning

The typical approach to the iliopsoas muscle involves fluoroscopy. The patient is placed in the prone position (Figure 19-4). For the lateral view, see the position of the C-arm in Figure 19-5.

Site of Needle Entry

PSOAS MAJOR MUSCLE

Select a point approximately 5 cm lateral to the spinous process at the L3 level (or the top of the iliac crest). With a 22-gauge, 5-inch, B-bevel needle, insert the needle using a "gun-barrel" technique until the needle is approximately at the anterior one third of the vertebral body in the lateral view.

QUADRATUS LUMBORUM MUSCLE

Injection of the quadratus lumborum is safely done at the L3-L4 level above the iliac crest. The muscle can be injected approximately 2 cm above the iliac crest and posterior superior iliac spine. A 22-gauge, 1.5–2-inch B-bevel needle is inserted in a gun-barrel technique after skin infiltration following local anesthetics. The needle advancement is stopped when the needle tip is at the level of the neuroforamen (Figure 19-6). In the lateral view of the lumbar spine, the tip of the needle should be behind the transverse process (see Figure 19-5). Note the position of the C-arm inFigure 19-4.

Confirmation of Correct Needle Position

PSOAS MAJOR MUSCLE

In the lateral view, the psoas major muscle spreads vertically over the anterior one third of the lumbar vertebral body when the contrast (iohexol) is injected. Note that it is always anterior to the foramina (Figure19-7, arrow B). After the correct needle placement is confirmed, 8–10 ml of a local anesthetic-steroid mixture is injected into the psoas muscle on one side.

QUADRATUS LUMBORUM MUSCLE

The quadratus lumborum muscle is seen posterior to the foramina at the level of the transverse processes (Figure 19-7, arrow A). The needle should be at or below L3. Inject 4–6 ml of local anesthetic-steroid mixture. If botulinum toxin A or B is injected, the manual guidelines should be followed.

MONITORING OF CHANGES DUE TO PROCEDURE

Pain relief after the injection of the local anesthetic should occur after 30 minutes.
Botulinum toxin injection will give relief in 2 to 3 days.

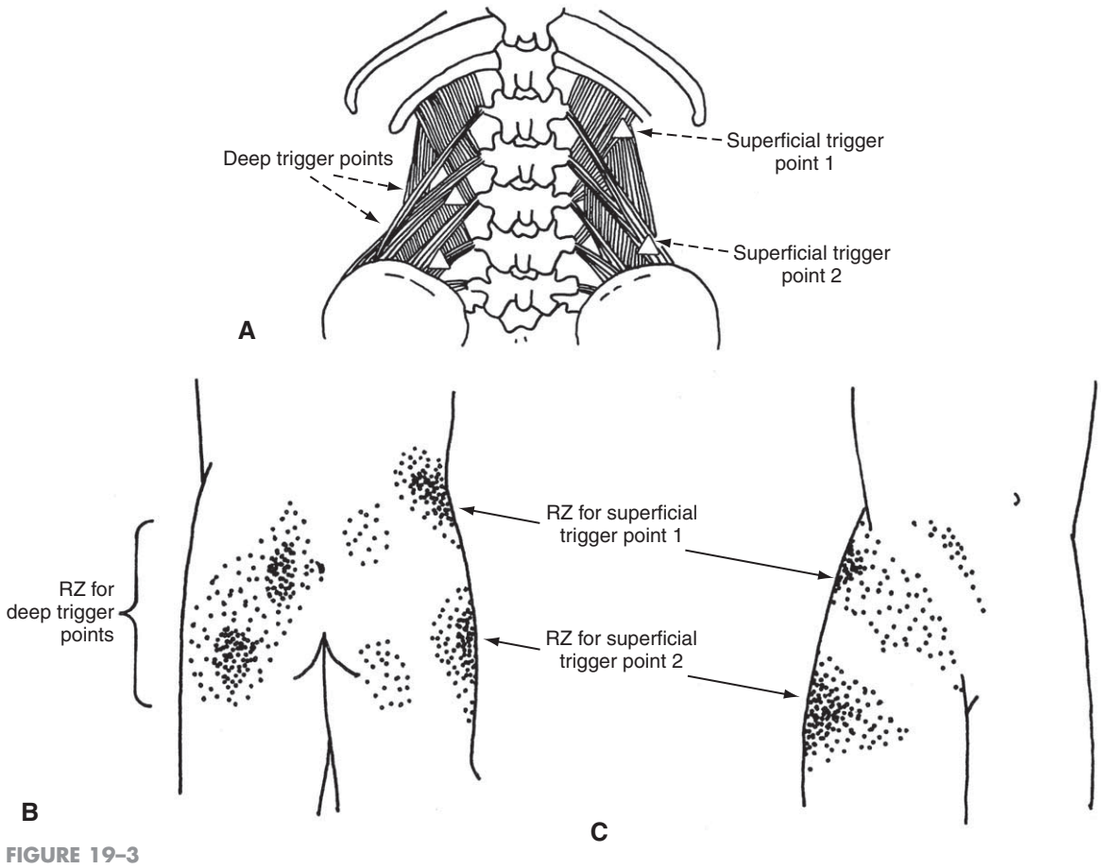


FIGURE 19–3

(A) The course of the quadratus lumborum muscle from its origin to insertion. Note the location of the TPs in the muscle. (B, C) Reference zones of the quadratus lumborum muscle.

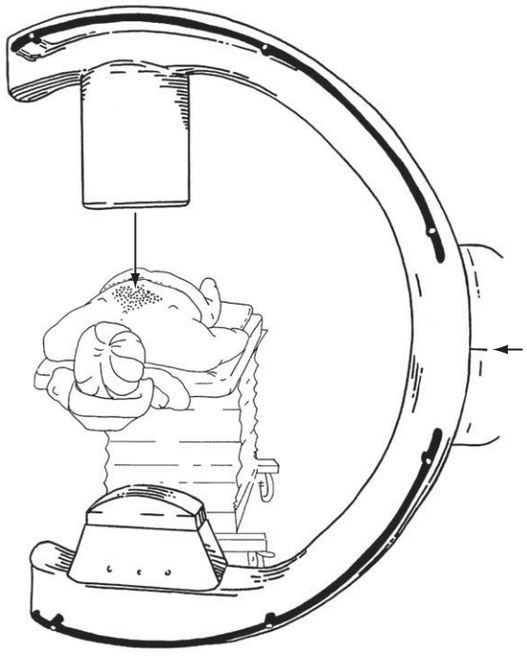


FIGURE 19–4

Patient lies in the prone position. The fluoroscope is positioned initially in the posteroanterior position to view the L3, L4, and L5 vertebrae.

On examination, pain should be gone on flexion and extension of the hip.

Quadratus lumborum muscle pain should be gone with flexion of the lumbosacral spine and rotation as if to tie the shoe or pick up the newspaper from the floor.

COMPLICATIONS

The complications of psoas and quadratus lumborum muscle injection include increased pain in the area of the injection, infection, or hematoma in the muscles.

CLINICAL PEARLS

When injecting the quadratus lumborum muscle, avoid potential damage by using needles that are not too long.

Lateral views should be used to confirm the needle tip in the muscular tissue.

Psoas muscle injection should be at the lateral aspect of the transverse processes to avoid the nerve roots and the epidural space.

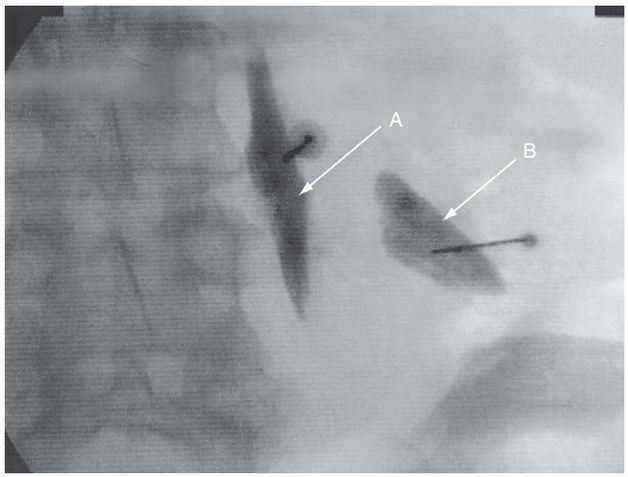


FIGURE 19-5
Position of the C-arm for viewing the lateral aspect of the lumbar spine.

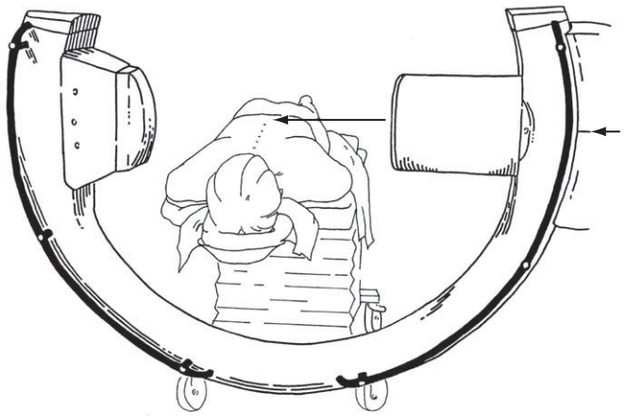


FIGURE 19–6
Fluoroscope image shows the posteroanterior view of the lumbar spine. Note that arrow A indicates the spread of the contrast material in the psoas muscle at L3-L4. Arrow B shows the spread of the contrast material in the quadratus lumborum muscle.

EFFICACY

Aside from anecdotal reports suggesting that psoas and quadratus lumborum muscle injections are helpful to relieve pain, several studies have been conducted to evaluate the effectiveness of various treatment modalities. In a small-scale, randomized double-blinded study, the effect of botulinum A for the treatment of myofascial pain was found to be superior to a placebo.[17] In 2000, Porta[17] reported the results of a single-blind study investigating myofascial pain treatment with botulinum A and concluded that botulinum A was more effective than methylprednisolone. A randomized, double-blind study conducted in 2001 confirmed the efficacy of botulinum A

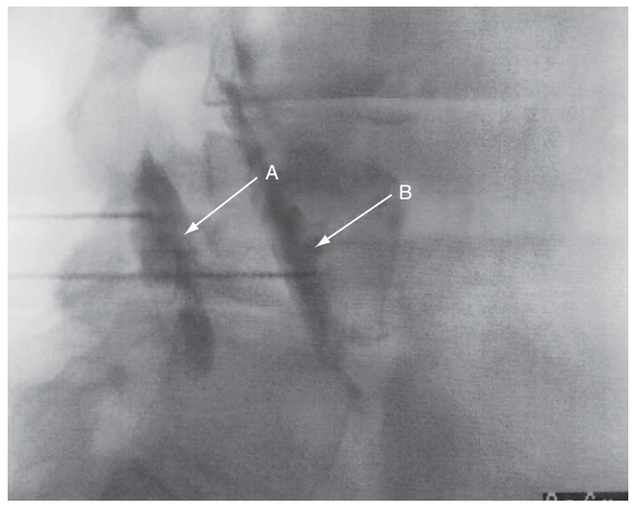


FIGURE 19–7
Fluoroscope image shows the lateral aspect of the lumbar spine. Note that arrow A indicates the image of the contrast material in the quadratus lumborum muscle at the level of and posterior to the transverse processes. Arrow B shows the image of the contrast material in the psoas muscle at the mid and anterior one-third of the vertebral bodies.

injections into the paravertebral muscles to relieve pain and improve function.[18] Studies investigating the treatment of TP pain have reviewed the use of steroids, nonsteroidal anti-inflammatory drugs, and antidepressants with varying results.[19]

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SECTION

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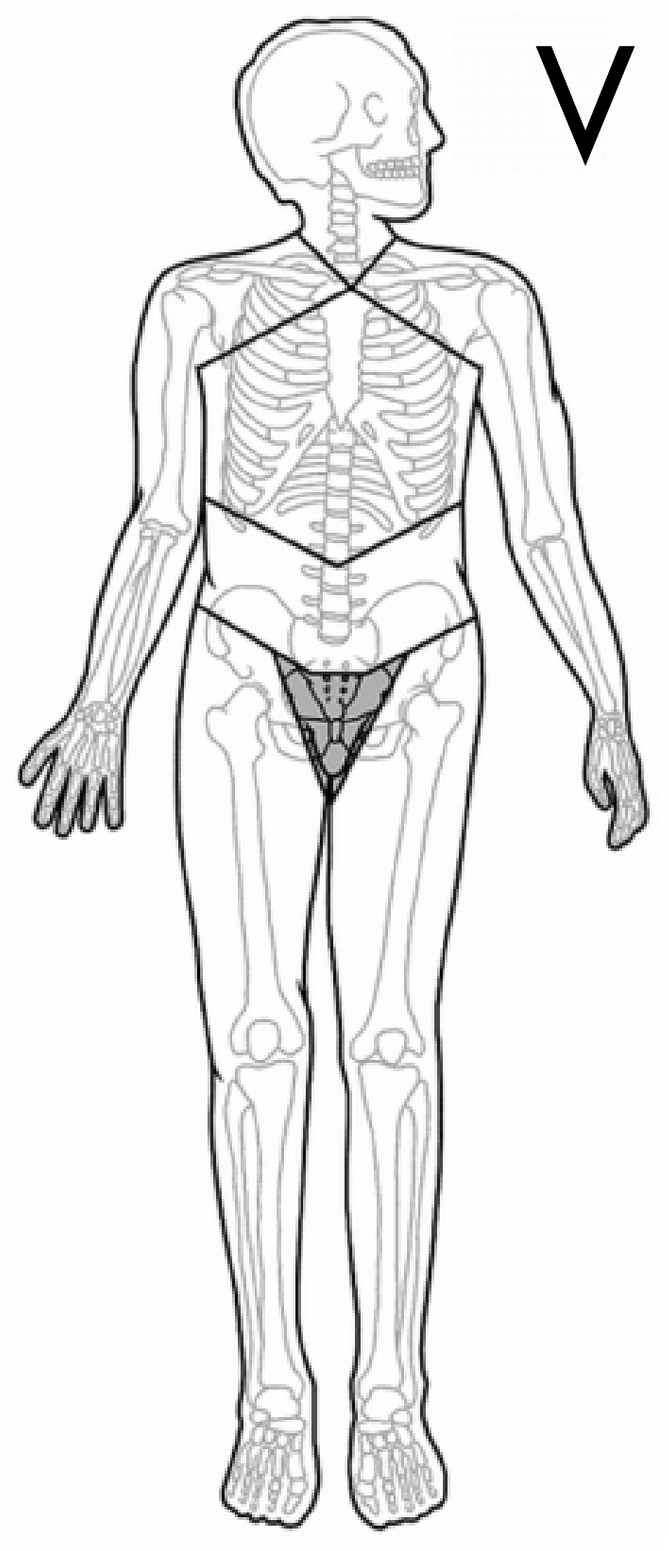

PELVIS

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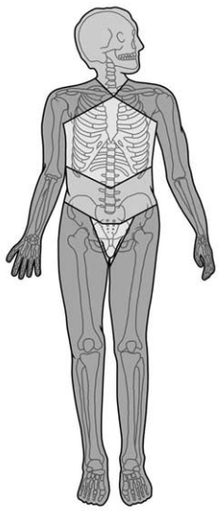


20 Pelvic Somatic Blocks

STEVEN D. WALDMAN

ANATOMY

The pudendal nerve is made up of fibers from the S2, S3, and S4 nerves (Figure 20-1). The nerve passes inferiorly between the piriformis and coccygeal muscles. Along with the pudendal vessels, the pudendal nerve leaves the pelvis via the greater sciatic foramen.[1] It then passes around the medial portion of the ischial spine to re-enter the pelvis via the lesser sciatic foramen. The pudendal nerve is amenable to blockade at this point via the transvaginal approach. The nerve then divides into three terminal branches: (1) the inferior rectal nerve, which provides innervation to the anal sphincter and perianal region; (2) the perineal nerve, which supplies the posterior two thirds of the scrotum or labia majora and muscles of the urogenital triangle; and (3) the dorsal nerve of the penis or clitoris, which supplies sensory innervation to the dorsum of the penis or clitoris.

INDICATIONS

Pudendal nerve block via the transperineal approach is used for pudendal nerve block in men and in women when tumor or radiation-induced scarring precludes use of the transvaginal approach. It can be used in the evaluation and management of pelvic pain believed to be subserved by the pudendal nerve. The technique is also useful to provide surgical anesthesia for surgery on the labia or scrotum including lesion removal and laceration repair. Pudendal nerve block with local anesthetic can be used as a diagnostic tool when performing differential neural blockade on an anatomic basis in the evaluation of pelvic pain when peripheral nerve injury or entrapment versus radiculopathy or plexopathy is being evaluated.[1] If destruction of the pudendal nerve is being considered, this technique is useful as a prognostic indicator of the degree of motor and sensory impairment that the patient may experience. Pudendal nerve block with local anesthetic may be used to palliate acute pain emergencies, including postoperative pain relief, while waiting for pharmacologic methods to become effective. Pudendal nerve block with local anesthetic and a steroid is also useful in the treatment of persistent pain after perineal trauma when the pain is believed to be secondary to inflammation or entrapment of the pudendal nerve. Such problems can occur after "straddle injuries" or during forceps deliveries. Pudendal nerve block with local anesthetic and steroid is also useful in the palliation of pain of malignant origin arising from tumors invading the labia or scrotum or the pudendal nerve itself. The technique may also be useful in palliation of persistent rectal, vulvar, or vaginal pain itching that has not responded to topical therapy.[2,3]

Destruction of the pudendal nerve is occasionally indicated for the palliation of persistent pelvic or rectal pain after blunt or open trauma to the pelvis or persistent pain mediated by the pudendal nerve after obstetric deliveries or transvaginal surgery or in the palliation of pain of malignant origin. Pudendal nerve block using a 25-gauge needle may be carried out in the presence of coagulopathy or anticoagulation, albeit with an increased risk of ecchymosis and hematoma formation.

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represents a relatively strong contraindication to the performance pudendal nerve block. Local infection involving the area of the pudendal nerve is also a contraindication to the performance of pudendal nerve block.

EQUIPMENT

- Peripheral nerve block tray
- 22-gauge, 13-cm styletted needle
- 25-gauge, 1-1/2-inch needle
- 18-gauge, 1-1/2-inch needle
- 12 ml sterile syringe

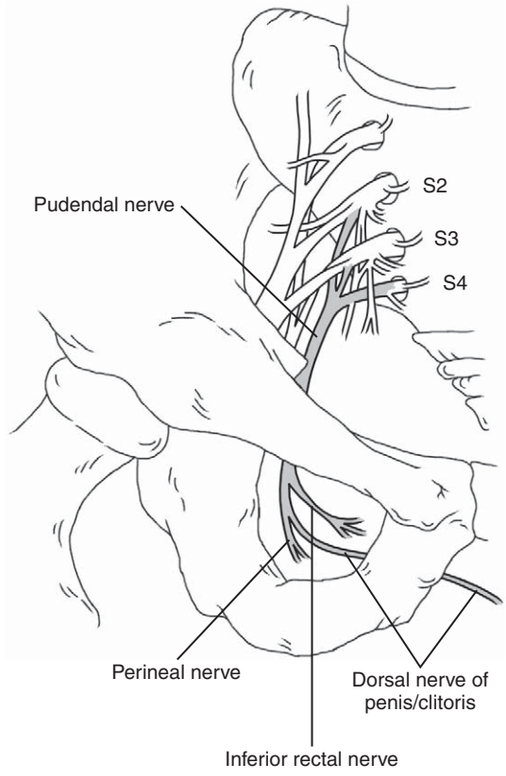


FIGURE 20–1
Anatomy of the pudendal nerve.

DRUGS

- 1% preservative-free lidocaine (for diagnostic or prognostic block)
- 0.25% preservative-free bupivacaine (for therapeutic block)
- Depot methylprednisolone (for therapeutic block)
- 6.5% aqueous phenol (for chemical neurolytic block)

PROCEDURE

The patient is placed in the lithotomy position. The ischial tuberosity is identified by palpation via transvaginal or transrectal palpation, and an area 1 inch lateral and 1 inch posterior to the tuberosity is then prepared with antiseptic solution. A skin wheal is raised at this point with local anesthetic. The index finger of the pain specialist's nondominant hand is inserted into the rectum to identify the ischial spine. A 6-inch needle is then placed through the previously anesthetized area and directed toward the ischial spine. The finger placed in the rectum will help guide the needle just beyond the ischial spine (Figure 20-2). After careful aspiration for blood is negative, 10 ml of 1.0% lidocaine are injected. An additional 3–4 ml of local anesthetic may be injected as the needle is withdrawn to ensure blockade of the inferior rectal nerve.

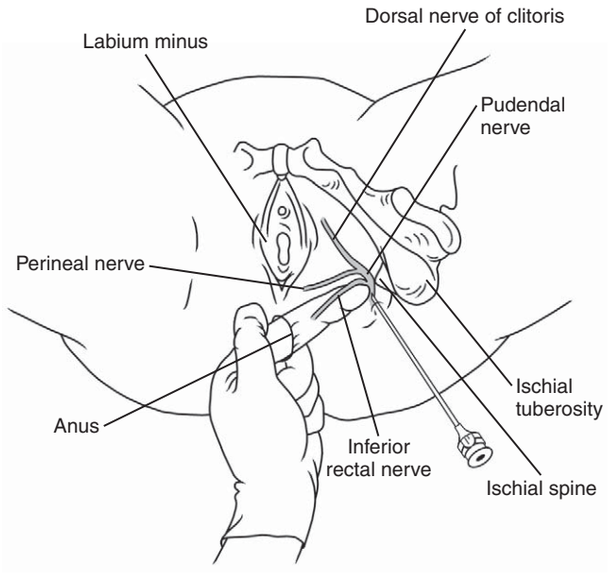


FIGURE 20–2
Palpation of the ischial spine via the rectum.

Computerized Tomography–Guided Technique

The patient is placed in the prone position on the computerized tomography (CT) table, and 5-mm transverse CT images are obtained from the head of the femur to the ischium.[4] The ischial spine, sacrospinous, and sacrotuberous ligaments are then identified (Figure 23-3).

The skin is then prepped with antiseptic solution, and after adequate analgesia of the skin and subcutaneous tissue with local anesthetic, a 22-gauge, 13-cm needle is placed transgluteally under CT guidance aiming toward ischial spine to block the pudendal nerve. After careful aspiration, 5 ml of local anesthetic are injected (Figure 20-4 A and B).

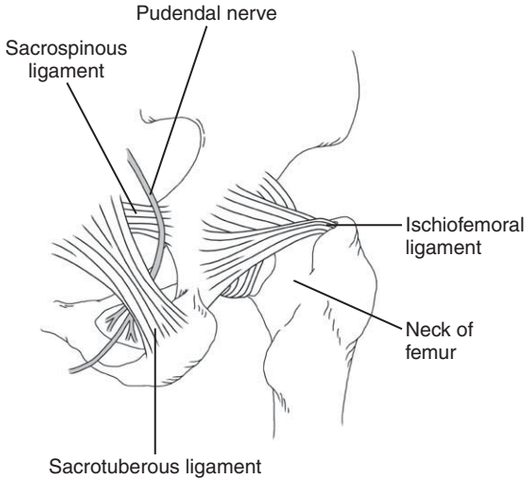
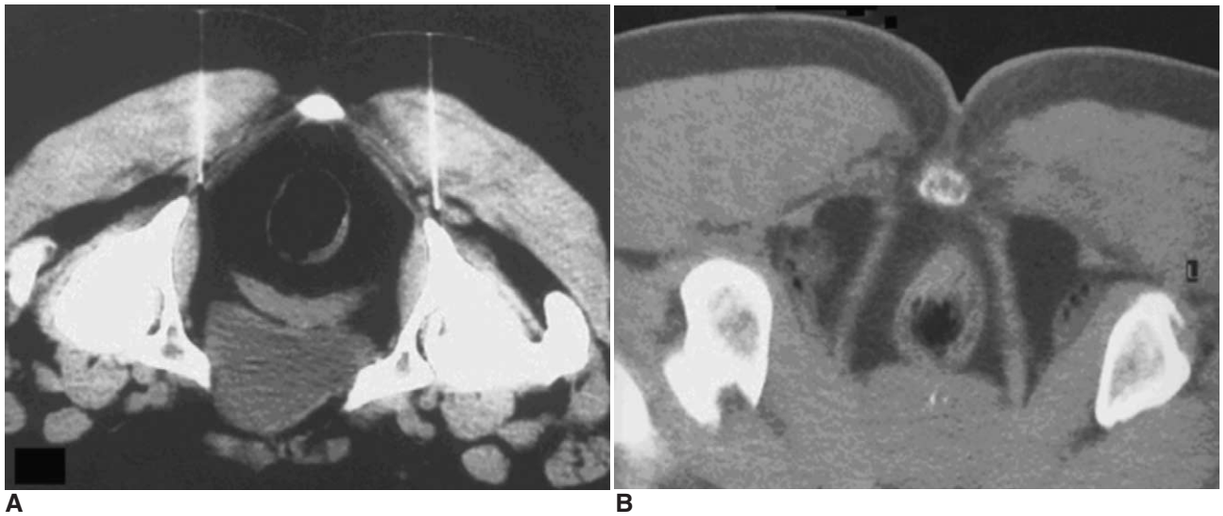


FIGURE 20–3
Landmarks to identify the course of the pudendal nerve.



A B

FIGURE 20–4

Site of injection of local anesthetic. (A) Preinfiltration. (B) Postinfiltration. (Images courtesy of Denis Thoumas, MD.)

If the pain has an inflammatory component, the local anesthetic is combined with 80 mg of methylprednisolone and is injected in incremental doses. Subsequent pudendal nerve blocks are carried out in a similar manner, substituting 40 mg of methylprednisolone for the initial 80-mg dose.

COMPLICATIONS

The proximity of the pudendal nerve to the pudendal artery and vein makes the potential for intravascular injection a distinct possibility. In spite of proximity to the rectum, infection after pudendal nerve block does not appear to be a problem, although, theoretically, infection and fistula formation, especially in patients who are immunocompromised or have received radiation therapy to the perineum, could represent a devastating and potentially life-threatening complication to this block.

CLINICAL PEARLS

Pudendal nerve block is a simple technique that can produce dramatic relief for patients suffering from the previously mentioned pain complaints. Destruction of the pudendal nerve has been shown to provide long-term relief for patients suffering from pain secondary to invasive tumors of the vulva and scrotum.

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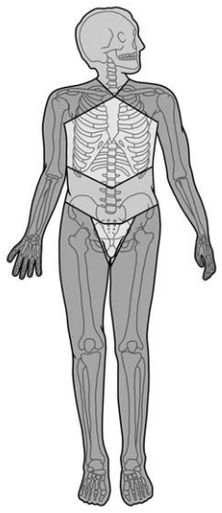
C H A P T E R



21 Pelvic Sympathetic Blocks

SERDAR ERDINE AND SULEYMAN OZYALCIN

HYPOGASTRIC PLEXUS BLOCK

HISTORY

The first attempts to interrupt sympathetic pathways from the pelvis have been made by Jaboulay in France[1] and Ruggi in Italy[2] in 1899. In 1921, Leriche[3] performed a periarterial sympathetectomy of the internal iliac arteries on a patient with "pelvic neuralgia" with good results. Cotte[4] presented a more systematic review in 1925. The first investigators to report superior hypogastric plexus block were Plancarte and colleagues.[5] Most recently, laparoscopically performed transection of the superior hypogastric plexus and stripping of the hypogastric nerves were reviewed by Chen.[6] De Leon-Casasola and associates[7] repeated the same study on 26 patients, but with fluoroscopy. Waldman and Wilson[8] described the use of computerized tomography (CT) scan to optimize needle placement. Kanazi and Frederick[9] have proposed an anterior approach. Other approaches to the superior hypogastric plexus have been reported, including a transvaginal approach[10] and a transdiscal technique.[11,12]

ANATOMY

Superior Hypogastric Plexus

The superior hypogastric plexus is the extension of the aortic plexus in the retroperitoneal space, below the aortic bifurcation (Figure 21-1). Pelvic visceral afferent and efferent sympathetic nerves from the branches of the aortic plexus, and fibers from L2 through L3 splanchnic nerves form the superior hypogastric plexus.[13]

The plexus receives fibers from the lumbar sympathetic nerve of L5. This plexus is in continuity with the celiac plexus and lumbar sympathetic chains above and innervates the pelvic viscera (bladder, uterus, vagina, prostate, rectum, etc.) via the hypogastric nerves.

It is situated on the anterior aspect of L5-S1 and on the disc between L5 and S1. It lies close to the sympathetic chain at this level, the common and internal iliac arteries, and veins on each side. The ureter is located just lateral to these structures in close proximity to the anterolateral aspect of the L5 vertebral body. It contains almost exclusively sympathetic fibers. As it courses distally, the superior hypogastric plexus converges and forms the hypogastric nerve. The hypogastric nerve follows the internal iliac artery and vein and connects with the inferior hypogastric plexus at both sides of the pelvis. Hypogastric nerves carry sympathetic fibers only. Since the aorta is located more toward the left, the superior hypogastric plexus and the hypogastric nerves are shifted somewhat to the left as well. Anatomical location of the superior hypogastric plexus and the hypogastric nerves, sympathetic predominance of the fibers of these plexus, and the role of the plexus in transmission of the majority of pain signals from the pelvic viscera should make these structures an ideal target for neural blockade.

The inferior hypogastric plexus or pelvic plexus in turn consists of fibers from the hypogastric nerves (which are predominantly sympathetic), postganglionic sympathetic fibers from the sacral splanchnic nerves, and parasympathetic fibers from the pelvic splanchnic nerves, the cell bodies of which are located at S2, S3, and S4 levels.[14] The right and left inferior hypogastric plexus are intertwined with the viscera of the pelvis, and for this reason, they cannot be isolated and separately blocked.

INDICATIONS

Superior hypogastric plexus block is indicated for a group of patients that have gynecological disorders with pain. The most common disoders in these patients are inflammatory pelvic disorders endometriosis, adhesions, and chronic pain.

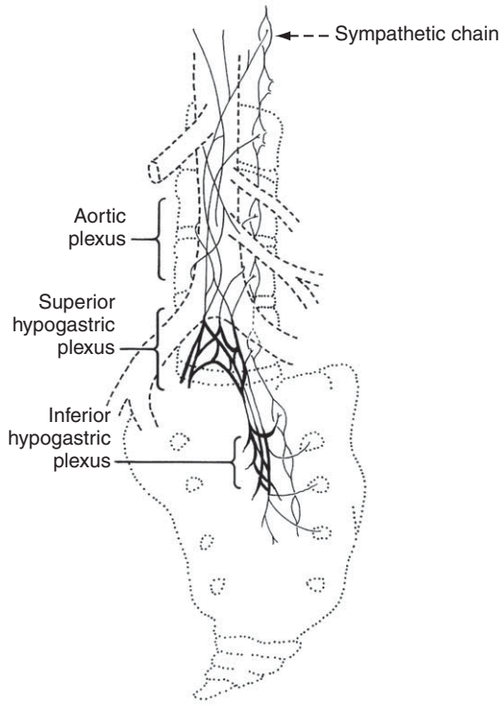


FIGURE 21–1
Anatomy of the hypogastric plexus.

The second group consists of nongynecological patients. Examples of disorders in this group are patients with interstitial cystitis, irritable bowel syndrome, and chronic pain after a surgery like suprapubic prostatectomy.

The third group of patients comprises those with neoplasms of the pelvic viscera.

CONTRAINDICATIONS

- Local infection
- Coagulopathy

EQUIPMENT

- 25-gauge, 3/4-inch infiltration needle
- 20-gauge, RFTC a curved blunt needle
- 16-gauge, 1-3/4-inch angiocath as an introducer needle
- 22-gauge, 6-inch needle
- 18-gauge, 1-1/2-inch needle for drawing up drugs and skin puncture
- 3-cc syringe
- 10-cc, three-ring syringe
- 10-cc syringe
- Intravenous T-piece extension set
- Metal Markell clamp

DRUGS

- Omnipaque (iohexol) radiopaque contrast solution
- Preservative-free normal saline (0.9% saline)
- 1.5% lidocaine for infiltration
- 0.5% bupivacaine or ropivacaine preservative-free
- 2% lidocaine preservative-free
- Water-soluble steroids (methylprednisolone, triamcinolone diacetate)
- 1-triple antibiotic ointment for skin

PREPARATION OF PATIENT

- Preprocedure medication: standard recommendations by the American Society of Anesthesiologists (ASA) for conscious sedation
- Monitoring: standard ASA-recommended monitoring (EKG, blood pressure, pulse oximetry, etc.)

PROCEDURE

Patients are placed in a prone position with the fluoroscope in the posteroanterior position (Figure 21-2).

Site of Needle Entry

The L4-L5 spinous processes are identified. The skin is marked 5–7 cm lateral from the midline at this point (Figure 21-3).

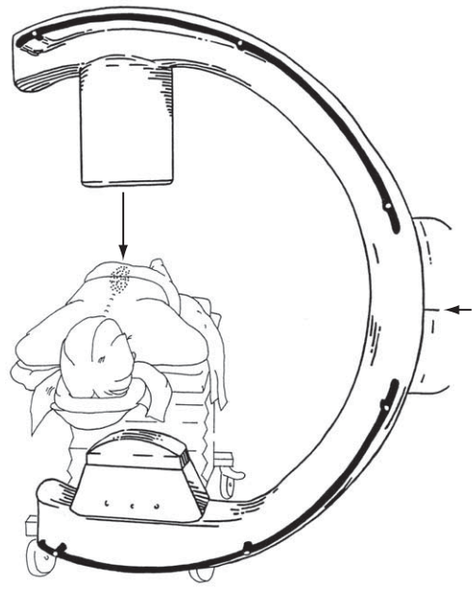


FIGURE 21–2
Patient is placed in the prone position. The beam of the C-arm fluoroscope is directed toward L5-S1 vertebral level in the posteroanterior view.

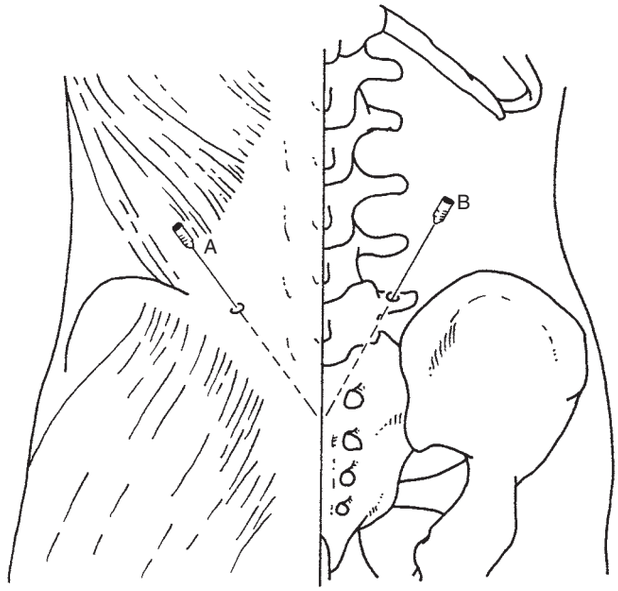


FIGURE 21–3

Lateral approach: The line drawing of the landmarks in the low back region to identify the needle point entry for hypogastric plexus block. (A) Note the needle direction parallel to medial iliac border traversing toward L5-S1. (B) Needle touching the transverse process and slipping below it to L5-S1. (Adapted from Plancarte R, et al: Hypogastric plexus block: retroperitoneal approach. *Anesthesiology* 71A:739, 1989, with permission.)

Technique of Needle Entry

LATERAL APPROACH

Draw a line lateral to the L4-L5 interspace. Use a 15-cm, 20-gauge needle. Direct the needle from midline to items lateral approximately 45 degrees medial and caudad to miss the transverse process of L5 and the sacral ala. In the lateral radiographic image, the needle tip needs to be at the anterior junction of L5-S1. In the anteroposterior view, the needle must be no more than 1 cm from the bony outline of L5-S1 (Figure 21-4).

MEDIAL APPROACH

Rotate the fluoroscope C-arm 15 degrees caudally so that the x-ray beam looks into the pelvis (Figure 21-5). This view enlarges the space between the L5 transverse process, sacral ala, and the posterior superior iliac spine. Under fluoroscopy using the 15-cm, 20-gauge needle, mark the most inferior and lateral part of this bone-free space. Place the needle and direct it medially and slightly caudad (Figure 21-6). Rotate the C-arm to a lateral view and observe the needle passing below transverse process and cephalad to the superior part of the L5 neural foramen. When the anterior edge of L5 vertebral body is reached, confirm this position by taking another AP view (Figure 21-7). Next, aspirate; and if negative for blood, inject 4–5 ml of water-soluble contrast material. The contrast should be contained within the lateral bony edge, anterior to the psoas muscle, and above the sacral nerve roots (Figure 21-8).

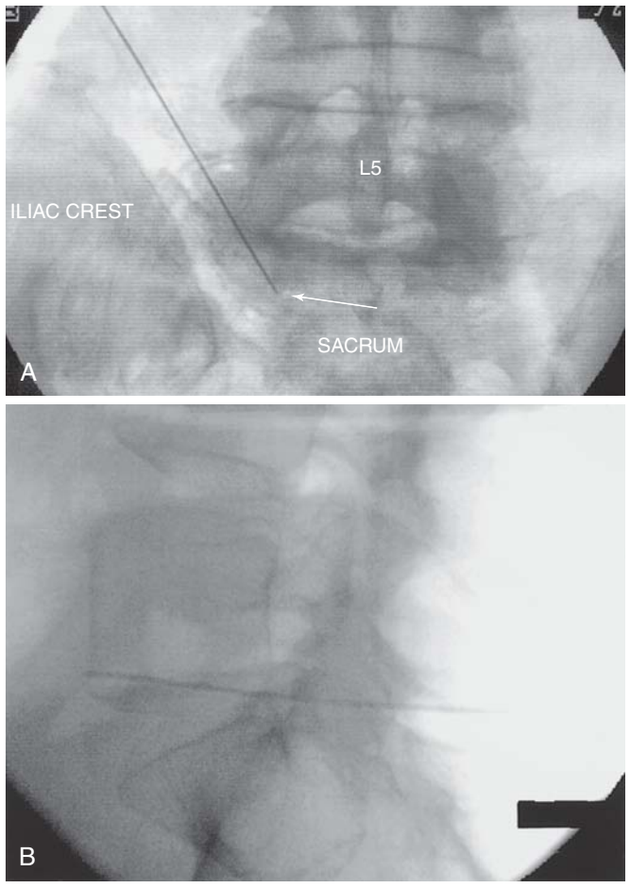


FIGURE 21–4

(A) Posteroanterior view of 6-inch, 22-gauge needle placed at the L5-S1 level parallel to the iliac medial border (lateral approach). (B) Lateral views with the needle in position.

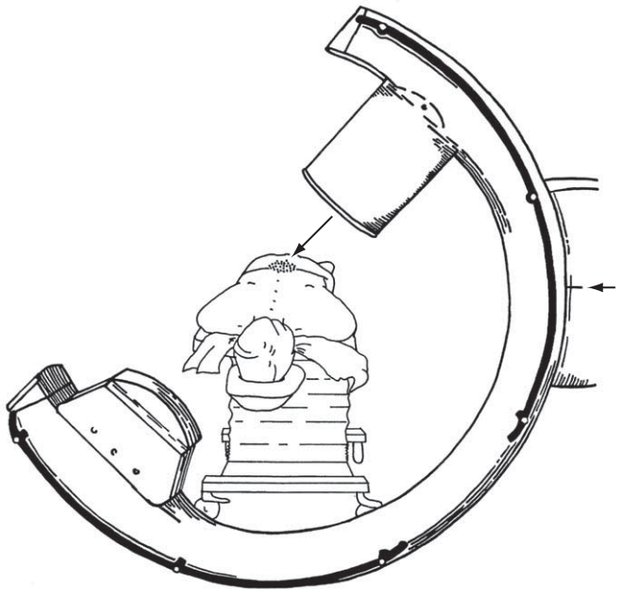


FIGURE 21–5

Patient in prone position and fluoroscope over L5-S1 for paraspinous (medial) approach.

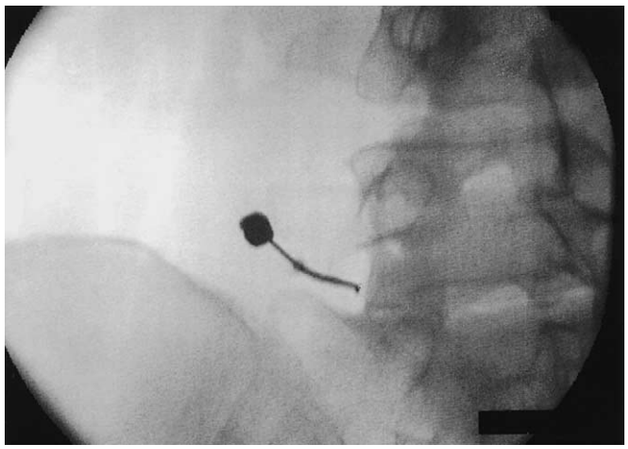


FIGURE 21–6

Medial paraspinous approach: Oblique view of the needle (curved blunt) at L5 level for hypogastric plexus block. The needle has traversed halfway to the site.

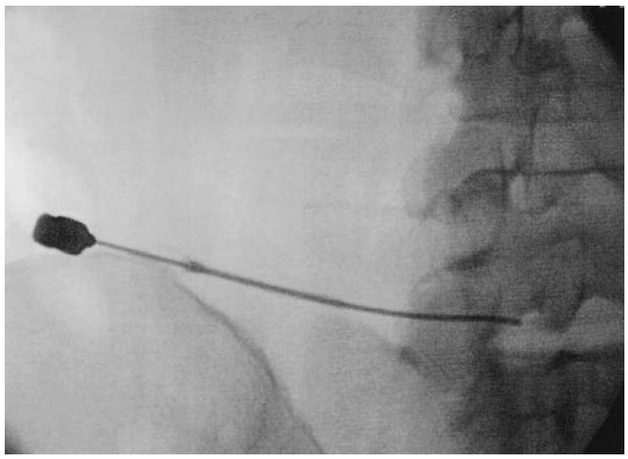
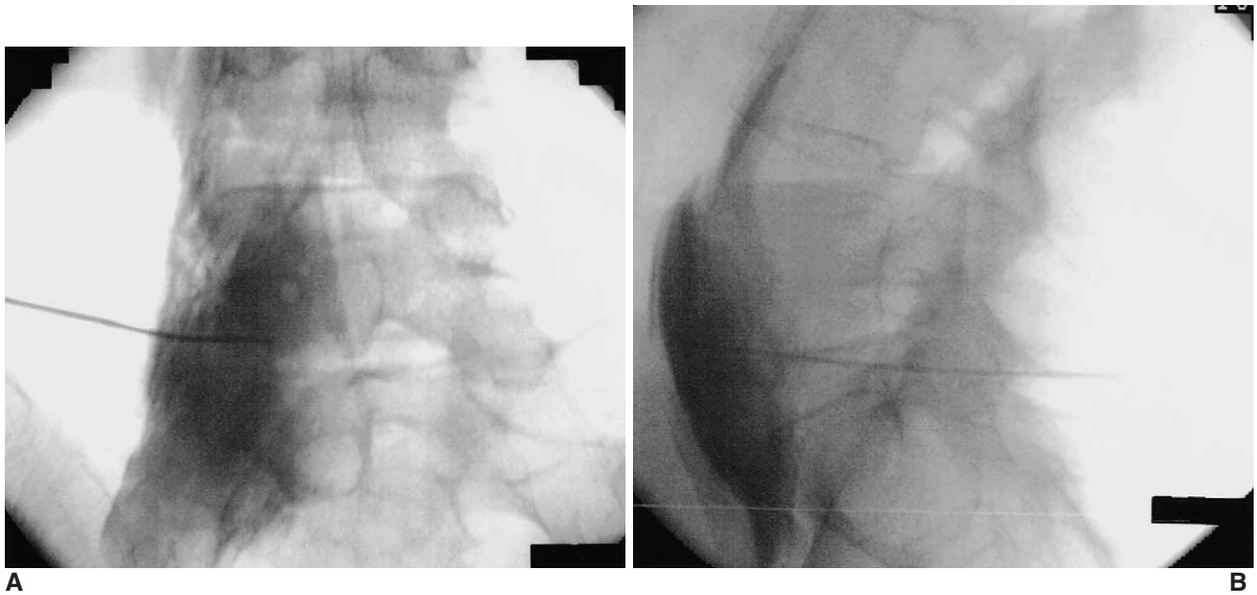


FIGURE 21–7

Medial paraspinous approach: final position of the needle at L5 for hypogastric plexus block (posteroanterior view).



A B

FIGURE 21–8

(A) Posteroanterior view showing the dispersion of contrast Omnipaque (iohexol) solution to confirm the correct needle position. Note the solution spreading vertically hugging the spine. (B) Lateral view showing contrast solution spreading over the L5-S1.

Transdiscal Approach for Hypogastric Plexus Block

The patient is placed in the prone position with a pillow beneath the iliac crest to facilitate opening of the interdiscal space. The L5-S1 interdiscal space is identified under fluoroscopy. Next, the fluoroscopy is placed in an oblique fashion and angled at 15–20 degrees or more for obtaining the best image of the disc to align the inferior endplates. In order to do so, a cephalad trajectory is needed. The entry point is approximately 5–7 cm from the midline.[12]

After local anesthetic infiltration of the skin and the subcutaneous tissues with 2% lidocaine, a 22-gauge, 10-cm block needle is introduced by tunnel vision lateral to the inferior aspect of the facet joint (Figure 21-9). The needle is advanced through the disc. While entering the disc, 0.5 ml of radiopaque iohexol solution is administered to verify needle position within the disc by lateral and anteroposterior views (Figure 21-10). The needle is then advanced further under lateral fluoroscopic control, and a 5-ml syringe with saline attached for loss of resistance (Figure 21-11). When the needle passes outside the L5-S1 interdiscal space, 3 ml of iohexol is administered to verify its final position. The dye is spread with a direct-line image (in a vertical plane) at that position (Figure 21-12).

Five milliliters of 10% aqueous phenol are given through the needle followed by 0.5 ml of air before drawing

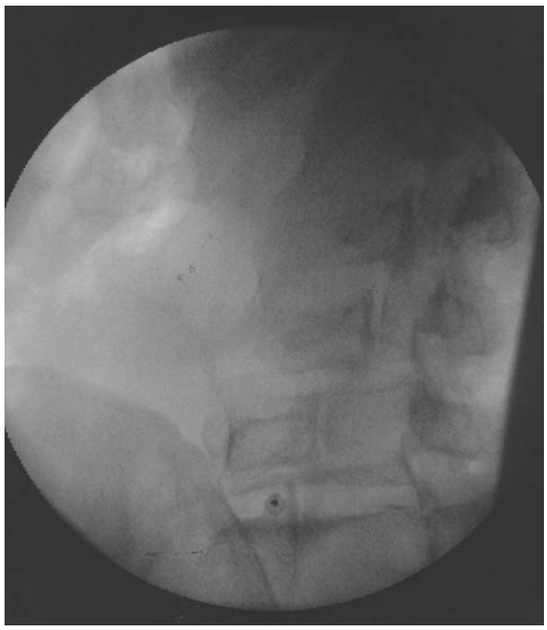


FIGURE 21–9
The patient is positioned prone; the fluoroscope is rotated from the AP position to the oblique view, opening the L5-S1 disc. The needle inserted in the lateral part of the disc in a tunnel view.

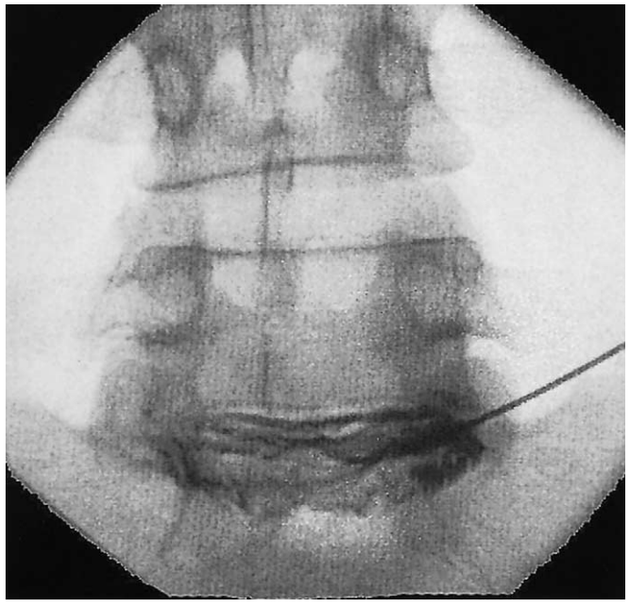


FIGURE 21–10
Contrast material in the disc confirming needle entry in the disc.

back to prevent the spread of the neurolytic solution within the disc material. While further drawing back the needle, cephazolin 50 mg in 1 ml are administered to the disc to prevent discitis. One gram of cephazolin as a prophylactic antibiotic is given intravenously 30 minutes before the procedure.[12]

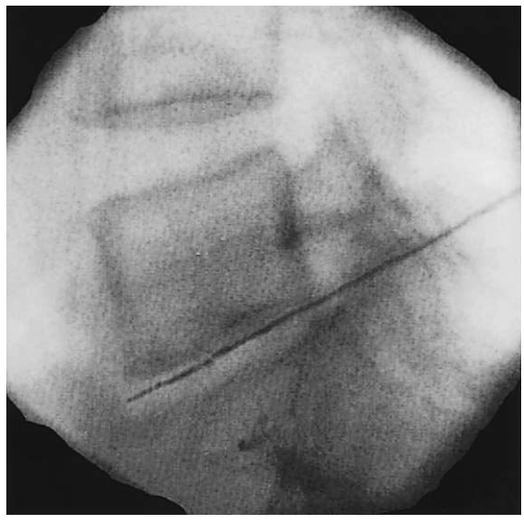


FIGURE 21–11
In this lateral view the needle has traversed through the L5-S1 disc to the anterior edge of the disc.

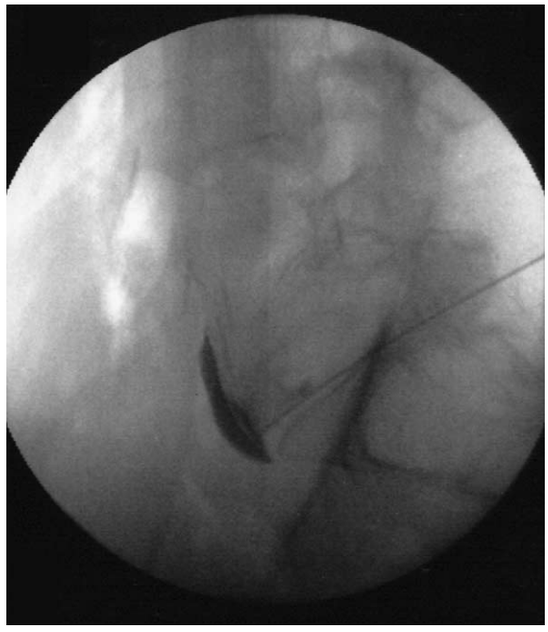


FIGURE 21–12
The needle has now passed the anterior edge of the disc. 3 mls of contrast material has been injected, which shows in this radiograph spreading vertically over L5-S1 region. This confirm the correct position for the Intradiscal approach.

Anterior Approach for Hypogastric Plexus Block

The patient is placed in the supine position with the table in 15 degrees of Trendelenburg. The vertebral body of L5 can occasionally be palpated. A skin wheal is placed just 3–5 cm below the umbilicus. A 6-cm, 22-gauge needle is placed perpendicular to the floor and advanced until bony contact is made. At this time, 20–30 ml of bupivacaine 0.25% are injected. Even though this technique has been

performed occasionally, it is not recommended due to high risk of infection.

For diagnostic blockade, 6–8 ml of bupivacaine 0.25% are injected through each needle.

For neurolysis, commonly 6–10% phenol with or without Omnipaque solution is injected on each side up to 10 ml.

CLINICAL PEARLS FOR SUPERIOR HYPOGASTRIC BLOCK

Measure the required needle length by placing the needle against the skin and taking a posteroanterior image. Bowel preparation prior to block is helpful to evacuate bowel content and gases.

EFFICACY

In the first published study on hypogastric block, patients with neoplastic involvement of pelvic viscera secondary to cervical, prostate, and testicular cancer or radiation injury were treated with neurolytic superior hypogastric plexus block. Pain was significantly reduced or eliminated in all cases, and no serious complications occurred.[5]

In a study by Erdine et al.[12] using the transdiscal approach, 20 patients—10 men and 10 women—were included. Twelve patients (60%) had statistically significant pain relief immediately after the block ($p < 0.05$). The mean visual analog scale (VAS) value was 7.25 (±1.11) before the procedure. VAS values decreased significantly at 24 hours and 1 month after the procedure (4.7 ± 1.03 and 2.8 ± 0.83, respectively; $p<0.05$). There was no significant difference in VAS values obtained 1, 2, and 3 months after the procedure.

The daily analgesic requirement decreased significantly after the block (180 ± 20.51 and 68 ± 15.07 mg/day of codeine, respectively; $p < 0.05$).

Fifteen patients (75%) were satisfied after the block. Patient satisfaction increased significantly 1 month after the block. In five patients (25%), no pain relief was observed and a spinal drug delivery system was implanted for spinal opioid administration 1 month after the procedure. Disc puncture was possible without difficulty in all patients, and there were no associated complications such as discitis or disc rupture.[12]

The largest study to date was carried out by Plancarte and De Leon-Casasola.[15] They studied 227 patients who had chronic pelvic pain due to cancer. By explicitly eliciting a history of vague, dull, poorly localized pain, the investigators attempted to select patients with predominantly visceral pain. The criteria for a successful diagnostic block were pain reduction of at least 50% lasting longer than 4 hours. Successful neurolysis was defined as a 50% pain reduction, a 40% reduction in use of opioid medication, and duration of effect of at least 3 weeks. Of 227 patients, 115 (51%) reported good pain relief after therapeutic neurolysis of the superior hypogastric plexus. Of the 227, a total of 159 reported good pain relief after diagnostic blockade. Limiting neurolysis to these positive responders, neurolysis was successful in 72%. A mean reduction in analgesic requirement of 43% was found in these patients. No major complications were reported. The investigators observed that effectiveness of the procedure depended mainly on the central position of the agent at L5-S1. Second neurolysis after initial failure of the procedure proved to be effective and increased the overall success rate.

At the Institute for Pain Management at the Texas Tech Health Sciences Center (TTUHSC), superior hypogastric nerve blocks have been implemented for more than 10 years. The technique used at the TTUHSC is similar to the technique described by Plancarte and De Leon-Casasola.[15] A unilateral or bilateral technique is used. Because of the predominance of the plexus on the left side, the left side is always included. A blunt curved needle is used to reduce the risk of trauma to neurovascular structures. A survey was performed on patients who had undergone superior hypogastric plexus block over a 4-year period. Twenty-two patients enrolled in this study. If these blocks were successful, most patients underwent therapeutic neurolysis with 6% phenol. Pain scores before and after treatments were obtained. A block was considered positive if more than 50% pain relief was provided for more than 4 hours. Therapeutic neurolysis was considered positive if pain relief was greater than 50% and lasted longer than 1 month. Information on reduction of narcotic medication, improvement in functional status, and the occurrence of complications was obtained as well. Causes of pelvic pain were diverse and included endometriosis, adhesions, interstitial cystitis, and postprostatectomy pain. Forty-five percent of the patients (10/22) had a positive response to diagnostic blockade (Table 21-1). Subsequently, 11 patients underwent 6% phenol injection.

In a recent case report, Rosenberg and coworkers[16] reported on the efficacy of this block in a patient with severe chronic nonmalignant penile pain after transurethral resection of the prostate. Although the patient did not receive a neurolytic agent, a diagnostic block performed with 0.25% bupivacaine and 20 mg of methylprednisolone acetate was effective in relieving the pain for more than 6 months. The usefulness of this block in chronic benign pain conditions has not been adequately documented.

TABLE 21–1 Number of Patients with Nonmalignant Visceral Pain Responding to Diagnostic and Neurolytic Superior Hypogastric Plexus Block

Pain Reduction (%)	0%	<50%	>50%	100%
Diagnostic block	8/22	4/22	6/22	4/22
Neurolytic block	3/11	4/11	3/11	1/11

COMPLICATIONS

Table 21-2 lists common problems and solutions associated with hypogastric plexus block. The proximity of the iliac vessels (arteries and veins) to the needle paths increases the potential for intravascular injection. This anatomic relationship also makes hematoma formation possible. If the position of the needle tip is not accurately verified, both intramuscular and intraperitoneal injection are possible. Even when the needle is inserted correctly, paraspinous muscle spasm may result owing to needle-induced paraspinous muscle irritation. This usually lasts only a few days. Less frequent problems are lumbar or sacral somatic nerve injury and renal or ureteral puncture. It is advisable to caution the patient about the potential for bowel or bladder habit changes, as well as decreases in sexual function following the neurolytic superior hypogastric plexus block, despite the rarity of these side effects.[17]

A potential risk of discitis can occur with the intradiscal approach. The only possible complication of the transdiscal technique is the risk of discitis. However, recent literature has shown the risk of infection to be low (1–4%). Some investigators recommend the use of a suitable broad-spectrum antibiotic in a single prophylactic dose whenever the intervertebral disc is entered.[18,19]

The combined experience of more than 200 cases from the Mexican Institute of Cancer, Roswell Park Cancer Institute, and M.D. Anderson Cancer Center indicates that neurologic complications do not occur as a result of this block.[16]

CONCLUSION

The superior hypogastric plexus, an extension of the preaortic plexus, is easily accessible to blockade by local anesthetics and neurolytic agents. Several techniques have been described. Long-lasting pain relief with this procedure has been achieved in patients with pelvic cancer pain. However, there is a discrepancy between diagnostic and therapeutic blockade in patients with nonmalignant pain. Since diagnostic blockade can give significant pain relief in a large variety of patients, it is worthwhile to investigate new methods that provide lasting neural blockade of the superior hypogastric plexus and long-lasting relief of this devastating condition.

TABLE 21–2 Common Problems

Problem	Solution
Touching L5 nerve root	Redirect needle.
Intravascular spread of contrast even in face of negative aspiration	Redirect; if problem persists, abort procedure and repeat another day.
Needle tip too lateral	Withdraw and redirect.

GANGLION OF IMPAR BLOCK

HISTORY

The first report of interruption of the ganglion impar block for relief of perineal pain appeared in 1990.[20] Since the ganglion impar was first introduced, various modified techniques were described such as transsacrococcygeal ligament technique,[21] sacrococcygeal transdiscal approach,[22] paramedian approach, and the two-needle technique[23] by using fluoroscopic[1] and CT guidance.[24,25] For therapeutic purposes, deposteroid and local anesthetic combinations,[22] neurolytic agents such as phenol,[20] cryolesioning,[26] and heat lesioning by radiofrequency thermocoagulation[23] can be used.

ANATOMY

Each sympathetic trunk in the pelvic area is situated in front of the sacrum, medial to the anterior sacral foramina. There are four or five small sacral ganglia, connected by interganglionic cords, and continuing above with the abdominal portion.[27,28] The ganglion impar (also known as the ganglion of Walther or the sacrococcygeal ganglion) is the most caudal ganglion of the sympathetic trunk. The impar ganglion has gray nerve fibers that connect the ganglion to the spinal nerve but seems to lack white nerve fibers, which connect the spinal nerves to the ganglion in the thoracic and upper lumbar region.[27–29] Visceral afferents from the perineum, distal rectrm, anus, distal urethra, vulva, and distal third of the vagina converge at the ganglion impar.[29]

Ganglion impar marks the end of the two sympathetic chains (Figure 21-13A and B). Commonly, it is a single ganglion produced by the fusion of the ganglia from both sides. Because of this, it is usually located in the midline; however, it may also be lateral to the midline. Its location has also been reported from the anterior to the sacrococcygeal joint 1–2" at the eoccyx.[21,29,30] Although an anatomical description of this ganglion is included in virtually every single book of anatomy,[27] we have been unable to find a description of the areas that send afferent fibers to this ganglion. However, clinical experience has shown that blockage at this point may be effective against some types of pain in the perineal region.

INDICATIONS

Ganglion impar block can be useful in the evaluation and management of sympathetically mediated pain of the perineum, rectum, and genitalia.[25] Visceral pain or sympathetically maintained pain in the perineal area associated with the malignancies of the pelvis may be effectively treated with neurolysis of the ganglion impar. Theoretically, this procedure can also be applied for benign pain

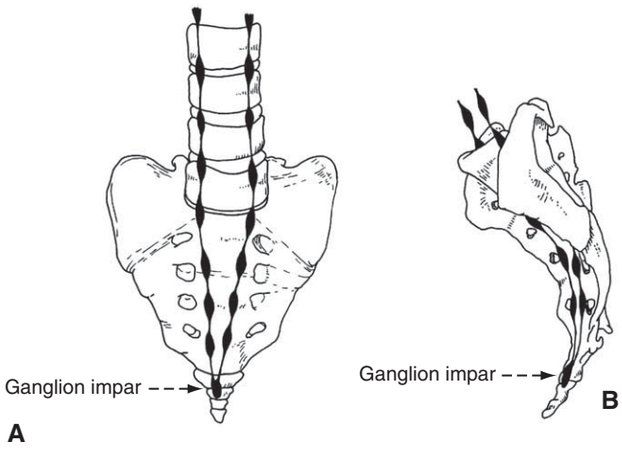


FIGURE 21–13

(A and B) The ganglion of impar is found in the area of sacrococcygeal ligament anteriorly. It is composed of the terminal confluence of the left and right sympathetic chains in the midline.

syndromes including pain secondary to endometriosis, complex regional pain syndromes, proctalgia fugax, radiation enteritis,[25] and postherpetic neuralgia.[31] Patients with a clinical picture of vague burning and localized perineal pain that is frequently associated with urgency may benefit from this block.

CONTRAINDICATIONS

- Local infection
- Coagulopathies
- Distorted anatomy

EQUIPMENT

- 25-gauge, 3/4-inch infiltration needle
- 22-gauge, 3-1/2-inch spinal needle. This needle can be shaped at the 60-degree angle or at 60- and 90-degree angles and curved[32] as shown in Figure 21-14
- Cryoprobe 8 for cryotherapy
- 15-cm radiofrequency needle (SMK) with 5-mm active tip,[23] for heat lesioning
- Intravenous T-piece extension

DRUGS

- 1.5% lidocaine
- 2% lidocaine
- 0.5% bupivacaine or ropivacaine
- Omnipaque 240 radiographic contrast solution (nonionic water-soluble contrast)
- Neurolytic: 6% phenol with contrast (5–10 ml)[20]

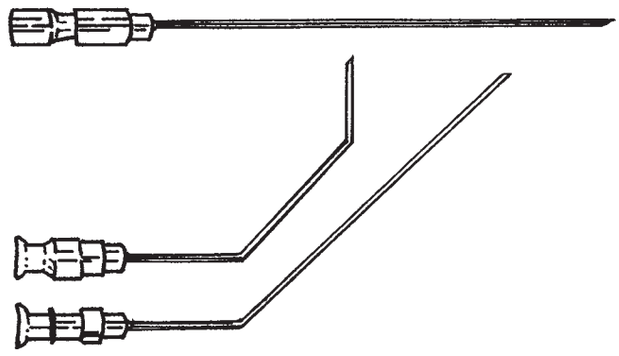


FIGURE 21–14

22-gauge spinal needles used for ganglion impar blocks. Note the alternative configuration of the needles depending on the angulation of the coccyx and the approach to be used.

PREPARATION OF PATIENT

Physical Examination

Perineum should be inspected for disease, infection, and ulceration. The patient must be evaluated for the ability to lie in either prone or lithotomy position.

Preoperative Medication

Use standard recommendations by the ASA for conscious sedation.

PROCEDURE

There are multiple approaches for this block.

Lateral Technique

This technique for performing the block is simple. As originally described,[20] the patient is placed in the lateral decubitus position with the hips flexed toward the abdomen (Figure 21-15). The right lateral decubitus is used if the operator is right-handed. Local anesthesia is injected at the level of the anococcygeal ligament, which is situated in the midway between the anus and the tip of the coccyx (Figure 21-16). A 22-gauge spinal needle that has been previously bent according to the curvature of the coccyx (see Figure 21-14) is then introduced, while efforts are made to maintain the tip of the needle in the midline and outside the posterior rectal wall. Inserting the index finger in the rectum facilitates placement of the needle's tip at the level of the sacrococcygeal junction. This technique can be quite uncomfortable in the patient with rectal pathology and also make it difficult to maintain sterility during the procedure.[31] Two milliliters of water-soluble contrast medium and biplanar fluoroscopy are used to verify the appropriate needle placement (Figures 21-17 and 21-18). Neurolysis is then performed with 4–6 ml of 6–10% phenol dissolved in radiographic contrast.

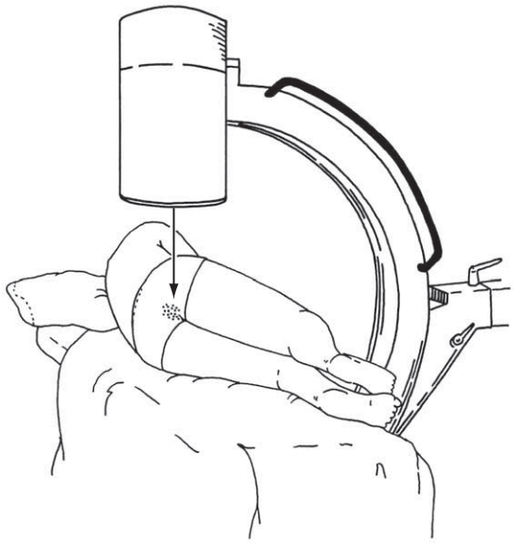


FIGURE 21–15
C-arm placed at the gluteal region in the lateral decubitus position.

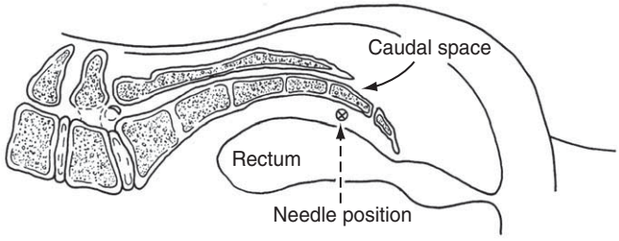


FIGURE 21–16
Lateral cross-section of the sacrococcygeal area illustrating the needle-tip position for ganglion impar block.

Prone Technique

Alternative techniques have been described for this block. In the transsacrococcygeal approach, a 22-gauge, 3.5-inch needle is placed directly in the retroperitoneal space, in the midline at the level of sacrococcygeal junction[2] in the prone position (Figure 21-19). An advantage of this approach is that the physician does not have to insert a finger in the rectum, which may be extremely painful for some patients, thus increasing the patient's tolerance. This approach may be challenging in patients with arthritis in the bones and calcification of the ligaments of the sacrum and coccyx.[31] This is particularly important in patients with postradiation proctitis.

A paramedian approach can be performed in the prone position for greater patient comfort. With this technique, a bent 3-1/2-inch spinal needle (see Figure 21-14) is used. The needle is inserted in the buttocks, inferior and lateral to the sacral hiatus. Initially, fluoroscopy in the anteroposterior (AP) position is used to confirm the direction. After changing to a lateral view, the needle is advanced until bone is contacted or the needle tip is in the perirectal space parallel to the sacrococcygeal ligament. After final AP image is obtained to ensure midline needle tip location, 1–2 ml of radiographic contrast solution is injected to avoid unintended spread. With confirmation of satisfactory spread, 5 ml of local anesthetic or neurolytic

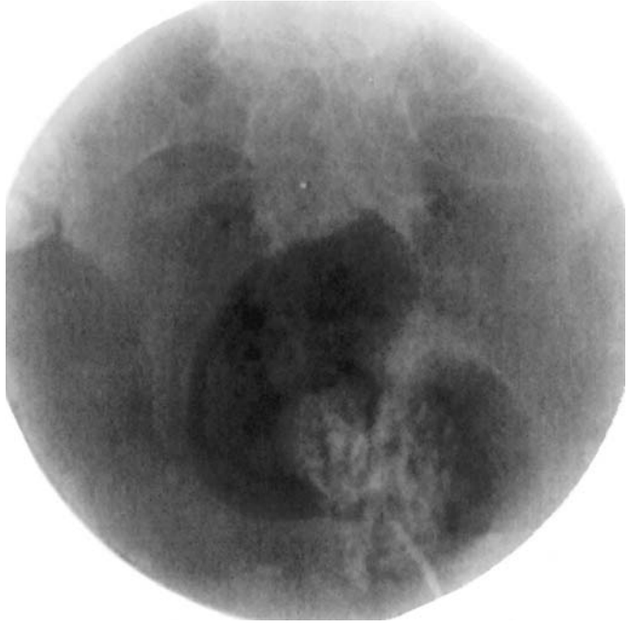


FIGURE 21–17
Anteroposterior fluoroscopic view that shows the contrast outlining the ganglion impar.

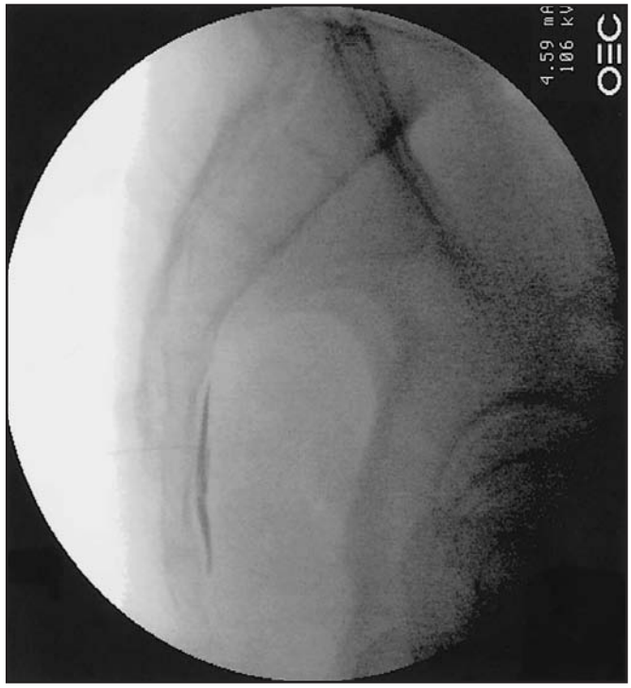


FIGURE 21–18
Lateral fluoroscopic view that shows the needle tip in the perirectal space between the rectum and the sacrum.

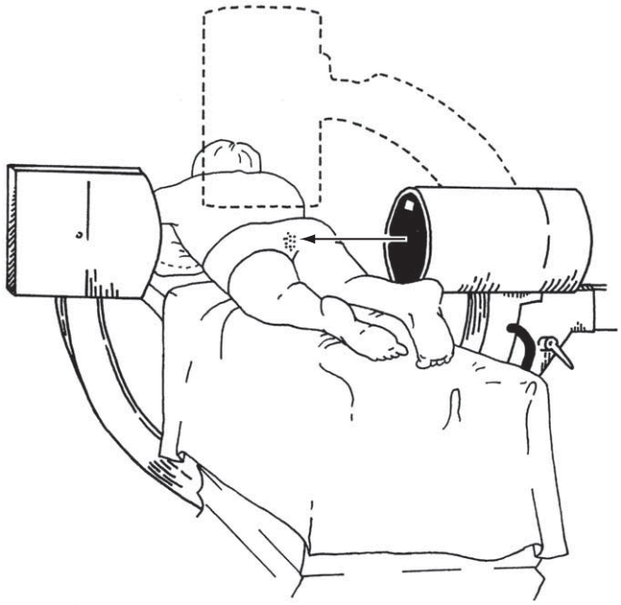


FIGURE 21–19
C-arm with patient in prone position for prone technique.

solution can be injected under fluoroscopy. Finger placement into the rectum is not needed for this approach. This technique may allow a greater tolerance of the procedure for the patient and physician.[31]

Huang[33] recently described a lateral approach in which the needle is advanced below the transverse process of the coccyx inferior to the level of the sacrococcygeal junction. The patient is placed at a lateral or prone position for this technique. This technique avoids the longer travel distance from needle entry point to the ganglion impar, and it causes less tissue damage. This technique can also be useful when the sacrococcygeal ligament is calcified.

The double-needle technique was described for radiofrequency lesioning of the ganglion impar.[23] In this technique, the patient is placed in the prone position. The first needle is placed through the sacrococcygeal ligament, and the second one is placed through the coccygeal disc. After confirming that both needles are placed correctly, sensory (1 V, 50 Hz) and motor (maximum 3 V and 2 Hz) testings are performed prior to the creation of radiofrequency lesioning.

Lithotomy Technique

In a third alternative approach, the patient is placed in the lithotomy position (Figure 21-20). The resulting curvature of the coccyx is decreased, allowing access to the ganglion impar with a straight 22-gauge spinal needle and facilitating needle positioning. However, placement of a finger in the rectum and fluoroscopy guidance are needed. The advantages of this approach include easy needle placement and a less cumbersome fluoroscopic evaluation of the needle's tip.

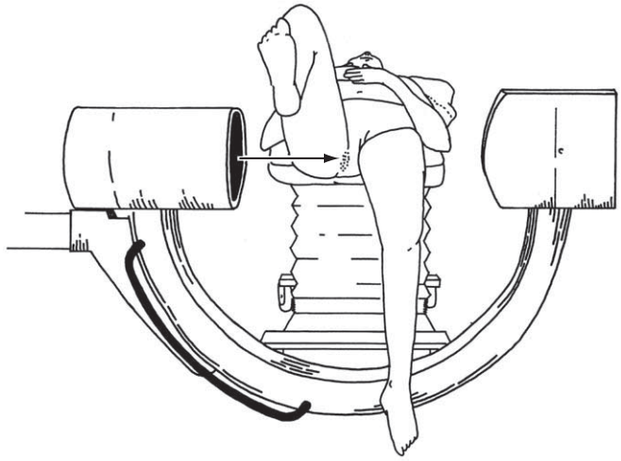


FIGURE 21–20
C-arm with patient in semi-lithotomy position for third alternative approach.

COMPLICATIONS

- Rectum puncture
- Neurolytic injection into nerve roots or rectal cavity
- Neuritis/nerve root injection
- Cauda equina syndrome

EFFICACY

Because the anatomic location of the ganglion impar is poorly described, the success rate of this procedure depends on the anatomical variability of the location of the ganglion.[26,30] Chang-Seok et al.[34] recently published a study to identify the location of the ganglion impar and to determine its shape and size, and topographic relation with the branch of the sacral nerve to facilitate a more successful blockade of the ganglion. Although a blind technique for this block was described,[25] fluoroscopic guidance is necessary for the success of the blockade of the ganglion impar. Some clinicians suggest CT guidance for the exact placement of the needle to this ganglion.[24,25]

Several studies have evaluated the ganglion impar block. As a first report, Plancarte and colleagues[20] evaluated 16 patients who experienced localized perineal pain associated with advanced cancer despite surgery, chemotherapy, radiation, and oral pharmacological therapy. The pain was reported as burning, and was associated with urgency in eight of the patients. Complete analgesia was obtained in eight patients, and 60–90% pain relief was reported for the rest. More than one block was performed in two of the patients, with further pain improvement. Follow-up was carried out for 14–120 days, depending on the patient's survival. No complications have been reported to date with this block.

Swafford and Ratzman[22] reported the efficacy of the transcoccygeal approach. In this study, 20 patients with

perineal pain unresponsive to previous treatment modalities were reported (18 with bupivacaine/steroid block and 2 with a neurolytic block). Five patients of the bupivacaine/steroid group reported complete pain relief, and 10 of this group reported greater than 75% pain reduction. Both neurolytic blocks resulted in complete pain relief. Duration of the pain relief varied from 4 weeks to permanent.

Vranken et al.[35] studied the efficacy of the ganglion impar block in long-lasting, treatment-resistant coccygodynia by using 5 ml of 0.025% bupivacaine. There was no pain reduction or increase of quality of life associated with the procedure.

Basagan and colleagues[36] evaluated the efficacy of the ganglion impar block in nine patients by using the transsacrococcygeal technique. They suggested that this technique was a safe and effective procedure in the treatment of perineal pain related to malignancy.

McAllister et al.[31] recently published a case report about sacral postherpetic neuralgia (PHN) and excellent results in treatment with the paramedian technique, by using corticosteroid (40 mg of triamcinolone) and repeated ganglion impar blockade by bupivacaine 0.025%. This study is very important, as it is suggesting that this procedure can be used effectively and safely in the treatment of non-cancer pain such as PHN. Reig et al.[23] described a new technique by using a double needle for radiofrequency application to the ganglion impar. They suggested that this technique was an effective and safe method, especially for some non-cancer perineal pain syndromes such as PHN, pain of the glans (unknown cause), post-traumatic perianal pain, and so on. However, further larger and randomized clinical studies are needed to confirm the acceptability of ganglion impar block in the treatment of non-cancer perianal pain.

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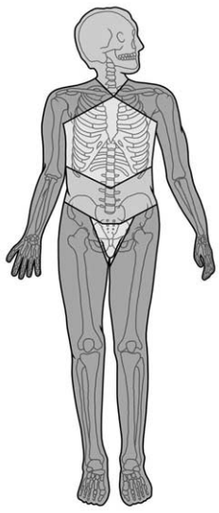
C H A P T E R



22

Pelvic Spinal Neuroaxial Procedures

GABOR B. RACZ AND CARL E. NOE

SACRAL EPIDURAL STEROID INJECTION

HISTORY

Cathelin[1] introduced the concept of caudal anesthesia in 1901. Controversy has surrounded the use of epidural steroid injections for pain related to disk herniations, spinal stenosis, and other causes for radicular pain. Winnie et al. reported efficacy with spinal steroid injections in 1973 with epidural steroids.[2] In the 1980s, Cuckler and colleagues[3,4] found no benefit. In Australia, concern about epidural injections of Depo-Medrol prompted a statement being issued encouraging interdisciplinary treatment for patients with chronic pain.[5] A review by Nelson and Landau[6] detail the history and argue against epidural steroid injections. Carette and coworkers[7] reported temporary benefit in patients with disk herniations but no reduction in the surgery rate.[7] Carette and Fehlings,[8] in a later review, suggested injections as an option for cervical radiculopathy. Meta-analysis has led to different conclusions regarding the effectiveness of epidural steroid injections.[9] Most, if not all, negative studies have employed either the single shot caudaltechnique or a translaminar approach without fluoroscopy. Both the single-shot caudal and nonfluoroscopic techniques are not recommended today.

Fluoroscopic guidance with anteroposterior and lateral views plus radiopaque contrast is now commonly used to confirm placement. Price et al.[10] reported a 36% incidence of inaccurate caudal placement without fluoroscopy. Bartynski and colleagues[11] reported a 25.7% incidence of incorrect needle placement without fluoroscopy for lumbar epidural injections. Single-shot caudal epidural steroid injections have become less common as fluoroscopy-guided procedures have become more common. However, Kim and coworkers[12] demonstrated that mid to lower lumbar levels could be reached with large-volume injections (50 ml) from the sacral approach.

The use of computerized tomography has been increasingly reported with injection procedures. Berger and colleagues[13] reported the use of CT-guided injections not only for needle placement but injectate distribution as well. Wagner[14] has advocated CT-guided injections to help avoid cysts and severely stenotic areas.

A debate exists over the use of saline, air, or both for the loss of resistance technique for epidural needle placement. Air injection could produce an air embolus or pneumocephalus. Others feel that cerebrospinal fluid (CSF) may be mistaken forsaline.

Historically, radiation safety has improved with reduced dosing, more effective shielding, and monitoring. Following guidelines for radiation safety, levels of exposure are now thought to be acceptable for patients and physicians.[15]

The rationale for the use of epidural steroids has been controversial and based mostly on an inflammatory concept of radiculitis. More recently, epidural corticosteroid has been shown to reduce neuropathic pain in laboratory models.[16] The clinical significance is unknown, but the anti-inflammatory effect on disk material may not be the only mechanism for analgesia.

Patients with multiple failed back surgeries and arachnoiditis represent a unique hazard to single-shot caudal epidural injections. The injected fluid may dissect through the surgical tear into the subdural space and loculate with sufficient pressure between the arachnoiditis scar formation and the epidural scar formation to compress arterial blood supply to the spinal cord.

ANATOMY

An important consideration is the proximity to the rectum for reasons of sterile conditions and potential rectal trauma with needle placement. Also, sacral nerve roots are critical for bladder, bowel, and sexual function.

The caudal epidural space is entered by penetrating superior to the sacrococcygeal ligament at the sacral hiatus. The subarachnoid and subdural spaces end at variable sacral levels, usually about sacral vertebral level S2. The fifth sacral roots and filum terminale exit the dura and continue through the sacral canal to exit at the sacral hiatus. The first through fourth sacral nerve roots are the only spinal roots that divide and change course into ventral and caudal rami within the vertebral canal, exiting via ventral and dorsal foramina. The sacral nerve roots are usually paired; however, the sacral nerve roots are the most variable nerve roots in the body. Occasionally, one or the other sacral ganglia or nerve root may be missing. This has specific relevance for neuromodulation techniques where trials of stimulation may reveal the presence or absence of nerve roots and the appropriate location of the electrode for optimal pain relief.

INDICATIONS

Most indications are for lumbosacral radicular syndromes.

- Disk herniations, or spinal stenosis
- Sacral nerve root scarring
- Coccygodynia
- Rectal pain
- Pudendal neuralgia
- Sacral fracture
- Post radiation sacral radiculopathy
- Sacral metastasis

CONTRAINDICATIONS

- Infection
- Altered coagulation, most commonly from therapeutic drugs for cardiovascular diagnoses requiring anti-coagulation.
- First trimester pregnancy
- Failed back surgery with arachnoiditis

PREPARATION OF PATIENT

Anticoagulant medications, including ginseng, gingko, garlic, and so on need to be discontinued prior to caudal procedures.

Neurologic history, physical examination, and radiographic diagnoses need to be well documented. This practice is particularly helpful in evaluating patients who suffer from complications following procedures.

Written informed consent including risks of paralysis, weakness, numbness, bowel, bladder, sexual dysfunction, bleeding, infection, and pain. Intravenous (IV) access should be established, and withhold food and fluids via oral ingestion (NPO).

EQUIPMENT

- Monitoring equipment for vital signs
- Pulse oximetry and EeG if sedation is used
- Local anesthetic, ropivacaine 0.2%, bupivacaine 0.25%
- Fluoroscopy
- Omnipaque, or other myelogram-grade contrast
- Epidural or spinal needle
- Syringes
- T-piece
- Alcohol-free corticosteroid

TECHNIQUE

Blind caudal epidural steroid injections are less common today and have largely been replaced by fluoroscopic-guided injection techniques.

The patient is positioned in the prone position and prepped and draped in a sterile fashion. A skin entry point is chosen only after an assessment of the curvature of the sacrum and depth to the hiatus. A lateral fluoroscopic view is sometimes helpful in the process. The more curved the sacrum and the deeper the hiatus, the more inferior the skin entry point needs to be. Also, the skin entry point may be best placed contralaterally to the patient's symptoms (Figure 22-1). This facilitates directing the needle toward the affected side.

Local anesthetic needs to be infiltrated along the potential path of the epidural needle rather than just at the skin and adjacent subcutaneous tissue. Once the skin is entered, lateral fluoroscopy is helpful in guiding the

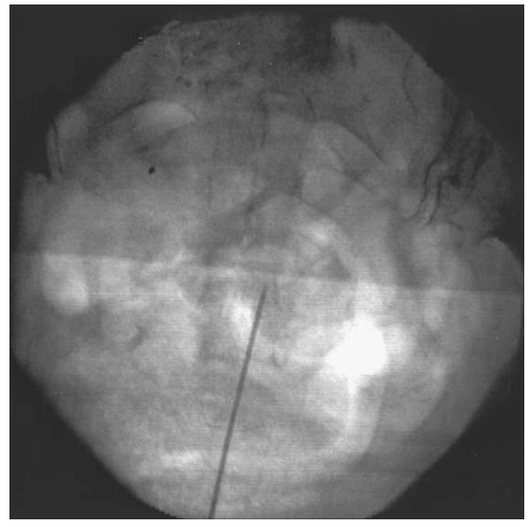


FIGURE 22–1

Sacral epidural RX Coude needle placement from skin entry on asymptomatic side toward symptomatic side.

needle to the hiatus (Figure 22-2). A loss-of-resistance technique and aspiration test may be helpful, but contrast injection may be most helpful in confirming placement.

Methylprednisolone 40 mg, triamcinolone 40 mg, dexamethasone 4 mg, betamethasone 15 mg, or equivalent doses of alternative corticosteroid can be administered.

Caution

In the 1950s, large-volume injections were used but abandoned because of loculation and pressure to the blood supply to the spinal cord. In cases of subcutaneous injection with injection going outside the sacral canal, sloughing off of skin over the sacrum occurred.

POSTPROCEDURE MONITORING

Patients should be monitored for 30 minutes or longer. Sedative drugs and local anesthetic may produce effects following the procedure, and patients should be advised of potential motor block effects for hours following the procedure. Patients should be given instructions to contact the physician for any problems before their follow-up visit.

COMPLICATIONS

- Hypotension
- Perianal numbness and sacral nerve root injury
- Spinal cord injury due to loculation of large volumes of injectate
- Avascular necrosis
- Bladder or bowel dysfunction
- Bleeding or hematoma

Reports of injections of isopropyl alcohol and other toxic solutions are a serious reminder to have a system in place to identify the contents of syringes to train personnel handling drugs and equipment.

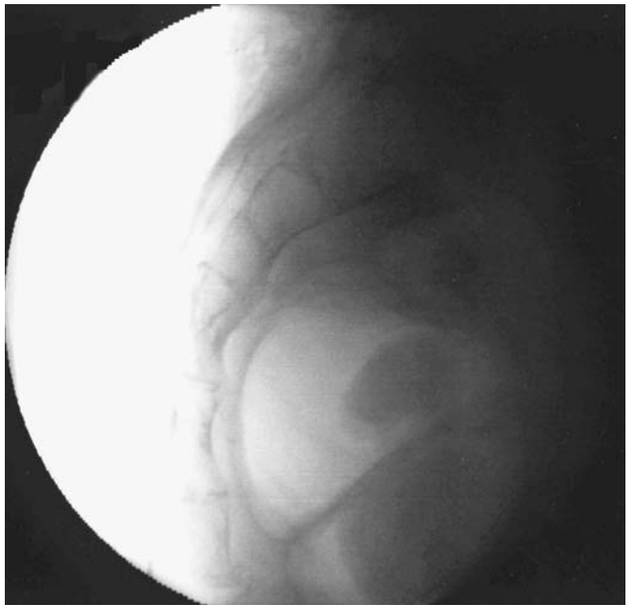


FIGURE 22–2
Lateral image confirming placement of needle in the caudal space.

METABOLIC COMPLICATIONS

Blood sugar can be significantly elevated in diabetic patients due to insulin suppression after corticosteroid administration and cortisol levels are suppressed.[17]

Cushing's syndrome has been reported following epidural injections of methyl prednisolone.[18] Lipomatosis, fatty proliferation in the epidural space, has been reported following epidural steroid injection. Resolution can occur after discontinuance of corticosteroids.[19] Lipomatosis can produce symptomatic spinal stenosis.

MECHANICAL COMPLICATIONS

Dural puncture can result in cerebrospinal fluid leak, hypotension and alterations of normal intracranial venous systems. Venous pooling and even venous tearing can occur, resulting in intracranial hematomas. Subdural injections of corticosteroid are of concern due to arachnoiditis.

Venous air embolism as well as venous air injection have been reported.[20] Air can occur in the cephalic region with subdural injections.[21]

INFECTIOUS COMPLICATIONS

A variety of infection problems have been reported. Many cases of meningitis, osteomyelitis, epidural abscess, and discitis have been reported. Arachnoiditis can occur after abscess formation. Arachnoiditis related to intrathecal injection of corticosteroid preparations is not recommended.

Exophiala dermititidis meningitis has been reported and associated with contaminated steroid preparations from compounded injected medications.[22]

Gaul et al.[23] reported a series of 128 cases of community-acquired purulent meningitis, and 6.25% had a history of spinal injections. *Staphylococcus aureus* was identified in 50% of the patients with a history of injection, and coagulase-negative staphylococci were present in 25%. No organism was cultured in the remaining 25% of cases. The authors make the point that even though infectious complications from injections are rare, they comprise a significant proportion of central nervous system infections.

OCCULAR COMPLICATIONS

Gibran et al.[24] reported vitreal hemorrhage following a caudal epidural steroid injection. The hypothesis was presented that increased cerebrospinal fluid leak pressure

from loculation of injectate caused increased venous pressure in the globe. Ocular complications seem to be related to multiple factors. Corticosteroid use, regardless of the route of administration, is associated with edema in the optic nerve and blurry vision.[25,26] This usually resolves without permanent vision change and is not common. Retinal necrosis, detachment, and vitreal hemorrhage have also been reported.[27] Injections may increase CSF pressure. Slow injections may help reduce pressure-related problems. Steroid myopathy has been reported from epidural steroid injections.[28] Facial flushing and generalized erythema can occur.[29] Pain following intra-arterial (Nicolau's syndrome) or IV (Tachon's syndrome) injection of corticosteroid produces pain syndromes that have been reported following epidural and other spinal steroid injections.[30]

Slipman and colleagues[31] reported two episodes of dysphonia in the same patient after a series of two epidural steroid injections. Vocal cord edema was visualized after the second injection but not after the first. Systemic corticosteroid may act as a procoagulant, and cerebral venous sinus thrombosis has been reported.[32]

Dietzel and Hedlund[33] suggested that athletes who experience analgesia following injections and return to sports may be at risk for worsened injury or delayed healing. The incidence of these complications is unknown. Willburger[34] reported a series of 7963 injections and complications: 10 patients with spinal headaches, 3 with numbness, 5 vasovagal reactions, 1 patient had a fall, 1 patient had a transient thoracic level block, and 5 patients had allergic reactions.

CASE HISTORY

A middle-aged female patient with a history of multiple failed back surgeries, back pain, and radiculopathy and documented arachnoiditis. Additional history included a severe anaphylactic reaction to contrast. The patient had an uneventful caudal epidural injection of 10 ml of local anesthetic and steroid. There was minimal change in the chronic pain status. A second injection was performed to see if a response could be achieved in an identical manner in the office setting. The second injection was followed by a motor block that never recovered, and the patient remained paralyzed at the time of malpractice litigation. There was a previous history of transient paralysis from which the patient recovered years before. The most likely explanation was the fluid dissection to the subdural space with sufficient pressure to compress and occlude the arterial blood supply to the conus and spinal cord.

EFFICACY

Meta-analysis studies have been conflicting. Various randomized trials were used in the analyses because of different criteria for study quality.[9] A meta-analysis of randomized trials using only studies employing fluoroscopic guided injections has not been reported. Rozenberg and colleagues[35] concluded that a determination could not be made with existing data. Valat et al.[36] reported no benefit in a randomized trial with injections without fluoroscopy. Most, if not all, negative studies have not employed the use of fluoroscopy.

In another study, aqueous betamethasone had no effect 1 month after translaminar epidural injections in patients with disk herniations or spinal stenosis, but Depo-Medrol, in equivalent doses, was helpful.[37] Aqueous corticosteroid may distribute rapidly and have a short-term local effect compared with Depo preparations.

Butterman,[38] in a randomized trial of injections versus surgical discectomy, found surgery to be associated with more rapid resolution of symptoms. A number of patients crossed over to the surgery group, but long-term pain scores were similar in both groups. Wilson-Macdonald and colleagues[39] reported superiority of epidural corticosteroid over intramuscular injections but no difference in the surgery rate between the two groups.

Two recent randomized trials have compared caudal injections to targeted injections. Hoppenstein et al.[40] found that single-shot caudal injections did not reduce pain scores and interlaminar injections, which were targeted with fluoroscopy, were helpful.[40] Dashfield and coworkers[41] found no difference between caudal injections compared with endoscopy-guided injections. Finally, Livesey et al.[42] reported similar improvement in a study comparing epidural steroid injections to laser discectomy.

American Society of Interventional Pain Practice (ASIPP) guidelines support the use of epidural corticosteroid injections.[43] No direct comparisons of caudal or translaminar epidural or nerve root blocks have been adequately studied.

CAUDAL DECOMPRESSIVE NEUROPLASTY

HISTORY

Caudal catheter anesthesia was introduced for obstetrics by Manalan in 1942.[44] Hatten reported epidurography and selective nerve block via the sacral hiatus using the Seldinger technique in 1980.[45]

The initial epidurography was performed serendipitously in 1921 by Sicard and Forestier.[46] Payne and Rupp[47] in 1950 combined hyaluronidase with local anesthetic in an attempt to alter the rapidity of onset, extent, intensity, and duration of caudal anesthesia. They demonstrated maximal efficacy in a group receiving local anesthetic, hyaluronidase, and epinephrine. Hyaluronidase concentration in this study was relatively dilute at 6 U/ml, with an average volume of injection of 24 ml. In 1951 Moore[48] added 150 U of hyaluronidase in 1309 nerve blocks, including 20 caudal blocks, to enhance the spread of local anesthetic. He showed hyaluronidase to be relatively nontoxic. Lievre and coworkers[49] reported the first use of

corticosteroid injected into the epidural space for the treatment of sciatica in 1957. They injected a combination of hydrocortisone and radiopaque dye in 46 patients with 31 positive results. In 1960, Goebert et al.[50] injected procaine and hydrocortisone into the caudal epidural space. The majority of patients derived benefit from their injections of 30 ml of 1% procaine hydrochloride with 125 mg of hydrocortisone acetate. In the same year Brown[51] injected larger volumes, 40 to 199 ml, of normal saline followed by 80 mg of methylprednisolone in an attempt to mechanically disrupt and prevent reformation of presumably fibrotic lesions in patients with sciatica. He reported complete resolution of pain for 2 months in the four patients he treated. It is notable that this investigation in 1960 laid the theoretical foundation for current therapies in which specific catheter placement is crucial to the effective treatment of epidural adhesions.

Hypertonic saline was first administered by Hitchcock[52] in 1967 for the treatment of chronic pain when he injected cold saline intrathecally. He later reported, in 1969, that it was the hypertonicity rather than the temperature of the solution that was the determining factor in its therapeutic effect.[53] Hypertonic saline was subsequently employed by Ventafridda and Spreafico[54] in 1974 for intractable cancer pain by intrathecal administration. All 21 patients in this study had pain relief at 24 hours, although only three patients reported relief at 30 days.

Claude Duval presented the use of epidural hypertonic saline without fluoroscopy for chronic pain at the American Society of Anesthesiologists meeting in the late 1970s.[55] Racz and Holubec[56] in 1989 reported the first use of epidural hypertonic saline to facilitate lysis of adhesions, and hyaluronidase was introduced as an alternative agent by Stolker and associates in 1994.[57] The development of a radiopaque epidural catheter that could be steered with less risk of shearing, obstructing and migrating was essential. The Racz catheter was developed following several deaths from a combination of IV plastic catheter migration and catheter kinking preventing aspiration before injection of 0.75% bupivacaine in obstetrical anesthesia.[58] A soft-tipped, radiopaque catheter that did not kink and could be directed was developed for targeted injections.[59]

The authors developed an interest in the dorsal root ganglion (DRG) as a site for pain generation and blockade only to find that in many of these patients they could not get to the DRG and the lateral recess space due to presumed scar formation. Following the injection of contrast, saline, local anesthetic and steroids, many of these patients' pain would improve for months and have documented motor function recovery, such as reversal of a foot drop. The concept of epidural scar formation was confirmed by animal data and in the clinical arena by epiduroscopy.[60,61] The use of epidural hypertonic saline was added on previous clinical experience and publications, and the need for postlysis of adhesion pain reduction. The hyaluronidase was added on previously publications in its use in the epidural space, and in the authors first retrospective review they found that the use of hyaluronidase reduced the outright failure rate significantly as it facilitates the compartment spreading and opening up of the perineural space.[62] To maintain nerve root mobility, neural flossing exercises have been added for patients to continue subsequent to discharge.

In 1994, Stolker and associates[57] added hyaluronidase to the procedure but omitted the hypertonic saline. In a study of 28 patients, they reported greater than 50% pain reduction in 64% of patients at 1 year. They stressed pain patient selection criteria and suggested that the effectiveness of the procedure was based on the effect of the hyaluronidase on the adhesions and the action of the local anesthetic and steroids on the sinuvertebral nerve.

Lysis of adhesions in the epidural space is effective when simpler procedures fail, such as rest, nonsteroidal anti-inflammatory medications, muscle relaxants, physical therapy, activity programs, two or three single-shot epidural steroids, and transcutaneous electrical nerve stimulator unit. The more informed patients would selectively undergo procedures such as lysis of adhesions rather than surgery. The results clearly show a dramatic decline in further surgical interventions in the authors' patient population. In appropriately selected patients with clearly documented herniated discs and nerve root compressions caudal decompression and neuroplasty is recommended. This concept is gaining support by having the U.S. regulatory agencies and most insurance companies, as well as courts, support the recognized effective nature of the procedure of lysis of adhesions.[56]

PATHOPHYSIOLOGY OF EPIDURAL SCARRING

Scarring in the epidural space occurs frequently following surgery and causes no problems. Scarring can occur following leakage of nucleus pulposus material into the epidural space.[60] The pain associated with the scar formation originates from the nerve itself, which is irritated, swollen, and angry looking, and has no space in which to move freely. In the neural foramina, nerves are normally associated with epidural veins. Epidural scarring often obstructs these epidural veins. The obstruction raises IV pressure, leading to additional edema formation within the epidural space.

INDICATION

The ideal indication for decompressive neuroplasty is radiculopathy due to epidural fibrosis and nerve root entrapment. For chemically sensitive discs, failed back surgery syndrome, and associated epidural inflammations, the placement of the catheter in the anterior epidural space has been extremely effective. In spinal stenosis the neuroplasty techniques have been helpful in some situations by decreasing edema and venous congestion with

the expected effect of attenuating the compressive effects on the spinal cord and nerve roots.[63]

In the lumbar region the caudal approach is extremely beneficial for the L5-S1 radiculopathy owing to the ease of entering the area of natural lordosis in that region. On the occasion that L4 or higher is problematic, a transforaminal approach with placement of the catheter into the anterior epidural space can be done individually or in conjunction with a caudal catheter. Thoracic neuroplasty is rarely done but may be useful in situations such as acute herpes zoster or thoracic vertebral compression fractures. For the cervical region, radiculer symptoms related to failed neck surgery, discogenic pain, and associated fibrosis from inflammation are the predominant indications. Since its introduction, there is now a common acceptance of this technique.[64,65]

DRUGS USED FOR NEUROPLASTY

Iohexol is a second-generation, nonionic, low-osmolar, radiographic contrast agent. The iodine content is 46% by weight, buffered by tromethamine to a pH of 6.8–7.7, and preserved with 0.1 mg/ml of edetate calcium disodium. The uniform coverage of the iodine atoms by the hydrophilic groups is responsible for its low toxicity.

Many concentrations (140, 210, 240, 300, 350 mgI/ml) of iohexol are available for subarachnoid, intravascular, and body cavity injections. For subarachnoid administration, the concentration should never be greater than 300 mgI/ml in adults and 210 mgI/ml in children. Because of the toxic effects of iodine, the total dose is 3.06 mg for adults and 2.94 mg for children. There is minimal protein binding in serum. Eighty-eight percent of an intrathecal dose is excreted renally in its unmetabolized form and can be found in the urine at 24 hours.[66] Chemotoxic reactions are often dose dependent and present as hypotension, dyspnea, cardiac arrest, organ failure, and/or loss of consciousness. The incidence of chemotoxic reactions with these radiographic contrasts occurs in only 1 in 100,000 patients. Intravascular injection of iohexol has been reported to have a low risk for causing renal failure.[67] There are some concerns that this risk may be increased with patients on oral hypoglycemic agents. Fortunately, these complications are infrequent with nonionic contrast agents.

Idiosyncratic reactions consist of headaches, myalgias, nausea, vomiting, dizziness, aseptic meningitis, and other neurologic disturbances. The most common reaction is a headache, which occurs in 18% of the patients after intrathecal iohexol.[66] Aseptic meningitis and neurologic disturbances are much less common. Allergic or anaphylactoid reactions with non-ionic contrast agents are rare and less frequent than those with ionic contrast agents.[68] Ndosi and associates[69] reported that the risk of significant reaction with concentrations of 240 mgI/ml of iohexol for myelography in a placebo-controlled, double-blinded study was no greater than that related to the lumbar puncture itself. This same study reported that headaches, dizziness, nausea, vomiting, and seizures are more likely to occur with 180- and 300-mgI/ml concentrations.

Hyaluronidase

Hyaluronidase is a lyophilized, white, odorless, amorphous powder that is commercially available in 150 and 1500 U. The smaller dose is supplied fully hydrated, whereas the larger amount is a solid with 1 mg of thimerosal preservative and 13.3 mg of lactose for mixing with a solute. Duran-Reynals[70] first described its spreading factor by strains of *Streptococcus* bacteria. It can be found in bee and snake venom, mammalian tissues, and sperm.[71] Its primary function is to depolymerize hyaluronic acid and to a lesser degree chondroitin-6-sulfate and chondroitin-4-sulfate. Hyaluronic acid is a large-molecule glycosaminoglycan that binds ground substance proteins that form proteoglycans. Not only are these proteoglycans found between the ground substances between cells, they are also in cheloids (dense scar tissue) and epidural adhesions.[72] Disruption of these proteoglycans is accomplished by cleaving the b-1, 4 glycosidic bonds.

The breakage of the proteoglycans has been found to accelerate the diffusion of injected drugs.[73] This hypodermoclysis was subsequently found to increase the efficacy of the local anesthetic infiltrations.[74] In the epidural space, the dura is composed of collagen, elastin, and surface fibroblasts that are not affected by hyaluronidase. Intrathecal use of hyaluronidase has been documented for chronic arachnoiditis with no report of any serious adverse effects in 15 patients.[75]

Nicoll and colleagues[76] reported on a prospective study of 6000 retrobulbar blocks with the adverse effects attributed to the local anesthetic spread into the central nervous system. Because of the known homology of mammalian hyaluronidase to insect hyaluronidase, attention should be given to possible complications with patients having venom allergies.[77] Anaphylactic-like reactions have occurred in isolated cases.[78]

Local Anesthetics

The local anesthetic used is limited to a concentration that provides only sensory blockade. With the local anesthetic, immediate pain relief is possible in addition to preparation of the epidural tissues for infusion of the hypertonic saline. Hypertonic saline tends to "burn" when infused without preinjection of local anesthetics. As an additional safeguard against subdural or intrathecal injection, the local anesthetic is delivered in divided doses to monitor for nonepidural spread. For the neuroplasty technique, it also serves as a diluent for the steroids.

Bupivacaine is significant for its cardiotoxicity, which has now been attributed to the S-enantiomer.[79] Levobupivacaine is exclusively composed of the S(-) monomer of racemic bupivacaine and has proven itself to have essentially the same clinical properties and potency as racemic bupivacaine with significantly fewer adverse effects.[80]

Ropivacaine is a third-generation S(-) monomer local anesthetic of the propyl-pipecoloxylidide amino-amide series with less cardiotoxicity, but with potency and duration of action similar to racemic bupivacaine. Significant differences of ropivacaine compared with bupivacaine are its vasoconstrictive properties and documented ability to reduce epidural blood and pial blood vessel size.[81,82]

Steroids

Methylprednisolone and triamcinolone are two of the more popular choices for epidural injection. Both interact with two different receptor types: glucocorticoid and mineral corticoid. The glucocorticoid is primarily responsible for regulation of carbohydrate metabolism and the inflammatory and immune responses, whereas the mineralocorticoid is responsible for regulation of electrolyte balance. Attempts to synthesize a steroid with anti-inflammatory properties have been difficult.

Collectively, methylprednisolone and triamcinolone are considered intermediate acting in duration with equipotent anti-inflammatory effects. Triamcinolone is a more glucocorticoid-specific agonist than methylprednisolone. Both of these drugs exhibit lower protein binding and metabolism than endogenous corticosteroids. Unfortunately, they all are potential suppressive agents of the hypothalamic-pituitary-adrenal axis.

Triamcinolone diacetate is suspended in a solution consisting of polysorbate 80 0.20%, polyethylene glycol 3350 3%, sodium chloride 0.85%, and benzyl alcohol 0.90% as a preservative. Its pH is adjusted to approximately 6.[83] This steroid is compatible with a variety of diluents unless a preservative is present. Flocculation and clumping of triamcinolone are reported when mixed with a preserved diluent or if the steroid has been frozen and thawed. Methylprednisolone has been shown to flocculate when mixed with lidocaine.

Although systemic toxicity from epidural injection is not exactly known, suppression of the hypothalamic-pituitary-adrenal axis from triamcinolone has been shown to persist for 21 days.[84] Examples of the toxic effects are metabolic disturbances, electrolyte imbalances, fluid shifts such as edema, muscle wasting, peptic ulcers, impaired wound healing, and immunologic dysfunction[85]; allergic reactions to the steroids are rare.[86] The preservative polyethylene glycol in many commercial formulations may cause arachnoiditis when injected intrathecally.[87] Case reports of epidural abscesses, aseptic meningitis, and bacterial meningitis have also been published.[88,89]

The commonly used corticosteroid preparations do not pass through a 0.2-micron filter, although local anesthetic does.

Hypertonic Saline

Hypertonic saline was first reported as a cold saline injection into the intrathecal space for chronic back pain in 1967.[50] Hitchcock[52] later reported that the hypertonicity of the solutions rather than the temperature was responsible for its effects. Computerized axial tomography studies showed a selective C-fiber blockade of dorsal rootlets that appeared to be related to the high chloride ion concentration.[90] Lake and Barnes' work[91] on frog spinal neurons showed that hypertonic saline decreased the spinal cord water content and depressed the lateral-column evoked ventral root response by affecting the gamma aminobutyric acid (GABA) receptors. Racz and associates[59] performed a study on dogs looking at the effects of hypertonic saline in the epidural space and showed that it took 20 minutes for the cerebrospinal fluid (CSF) to equilibrate, with resultant doubling of the CSF sodium concentration.[92]

Most of the complications cited are related to intrathecal hypertonic saline. Clinical complications of intrathecal injection of hypertonic saline consist of cardiac, respiratory, and neurologic sequelae such as hypertension, tachycardia, and tachypnea with pulmonary edema.[93] The changes can occur rapidly with associated hemorrhaging.[54] Regarding complications directly related to epidural injection, two cases of arachnoiditis from possible subdural or subarachnoid spread have been reported.[94]

Subdural injection of contrast is not easy to recognize. Subdural placement of a catheter should be easier to recognize because the catheter cannot be steered to the lateral recess of the nerve root. The dural sac will confine the movement of the catheter to the subdural space. The use of local anesthetic test dosing is important. Our most commonly used medications are 0.2% ropivacaine or 0.25% bupivacaine. Subarachnoid placement will give rise to a rapid motor block within 1–2 minutes. Subdural placement of the same medication will give rise to a sudden onset of motor block but 10–20 minutes later. Epidural injection of the same medication should not give rise to a motor block except in rare instances such as in patients with demyelinating disease, that is, multiple sclerosis. The motor block complication is extremely important because the treatment is support of ventilation and circulation. These patients must have an IV access site and equipment readily available and personnel available to administer ventilatory support and/or carry out endotracheal intubation.

ANATOMY

The sacrum is a large, triangular bone, situated below L5. Its apex articulates with the coccyx. Its anterior surface is concave. Anteriorly, four transverse ridges cross its median part. The portions of the bone between the ridges are the bodies of the sacrum. There are four anterior sacral foramina through which the sacral nerves exit and lateral sacral arteries enter. The posterior surface of the sacrum is convex. There are rudimentary spinous processes from the first three or four sacral segments in the midline. The laminae unite to form the sacral groove. The sacral hiatus is formed by the failure of the laminae of S5 to unite

posteriorly. The tubercles that represent remnants of the inferior articular processes are known as the sacral cornua; they are connected inferiorly to the coccygeal cornua. Laterally one can identify four dorsal sacral foramina. They transmit the posterior divisions of the sacral nerves. The sacrum may have many variations. The bodies of the S1 and S2 may fail to unite or the sacral canal may remain open throughout its length.

The caudal canal has a variable orientation in the anterior-posterior plane, necessitating an epidural needle skin entry point inferior to the sacral hiatus. The sacrum may curve in, resembling a kyphotic shape, placing the inferior sacral canal posterior to the sacral hiatus.

Radiologic Landmarks of Caudal Canal

In a lateral view, the caudal canal appears as a slight step off on the most posterior part of the sacrum. The median sacral crest is seen as an opaque line posterior to the caudal canal. While still in the lateral view, the sacral hiatus is usually visible as a translucent opening at the base of the caudal canal. To aid identification of the sacral hiatus, the coccyx can be seen articulating with the inferior surface of the sacrum.

On the anteroposterior view, the intermediate sacral crests are seen as opaque vertical lines on either side of the midline. The sacral foramina are seen as translucent near-circular areas lateral to the intermediate sacral crests. Note that the presence of bowel gas can make recognition of these structures difficult.

INDICATIONS

- Failed back surgery syndrome
- Epidural fibrosis
- Lumbar radiculopathy
- Spinal stenosis
- Lateral recess stenosis
- Back pain and radiculopathy
- Herniated disks
- Radicular neuropathic pain
- Postradiation neuropathy
- Postmeningitis epidural scarring

CONTRAINDICATIONS

- Infection
- Coagulopathies
- Unstable lumbar spine
- Inability to lie in prone position
- Arachnoiditis

EQUIPMENT

- 25-gauge, 3/4-inch infiltration needle
- 18-gauge, 1-1/2-inch needle
- 15–16-gauge Epimed RX Coudé epidural needle
- Epimed Tun-L or Epimed Brevi-stiff catheter for dense scarring
- Loss-of-resistance syringe
- 3-ml syringe
- Two 10-ml syringes
- Needle holder
- 3-0 nylon on cutting needle
- Scissors

DRUGS

- 1.5% lidocaine for skin infiltration
- 2% preservative-free lidocaine
- 0.2% preservative-free ropivacaine or 0.25% levobupivacaine or bupivacaine
- 0.9% preservative-free normal saline
- 10% preservative-free hypertonic saline
- 1500 U of hyaluronidase

PREPARATION OF PATIENT

Documentation of deficits from the history and physical examination, diagnoses, and radiological studies is important prior to this procedure.

Physical Examination

- Straight leg raise: positive radicular signs at less than 60 degrees
- Identification of pain to nerve root levels
- Functional evaluation
- Confirm stable vital signs
- Ability to lie prone for 60 minutes

Informed Consent

Written informed consent including risks of paralysis, weakness, numbness, bowel, bladder dysfunction, infection, bleeding, and worsened pain should be obtained.

Catheter shearing is included in the informed consent to allow surgical removal of a sheared catheter. Catheters are made from material biologically tested for implantation and for durability and safety. Passing any catheter through a needle predisposes to shearing. Catheter shearing usually occurs in the hands of the inexperienced. We have noted at Texas Tech that more shears occur during the first 4 months of fellowship training. The tip of the needle opening must match the direction the catheter is going. If the catheter is going in a different direction than the needle is pointing, and the catheter is pulled back, the tip of the needle can cut into the plastic wall of the catheter. This partial shear can be felt as resistance. At this point, the catheter and needle must be removed together as a unit. If the cut into the catheter wall is not recognized, the “fish hook”–like effect will hang up in the subcutaneous tissues and hard pulling against resistance can tear the

rest of the catheter. The catheters are visible under x-ray because of a metal spring within the core. Based on experience in chronic pain patients who are looking for explanations for their pain, we obtain an informed consent to have sheared catheters surgically removed. If the catheter is hanging up and has not been completely sheared, our first approach is to stop pulling and walk away from the problem for a few minutes, then go back and push the catheter to disengage the "fish hook," and twist the catheter and gently withdraw.

This method works because the size of the hole is larger than the catheter and one needs to find the position in which the catheter will slide out easily. The other common reason for shearing is the use of smaller-than-recommended Tuohy-type needles that have a sharp V-shaped cutting back end of the opening that can cut into the catheter. The newer RX Coudé needle has a wide-open back end that has a low risk of shearing as long as the direction and catheter are similar.

Laboratory Studies

Indicated laboratory and radiological studies should be obtained. Prothrombin time, partial thromboplastin time, bleeding time, white blood cell count with differential, urine analysis, and magnetic resonance imaging of the affected area are usually reviewed.

Radiological Studies

Diagnostic findings should be documented prior to the procedure.

Preprocedure Medication

It may be necessary to sedate the patient with 1–2 mg midazolam and 25–50 µg of fentanyl. IV injection of 1 g of ceftriaxone (Rocephin) is recommended.

Monitoring Procedures

Usual monitoring includes an automated blood pressure cuff, electrocardiogram, and pulse oximeter; the patient should have a patent IV catheter. Fluoroscopy is essential to the performance of safe adhesiolysis. To minimize radiation exposure, it is preferable to use a fluoroscope with memory capabilities and efficient computed image processing. For documentation purposes, videotaping the fluoroscopy screen during the procedure or printouts for subsequent review is often carried out. For personal safety, the physician should use appropriate protective measures such as leaded gloves, apron, thyroid shield, and leaded glasses. An addition of a leaded skirt around the fluoroscopy table can further decrease radiation exposure. Lastly, fluoroscopy is important in obtaining the maximum benefit from this procedure, that is, for verification of needle placement, visualization of dye spread, and proper catheter placement.

TECHNIQUE

On the fluoroscopy table, the patient is placed prone with a pillow under the abdomen to straighten the lumbar spine. Monitors are applied, including an electrocardiogram, pulse oximeter, and a blood pressure–monitoring device, preferably an automated one. The sacral area is then sterilely prepared and draped from the top of the iliac crest to the bottom of the buttocks. Abduction of the legs and internal rotation of the feet facilitates entry into the sacral hiatus. The sacral cornua and the sacral hiatus are palpated with the index finger of the nondominant hand rolled laterally over the sacral hiatus. For entry through the skin, a spot approximately 1 inch lateral and 2 inches inferior to the sacral hiatus in the contralateral gluteal region on the affected side for treatment is accessed. This point of entry inherently allows the needle, as well as the catheter, to be directed toward the affected side. The entry point is infiltrated with a local anesthetic, such as 1% lidocaine. A 15–16-gauge epidural needle, preferably an Epimed RX Coudé, is used in the following manner. The nondominant index finger is used to establish the location of the sacral hiatus, and the finger remains at that location. Contralateral from the symptomatic side, 1 inch from the midline and 2 inches from the sacral hiatus on the gluteal mound, the skin is infiltrated with a 25-gauge 1-1/2-inch needle with local anesthetic and is carried out along the tract to the sacral hiatus. An 18-gauge needle is used to perforate the skin. A 15-gauge RX Coudé needle is pushed through this perforation and advanced toward the sacral hiatus with the curve tip directed anteriorly directly beneath the palpating finger to the sacral hiatus. Anteroposterior and lateral fluoroscopic views are taken and saved to make a three-dimensional assessment if adjustment of depth or direction is needed of needle placement. With the appropriate correction, the needle is popped into the sacral canal through the sacral hiatus, and rotated 180 degrees so that the curve tip matches the direction of the sacral canal. The needle is advanced to a level no higher than the S3 foramen to avoid damaging the sacral nerve roots.

After negative aspiration for blood and CSF, 10 ml of iohexol (Omnipaque 240) or metrizamide (Amipaque) is injected under fluoroscopy for an epidurogram (Figure 22-3). If venous runoff is noted, the needle tip is moved during injection until contrast media is seen spreading within the epidural space. As contrast media is injected into the epidural space, a "Christmas tree" shape will be noted as the dye spreads into the perineural structures inside the bony canal and along the nerves as they exit the vertebral column. Epidural adhesions will prevent dye spread, such that there will be a marked absence of dye outlining the involved nerve roots. A lateral view will also show a lack of dye outlining the scarred nerve roots.

If the needle tip is subarachnoid, dye spread will be noted centrally and cephalad many levels above L5. If the

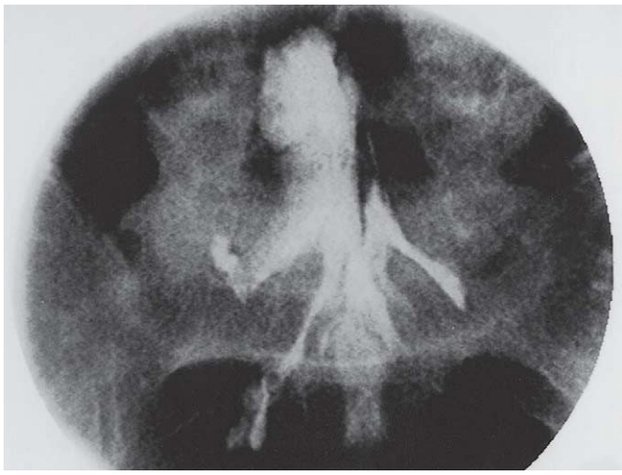


FIGURE 22–3

Radiographic image of the caudal canal after contrast material (iohexol) has been injected during neuroplasty. One can discern a "Christmas tree" appearance of the contrast solution.

needle tip is subdural, dye spread will also be central and cephalad but will not be as wide as that of a subarachnoid injection (Figure 22-9). The contrast will enhance the view of the outline of the nerve roots and the dura from the circumferential spread within the less-resistant subdural space. Injection of local anesthetic into the subarachnoid or subdural space will result in a motor block that is notably more profound, with a more rapid onset of the block than that seen after injection into the epidural space. A subdural block is often typified by a segmental motor block with a diffuse sensory block to the level expected from a subarachnoid injection of local anesthetic.

If CSF is aspirated, it is best to abort the procedure and repeat it another day. If blood is aspirated, the needle is first retracted caudally in the sacral canal until no blood can be aspirated. If this is unsuccessful, an attempt can be made to proceed with catheter placement into the proper location. Aspiration of this catheter should be negative for blood, and lack of venous runoff should be confirmed through the injection of contrast agent.

There is a learning curve that usually takes 6–7 months to develop the necessary three-dimensional skills in accessing the sacral canal as described above. To thread the X-L catheter the steering is made easier if a 1-inch, 15-degree bend is placed near the tip. Early in the learning curve, a common problem develops from the tip of the needle being too close to the side wall of the sacral canal. The needle can be pushed or the tip rotated so that the threaded catheter is advanced just short of the midline on the affected side. The target site is inevitably the ventrolateral epidural space. Steering the catheter with the tip of the catheter pointing toward the neural foramen and the bent-tip elbow pointing medially usually represents ventrolateral catheter tip placement. This can be verified on lateral fluoroscopic visualization.

The ideal epidural catheter for use is a stainless-steel, fluoropolymer-coated, spiral-tipped Racz Tun-XL-24.[59,98] A Racz catheter is passed through the needle into the scar tissue. The bevel of the needle should be facing the ventrolateral aspect of the caudal canal of the affected side. This turning of the needle facilitates passage of the catheter to the desired side, and decreases the chance of shearing the catheter. Because scar formation is usually uneven, multiple passes may be necessary to place the catheter into the scarred area. For this reason, it is best to use a 15-gauge RX epidural needle, which has been specially designed to allow multiple passes of the catheter.[99] To facilitate steering of the catheter into the desired location, a 15-degree bend is placed at the distal end of the catheter. After final placement of the catheter and negative aspiration, another 3–5 ml of contrast medium (maximum of a total of 20 ml) is injected through the catheter (Figure 22-4). This additional dye should be seen spreading into the area of the previous filling

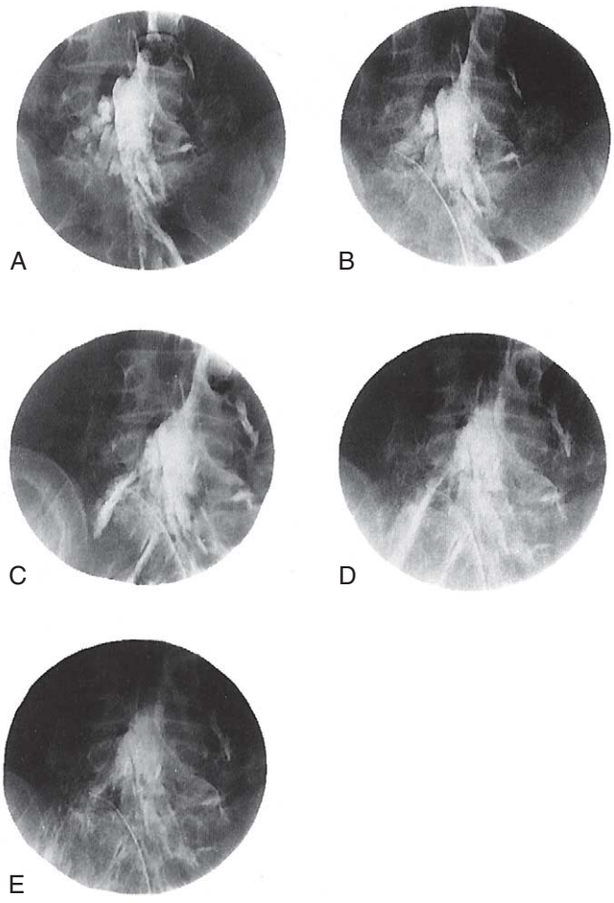


FIGURE 22–4

Radiographs from a patient with left lower extremity pain and foot drop. (A) After 10 mL of iohexol (Omnipaque) 240. Note complete filling defect on left at L4 and S1 and partial filling of the L5 spinal nerve. (B) Racz Tun-L-Kath-SL threaded into the L5 neural foramen. (C) After injection of another 10 mL of iohexol, note the opening of the L5-S1 nerve root and cephalad spread of contrast medium. (D-, E) Further opening of filling defect and cephalad spread of contrast medium. (From Waldman SD [ed]: Interventional Pain Management, 2nd ed. Philadelphia, WB Saunders, 2001, pp. 441.)

defect with outlining of the targeted nerve root. Next, 1500 U of hyaluronidase (Wydase) in solution with 10 ml of preservative-free normal saline is injected rapidly. Afterward 10 ml of 0.2% ropivacaine and 40 mg of triamcinolone are injected through the catheter in divided doses after negative aspiration. This additional volume is helpful in further lysis of adhesions because the catheter tip is in the scar tissue. The area of scarring and subsequent scar dissection should be noted and recorded. The steroids cannot be injected through the 0.2-micron bacteriostatic filter, so the steroid must be injected prior to placement of the in-line filter.

If contrast media is not used because of an allergic history, the procedure is the same except for the absence of dye. Aspiration should be negative for CSF and blood prior to any injection. Additionally, a test dose of local anesthetic should be given to verify that the needle and subsequently the catheter are not subarachnoid or subdural. When properly placed, the patient often reports pain with injection in the dermatomal distribution of the scarred area.

When the procedure is completed, the catheter should be secured to the skin with 3-0 nylon on a cutting needle. Caution must be taken to not puncture the catheter with the needle, as well as not cut the catheter coating while wrapping it. Triple-antibiotic ointment, such as polymyxin, and two 2×2-inch split gauze pads are used to cover the catheter exit site. The surrounding skin is sprayed or covered with tincture of benzoin and, with a single loop of the catheter toward midline, all of the above is covered with a 4×6–inch size sterile transparent surgical dressing. On top of the transparent dressing, we place two 4×4–inch gauze pads over the puncture site and apply four pieces of 6-inch-long Hypafix tape over the area. This Hypafix tape has the unique ability of being elastic yet porous, so that the patient does not "sweat it off" during the 3 days that the catheter is kept in place. Prior to undraping the sterile field, the catheter is connected to an adapter and a 0.2-micron bacteriostatic filter that are not removed during the duration of the three daily injections. The filter is capped, and the catheter is taped to the flank of the patient. In the preoperative area and during hospitalization, the patient is given IV antibiotics in the form of cephalosporins, such as ceftriaxone (Rocephin), 1 g daily intravenously. Prophylactic antibiotics are given to prevent bacterial colonization, which is especially hazardous in view of the epidurally administered steroid. It is also our practice to send a patient home on an oral antibiotic for 5 additional days as epidural abscess prophylaxis.

POSTPROCEDURE MONITORING AND INJECTION

Once the patient is taken to the recovery room and vital signs are obtained for 20–30 minutes and there are no signs of motor block, including the ability to flex the hip and raise the leg to 90 degrees, 10 ml of the 10% hypertonic saline is infused over 20–30 minutes. For the infusion, the patient is placed in the lateral position with the painful side down and remains in this position for 30 minutes following the completion of the infusion. The purpose of this positioning is to have a prolonged hyperosmolar effect on the swollen painful nerve roots in order to reduce the edema and facilitate functional recovery. Please note that the volume of hypertonic saline is the same or less than the volume of local anesthetic previously injected. Occasionally, the patient may complain of severe burning pain during the infusion. The burning is usually from the introduction of hypertonic saline to unanesthesized epidural tissue. Should this occur, the infusion must be stopped and a 3–5-ml bolus of additional local anesthetic is injected. After 5 minutes, the hypertonic saline infusion can be restarted without incident. Following completion of the hypertonic saline infusion, 1.5 ml of preservative-free normal saline is used to flush the catheter. Once this task is completed, the cap is replaced on the filter.

The hypertonic saline has a mild, reversible local anesthetic effect and also reduces edema of previously scarred or inflamed nerve roots.[100,101] Injection of hypertonic solutions into the normal epidural space is quite painful unless preceded by local anesthetic. If the hypertonic saline spread is greater than the coverage area of the local anesthetic, the patient may have severe pain. The pain caused by the hypertonic saline in the epidural space rarely persists more than 5 minutes.

The absence of motor block from local anesthetic is an assurance of epidural placement. Hypertonic saline will expand by osmotic effect to 11 times the original injected volume, so that if it is injected in the subdural or subarachnoid spaces, the hypertonic saline would exert pressures on surrounding subdural structures secondary to the volume expansion. Therefore, it is important to use radiological, contrast, and pharmacological confirmation of placement to prevent the volume-related compression of structures within the dural sac.

The catheter is left in place for 3 days. On days 2 and 3, the catheter is injected once a day with 10 ml of 0.2% ropivacaine after negative aspiration from the catheter. Fifteen minutes later, 9 ml of 10% saline is infused over 20 minutes for patient comfort. As with all hypertonic saline infusion series, the catheter must be flushed with 1.5 ml of preservative-free normal saline. On day 3, the catheter is removed 10 minutes after the last injection. A triple-antibiotic ointment is placed on the wound and is covered by a bandage or other appropriate dressing.

CLINICAL PEARLS

We inject only one dose of steroid and do so in the operating room under total sterile conditions. After the bacteriostatic filter is placed, it is not removed during the series of reinjections. We have demonstrated in our laboratory that when methylprednisolone (Depo-Medrol) plus local anesthetic or triamcinolone (Aristocort) and local anesthetic are injected through a bacteriostatic filter, the filter screens out virtually all of the steroid.[102]

During the time that the catheter is indwelling, the patient should keep the insertion site dry. We also recommend

that the patient keep the area dry for 48 hours after removal to decrease the chance of infection. Showering is permitted after this period, but immersion of the wound such as in a bath or pool therapy should be avoided for a minimum of 7 to 10 days.

This procedure is usually followed by significant improvement in pain and motor function. With improvement of pain, it is important to initiate aggressive physical therapy to improve muscle strength and tone, which is usually decreased from lack of use secondary to pain. Often it is not possible to completely lyse existing epidural adhesions because of the extensive amount of scar tissue. If necessary, we repeat the procedure. Because of the steroids used, a 3-month delay between procedures is necessary, during which time the patient should be encouraged to continue intense physical therapy. This therapy should begin immediately, when possible. Initiation of neural flossing techniques, especially while the local anesthetic is still active, provides a prime opportunity to maximize the adhesiolysis process with the least discomfort to the patient. One month of aquatic therapy followed by aggressive, graded physical therapy and work hardening is also recommended.

After negative aspiration is noted, all solutions should be injected slowly. Observation of the fluoroscopies often initially reveals massive epidural scar formation, as seen in the series of radiographs shown in Figure 22-3. In Figure 22-3A, after injection of 10 ml of Omnipaque 240, one can see the dye preferentially spreading toward the right side, opening up the right L4-L5 and S1-S2 nerve roots, whereas there is a complete filling defect of the left L4 and S1 and partial filling of the L5 nerve root. In Figure 22-3B, a Racz Tun-L-XL catheter is threaded into the L5 neural foramen area, and through this, an injection of an additional 10 ml of Omnipaque is seen to open up the L5, S1 nerve root, as well as to spread cephalad as evidenced by the disappearance of the L4-L5 disc space because this space is masked by the spreading contrast. This is followed by the injection of 10 ml of preservative-free saline, 1500 U of hyaluronidase spreading to L4 and L5, and finally the 10 ml of 0.2% ropivacaine and 40 mg of triamcinolone. The contrast is spreading up to L4, L5, and then to S1, evenly, almost like a Christmas tree appearance. The foot drop dramatically improved the following day as a result of the decompression of the L4-L5, S1 nerve roots by dissection of the perineural space with the injected material.

Immediate monitoring is for acute effects of spinal local anesthetic and mechanical complications. The initial 4 hours should include bed rest or supervised walking, as partial motor block may be present. Bladder function and motor recovery should be present before the patient is allowed to ambulate independently.

Patients should be warned that perianal numbness may be transiently present, especially in patients with spinal stenosis due to a transient neuropraxia. In the presence of persistent neuropraxia, an MRI should be considered to evaluate for possible complications, especially loculation and spinal cord compression.

Delayed monitoring is aimed at hematoma formation and infection.

COMPLICATIONS

Injection of local anesthetic into the subarachnoid or subdural space results in a motor block that is notably more profound and of more rapid onset than that subsequent to injection into the epidural space. A subdural block is often typified by a segmental motor block with a diffuse sensory block to the level expected from a subarachnoid injection of local anesthetic.

If CSF is aspirated, it is best to abort the procedure and repeat it another day. If blood is aspirated, the needle is first retracted caudad in the sacral canal until no blood can be aspirated. If this is unsuccessful, an attempt can be made to proceed with catheter placement into the proper site. Aspiration through this catheter should be negative for blood, and lack of venous runoff should be confirmed with injection of contrast medium. The adverse effects include bruising, transient hypotension, transient breathing difficulty, numbness of the extremities, bowel or bladder dysfunction, paralysis, infection, sexual dysfunction, and the possibility that the catheter might shear.

The most common idiosyncratic reaction occurring after intrathecal iohexol is headache, which occurs in approximately 18% of patients.[66] Myalgias, nausea, vomiting, and dizziness may also occur. Aseptic meningitis and neurologic disturbances have been reported as infrequent complications. In addition, allergic or anaphylactoid reactions may occur rarely with nonionic agents, but far less frequently than with their ionic predecessors.[68]

The exact complication rate of iohexol epidurography is unknown; however, a number of studies on the complications of iohexol myelography have provided some indications regarding the safety margin of epidurography. Ndosi and colleagues[69] determined in a double-blinded, placebo-controlled trial that myelography with appropriate concentrations of iohexol (240 mgI/ml) carried no more risk of significant reaction than a diagnostic lumbar puncture. The same study demonstrated that iohexol concentrations of 180 and 300 mgI/ml were more likely to result in headache, dizziness, nausea, vomiting, and seizures.

Insect hyaluronidase is an allergen in stinging-insect venoms and has a known homology to mammalian hyaluronidase.[77] Anaphylactic-like reactions have occurred in isolated cases.[78] Heightened awareness of this complication should be considered when treating patients with a history of venom allergy.

The significant toxicity of bupivacaine is cardiotoxicity. This usually results from accidental intravascular administration of large doses of bupivacaine. Bupivacaine disassociates from sodium channels more slowly than lidocaine during cardiac diastole; therefore, its effect is more

pronounced and cumulative. This leads to severe cardiac arrhythmias and myocardial depression.

Systemic toxicity of triamcinolone diacetate depends on the dose and duration of treatment and the rapidity with which it is absorbed from the epidural space. These kinetics have not yet been elucidated; however, after epidural administration, suppression of the hypothalamic-pituitary-adrenal axis has been shown to persist for 21 days.[84] The potential systemic toxic effects, although rare, are related to its glucocorticoid activity and include fluid, electrolyte, and metabolic disturbances; muscle wasting; peptic ulcer; and impaired wound healing and immunologicfunction.[85] Allergic reactions have been reported in rare instances.[86] Intrathecal corticosteroid administration may be a serious complication of epidural corticosteroid injections since depot formulations commonly contain polyethylene glycol, which may cause arachnoiditis when administered intrathecally. Cases of aseptic and bacterial meningitis, as well as epidural abscess, have been reported rarely.[88,89]

Clinical complications of intrathecal hypertonic saline have been well described in multiple investigations. They include cardiac, respiratory, or neurologic sequelae in approximately 10% of patients.[52] In addition, the discomfort associated with intrathecal administration requires general anesthesia. Serious complications can occur when osmotic effects of hypertonic saline cause elevated CSF pressure, which in turn results in hypertension, tachycardia, and tachypnea with pulmonary edema. These changes can occur precipitously and with hemorrhagic consequences.[52]

Conversely, such complications from hypertonic saline injected epidurally are not observed, although two cases of arachnoiditis were reported by Aldrete and colleagues.[94] It was suggested the solutions may have been injected into the subarachnoid space in these cases. These researchers' results were based on a survey of 72 patients who were randomly selected from a pool of approximately 200 patients who had the procedure performed, as well as follow-up with these patients, which occurred 6 months to 1 year later. They found that 25% of the patients did not decrease their use of pain medication, 43% decreased their dosage and frequency of their medication use, 16.7% discontinued pain medication, and only 1.4% increased their use of pain medication. Although 72.2% of the patients reported pain relief on discharge, 25% reported no relief and 2.8% reported worse pain on discharge; 37.5% of the patients reported less than 1 month of relief, 30.5% reported 1 to 3 months relief, and 12.5% reported 3 to 6 months relief. In total, 30.6% of the patients returned to work or returned to daily functions.

Arthur and colleagues[103] described a study at the 7th World Congress on Pain in which the lysis of adhesions technique was identical to the present technique in a randomly selected group of patients for a retrospective review. The selection criterion was hyaluronidase injection in 50 patients, and the results compared with 50 patients who were not administered hyaluronidase. The results showed 81.6% of the hyaluronidase group had pain relief, with 12.3% having persistent relief, but 6.1% reported no response; 68% of the no-hyaluronidase group had relief of pain, with 14% having persistent relief, but 18% reported no response at the end of 3-year follow-up.[103] The hyaluronidase group had a threefold reduction in the failure rate.

A study by Devulder and coworkers[104] in 1995 was based on 34 patients in whom epidural adhesions were suspected based on either magnetic resonance imaging or their history of back surgery. In their protocol, in which hyaluronidase was not employed, an epidural catheter was placed via the sacral hiatus under fluoroscopy but without direction toward the affected site. The catheter was simply advanced 10 cm into the epidural space, and 10 ml of contrast agent (10 hexol 240 mgI/ml) was injected. Defects that corresponded to the patients' pain were demonstrated in the resulting epidurograms of 30 of the 34 patients. Injection of 20 ml of 2% lidocaine with 80 mg of methylprednisolone added was followed by 10 ml of 10% hypertonic saline. The procedures were repeated on days 2 and 3 via the indwelling catheter. The researchers noted a regression of adhesions in 14 of the 30 patients who had had defects. Seven of these patients reported marked improvement of their pain, defined as a visual analog scale score of less than 4 at 1 month. Only two of these patients reported this level of improvement at 3 months, and at 1 year this entire group of patients had undergone a different treatment because their pain returned. Only four of the patients without any improvement of contrast spread reported marked pain relief at 1 month, two at 3 months; and one remained pain free at 1 year. Chi-square analysis of these data showed no statistically significant correlation between enhanced contrast spread after the injections and a better outcome. This procedure has been criticized for the lack of guidance of the catheter tip into the lesion and demonstrates the importance of directing the catheter tip into the lesion.

Manchikanti et al.[105] used the same approach but as a control group in a prospective randomized trial of lysis of adhesions of targeted lesions in the ventrolateral epidural space, and found no improvement with the technique described by Devulder and colleagues[104] but reported significant long-term improvement with the Racz technique.

TRANSFORAMINAL NEUROPLASTY

HISTORY

Lumbar transforaminal injections were first developed as a method to inject the DRG. The idea of transforaminal needle and catheter technique was first presented by Michael Hammer.[106–111] More recent techniques shifting the emphasis to the superior pars are described in this chapter.For a detailed description of this technique see also chapter 17.

ANATOMY

The lumbar spine consists of five lumbar vertebrae. The borders of the lumbar foramen consist of the vertebral body and disc anteriorly, the pedicles superiorly and inferiorly, and the facet particular processes posteriorly. Within the foramen the nerve root exits in an anterocaudal direction. Anterior to the nerve root, radicular vessels can be found to follow the nerve root into the epidural space. Posterior to the nerve root is the DRG.

INDICATIONS

Transforaminal neuroplasty may be indicated when the nerve roots are difficult to "open" or when access to the anterior space is needed.

CONTRAINDICATIONS

The main contraindications to transforaminal neuroplasty are local infection and coagulopathies.

DRUGS AND EQUIPMENT

See drugs described in caudal neuroplasty section.

PATIENT POSITION

The patient is in the prone position with enough table clearance to provide full range of rotation of the fluoroscopy.

TECHNIQUE

After consent, the patient is placed in the prone position. Using sterile preparation and technique, the back is cleansed with a sterilizing solution from just below the scapula to the lower margin of the buttocks. Preparation of the lumbar region is appropriate only if the upper lumbar region is the source of the problem, without sciatic involvement. Using fluoroscopy the desired lumbar level and side are identified. The fluoroscope is then oblique to the ipsilateral side of the desired foramina to 15–20 degrees. At this point the spinous process moves to the other side. Once a "Scottie dog" image is obtained, the fluoroscope is then rotated in a caudal-cephalad direction for 15–20 degrees to optimize the fluoroscopic view of the neural foramen, and the tip of the dog's ear is just overlapping the disk space. It is worthwhile to remember that the fluoroscope movement and the Scotty dog's ear movement, the superior pars, occur in the opposite direction. A caudal-cephalad rotation elongates the superior articular process ("ear of the Scottie dog"). The tip of the ear or superior articular process in a "gun-barrel" technique is marked on the skin. This spot is the skin entry site, and local anesthetic is injected for skin infiltration. An18-gauge needle is used to make a puncture wound. Through this wound, a 15–16-gauge Epimed RX Coudé epidural needle is advanced anteriorly, until the tip of the dog's ear (superior pars) is contacted curving medially. Next, a lateral fluoroscopic view is obtained prior to further introduction of the needle. To facilitate passage of the needle past the articular process, the epidural needle is turned laterally to slide past the bone and turned medially and slowly advanced until a "pop" is felt. This "pop" represents the penetration of the intertransverse ligament. The needle tip on a lateral view should be in the posterior aspect of the foramen. An Epimed Tun-L-XL or Brevi-XL epidural catheter is then inserted through the epidural needle. Occasionally, the epidural needle must be tilted at the hub laterally to aid entry of the epidural catheter into the anterior epidural space. The catheter is advanced medial to the pedicle. After catheter placement is confirmed to be in the anterior epidural space under lateral view, the stylette is removed from the catheter and a connector is placed on the proximal end of the epidural catheter (Figure 20-5). Aspiration should be negative before 3 ml of iohexol (Omnipaque 240) radiographic contrast is injected. The contrast injection should show opening of the entered neuroforamen with contrast agent exiting along the path of the nerve root. When satisfactory contrast spread is seen, 6 ml of 1500 U of hyaluronidase in 10 ml of preservative-free normal saline are injected to facilitate the opening of other adhesions. Lastly, 2 ml increments of 0.2% ropivacaine or 0.25% bupivacaine with 40 mg of triamcinolone diacetate solution are injected to a total volume of 6 ml of local anesthetic/steroid mixture. During the injection, the contrast must be seen spreading out through the neural foramen, and hyaluronidase has a significant role in opening up the scarred-down epidural space. If all the injection volume loculates within the epidural space without spreading outside, it could represent a significant loculation hazard. This occurrence is prevented by ventral epidural catheter placement and multiple fluoroscopic views to verify lateral spread.

The catheter is then secured in place with 2-0 nylon on a cutting needle, and a 0.2-micron bacterial filter is connected. The dressing consists of triple-antibiotic ointment over the wound site, a 2 × 2–slotted gauze pad over the ointment, small inferior loop of the catheter, and cover with a transparent surgical dressing. For added security, the dressing is further covered by Micropore tape.

After the patient is transported to the recovery room and negative aspiration of the catheter is confirmed, 5 ml of 10% hypertonic saline are infused over 20 minutes. Once the infusion is completed, the epidural catheter is cleared with 1–2 ml of preservative-free normal saline. The epidural catheter is then left in place for reinjections two and three on days 2 and 3, or in some cases the second and third injections are performed on day 2. Because of patient movement, the transforaminal catheters often migrate out prior to the second and third injections; therefore, prior to reinjections, fluoroscopic verification is carried out. If the catheter has migrated out, it is removed. For second or third injection, the catheter is checked for correct placement. The local anesthetic is

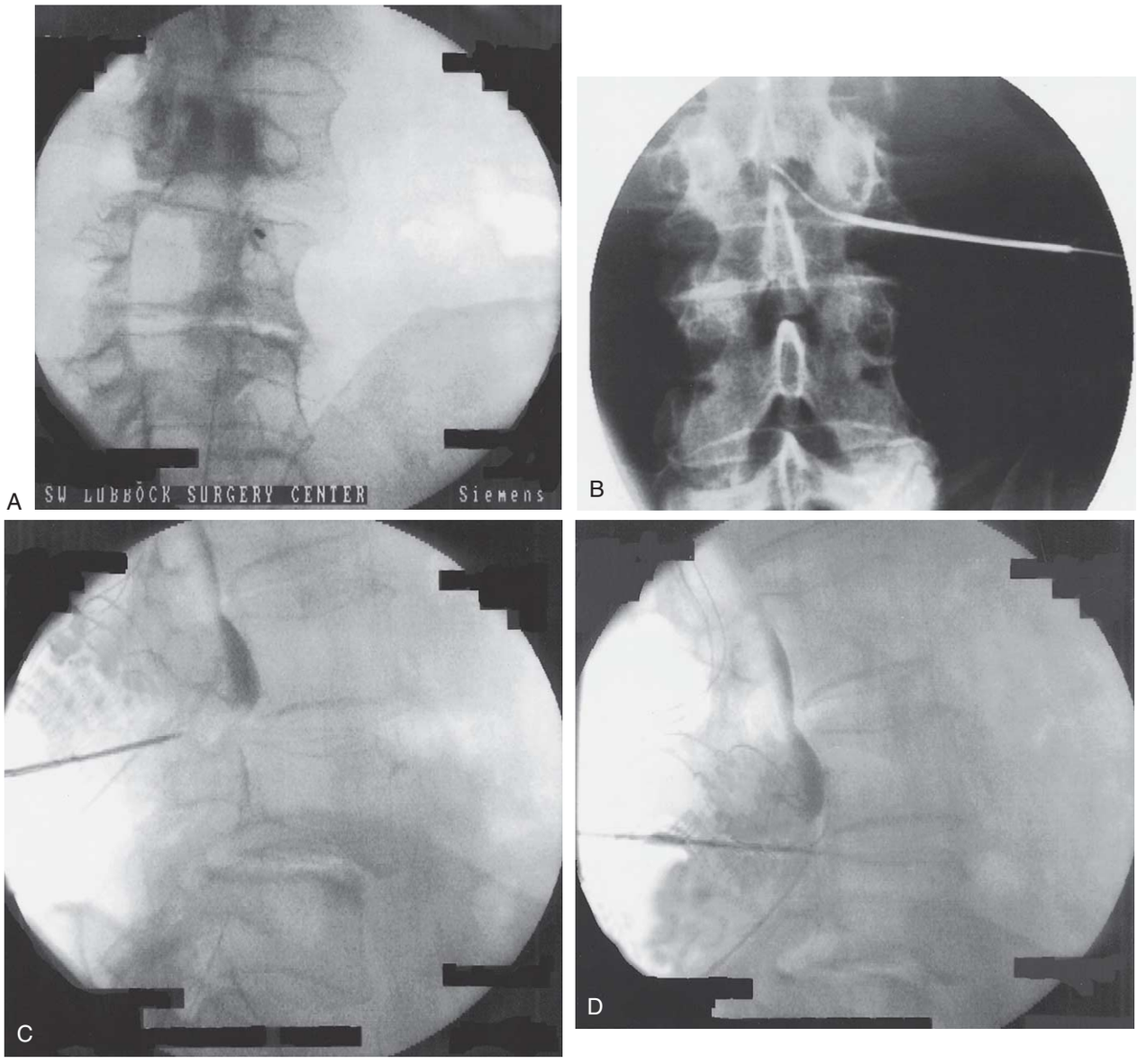


FIGURE 22–5

(A) Oblique view of the lumbar spine showing the needle entry point for the transforaminal technique. (B) Anteroposterior view of the transforaminal technique with catheter entering the epidural space. (C) Lateral view of the transforaminal technique with the catheter entering the epidural space. (D) Lateral radiographic imaging of the lumbosacral region in the transforaminal technique showing the passage of the catheter anteriorly with contrast spread from the tip.

given in divided doses (2 ml and 4–6 ml) at 5-minute intervals. As with the first injection, the hypertonic saline (5 ml) is infused over 20 minutes and flushed with 1–2 ml of normal saline.

The possibility of subdural or intravascular injection should be monitored. Removal of the catheter is performed after the third infusion. Care must be taken to remove the epidural catheter intact.

EFFICACY

Igarashi and colleagues[64] reported better results in patients with single level pathology compared with patients with multilevel pathology. Gerdesmeyer et al.[65] reported dramatic improvement in Oswestry scores from 64 to 22 in a pilot study. A multicenter trial is ongoing.

Heavner and colleagues[106] reported a randomized trial with four groups: hypertonic saline plus hyaluronidase, hypertonic saline, isotonic saline, and isotonic saline plus hyaluronidase. Patients in the two hypertonic saline groups required less treatment for pain in the long-term follow-up period. At the 12-month follow-up, the effect of hypertonic saline plus hyaluronidase was most effective.

Manchikanti and coworkers[105] reported results from comparing three groups: (1) nonfluoroscopic-guided local anesthetic and steroid, (2) fluoroscopic-guided local anesthetic plus steroid without hypertonic, and (3) fluoroscopic-guided local anesthetic plus steroid with hypertonic saline.

Outcomes from group 3 were better than group 2, which was better than group 1.

SACRAL NERVE ROOT INJECTION

HISTORY

The origin of selective nerve root blockade (SNRB) can be traced back to 1906, when it was performed for urologic surgery. A few years later, Kappis[112] described the blockade of the brachial plexus via the cervical nerve roots. Paucet is credited for injecting sacral nerve roots (trans-sacral anesthesia) to salvage inadequate caudal anesthesia for obstetrics in 1914.[113] Krempen and Smith[114] advocated nerve root blocks as a diagnostic tool in 1974.

Lysis of adhesions of lumbosacral nerve roots was reported in 1989 by Holubec and Racz,[115] who noted that loculation can occur within the sacral canal not just in the spinal canal, causing compression and neurological deficits.

Nerve root blocks are used for three purposes: diagnostic, prognostic, and therapeutic. The sensitivity and specificity of diagnostic and prognostic blocks have been an issue. Clark and Awad[116] reported that 1 ml of injectate would adequately and selectively block 7 out of 9 nerve roots and 2 ml, 8 out of 9, but that 2 ml could spread to other levels.

The question exists as to whether a selective nerve block can be reliably performed.[117] A number of techniques have recently been described with interesting theoretical advantages. Catheter placement for selective nerve blocks has gained some popularity. Vranke et al.[118] reported placing a catheter via the inferior first sacral foramen for continuous infusion in a patient with ovarian cancer and sacral involvement. This technique seems useful in patients with metastatic disease affecting primarily one nerve root because it allows for very localized effects of local anesthetic. Friedman and colleagues[119] used a catheter for a selective nerve root block. Catheter injections may have a safety advantage over needle injections.

Computerized tomography is being used more frequently for injections. Quinn et al.[120] reported diagnostic benefit using CT-guided blocks in patients with inconclusive radiologic studies. Morel and coworkers[121] use CT in conjunction with neurostimulator guidance. Berger and colleagues[122] advocate the use of CT not only for needle placement but visualization of injectate spread as well.

ANATOMY

The sacrum is a large, triangular bone composed of five fused sacral vertebrae. There are eight sacral foramina, each with a ventral and a dorsal opening. The dorsal sacral foramina are located just lateral to the intermediate sacral crest that represents the fused articular processes of the sacral vertebra.[123] The dorsal S1 foramen is located approximately 1 cm medial to the posterior superior iliac spine (PSIS), while the S2 foramen is 1 cm medial and 1 cm inferior to the PSIS. The S3 foramen is located at the level of the posterior inferior iliac spine midway between the S2 and S4 foramina. The S4 foramen is immediately lateral to the sacral hiatus and just superior to the sacral cornua. The sacral foramina are somewhat rounded in form and diminish in size from above downward. The posterior sacral foramina are superior to the corresponding anterior foramina. Also, the posterior S1 foramen has an elevated inferior lip.

Nerve roots divide into anterior and posterior divisions that exit the sacrum through their respective sacral foramina (S1-S4). The fifth sacral nerve and the coccygeal nerve exit inferiorly through the sacral hiatus.

Lateral or median sacral arteries give off branches that pass into the sacral canal via the sacral foramina (Figure 22-6).

INDICATIONS

The most common sacral nerve root block is the S1 root for lumbosacral radiculopathy.

- L5-S1 spondylosis or disk protrusion
- Epidural scarring at L5-S1
- Post-traumatic lumbosacral radiculopathy
- Postsacral fracture radiculopathy

Based on years of experience and thousands of cases of epidural scarring, we have learned that epidural scarring commonly involves the lumbosacral area; however, the S3 and S4 nerve roots are rarely involved with dense scar tissue formation. Addressing the issue of scarring, most commonly the S1 root block is performed.

There has been a report of paralysis from an S1 root block with a sharp needle.[124] Injury to nerve roots from sharp needles can be followed by chronic and significant sacral neuropathy. Fortunately, these are rare events and we cannot determine the incidence.

CONTRAINDICATIONS

- Local infection
- Coagulopathy
- Bony abnormality of the sacrum
- Tarlov cyst in sacral canal
- Arachnoiditis

EQUIPMENT

- Nerve block tray
- 25-gauge needle for skin infiltration
- 3-inch, 20–22-gauge, blunt Coudé needle
- 16-gauge IV introducer needle

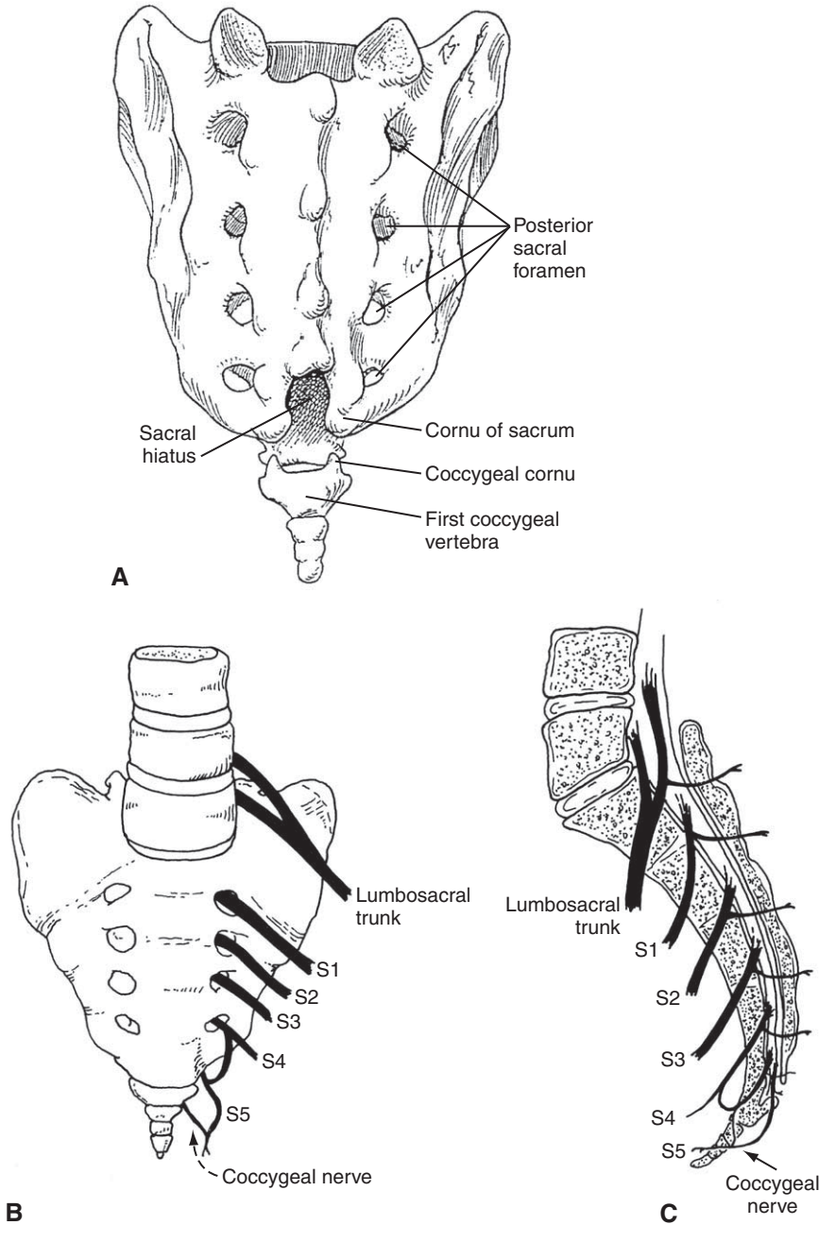


FIGURE 22–6

(A) Anatomy of the sacral hiatus and dorsum of the sacrum. (From Raj PP, editor: *Clinical Practice of Regional Anesthesia*. New York, Churchill Livingstone, 1991, p. 328, with permission.) (B) Anterior view of the sacrum with anterior sacral nerve root exiting. (C) Lateral view of the sacrum that shows the sacral nerves exiting the foramen both anteriorly and posteriorly.

- Three 10-ml syringes
- IV T-piece extension set

DRUGS

- 1.5% lidocaine
- 2% lidocaine
- 0.25% bupivacaine/0.2% ropivacaine
- Iohexol (Omnipaque 240) radiographic contrast
- 500 U hyaluronidase
- 10 ml preservative-free saline
- 4 mg dexamethasone

Pulsed Electromagnetic Field or Radiofrequency Thermocoagulation—Additional Equipment

- 10-cm, curved, blunt radiofrequency thermocoagulation (RFTC) needle
- RFTC electrode and connecting cables
- Grounding pad

PREPARATION OF PATIENT

One should examine for local infection and distorted anatomy that may interfere with performance of the procedure.

Laboratory Studies

Perform routine laboratory studies as indicated to rule out infection and coagulopathy.

Preprocedure Medication

For preoperative medication, use the standard recommendations by the American Society of Anesthesiologists for conscious sedation.

PROCEDURE

The patient is positioned in the prone position. Block of the sacral nerve roots is accomplished through the posterior sacral foramen. The site of entry is visualized by adjusting the fluoroscopic beam to align the chosen posterior foramen with the anterior foramen by rotating the C-arm cephalad and slightly laterally.[125] Use a marker to mark the inferomedial part of the neuroforamen and insert the IV cannula to the posterior neural foramen in a slightly superolateral direction (Figures 22-7 and 22-8). Remove the metal needle from the cannula. Insert the blunt Coudé needle curving in a caudal direction, which should pop through the foramen into the sacral canal (Figure 22-9). Lateral fluoroscopic visualization is used to confirm needle placement within the sacral canal (Figure 22-10). The needle is rotated 180 degrees to a cephalad direction and is advanced in the sacral canal and connected to the contrast-filled syringe. Aspiration is carried out, and if it is negative for blood, 2 ml of Omnipaque 240 contrast are injected under fluoroscopic visualization. The C-arm is rotated to the anteroposterior direction and aspiration is repeated; then 1–2 ml local anesthetic and 1 ml steroid are injected.

Radiofrequency of Sacral Roots

The goal is to position the tip of the needle directly adjacent to the DRG of the desired nerve root. A sensory paresthesia should be felt in the desired dermatome at less than 1.0 V at 50 Hz stimulation. Ideal stimulation should be felt between 0.4 and 0.6 V. If stimulation is felt at less than 0.4 V, the tip of the needle is too close to the DRG; and if stimulation is felt at greater than 0.6 V, the tip is too far away from the DRG. Motor stimulation is then performed at 2 Hz. There should be a clear dissociation between motor and sensory stimulation; that is, the voltage required to see motor fasciculations at 2 Hz should be at least two times the voltage that produces sensory stimulation at 50 Hz.[126] Thus, if good sensory stimulation at 50 Hz is noted at 0.5 V, the motor fasciculations at 2 Hz should not be seen at voltages less than 1.0 V. The point of dissociation defines the position of the DRG. If dissociation between sensory and motor stimulation cannot be obtained, the tip of the needle is not in alignment with the DRG, and lesioning at this point is not recommended.

Once the proper stimulation parameters have been achieved, inject 2 ml of local anesthetic with 40 mg of triamcinolone diacetate. Wait 3–5 minutes, and then lesion at 67°C for 90 seconds with conventional radiofrequency. The use of the procedure is less frequent due to cases of neuritis related to thermocoagulation.

Pulsed Electromagnetic Field

For pulsed electromagnetic field (pEMF), the position of the curved tip of the needle should be similar to that placed for radiofrequency of the DRG. Once the correct site of needle is confirmed by fluoroscopy, then the sensory stimulation at 50 Hz should be done. One expects paresthesia in

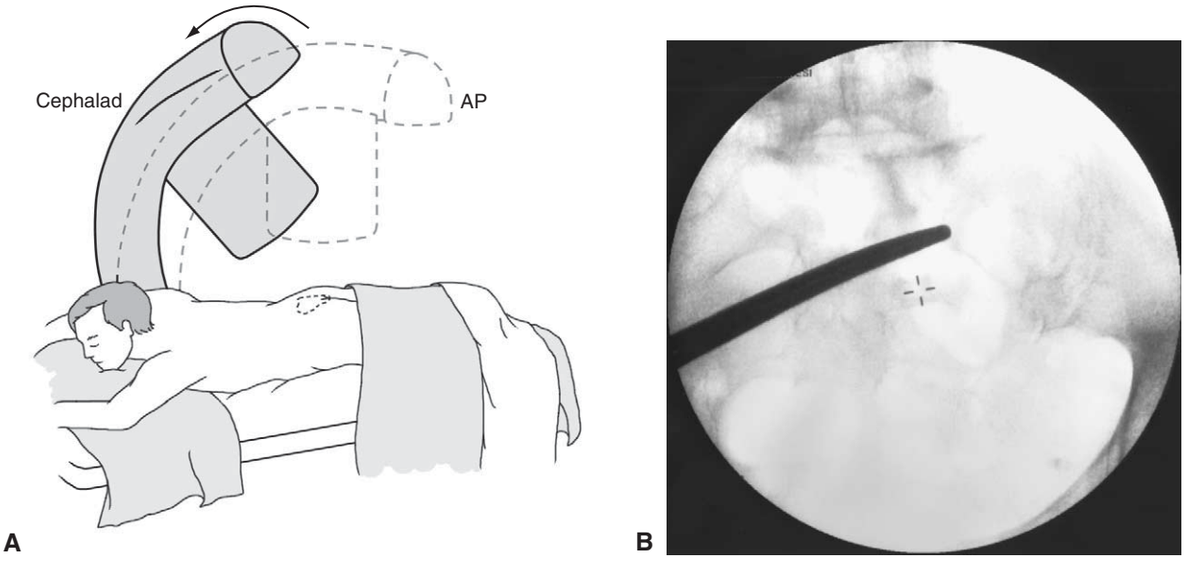


FIGURE 22–7

(A) C-arm position. (B) Sacral nerve root: surgical forceps used to locate skin entry point for first sacral foramen.

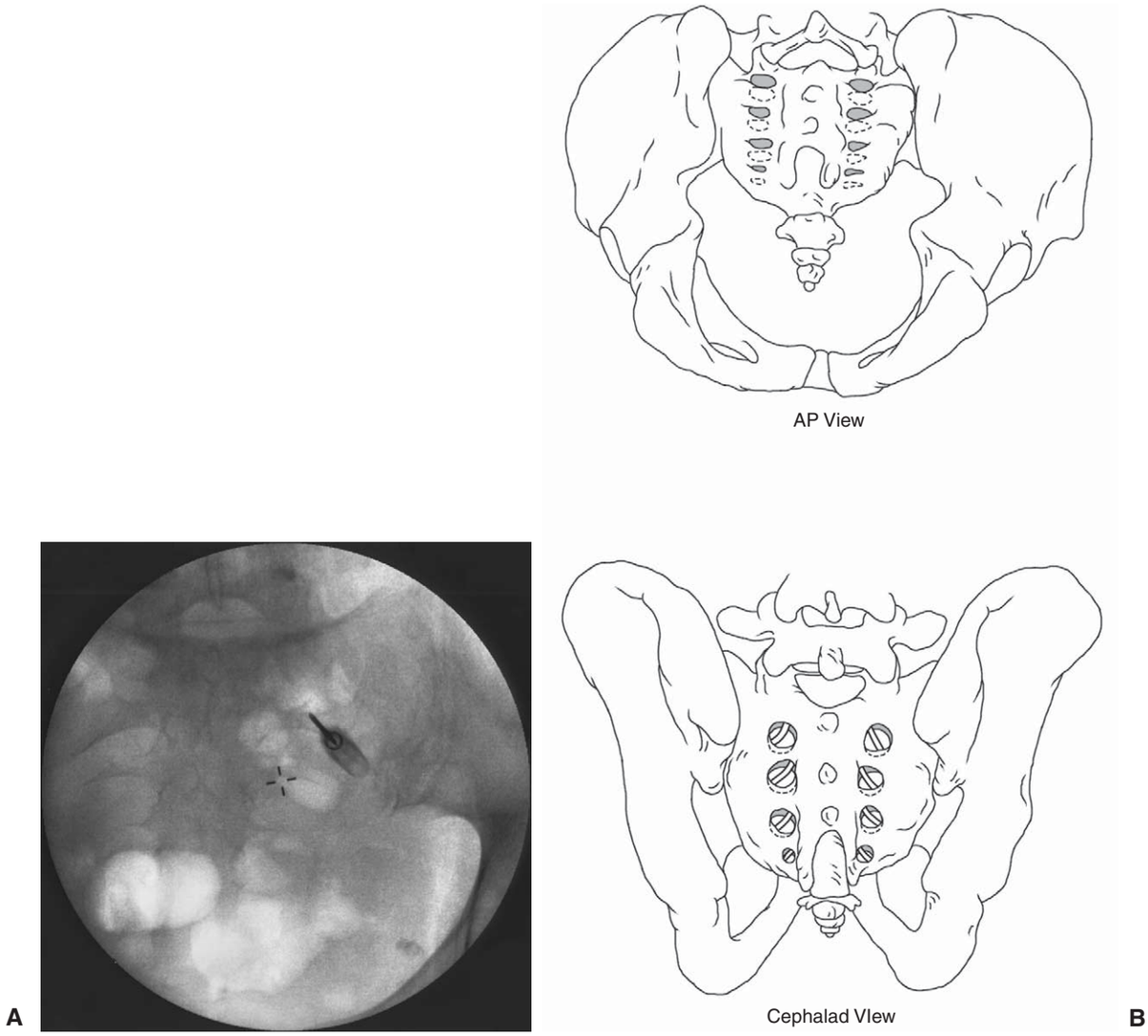


FIGURE 22–8

(A) Introducer cannula placement at SI foramen. (B) Views of sacrum from C-arm positions (cephalad and anteroposterior).

the distribution of that nerve root at about 0–3 V. pEMF is done at this site two or three times for 120 seconds and at 42°C. After pEMF, patient care is similar to conventional radiofrequency.

The clinical impression for outcome following pulsed radiofrequency procedures is better when local anesthetic and steroid are injected after the procedure. The injected local anesthetic and steroids lower the impedance during the procedure. There is no evidence to substantiate this clinical impression other than the completely different outcomes personally communicated by practitioners who do and do not use local anesthetic and steroid.

CAUTION

Injection of local anesthetic and steroid following pulsed radiofrequency, in very rare cases, is followed by paralysis in the lumbar area related to intra-arterial injection and cord infarction. Perceived incidence is approximately 1 in 10,000; therefore, our use of the blunt curved radiofrequency needle is recommended.

POSTPROCEDURE MONITORING

Patients should be monitored for at least 30 minutes for signs of spinal block or other complications.

COMPLICATIONS

One of the biggest concerns is damage to the nerve root while positioning the needle. Using a blunt-tip needle, one can reduce this complication. Neuritis after conventional radiofrequency lesioning is another concern. Sensory and motor testing is important to confirm safe and accurate electrode placement. If the proper parameters are not met, there is an increased

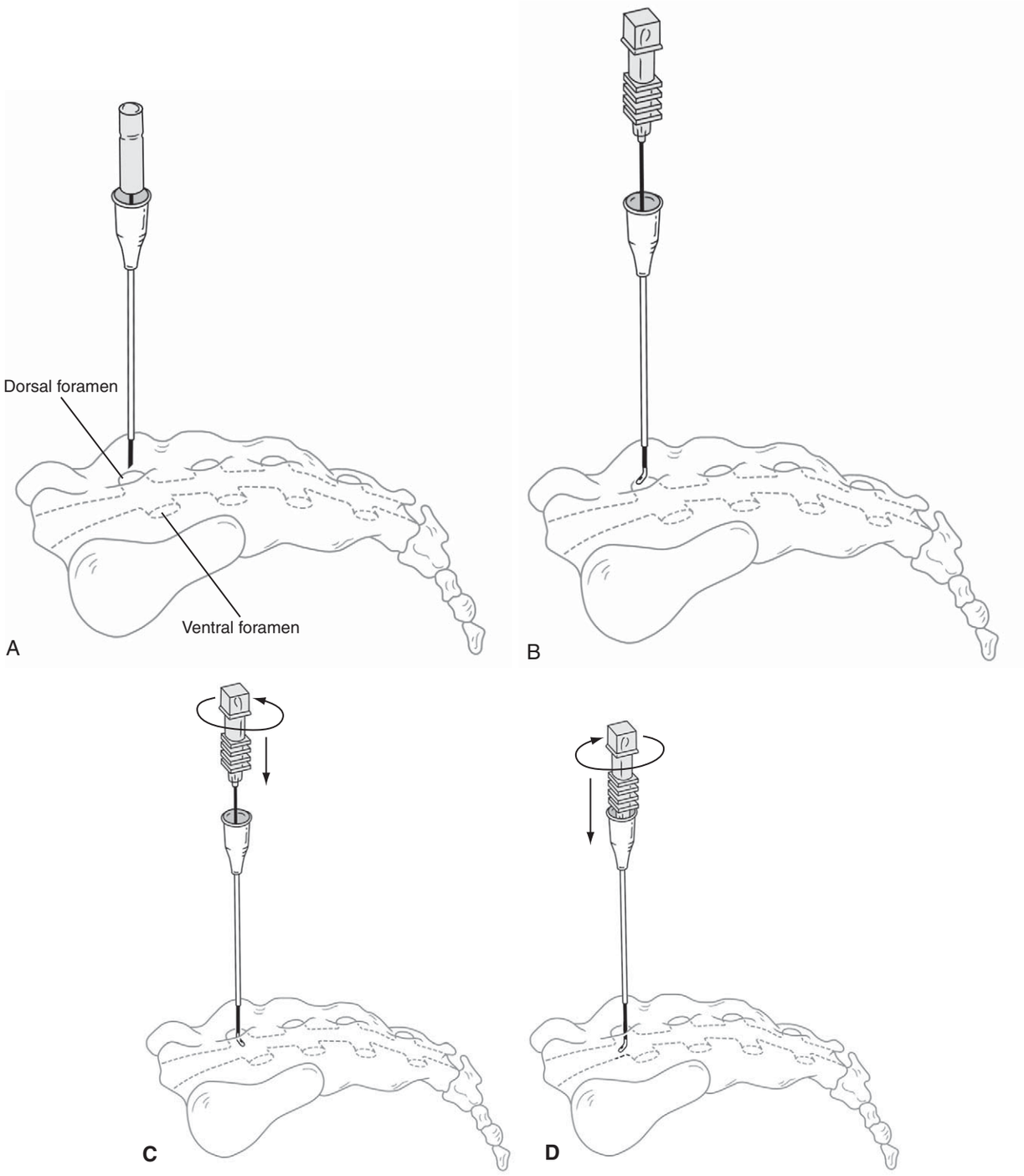


FIGURE 22–9

Curved blunt needle placement. (A) Introducer needle placement. (B) Blunt Coudé needle through introducer cannula. (C) Rotate blunt needle downward and enter posterior S1 foramen. (D) Rotate blunt needle cephalad within sacral canal.

incidence of postprocedure neuritis (30%). Injection of steroid prior to lesioning will help reduce, but not eliminate, the incidence of neuritis.

Other complications include intravascular and intrathecal injection of medication, paralysis, bowel and bladder incontinence, bruising, bleeding, increased pain, and infection.[127] Huntoon and colleagues[124] reported a case of paraplegia and spinal cord infarct after a transforaminal injection in a patient with previous spinal surgery.[124] Houten and Errico[128] reported three cases of sudden paraplegia after

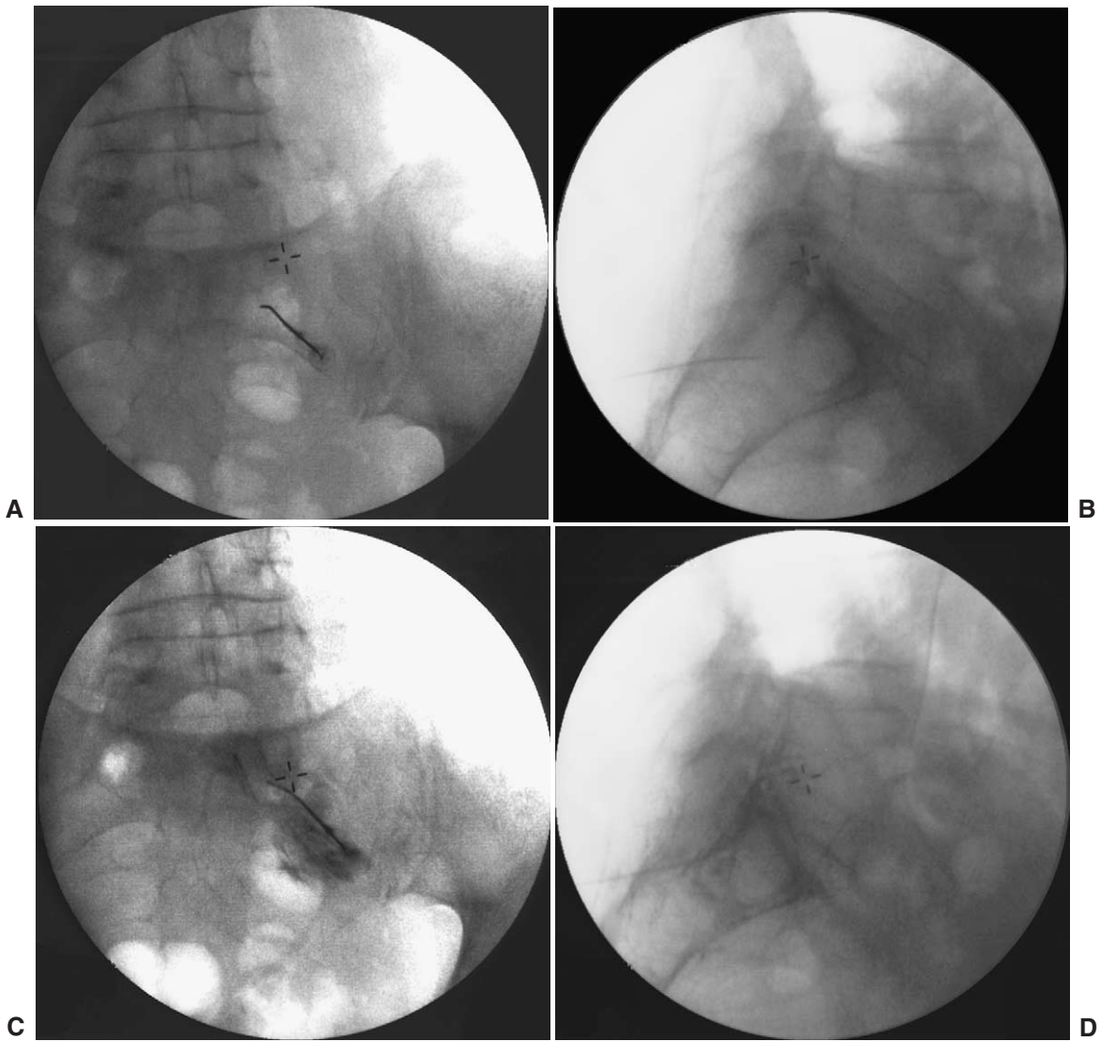


FIGURE 22–10

(A) Curved blunt needle placement. (B) Lateral image of placement in sacral canal. (C) Anteroposterior view: injection of contrast outlines S1 nerve root. (D) Contrast injection on lateral image.

steroid injection during nerve root block procedures. Some overlapping risks exist among nerve root, transforaminal, and interlaminar blocks. A review of the complication section of the chapters on each block is worthwhile. Huston and colleagues[129] reported a series of 151 patients and 306 selective nerve root injections with no major complications.

The incidence of major complications may be low, but the potential for devastating complications is such that the risk–benefit for each patient should be thoughtfully considered. Is a test dose of local anesthetic needed after contrast injection and before steroid injection?

EFFICACY

Diagnostic blocks are used to confirm clinical diagnoses when radiographic studies are not definitive. Jonsson and coworkers[130] advocated diagnostic blocks for patients with clinical radiculitis but inconclusive radiographic findings. In a number of patients, the correlation with diagnostic blocks is better than with radiographic studies.

Taguchi et al.[131] described results from diagnostic radiculography. Nerve root injection with contrast alone correlated very well with nerve root blocks with local anesthetic and intraoperative findings, especially with foraminal and extraforaminal problems.

Prognostic blocks are usually used to predict surgical outcome and select patients who are most likely to benefit from surgery.[132–134] Sato and Kikuchi[135] reported diagnostic and prognostic utility of nerve root blocks in patients with multilevel stenosis to determine the primary level of symptomatology in patients who eventually underwent surgery. The surgeons focused the surgical procedures on one symptomatic level, thus sparing the patients multiple-level procedures. However, Herron[136] found selective nerve root blocks to be poorly predictive of surgical outcome in patients with multiple surgeries.

The literature of efficacy for therapeutic nerve root blocks overlaps with the transforaminal injection literature. Kim et al.[137] reported success with nerve root blocks for pain relief in patients with vertebral compression fractures. Kolsi and colleagues[138] found no difference in a randomized trial comparing nerve root injections with interspinous steroid injections. In another randomized trial, Thomas and coworkers[139] found transforaminal injections to be helpful compared with interspinous injections. Karppinen et al.[140] demonstrated short-term efficacy in a randomized, controlled trial with transforaminal blocks. Vad et al.[141] reported benefit with transforaminal injections, but the methods section did not describe a randomized trial as indicated in the title of the paper. Patients chose between a transforaminal injection and a soft tissue injection. Riew and coworkers[142] reported pain relief and a reduction in the surgery rate in a randomized controlled trial. In a review, DePalma and colleagues[143] found evidence in the literature of effectiveness for transforaminal injections for radicular pain.

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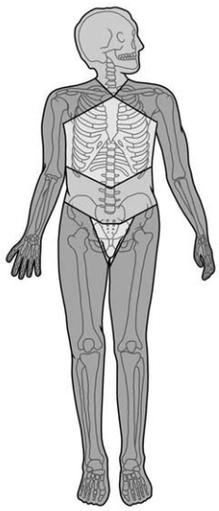
C H A P T E R



23 Sacroiliac Joint Blocks

LEE ANN PARADISE AND P. PRITHVI RAJ

HISTORY

The history of the sacroiliac joint (SIJ) and its painful syndromes have been controversial for hundreds of years.[1–10] Although Meckel first described motion in the joint in 1816, the quantity and quality of motion within the articular mechanism is still debated.[11] There is also controversy about the etiology of the gradual anatomical changes observed in the articular surfaces throughout life. Some believe these changes represent pathological degradation, and others believe the changes are a physiological adaptation. These controversies plague the physician who wishes to determine the role of the SIJ in a patient's pain with a low back pain history.

ANATOMY

BONY ARCHITECTURE OF SACROILIAC JOINT

There is great variability in the external contours of the SIJ articular surfaces. They are generally auricular in form, tending to be more of a C-shape in males and an L-shape in females. The articular surface spans from the S1 to S3 levels in both men and women and sometimes extends to S4 (Figure 23-1).[9,12] The joint surfaces at the S1 level are the largest, whereas the S3 surfaces are smallest. Each entire SIJ surface demonstrates a surface area of approximately 17.5 cm^2, allowing for shock absorption and a transfer of large bending forces.[13,14]

In the upright position, the joint orientation of the sacral propeller-form is about 20 degrees ventrolateral to dorsomedial at the S1 level. At the S2 level, orientation of the surface is less oblique. The precise degree of orientation differs from person to person, while the propeller form remains consistent. Ligaments restrain the movement during nutation, whereas architecture restrains counter-nutation movement as the joint surfaces approximate one another.[10]

The SIJ itself lies deep between the sacrum and ilium. The sacral cartilage is thick (3 mm), white, shiny, and smooth, whereas the iliac cartilage is thin (0.5 mm), bluish, dull, and rough. Although the sacral cartilage is thicker in women versus men, the iliac cartilage demonstrates the same thickness in both genders (Figure 23-2).[15]

The "joint" space is filled with fibrous cartilage, as there is very little congruence between the ilium prominence of this joint and its smaller concave sacral counterpart.

LIGAMENTS OF SACROILIAC JOINT

The ligamentous structures associated with the SIJ serves two functions. First, it enhances stability by increasing the friction in the SIJ and thus contributes to the self-locking mechanism.[16,17] Second, the system offers proprioceptive feedback in context with the rich plexus of articular receptors.

The ligaments surrounding the SIJ can be divided into four different layers, with the most superficial layer composed of the laminae of the thoracolumbar fascia. This fascial system can be divided into superficial and deep laminae.[17] The lower trapezius, latissimus dorsi, and external abdominal oblique all attach into the superficial lamina cranial to the SIJ, while the gluteus maximus attaches caudally. The fibers from one side of the superficial lamina attach to the spinous processes of L2 through L4, while fibers below the L4 region cross the midline and blend into the fibers of the contralateral gluteus maximus.

The next layer associated with the SIJ includes the sacrospinous and the sacrotuberous ligaments. These ligaments work in unison to restrain nutation movements at the SIJ. The sacrospinous ligament lies anterior to the sacrotuberous ligament and courses superiorly, medially, and posteriorly from the ischial spine to a broad insertion on the sacrum and coccyx[12,18] (Figures 23-3 and 23-4).

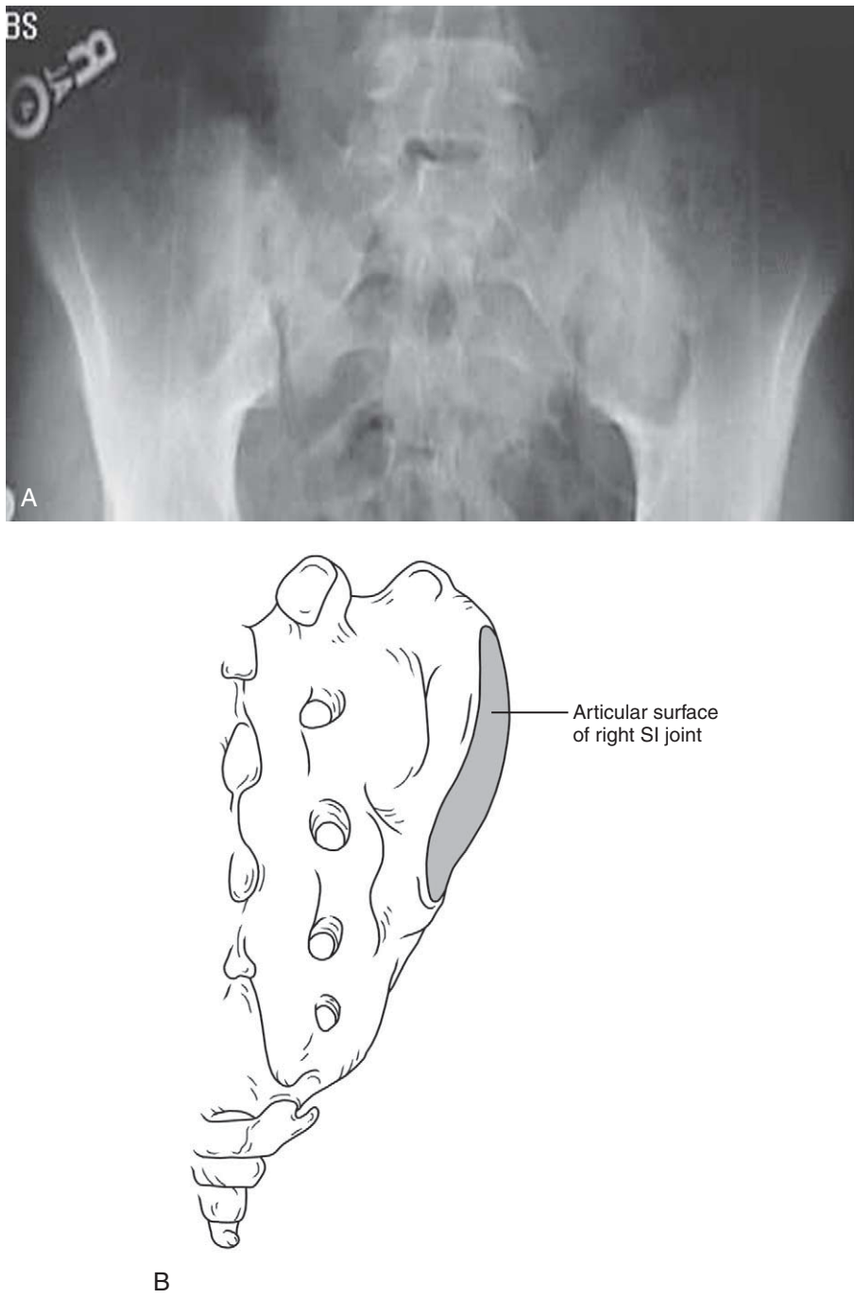


FIGURE 23-1

(A) Radiographic imaging of the sacroiliac joint (arrows) and relation with the sacrum and ilium. (B) Dorsal view of the sacrum on the right side. Shaded areas represent the sacral articular surface. Surface courses dorsal-medial to ventral-lateral at S1 and S2 levels. (Adapted from Sizer PS Jr, Phelps V, Thompsen K: Disorders of the sacroiliac joint. *Pain Pract* 2:17–34, 2002, figure 1, with permission.)

The sacrotuberous ligament connects the sacrum with the ischial tuberosity, as well as the coccyx via numerous tendinous slips. More specifically, the medial fibers converge on the cranial sacrum and posterior superior iliac spine (PSIS), whereas the lateral fibers converge on the caudal sacrum. The lateral fibers are therefore more influential in controlling nutation due to their greater distance from the axis of motion. The lowest fibers of the gluteus maximus and the tendinous portion of the long head of the biceps femoris blend in with this ligament.[18] The sacrotuberous ligament demonstrates a spiral fiber configuration along its course, which lends to the storage and release of elastic energy during the landing and propulsion phases of gait, respectively. This ligament restrains sacral nutation and indirectly stabilizes the pubic symphysis anteriorly by reducing motion between the two innominates posteriorly. This ligament is perforated by sensory branches of S2 and S3. These nerves might be entrapped at this site.

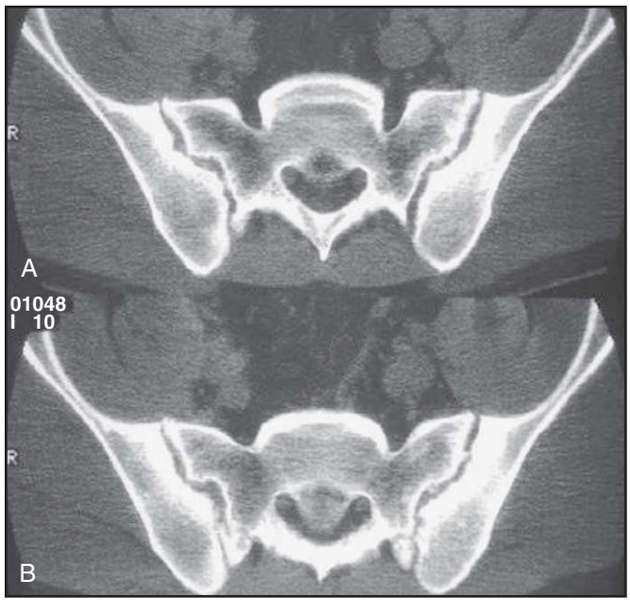


FIGURE 23-2

Transverse section at the sacroiliac joint. Note the torturous course of the joint from anterior to posterior direction. (A) The CT scan of the male pelvis showing thinner sacral cartilage. (B) The CT scan in the female pelvis showing thicker sacral cartilage compared with males.

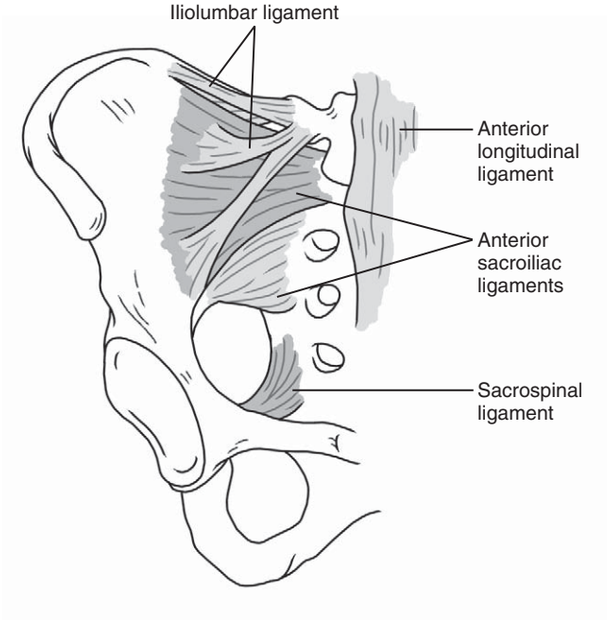


FIGURE 23-3

Line drawing of the anterior view of the sacrum and pelvis. Note the ligaments surrounding the anterior surface of the sacroiliac joint.

Deeper in the ligament system arises the posterior SI ligament network and the iliolumbar ligament. The posterior SI ligaments consist of both short and long branches. Irritation of a short branch can refer pain to an area from the posterior thigh to the knee, whereas irritation of the long branch can refer pain to the lateral calf and foot.

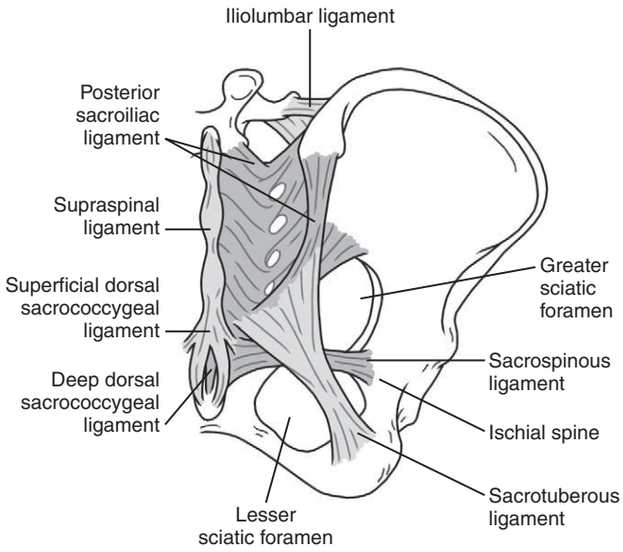


FIGURE 23-4

Line drawing of the posterior surface of the sacrum and pelvis. Note the ligamentous attachments surrounding the sacroiliac joint.

The iliolumbar ligament travels from the transverse process of L5 to the iliac tuberosity. Bilaterally, it prevents anterior shear of the fifth vertebrae. Unilaterally, it provides stability for L5 in the frontal plane via the posterior bands[18] and lateral stability via the anterior bands of the ligament.[19] The iliolumbar ligament is intimately connected with L5 such that movement of the ilium causes immediate motion in the L5-S1 segment and therefore contributes to the coupling behaviors at L5-S1.

The deepest layer of the ligament system consists of the interosseous ligament. This structure is 8–10 cm deep to the dorsal skin and is thus not palpable. This deep structure tightens with nutation, contributing considerably to the self-locking mechanism.[17,20] The anterior SI ligament is located on the anterior aspect of the SI joint. It is considerably weak and thin, averaging approximately 2 mm in thickness. It acts as a shock absorber for the capsule and allows increased SIJ motion with pregnancy.[21] It appears that the anterior SIJ ligament is subject to tearing during trauma and/or joint diseases.[22]

DEVELOPMENTAL CHANGES IN SACROILIAC JOINT

At Birth

Anatomical changes are seen in the SIJ throughout a person's lifetime.[23] At birth, the sacrum completes the pelvic ring along with the innominates to form a Roman arch.[24] At this period, the orientation of the SI joints is likened to that of quadrupeds, with the articular surfaces essentially parallel to each other and the lumbar zygapophyseal joint surfaces.[9] There is a considerable difference between the

cartilaginous surfaces of the ilium and sacrum. The sacrum has hyaline cartilage that is glossy, smooth, white, and about three times thicker than that of the ilium. Conversely, the ilium possesses a specialized form of hyaline cartilage that is dull, striped, bluish, and thin.[25]

Change at 1 Year

The sacrum enlarges laterally after approximately 1 year of age, and the articular surfaces fold in the transverse and sagittal planes to resemble a propeller.[9,17] Sacral widening enhances the sacral articulation with the ilium and offers a firm base of support for the trunk during upright standing and locomotion. Large interindividual differences continue to be observed throughout the first decade of life, accompanied by advancements in the sacral wedge shape and propeller form.

Change at Second Decade of Life

In the second decade, gender-related changes are seen.[13] During this period, the male synovial capsule thickens and an intra-articular sacral bony tubercle develops (seen in 88% of males, and only 15% of females). Additionally, males demonstrate an accentuation of the propeller shape, which leads to decreases in SIJ range of motion. Conversely, females demonstrate decreased SIJ mobility until the age of 14 years, followed by a gradual increase in the range of motion as hormones change and the soft tissue adapts.

Change at Third Decade of Life

The sacral vertebrae begin to ossify in the third decade, while disc material can remain present and pliable into older age groups. In addition, the mobility of the joint continues to increase in females, in contrast to a gradual decrease in males. The resulting ratio of mobility proceeds to approximately 5 to 1. Pregnancy can increase mobility of the SIJ 2.5 times.[13,26]

Change at Fourth Decade of Life

Females typically demonstrate persistent movement at the SIJ in the fourth decade of life, whereas the male SIJ demonstrates a decline in movement. Complementary ridges and depressions can be observed on the iliac and sacral cartilages, respectively. During this decade the sacral cartilage becomes yellow and dull, while both surfaces roughen. The synovial vascular supply tends to decrease while the synovial membrane itself thickens in both men and women. In this decade, there is also an increased potential for osteophytes, especially in males.[7,13,27]

BIOMECHANICS OF SACROILIAC JOINT

Most joints in the musculoskeletal system can be classified as "stable mobilizers" due to their functional priority of movement in the context of stability at the end of the available range of motion (e.g., the glenohumeral joint). Conversely, the SIJ can be classified as a "mobile stabilizer" due to its priority of stability in the context of limited available movement.[28] This stability is achieved through both the form- and force-closure observed in the joint mechanism.[24] Form-closure or architectural stability requires no additional force to maintain the equilibrium in the system[29] and is achieved through both macro structural and micro structural features. The propeller-shaped architecture provides a curved surface congruency that is oriented in numerous oblique planes (Figure 23-5). This macrostructural feature reduces translatory motions, lending to joint surface stability in the context of numerous force vectors.[30,31]

FUNCTION OF SACROILIAC JOINT

SHOCK ABSORPTION

The SIJ serves as a major shock absorber and force transducer that is implemented during weight-bearing activities. Along with the symphysis pubis, the sacrum serves as

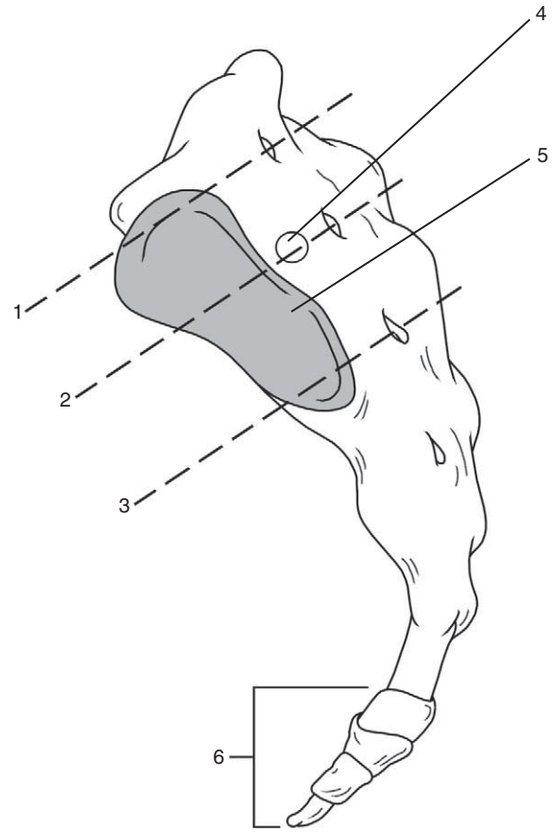


FIGURE 23-5

Lateral view of the sacrum (1) S1 level, (2) S2 level, (3) S3 level, (4) location of the axial joint, (5) sacral articular surface of the sacroiliac joint (note the inverted surface shown), (6) coccyx. (Adapted from Sizer PS Jr, Phelps V, Thompsen K: Disorders of the sacroiliac joint. *Pain Pract* 2:17–34, 2002, figure 6, with permission.)

a principal area of support for the pelvis.[32] In addition, the sacrum serves as a primary site for the storage of elastic energy during the landing sequence of gait. The delayed closure of the sacral epiphyseal plates lends the structure to elastic energy storage, which is released during each subsequent propulsion phase.

FORCE TRANSDUCTION: FROM LOWER EXTREMITIES

In order to achieve force transduction, the SIJ serves as a component of three different kinetic chains.[33–35] First, the lower extremities transfer forces into the sacrum through the innominates at the hip joints. Thus, ground reaction forces can be transmitted through the lower extremities, resulting in movements at the SIJs. Second, one may observe a true closed kinetic chain in the pelvic girdle, as forces are transferred through one innominate, into the sacrum and the other innominate through the two SIJs, and back into the original innominate through the pubic symphysis.

FORCE TRANSDUCTION: FROM HEAD AND TRUNK

Finally, forces from the head, neck, and trunk are transmitted from the trunk through the lumbar spine into the sacrum and innominates. Consequently, movement of the lumbar spine can have a direct effect on movement in the SIJ.[36] In this transduction, forces are transmitted from the lumbar spine into the sacrum through the L5-S1 intervertebral disc. These forces continue into the innominates through the SIJ on each side. However, it appears that forces can also be directly distributed from the lumbar spine into the innominates through the iliolumbar ligament. While this ligament was historically interpreted as a primary restraint to lumbar movement with respect to the sacrum, recent investigators have discovered that ligament transection can produce a significant increase in movement at the SIJ.[36] The greatest rotatory motion in the SIJ occurs in the sagittal plane[35,36] and can be labeled as nutation and counter-nutation. With nutation, the upper sacral segment (sacral base) rotates ventrally, the middle segment (in proximity of the axis) demonstrates very little movement, and the lower segments (sacral apex) rotate dorsally (Figure 23-6A). Thus, the pelvic inlet narrows and the outlet widens in the anterior-posterior direction. Nutation can be observed during trunk forward bending, where the sacrum tilts ventral and the ilia appear to move toward one another.[34] Nutation also occurs when individuals are positioned in standing and/or in a lordotic posture.[37]

On the contrary, counter-nutation produces a dorsal rotation of the sacral base and ventral rotation of the sacral apex (Figure 23-6B). This movement unwinds the ligaments involved in the self-locking mechanism, requiring the bony architecture to produce opposition and restraint to the movement. The pelvic inlet widens in the anteroposterior direction with this movement, while the outlet narrows. Counter-nutation can be observed at the SIJ during supine positioning[38] and the third trimester of pregnancy. This behavior lends greater mobility to the SIJ and a wider passageway for the fetus to descend in preparation for delivery.[24,25]

INNERVATION

The SIJ demonstrates a complex neural network. Portions of the sacral plexus from S1 and S2 innervate the posterior SIJ. Moreover, segments from L3 to S2 innervate the

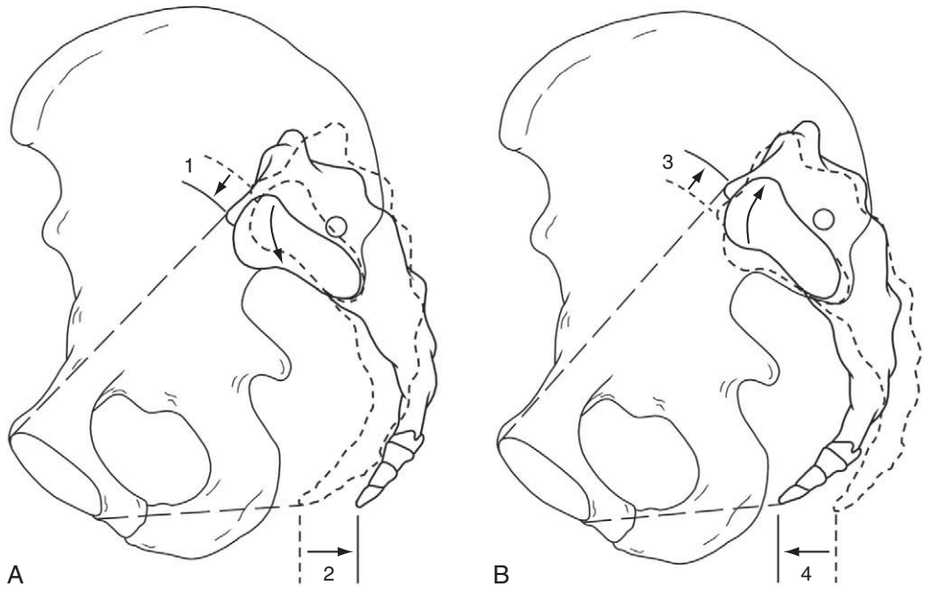


FIGURE 23-6

Sacral iliac movement in the sagittal plane: (A) nutation, (B) counter nutation, (1) Pelvic inlet closure with nutation. (2) Pelvic outlet opening with nutation. (3) Pelvic inlet opening with counter nutation. (4) Pelvic outlet closure with counter nutation. (Adapted from Sizer PS Jr, Phelps V, Thompsen K: Disorders of the sacroiliac joint. *Pain Pract* 2:17–34, 2002, figure 6, with permission.)

ventral side.[9,39] Thus, the nociceptors to the SIJ are derived from levels L2 to S4, contributing to pain in these dermatomes with SIJ problems.[40] This complicated neural network can give rise to the nondescript pain patterns and distinctive reference distributions.

CLINICAL EXAMINATION OF PATIENT WITH SACROILIAC JOINT PAIN

Painful sacroiliac conditions can be infectious, metabolic, inflammatory, neoplastic, degenerative, and traumatic in nature.[38,41] SIJ symptoms are acute and chronic. Acute symptoms can be bacterial or nonbacterial in nature. Bacteria (*Staphylococcus*, *Streptococcus*, *Enterococcus*, and *Pneumococcus*) can be communicated to the SIJ through the adjacent venous plexi associated with the bowel, resulting in severe sacroiliitis. Nonbacterial, acute sacroiliitis can be associated with ankylosing spondylitis, gout, rheumatoid arthritis, psoriasis, and trauma. Conversely, chronic sacroiliitis can be unilateral or bilateral in nature. Unilateral, chronic SIJ pain can be associated with ankylosing spondylitis, tuberculosis, psoriasis, Reiter's syndrome, and inflammatory bowel disorder. Bilateral, chronic, nontraumatic sacroiliitis is most frequently associated with ankylosing spondylitis and/or psoriasis. The first clinical manifestation of ankylosing spondylitis is commonly pain from the SIJ. Skin lesions can accompany psoriatic arthritis of the SIJ, and Rieter's will be accompanied by urethritis and episodic conjunctivitis. In the absence of a traumatic history, clinicians can turn to laboratory testing for HLA-B27, uric acid levels, ANA, and rheumatic factor for assistance in the diagnosis of these painful conditions.

The most common painful SIJ presentation seen in a clinic is a mechanical lesion associated with trauma, as well as adjacent lumbar arthropathy and/or fusion. Symptoms that result from a primary mechanical SIJ lesion are the consequence of either joint hypermobility or fixed subluxation (locking).

SIGNS AND SYMPTOMS

Patients frequently complain of painful catching and increased pain with lower extremity loading, such as unipodal standing or landing and propulsion with gait.[42] Pain may be induced by simple movements, such as the trunk shift with sneezing or turning in bed. Ascending and descending stairs may also be provocative, as well as landing after a jump or hop. Pain is not typically provoked with Valsalva. Paresthesias, numbness, or weakness is not commonly associated with painful mechanical SIJ states.

EVALUATION

The pain associated with an SIJ lesion can be elicited by selected manual provocation tests and diagnostic injections.

Manual Compression Test

For the dorsolateral pelvic compression test, the patient should be positioned supine with the knees flexed 15 degrees over a pillow, placing the SIJ in a maximum loose position. Then, the patient should place one forearm behind the lumbar spine to support the lordosis. This lumbar pre-position can reduce movement and subsequent symptoms from the lumbar segments during the test procedure. The clinician should cross his or her arms and then exert dorsolateral compression on the patient's anterior superior iliac spine (ASIS). To perform the axial femoral compression test, the patient is again positioned supine. The clinician stands on the patient's painful side, positions the patient's hip at 90 degrees flexion and the knee in relaxed flexion, reaches across with one hand to stabilize contralateral ASIS, secures the femur up against his or her body, and finally uses the other hand to exert an axial compression load through the femur. For each test, compression should be sustained for as long as 2 minutes in order to ensure gradual creep deformation and potential provocation.[42]

If pain is immediately produced with the manual provocation tests, then the clinician should suspect either clinical instability and/or an acute sacroiliitis that is associated with infection or an inflammatory disease. Conversely, if the provocation is produced only when the test compression is held for considerable time (1–2 minutes), then the clinician should suspect a primary mechanical condition, such as a locked subluxation.[38]

Once a positive provocation test is observed, the clinician can implement the forward flexion test (also termed "Vorlauf" phenomenon) in standing and sitting in order to further discern a clinical instability versus lock subluxation.[38] However, this test has been viewed as controversial in terms of validity and clinical utility. Dreyfuss et al.[14] observed asymmetries in the standing flexion and sitting flexion tests in 13% and 8% of normal asymptomatic individuals, respectively.

For the test, the physician palpates the caudal aspect of both PSISs with the thumbs while the patient stands upright. The patient flexes forward, and the examiner observes for changes in the relative position of each PSIS. Normally, the PSISs should migrate cranially in an equal fashion on both sides as the sacrum nutates, the ligaments tension load, and the self-locking mechanism engages. The position of the PSISs should be evaluated only at end range of forward flexion. Matthijs suggested that patients stand with a separate weight scale under each foot so that weight distribution can be monitored and symmetry can be ensured throughout the test. While more-cranial PSIS indicates a positive "Vorlauf" phenomenon,[40] the clinical interpretation of this finding depends on its correspondence with the location of pain.[41] For a locked subluxation, the positive "Vorlauf" occurs on the painful side as a hypo mobile state induces the joint to reach end range early and the ilium is moved cranial with the nutating sacrum. Conversely, the positive

"Vorlauf" occurs on the nonpainful side during a painful hypermobile state (clinical instability), as the ligaments tension load and the self-locking mechanism engages earlier on the nonpainful side.

Interpretation of the Evaluation

Realistically, the examination and diagnosis of SIJ painful conditions can be challenging. This challenge is due to the complexity of the nerve supply, as well as the paucity of sensitive and specific tests that can be utilized to identify painful SIJ states.[41] While SIJ pain may arise in isolation or in concert with problems in the lumbar region, the physician must discriminate a patient's SIJ symptoms from other pain states that refer pain to similar areas.[43] Maigne et al.[44] suggested that a so-called "sacroiliac syndrome" presents with no specific, distinctive clinical features. Yet, while the topography of the pain remains the best criterion, symptoms associated with the SIJ are typically vague and diffuse in the hip and pelvic regions.

Popularized testing of the SIJ has included many approaches, including palpation for tenderness, hyperirritability, and tissue texture changes; assessment of myofascial and musculotendinous restrictions; regional analysis of muscle imbalance and leg length discrepancy; postural analysis and evaluation for asymmetries; joint motion tests; and provocation maneuvers.[44] Reliability and validity of clinical SIJ testing are questionable,[43] and clinicians may need to re-evaluate the use of many of these tests. Tests that evaluate SIJ movement upon manual sacral springing are most likely evaluating lumbar mobility or sacral osseous deformation, due to incomplete fusion of sacral vertebral growth plates and persistent interposing disc material. One can observe intraindividual anatomical differences in joint surface shape and orientation between the two SIJs in both the parasagittal and transverse planes.[41,45] When comparing left with right, these anatomical differences can produce motions that can be confused as clinical hypomobility versus hypermobility, further distorting the diagnosis. For example, Potter and Rothstein[46] observed less than 50% agreement between two experienced clinicians when performing the majority of position and mobility tests at the SIJ. Moreover, Dreyfuss et al.[47] concluded that the Gillet test, Patrick's test, Gaenslen's test, and midline sacral thrust demonstrated little diagnostic value when compared with diagnostic SIJ infiltration outcomes.

Investigators have observed more promising results when using tests that modify symptoms, including injections and manual pain provocation tests. Diagnostic intra-articular injections have been commonly implemented and numerous investigators have endorsed these diagnostic block procedures. Calvillo et al.[43] suggested that the most reliable method to establish the diagnosis of SIJ arthralgia is a fluoroscopically guided intra-articular injection preceded by an SIJ arthrogram. Schwarzer et al.[22] also advocated this procedure and recommended a double-injection technique to reduce the incidence of false-positive responders. Maigne et al.[44] incorporated a double intra-articular injection procedure using lidocaine and bupivacaine on separate occasions.

Manual provocation tests have been recommended as screening tools, especially when diagnostic blocks are not readily available.

MANAGEMENT

Management strategies for SIJ pain states are diagnosis specific. Pain that arises from systemic disease merits pharmacological interventions directed at reducing inflammation and curbing the pathological processes, while pain that arises from infection merits antibiotic therapy. Patients who suffer from symptoms that are related to a primary mechanical SIJ pain state can benefit from measures that are intended to normalize the mobility status of the joint.

DIAGNOSTIC SACROILIAC JOINT INJECTIONS

An SIJ injection can serve to diagnose the source of pain, as well as treat the pain. Injecting the SIJ with a local anesthetic, such as lidocaine, can help a clinician determine whether the source of the pain originates in the joint or from outside the joint. Bupivacaine is another anesthetic used to numb the joint. Bupivacaine is slower to take effect than lidocaine, but it also takes more time to wear off, which means it provides longer-lasting pain relief for the patient (Figure 23-7).

If the joint is injected and the pain does not go away, the source of the problem is probably somewhere other than the joint. If the pain immediately ceases, cortisone may be added before the needle is removed in order to reduce inflammation, which may be causing the pain. Because cortisone is long lasting and can be slow releasing, it tends to provide effective pain relief. Although it may take several days to reduce the inflammation, the pain-relieving effects of injecting cortisone can last for weeks or even months. In some cases, it may be beneficial to add morphine or fentanyl to the cortisone for greater pain relief. However, this is usually reserved for very serious cases.

The injection requires the use of imaging, such as fluoroscopic guidance or a computerized axial tomography scan, so that the clinician can be sure the needle is placed correctly in the joint.

CONTRAINDICATIONS

- Local infection
- Sepsis

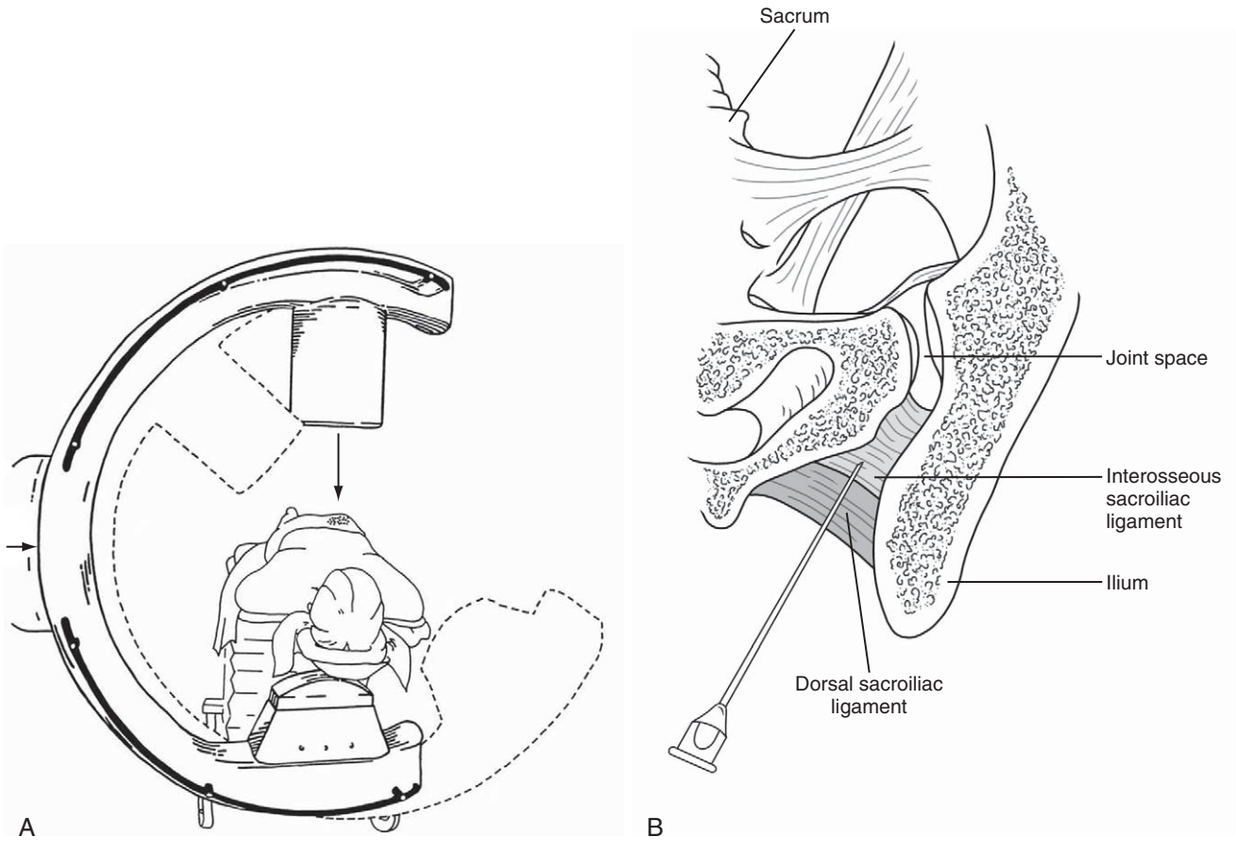


FIGURE 23-7

(A) The patient lies prone. The C-arm is started with the posteroanterior view and rotated toward the oblique view until a clear view of the sacroiliac joint is obtained. (B) A cross-section of the sacroiliac joint showing the entry of the needle at the dorsal inferior aspect of the joint. (C) Sacroiliac joint enhanced fluoroscopically by oblique positioning of the C-arm. Note the needle entry at the dorsal inferior surface of the joint.

- Coagulopathy
- Allergy to potential drugs being used

EQUIPMENT

Local nerve block
25-gauge, 3/4-inch needle
22-gauge, 1-1/2-inch needle
23–25-gauge, 3-inch needle
3-ml syringe
Intravenous T-piece extension
Radiofrequency thermocoagulation
16-gauge, 1-1/4-inch angiocatheter
20-gauge, curved Racz-Finch radiofrequency thermocoagulation needle, SMK 100-mm (active tip 0.5 cm), or SMK 145-mm (active tip 0.5–1.0 cm)

DRUGS

- 1.5% lidocaine for skin infiltration
- 0.5% bupivacaine/ropivacaine
- 2% lidocaine
- Steroids (optional)
- Hyaluronidase sodium (Hyalgan) (optimal)

TECHNIQUE

PREPARATION OF PATIENT

In order to evaluate the patient's general health, a complete patient history should be taken that includes determining whether a pre-existing disease or injury is present. Some symptoms, such as bladder or bowel dysfunction or numbness, may suggest an emergency that requires immediate care. The patient's pain history should include how long the problem has been present and the various treatments the patient received, including any medications, injections, modalities, bracing, or manipulations. The treatment outcomes should also be noted. Provocative and palliative positions or activity can be used to help guide the course of future treatments. Function loss is significant because it can be an indication of suffering and a measure of treatment success as the patient begins to resume activities.

DIAGNOSTIC PROCEDURE

Fluoroscopy Guidance

Due to the anatomical complexity of the SIJ, the procedure is difficult. Correct positioning of the patient is critical. While the patient lies prone, the C-arm is started in the posteroanterior view and rotated toward the oblique view until a clear view of the SIJ is obtained. Initially, it is helpful to find the L5-S1 disc space at L5-S1. The cephalic is rotated 15–25 degrees in order to open the disc space at L5-S1. It is important to start with the oblique view and rotate toward the anteroposterior view to visualize the widest space at the most inferior aspect of the S1 joint.

The scout image must show the entire S1 joint visualized for needle entry at the most inferior aspect. The C-arm is angled in such a way that the lines of the posterior and the anterior aspects of the joint are seen to overlap. Injection of the contrast material spreads throughout the SIJ in an inferior to superior fashion. It is important to note that the entry should be made in the posterior part of the joint rather than the anterior part of the joint; otherwise, the result may be a failed procedure.

Five percent to 0.5% bupivacaine/ropivacaine with or without 40 mg of triamcinolone is injected for the diagnostic and therapeutic block. The patient should be monitored for at least 30 minutes prior to discharge. If the local anesthetic block is successful, 2–5 ml of Hyalgan can be injected, twice weekly, up to five times. After the radiofrequency needle is inserted in the S1 joint, sensory and motor testing is done.

Computed Tomography Guidance

Because of the difficulty one can encounter entering the joint under fluoroscopy guidance, the injection can be performed under computed tomography (CT) guidance.

PROCEDURE

The patient should be placed prone on the CT gantry, and the appropriate SIJ identified by CT. The area is prepped and draped, and local anesthetic is injected into the skin. A 22-gauge, 3-1/2-inch spinal needle is placed into the joint under intermittent CT guidance (Figure 23-8A). Omnipaque-180 is injected to outline the joint at both anterior and posterior borders (Figure 23-8B). Bupivacaine with or without triamcinolone is injected. The inferior third of the joint can be very narrow, which usually prevents needle entry under fluoroscopy.[48]

Ten minutes after the procedure, the patient should be evaluated for pain relief.

Therapeutic Procedure: Technique of Dussault et al.

With informed consent, the patient is positioned prone on the C-arm fluoroscopic table. With the x-ray tube perpendicular to the table, the skin is marked over the distal 1 cm of the SIJ (Figure 23-9 A). The tube is then angled about 20–25 degrees in a cephalic direction to displace the posteroinferior portion of the SI joint in a caudal direction (Figure 23-9B). Using a sterile technique, the skin should be anesthetized at the previously marked site. A 22-gauge, 3- or 5-inch (depending on patient size) straight or 10-degree curved-tip spinal needle is then advanced perpendicular to the fluoroscopic table. With the tube in the cephalic position, the needle is directed toward the posterior SIJ, without angling the needle in either a cephalic or caudal direction (Figure 23-9C). The 10-degree curved-tip needle is made by hand, with the needle bevel centered on the convex or outer part of the curve (Figure 23-9D). The tip of the curved-tip needle is oriented in a cephalic direction, and the convex portion of the curve is oriented downward (closest to the joint). The curved-tip needle may be advanced either vertically or angled 10 degrees downward to initially compensate for the 10-degree curve until the needle reaches the joint. As the needle contacts firm tissues on the posterior aspect of the joint, it should be maneuvered through the ligaments and capsule into the joint by advancing it about 5–10 mm, usually by angling the needle tip slightly laterally to follow the natural curve of the joint. Intra-articular position is confirmed by injecting 0.2–0.5 ml of contrast material (Omnipaque [300 mg iodine/ml], iohexol; Nycomed, Princeton, NJ) through the needle. After the contrast material outlines the joint, 6 mg (1 ml) of betamethasone sodium phosphate and betamethasone acetate solution and 1 ml of 0.5% bupivacaine hydrochloride should be injected. Dussault et al.[49] took note of each patient's pain level just prior to the procedure with use of a numeric grid line graded in centimeters from 0 (no pain) to 10 (the maximum tolerable pain). Approximately 10 minutes after the injection, the pain level was recorded again. The fluoroscopy technique has also been used with injection of 6% phenol for persistent sacroiliitis by Ward et al.[50]

Radiofrequency Technique as Described by Buijs and Colleagues

RADIOFREQUENCY SACROILIAC JOINT DENERVATION TECHNIQUE

While in a prone position, the patient's sacrum is positioned horizontally. With a C-arm fluoroscope (Philips BV 25, Philips Eindhoven, The Netherlands), the S1 dorsal foramen is visualized in "tunnel vision." After sterilization of the skin, a Sluijter-Metha 22-gauge, 10-cm needle with a 5-mm tip is placed under bone contact in the lateral upper quadrant of the S1 dorsal foramen. The needle must be placed alongside the nerve in the foramen in order to increase the efficacy of the radiofrequency denervation (Figure 23-10). Next, a lateral view is taken to ascertain that the needle is not too close to the ventral foramen. The procedure is repeated at the S2 and S3 foramen. Caudal movement of the fluoroscope is needed to show the foramen of S2 and S3 in the correct way. The needles form a straight line, parallel to

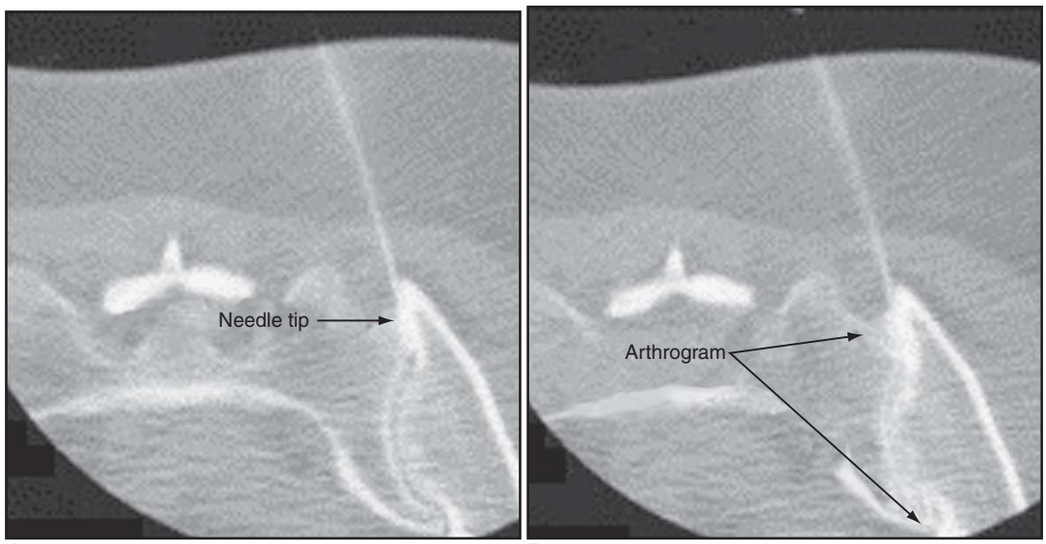


FIGURE 23–8

Computed tomography (CT) images from sacroiliac joint injection. (A) Needle (22 gauge) directly inserted into the sacroiliac joint. (B) CT image after injection of 1 ml Omnipaque 300. (From Block BM, Hobelmann JG, Murphy KJ, Grabow TS: An imaging review of sacroiliac joint injection under computed tomography guidance. *Reg Anesth Pain Med* 30:295–298, 2005, figure 2B-C, with permission.)

the position of the dorsal sacral foramina. Subsequently, electrostimulation is used to verify the correct position of the needles. The needle stylette is replaced with a thermistor electrode. A 50-Hz current with a pulse duration of 2 milliseconds is used. The voltage is increased until the patient recognizes paresthesia or a sensation of pressure in the painful region. Stimulation values up to 0.5 V are acceptable. If there is no effect at 0.5 V, the needle is not correctly placed and should be repositioned. The absence of motor involvement is verified by stimulation with a 2-Hz current (2-millisecond pulse duration) up to 2 V. During 2-Hz stimulation, no muscle contractions should be palpated at the S1, S2, and S3 innervated muscles, nor recognized by the patient in the urogenital or anal region. If these criteria are fulfilled, the needles are in the correct position and the thermistor electrode is removed, followed by injection of a small amount of local anesthetic through each needle. Then the thermistor electrode is reinserted. After 2 minutes, a neurotomy is performed with the tip temperature at 80°C for 60 seconds.[51]

Pulse Radiofrequency Neurotomy

With the early reports of deafferentation pain syndromes and motor deficit with the application of thermal radiofrequency lesion, pulse radiofrequency represents the most recent advance in clinical practice. The initial clinical data on pulse radiofrequency neurotomy demonstrate a response rate similar to conventionalthermal radiofrequency lesions for sacroiliac arthropathy.[52]

COMPLICATIONS OF RADIOFREQUENCY

Following SIJ radiofrequency denervation, some patients can have gluteal discomfort, hip pain, or referred posterior thigh pain that usually resolves in 10–15 days. It is advisable to provide adjunct analgesic oral therapy. Patch hypoesthesia in the buttocks can be referred that resolves spontaneously within 2 to 4 weeks.

CLINICAL PEARLS

The facets and root ganglia should be lesioned for the complete treatment to be performed. It is recommended to perform pulsed radiofrequency lesions in the dorsal root ganglions of S1, S2, and S3, and conventional radiofrequency in L4-L5 and L5-S1 medical branches. The S2 segmental root largely contributes to the innervation of the SIJ. S2 dorsal root ganglion–pulsed radiofrequency can alleviate residual symptoms after sacroiliac denervation.

EFFICACY

Diagnostic and Therapeutic Injections

In the literature there are some data on the efficacy of SIJ injections and RF procedures. In the fluoroscopic-guided technique of SIJ injection by Dussault et al.,[49] all but one of the SIJs were successfully injected intra-articularly, as confirmed by means of injection of contrast material and fluoroscopic spot imaging. The mean fluoroscopic time for the procedure was 108 seconds (range, 36–328 seconds), and

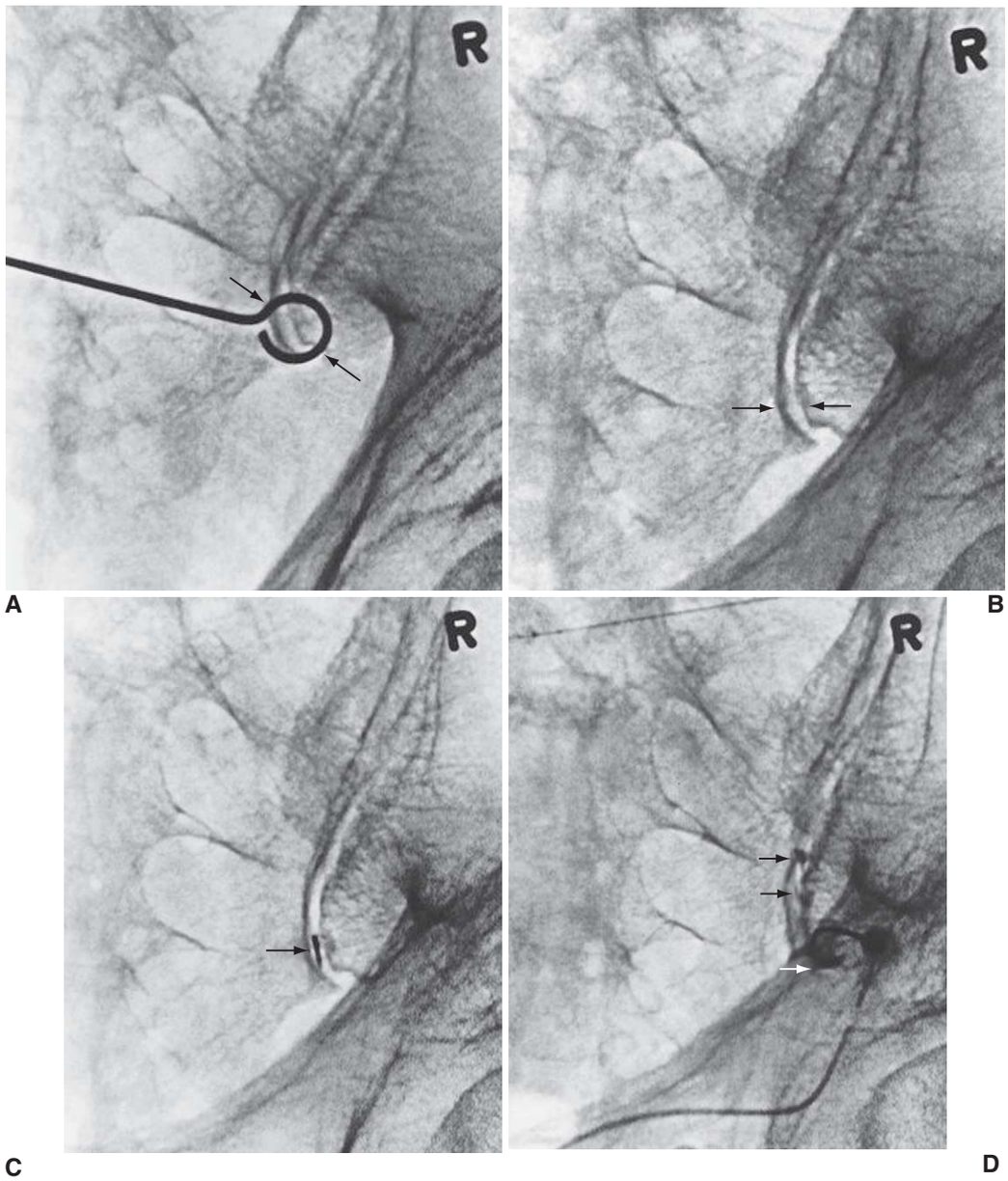


FIGURE 23–9

Posteroanterior fluoroscopic radiographs depict technique for sacroiliac (SI) joint injection, with patient prone. (A) With the x-ray tube perpendicular to the fluoroscopic table, a localization probe (arrows) is centered over the distal 1 cm of the right SI joint, and the skin is marked. (B) With the x-ray tube angled 20 degrees cephalad, the posterior aspect of the inferior SI joint (arrows) is clearly depicted caudally. (C) Straight needle (arrow) is advanced perpendicular to the fluoroscopic table into the posterior portion of the SI joint. (D) The x-ray tube is angled 25 degrees cephalad, and the SI joint is opacified with contrast material (arrows). R, right. (From Dussault RG, Kaplan PA, Anderson MW: Fluoroscopy-guided sacroiliac joint injections. *Radiology* 214:273–277, 2000, with permission.)

70% (22 of 31) were performed in less than 2 minutes. Data for pain relief were available for 28 of the 31 injections. Two patients had no pain at the time of the procedure; therefore, an immediate change could not be evaluated in them. In a third patient, the pain level after the procedure was not available. After injection, pain decreased by 80% or more in 7 of the 28 joints (27%); by 50–70% in 11 joints (39%), including the patient with bilateral sacroiliitis; and by less than 50% in 10 joints (36%). Pain relief of 50% or more after intra-articular injection of local anesthetic was obtained in 55% (10 of 18) of the joints with normal conventional radiographs, in 62% (5 of 8) of the joints with degenerative joint disease, and in the one patient with bilateral sacroiliitis as a result of ankylosing spondylitis. Thus, pain decreased 50% or more in 64%(18 of 28) of the joints after intra-articular injection of local anesthetic.

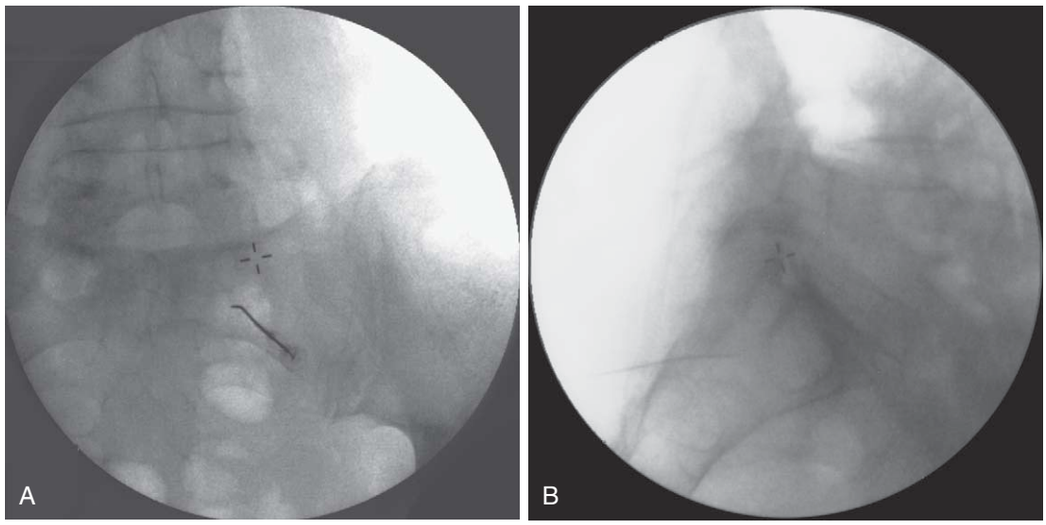


FIGURE 23–10

AP view of the sacral foramin with the needle in place for radiofrequency of S1 nerve root. (B) Lateral view of the needle entering from posterior aspect in the foramen on the nerve root.

Advantage of Computerized Tomography Guidance

Needle placement is easier and less painful when performed under CT guidance. The patient can have a large overhanging iliac bone that usually obscures the entry into the SIJ. This may be the reason for the difficulty entering the joint under fluoroscopy. Tomographic marker tape can be used to facilitate a CT-guided needle-placement procedure (Figure 23-11A). The tape's radiopaque marks are visible in the scan and, thus, mark skin entry points. On the CT image, one can see that the approach needed to enter the joint is more oblique (medial to lateral) than is apparent on fluoroscopy. By use of CT guidance and tomographic marker tape, one can easily place the needle into the joint (Figure 23-11B).

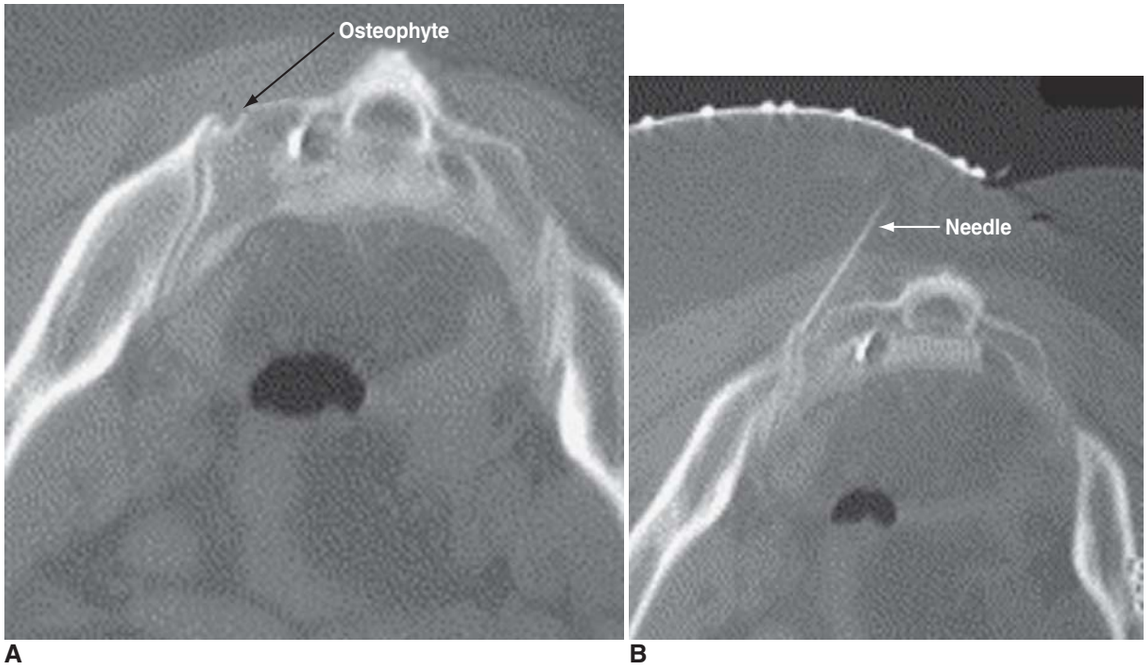


FIGURE 23–11

(A) CT image of left sacroiliac joint (SIJ) with osteophyte preventing usual needle placement. (B) Spinal needle (22 gauge) inserted into SIJ. (From Block BM, Hobelmann JG, Murphy KJ, Grabow TS: An imaging review of sacroiliac joint injection under computed tomography guidance. *Reg Anesth Pain Med* 30:295–298, 2005, figure 3B-C, with permission.)

Use of CT guidance for injections has potential advantages. CT-guided blocks can potentially enhance the performance of non–CT-guided procedures. The ability to use CT guidance gives pain medicine specialists another tool. Recent advances in CT technology allow dynamic CT evaluation (live-CT), which can be used like fluoroscopy. Live CT can image the patient during injection to control the precise placement of the injectate and, thus, improve patient safety.

In a study conducted in 2001 by Slipman and colleagues,[53] nearly two out of three severely impaired patients obtained significant relief from their back and leg pain following a therapeutic SIJ injection. Long-lasting relief has been reported by Calvillo and associates[54] by using viscus hyaluronate. Ward and colleagues[50] reported on the efficacy of fluoroscopy-guided SIJ injections with phenol in 10 patients with known sacroiliitis. Initially the patients were injected with 0.5% bupivacaine and 80 mg of Depo-Medrol. All the patients experienced pain relief for 2–4 weeks after the injections. Then they had fluoroscopy-guided SIJ injections with 6% phenol. The authors reported that 20% of the patients had a greater than 70% improvement with an average duration of 24 weeks. Sixty percent of the patients had a 50–70% improvement with an average duration of 20 weeks. Ten percent had a 20–50% improvement that lasted 12.5 weeks, and the remaining 10 percent had less than 20% improvement. The authors, therefore, concluded that substantial and prolonged pain relief is possible by providing injections of phenol for the ablation of the SIJ.

Radiofrequency of Sacroiliac Joint

In the study by Buijs et al.,[51] the following results were reported: after 12 weeks, 15 of the 43 procedures resulted in complete pain relief, whereas after another 14 procedures patients reported pain relief of 50% or more. Specifically, after the first round of procedures, 10 patients reported pain relief of 50% or more and 13 patients reported the same level of pain relief after the second round of procedures. One patient, who was treated on both sides, had complete pain relief on one side and 50% pain relief on the other side. Therefore, in total, 24 patients claimed a 50% or greater decrease in pain. One patient complained of increased pain. There were no other complications.

In the study by Yin and colleagues[55] on sensory stimulation–guided SIJ radiofrequency neurotomy, it was concluded that the stimulation-guided approach toward the identification and subsequent radiofrequency and thermocoagulation appeared to offer significant therapeutic advantages over existing therapies. In a total of 14 patients, 64% experienced a successful outcome, with 36% experiencing complete relief. No patient experienced a worsening of pain.

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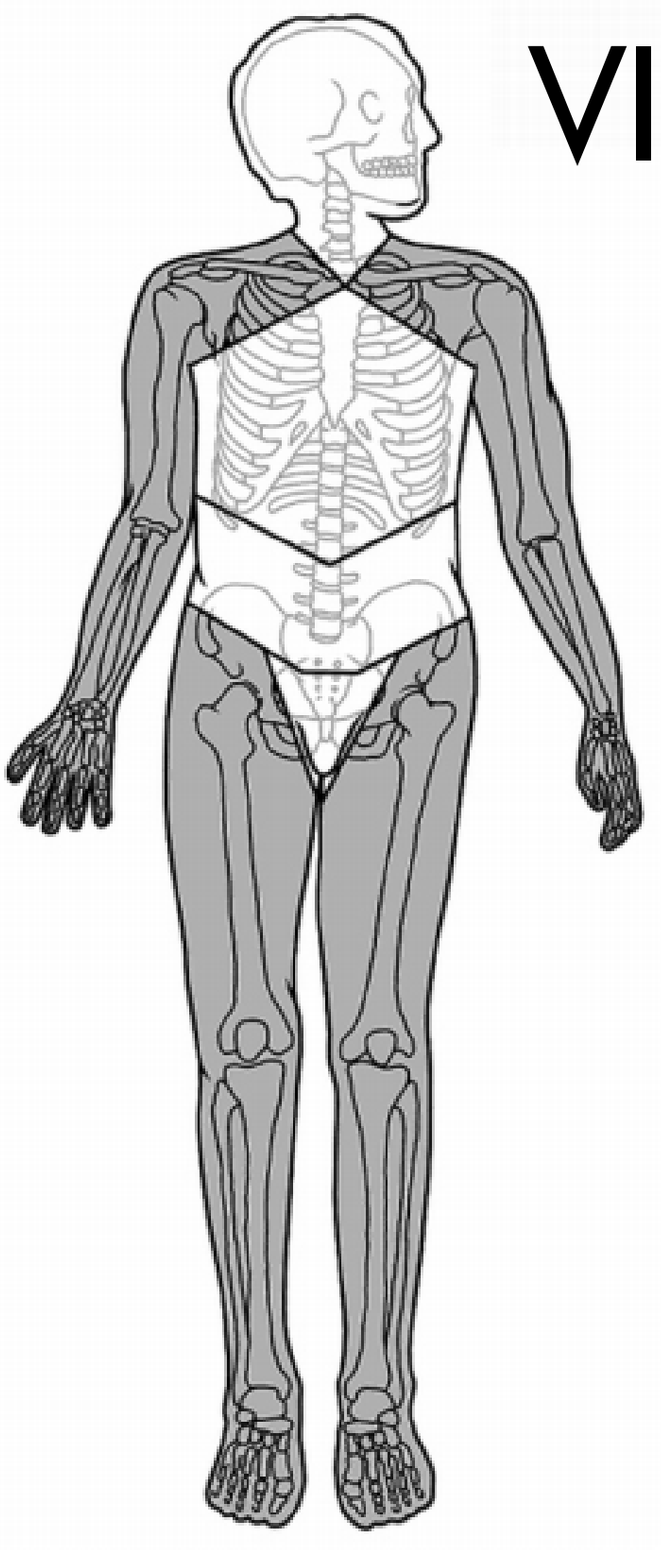
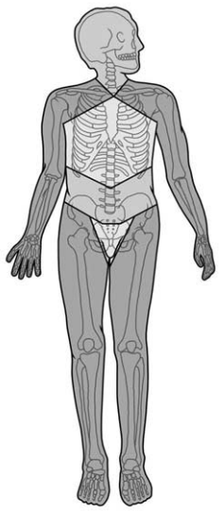
SECTION

VI



EXTREMITIES

C H A P T E R



24 Somatic Blocks of the Upper Extremity

LEE ANN PARADISE, JASON ROBERT FRYER, AND P. PRITHVI RAJ

HISTORY

Halsted performed the first brachial plexus block in 1885.[1–3] He did so by surgically exposing the roots of the brachial plexus and individually injecting them with cocaine. Crile developed the intraneural brachial plexus block, also known as the "Crile's technique," in 1897. Originally used in the therapeutic treatment of a 12-year-old boy, Crile eventually used the technique to provide anesthesia of the upper extremities.[1] As the techniques required the surgical exposure of the plexus, they were not widely used and focus shifted to the development of percutaneous measures. Four key approaches to the brachial plexus block have been developed and modified since Halsted's initial discovery: the axillary, the infraclavicular, the interscalene, and the supraclavicular. Each approach is discussed in detail below.

AXILLARY BLOCK

The first percutaneous brachial plexus block was reported by Hirschel in 1911.[1,3] Unfortunately, despite Hirschel's personal success with the procedure, others found it difficult to use and criticized its efficacy.[1] In 1917, Capelle described what would become the precursor of the axillary perivascular technique. Subsequently, Reding (1921), Labat (1922), Pitkin (1927), Accardo and Adriani (1949), Burnham (1958), Hudson and Jacques (1959), Eriksson (1962), and de Jong (1965) would modify the technique until it would become one of the most commonly practiced blocks used by anesthesiologists today.[1,3,4]

SUPRACLAVICULAR BLOCK

Only months after Hirschel performed his axillary technique in 1911, Kulenkampff described the first percutaneous supraclavicular.[1,3] The popularity of Kulenkampff's technique continued for over a decade, despite the serious complications associated with its administration, such as a high incidence of pneumothorax.[1,2] Labat made the first significant change to the technique in 1922, by advocating injections of the local anesthetic agent at three separate points.[3] In 1929, Livingston described what is now known as the subclavian perivascular approach. In 1940, Patrick departed from these techniques and described an entirely new way of laying down a wall of anesthetic through which the plexus passed. This "standard" or "classical" technique of supraclavicular brachial plexus block underwent further development over the years.[1–3]

INFRACLAVICULAR BLOCK

In 1917, Bazy offered an alternative to the techniques used by Hirschel and Kulenkampff. By inserting the needle below the clavicle, medial to coracoid process, and advancing toward the Chassaignac, Bazy's technique blocked all the nerves derived from the plexus while minimizing the risk of pleural injury.[1,3,5] Minor modifications to the technique were proposed by Babitzki (1918) and Balog (1924) as alternatives to the Kulenkampff technique when it could not be used due to the presence of deformities or injuries in the subclavian area.[1,5] A modified infraclavicular approach was reintroduced in 1973 by Raj, in which the needle was inserted more medially and directed laterally from the point of entry, thus greatly reducing the risk of pneumothorax.[6] Further modifications were proposed by Sims (1977) and Whiffler (1981) to overcome some of the disadvantages of the Raj technique.[1] Research has shown that use of a peripheral nerve stimulator helps provide consistently positive results with the infraclavicular approach.[3]

INTERSCALENE BLOCK

In 1912, Kappis described the use of "paravertebral conduction anesthesia" of the brachial plexus, injecting just lateral of the spinal column through a single skin wheal.

Santoni modified this technique in 1916, using injections into separate skin wheals. Both of these posterior paravertebral approaches, however, were extremely painful for the patient, which greatly diminished their popularity.[1] In 1919, Mulley performed the first interscalene approach, although credit went to Etienne when he described the first true interscalene approach in 1925. Modifications were made to both the anterior and posterior approaches for the interscalene brachial plexus block over the years, including Winnie (1970) and Pippa (1990).[1,7] Winnie is typically credited with the current popularity of the interscalene approach.[1]

ANATOMY

The brachial plexus consists of a plexus running from the spine (C5 to T1, with minor contributions from C4 and T2), through the neck, the axilla, and into the arm (Figures 24-1 and 24-2). With the exception of the intercostobrachialis nerve, all nerves in the upper extremity stem from the brachial plexus. From the proximal to the distal part of the plexus, it is divided as described in the following sections.[2,8]

ROOTS, TRUNKS, AND DIVISIONS

The nerve roots enter the interscalene groove between the scalenous anterior and scalenous medius muscles. The C5 and C6 nerve roots form the upper trunk, the C7 continues as the middle trunk, and the nerve roots of C8 to T1 unite to form the lower trunk at the lateral border of the scalenus anterior muscle. The trunks are sheathed by the prevertebral fascia and lie in the same plane as the subclavian artery. The upper and middle trunks lie above the subclavian artery, while the lower trunk lies posterior to the subclavian artery, near the first rib. Each trunk divides into anterior and posterior divisions.

CORDS

The lateral, medial, and posterior cords derive the name from their relation to the second part of the axillary artery behind the pectoralis minor muscle. The lateral cord is formed by the anterior divisions of the upper and middle trunks. The medial cord is formed by the anterior division of the lower trunk. The posterior cord is formed by the posterior divisions of all the trunks.

BRANCHES

Several branches arise from the brachial plexus and can be divided into those derived from the roots, the trunks, and the cords. From the roots, C5 contributes to the phrenic nerve and branches to the levator scapulae muscle, C5 to C7 branch to the serratus anterior, and C8 to T1 branch to the rhomboids and levator scapulae. From the trunks, C5 and C6 send a nerve to the subclavius, and

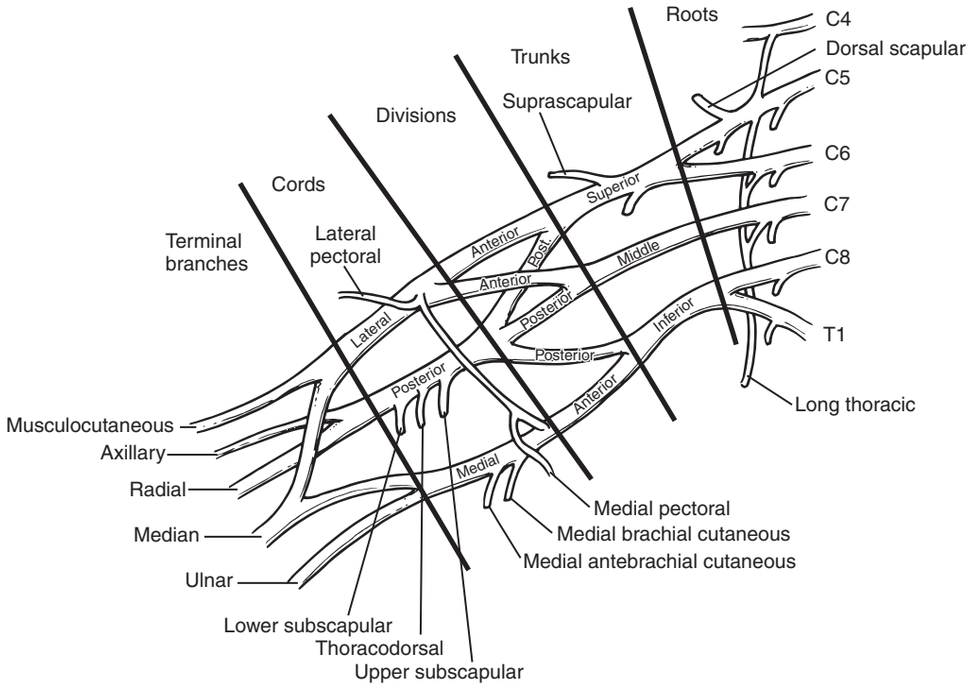


FIGURE 24–1

Anatomy of the brachial plexus. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, p. 343, with permission.)

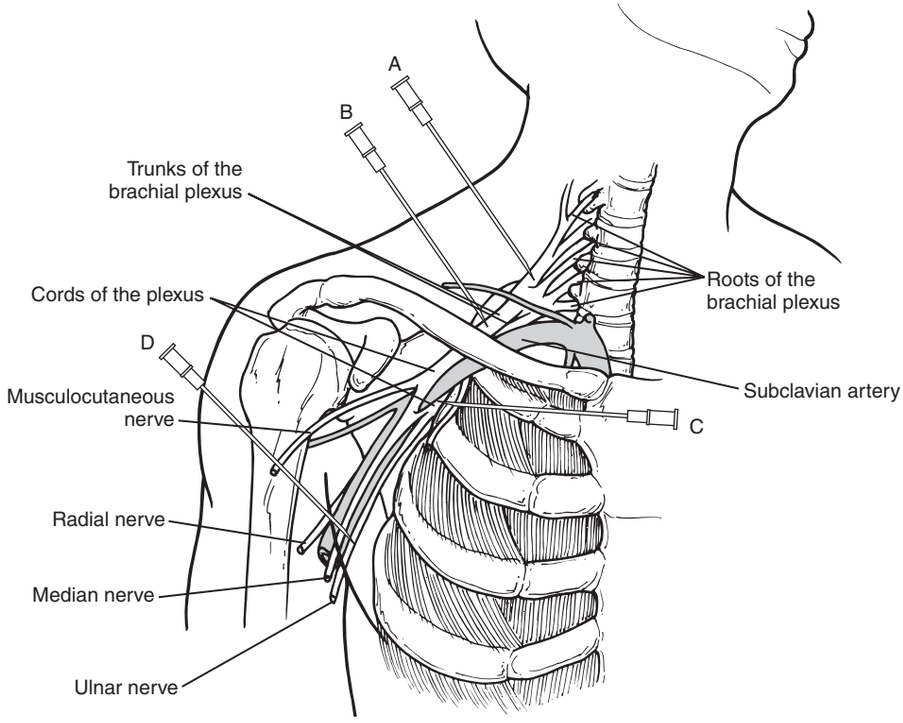


FIGURE 24–2

Anatomy of the brachial plexus, showing the axillary, infraclavicular, interscalene, and supraclavicular approaches. A. Interscalene approach. B. Supraclavicular approach. C. Infraclavicular approach. D. Axillary approach (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, p. 343, with permission.)

the suprascapular nerve contributes to the supraspinatus and infraspinatus. The lateral cord branches to the lateral pectoral nerve, the musculocutaneous nerve, and the lateral head of the median nerve. The medial cord branches to the medial pectoral nerve, the medial cutaneous nerve of the arm and forearm, the medial heads of the median, and ulnar nerves. Finally, the posterior cord divides into the upper and lower subscapular nerves, the nerve to the latissimus dorsi, the axillary nerve to the shoulder joint, which innervates the deltoid and teres minor, and the radial nerve.

Except for the innervation of the skin over the upper part of the shoulder (C3, C4) and the upper part of the medial arm (T2), all motor and sensory innervation to the upper extremity is derived from the brachial plexus. Sympathetic innervation stems from the T1 to T5 spinal segments. The T1 and T2 postganglionic fibers transverse the brachial plexus via the stellate ganglion, and the T3 to T5 postganglionic fibers join the vascular branches of the subclavian artery leading to the arm.

INDICATIONS AND CONTRAINDICATIONS

AXILLARY BLOCK

The axillary brachial plexus block is simple to perform, has few complications, and has a good cost-to-benefit ratio. As such, it is the most popular brachial plexus block in use for all surgeries of the elbow, forearm, and hand.[4,8–10] It is indicated for operations in the arm, continuous analgesia, pain syndrome, physiotherapy, and sympatholysis.[8]

Despite its general safety, the axillary brachial plexus block is contraindicated in the presence of systemic infection, pre-existing neuropathies, and coagulopathy. Strong contraindications include unfamiliarity with the procedure, patient refusal, infection at site, and allergies to the agents being used.[9]

SUPRACLAVICULAR BLOCK

The supraclavicular brachial plexus block, also known as the intersternocleidomastoid block, is indicated in the diagnosis and treatment of chronic shoulder pain conditions, as well as providing anesthesia for surgery of the shoulder, arm, and forearm. The block can also provide temporary relief from muscle spasm or strain in the supraspinatus and infraspinatus muscles.[10,11] The continuous supraclavicular brachial plexus block is indicated for treating synovectomy of the shoulder and hand found in patients with rheumatoid arthritis.[11]

The supraclavicular brachial plexus block is strongly contraindicated in the case of patient refusal, infection at site, allergies to the agents being used, coagulopathy, infection at site, moderate to severe respiratory disease, and ipsilateral apical bullae. Other contraindications include pre-existing neuropathy, particular stature (short neck, stiff neck, etc.), prior neck surgery or radiation, associated disease, and contralateral, recurrent laryngeal nerve palsy.[9,11]

INFRACLAVICULAR BLOCK

The infraclavicular brachial plexus block was developed to avoid the complications associated with supraclavicular blocks, as well as providing the advantageous benefit of being performed with the patient's arm in any position (unlike the axillary approach).[2] Properly administered, this block is indicated for treating sympatholysis and pain syndromes, as well as providing anesthesia and analgesia for elbow, hand, distal arm, and wrist surgery.[4,8] It can provide a consistent block of the axillary and musculocutaneous nerves, as well as treat patients with chronic pain of the upper extremity over prolonged periods.[2,10]

The infraclavicular brachial plexus block is strongly contraindicated by the unfamiliarity with the procedure, patient refusal, infection at site, coagulopathy, and allergies to the agents being used. Additional contraindications include thorax deformity, dislocated healed clavicular fracture, pre-existing neuropathies, and the presence of foreign bodies in the area (e.g., pacemakers).[8,9]

INTERSCALENE BLOCK

The interscalene brachial plexus block is primarily indicated for anesthesia and postoperative analgesia in surgery of the shoulder and clavicle. This block is also indicated for arm and forearm surgery, as well as the insertion of arteriovenous grafts for hemodialysis. The interscalene catheter is indicated for acromioplasties, carcinologic surgery and physiotherapy of the shoulder, rotator cuff repair, and total shoulder arthoplasty.[4,12]

The interscalene brachial plexus block is strongly contraindicated when there is unfamiliarity with the procedure, patient refusal, infection at site, moderate to severe respiratory disease, ipsilateral apical bullae, and allergies to the agents being used. Other contraindications include coagulopathy, prior neck surgery or radiation, ipsilateral/contralateral pneumothorax, contralateral phrenic and recurrent paresis, and chronic obstructive pulmonary disease.[8,9]

EQUIPMENT

AXILLARY BLOCK

A standard regional anesthesia tray is prepared with the following equipment[4]:

- Sterile towels, sponges, and 4-inch × 4-inch gauze packs
- Sterile gloves, marking pen, and surface electrode
- 20-ml syringes containing local anesthetic
- Three-way stopcock
- 1–1-1/2-inch, 25-gauge needle for skin infiltration
- 3–5 cm, short-bevel, insulated stimulating needle
- Peripheral nerve stimulator

SUPRACLAVICULAR BLOCK

A standard regional anesthesia tray is prepared with the following equipment[11,13]:

- Standard anesthesia monitoring and available resuscitation facilities
- Venous access with an intravenous drip started
- Sterile towels, sponges, and 4-inch × 4-inch gauze packs
- Sterile gloves, marking pen, tape meter for collar measure, and surface electrode
- 21-gauge, insulated stimulating needle; 6 cm long for narrow necks (<38 cm) to 10 cm for thick necks (>38 cm)
- Neurostimulator
- 10-ml syringes containing local anesthetics
- Additional equipment for continuous block: catheter set, 20 ml saline, and 5 ml contrast media

INFRACLAVICULAR BLOCK

A standard regional anesthesia tray is prepared with the following equipment[4,14]:

- Sterile towels, sponges, and 4-inch × 4-inch gauze packs
- Sterile gloves, marking pen, and surface electrode
- 20-ml syringes containing local anesthetic
  Peripheral nerve stimulator
- 1-1/2-inch, 25-gauge needle for skin infiltration
- 10-cm, short-bevel, insulated stimulating needle
- Additional equipment for continuous block: catheter set and 20 ml saline

INTERSCALENE BLOCK

A standard regional anesthesia tray is prepared with the following equipment[4]:

- Sterile towels, sponges, and 4-inch × 4-inch gauze packs
- Sterile gloves, marking pen, and surface electrode
- 20-ml syringes containing local anesthetic
- Peripheral nerve stimulator
- 1-1/2-inch, 25-gauge needle for skin infiltration
- 3–5 cm, short-bevel, 22-gauge, insulated stimulating needle
- Additional equipment for continuous block: catheter set, 20 ml saline, and 3–5-cm insulated stimulating needle (Tuohy-style or Quincke-tip)

DRUGS

For all approaches to the brachial plexus block, the concentration and type of anesthetic will depend on the size of the patient and available drugs, as well as whether the

block is intended for use as surgical anesthesia or for pain management. Although lidocaine and mepivacaine are commonly used for single-shot blocks, the most commonly preferred local anesthetics for use in long-term pain relief and continuous infusion are bupivacaine and ropivacaine.[3,4,8] To minimize the amount of local anesthetics and, theoretically, improve the efficacy of the block, adjuvant drugs such as opioids or clonidine may be used.[2]

Plasma concentration and pharmacokinetics of brachial plexus block infusion in a steady state are similar to those seen with epidural infusion. Once the steady state is achieved, the drugs infused do not accumulate if infusion continues at the same rate. The metabolites also remain at insignificant levels without causing any deleterious effect. However, continuous infusions should be used with caution when treating patients with liver and kidney disease.

AXILLARY BLOCK

The axillary brachial plexus approach requires a larger volume of local anesthetic (35–40 ml) to achieve complete anesthesia. Due to the increased risk of inadvertent intravascular injection in this area, the local anesthetic should be injected slowly and with frequent aspiration. Also, for this reason, use of bupivacaine is contraindicated due to its high cardiotoxicity.[4]

For short-duration blocks (<3 hours), 1.5% mepivacaine (1:200,000 epinephrine), 40 ml, is used. For medium-duration blocks (3–6 hours), 1.5–2% lidocaine (1:200,000 epinephrine), 40 ml, is used. For long-duration blocks (>6 hours), 0.5% ropivacaine, 40 ml, is used.[4,9] For continuous blocks, 0.2% ropivacaine, 20 ml, is administered approximately every 6 hours.[8]

SUPRACLAVICULAR BLOCK

The supraclavicular brachial plexus approach requires larger volumes of local anesthetic to achieve complete anesthesia. Due to the increased risk of inadvertent intravascular injection in this area, the local anesthetic should be injected slowly and with frequent aspiration. The risk–benefit ratio of using large concentrations of local anesthetic during this approach must be examined prior to performing the block.

For short-duration blocks (<1 hour), 2–3% 2-chloroprocaine, 40 ml, is used. For medium-duration blocks (>2 hours), 1.5–2% lidocaine (1:200,000 adrenaline), 40 ml, is used. For long-duration blocks (>3 hours), 0.5% ropivacaine, 40 ml, is used.[2,11] For continuous blocks, 2.5 mg/ml (0.25%), bupivacaine is administered at an infusion rate of 6–10 ml/hr.[15]

INFRACLAVICULAR BLOCK

The infraclavicular brachial plexus approach requires larger volumes of local anesthetic to achieve complete anesthesia. Due to the increased risk of inadvertent intravascular injection in this area, the local anesthetic should be injected slowly and with frequent aspiration. The risk/benefit ratio of using large concentrations of local anesthetic during this approach must be examined prior to performing the block.[4]

For short-duration blocks (<3 hours), 1–1.5% mepivacaine (1:200,000 epinephrine), 30–40 ml, is used. For medium-duration blocks (3–6 hours), 1.5–2% lidocaine (1:200,000 epinephrine), 30–40 ml, is used. For long-duration blocks (>6 hours), 0.5–0.75% ropivacaine, 30 ml, is used. For continuous blocks, 0.25% bupivacaine or 0.2% ropivacaine can be continuously infused at 10 ml/hr.[4,8]

INTERSCALENE BLOCK

The interscalene brachial plexus approach requires a larger volume of local anesthetic (35–40 ml) to achieve complete anesthesia, but smaller concentrations (15–20 ml) can be used to achieve successful analgesia. Due to the increased risk of inadvertent intravascular injection in this area, the local anesthetic should be injected slowly and with frequent aspiration.[4]

For short-duration blocks (<1.5 hours), 2–3% 2-chloroprocaine (1:200,000 epinephrine), 40 ml, is used. For medium-duration blocks (>2 hours), 1.5–2% lidocaine or mepivacaine (1:200,000 epinephrine), 30–40 ml, is used. For long-duration blocks (>6 hours), 0.5–0.75% ropivacaine, 30 ml, is used.[4,9] For continuous blocks, 0.2% ropivacaine, 15–20 ml, is administered approximately every 6 hours.[4,8]

PREPARATION OF PATIENT

PHYSICAL EXAMINATION

Before administering a brachial plexus block, it is important to conduct a preoperative evaluation of the area of infiltration for the presence of local infection and distorted anatomy, as well as conduct an assessment of the ability to properly position the arm. A neurological examination should be performed to document any deficits.

PREOPERATIVE MEDICATION

For preoperative medication, use recommendations for conscious sedation by the Standard American Society of Anesthesiologists.

PERIPHERAL NERVE STIMULATOR

These procedures are best done with the use of a peripheral nerve stimulator to confirm the tip of the needle to be on the brachial plexus. Proper use of the peripheral nerve stimulator is described in the following.[16]

The ground electrode is attached to the electrocardiogram pad and placed away from the region to be stimulated

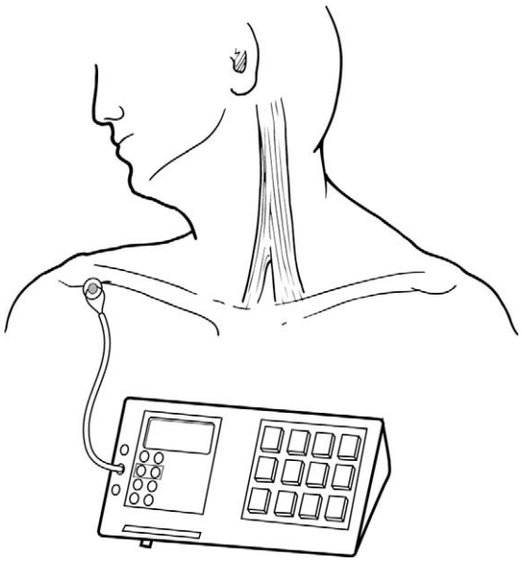


FIGURE 24–3

Ground electrode is placed in a region away from the block. In this example, it is placed on the opposite shoulder. (From Raj PP, editor: *Textbook of Regional Anesthesia.* Philadelphia, Churchill Livingstone, 2002, p. 262, with permission.)

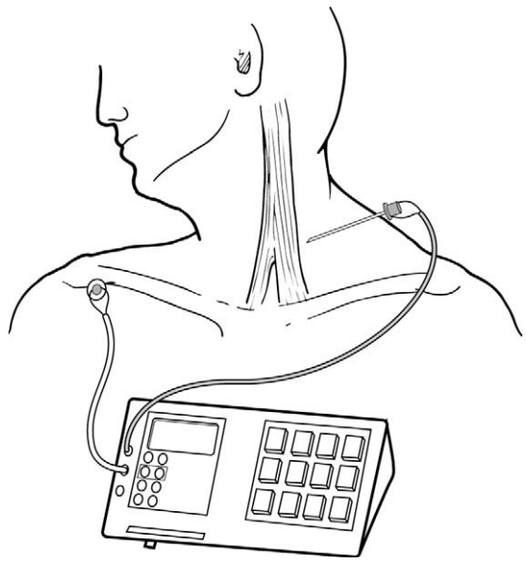


FIGURE 24–4

The needle penetrates the skin with the exploring electrode connected to the hub of the needle. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, p. 262, with permission.)

(Figure 24-3). Conventional electrocardiogram-type electrodes are suitable, but care must be taken to ensure that they make good contact with clean, dry skin. Due to current flow between the two electrodes, the ground should not be positioned over a superficial peripheral nerve and the current should not be allowed to pass through the myocardium.

The syringe containing the anesthetic solution is attached to an extension set filled with anesthetic solution. The end of the extension set is connected to a 22-gauge, 3.75-cm needle. Next, the needle is inserted through the skin and advanced a short distance. The exploring electrode is connected to the hub of the needle via the alligator clamp (Figure 24-4). The ampere control is set so that the current flows at 4–5 mA at a frequency of one pulse per second (Figure 24-5).

The needle is advanced slowly, while the forearm and hand are observed carefully for muscle movements. Flexion or extension of the elbow, wrist, or digits confirms that the needle is in close proximity to nerve fibers of the brachial plexus, usually within 1–2 cm of the nerve. As the needle is advanced toward the nerve, the twitch increases in intensity. The best results are obtained if the movements occur as distally as possible (i.e., fingers or hand) (Figure 24-6). If the twitch decreases with needle advancement, the needle is likely to one side of the nerve and should be repositioned.

The current is reduced (Figure 24-7), and the needle is moved deeper until a point is reached where there is optimal muscle twitch with minimal current, usually 0.5 mA to 1.0 mA (Figure 24-8). This marks the test injection point.

After careful aspiration, 1–2 ml of anesthetic solution is injected through the needle. The immobile needle technique described by Winnie is appropriate in this situation.[17] Within

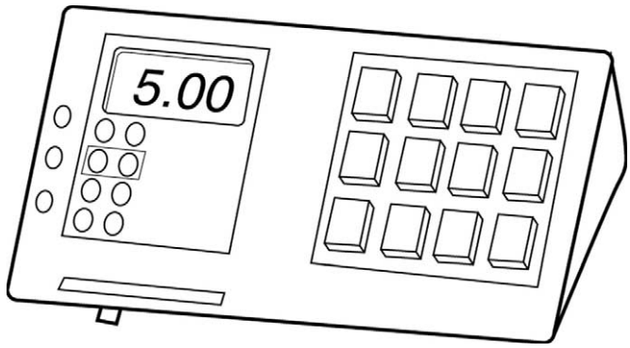


FIGURE 24–5

Initially, the stimulator is set at 4 mA to 5 mA and one pulse per second. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, p. 263, with permission.)

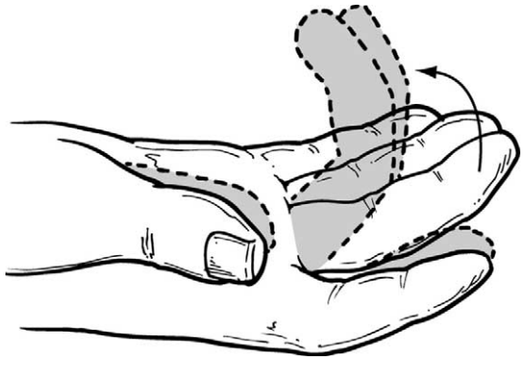


FIGURE 24–6

Flexion of medial 1-1/2 digits is seen as the needle approaches the nerve trunk in brachial plexus block. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, p. 263, with permission.)

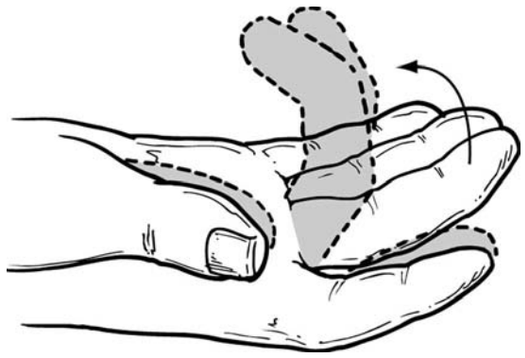


FIGURE 24–7

The current is reduced to between 0.5 mA and 1.0 mA, and the needle is stabilized when maximal stimulation of the contracting muscles is achieved. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, p. 263, with permission.)

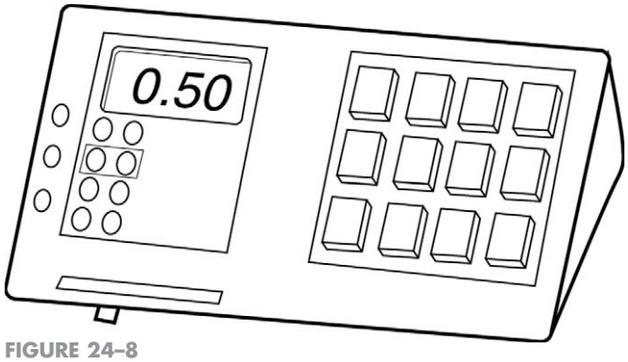


FIGURE 24–8

Maximal flexion of digits supplied by the median nerve is seen here with stimulation at 0.5 mA; this suggests that the needle is in close proximity to the median nerve fibers. (From Raj PP, editor: Textbook of Regional *Anesthesia*. Philadelphia, Churchill Livingstone, 2002, p. 263, with permission.)

10 seconds of the test injection, muscle movement should diminish considerably or disappear entirely (Figure 24-9).

If a reduction in muscle movement does not occur, the stimulation is probably coming from the side of the needle, in which case the needle should be repositioned and the procedure repeated until complete cessation of the muscle twitch can be obtained.

Immediately after muscle movement has ceased, the remaining anesthetic solution should be injected through the needle. Although it is possible to restore the muscle twitch after injection by increasing the stimulating current to 10 mA, this does not mean the block has failed. However, if further contractions continue at a low current, it is worth injecting more local anesthetic.

The onset of the block occurs initially in the region supplied by the nerve to the muscles that were twitching. Proximal muscle groups are typically paralyzed earlier than the occurrence of sensory loss or sympathetic block.[16] When stimulation of pure sensory nerves is performed, confirmation of needle tip location at the nerve is obtained by eliciting from the patient a radiating paresthesia with every pulsation in the distribution of the nerve. The

FIGURE 24–9

Complete cessation of movement within a few seconds is seen after 2 ml of local anesthetic solution is injected. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, p. 263, with permission.)

quality of the pulsation felt by the patient is very important and should occur at low current and be stopped instantaneously with a 1 ml-test dose.

It is important to check on the condition of the battery, as its failure may cause the muscle twitch to cease. In addition, muscle twitch cessation may occur due to a loose connection, improper needle placement, or some other unknown reason.[16]

PROCEDURES

INFRACLAVICULAR BLOCK

The patient is positioned supine with the head facing away from the side to be blocked. The patient's arm should be abducted and flexed at the elbow for better definition of the landmarks (Figure 24-10A and B). The landmarks used during the block include the following:

- Acromioclavicular joint
- Medial end of the clavicle
- Coracoid process
- Head of the humerus
- Sternoclavicular joint

The length of the clavicle is defined by locating the acromioclavicular junction and the sternoclavicular junction, and then determining the midpoint between them. From this point, a mark is made 2.5–3 cm caudal to the clavicle. The brachial artery is then palpated in the axilla. Using the previously discussed technique for using a peripheral nerve stimulator, the stimulating needle is inserted at a 45-degree angle to the skin below the clavicle and advanced toward the most proximal point at which the axillary artery can be palpated. After appropriate nerve stimulation is elicited (preferably the medial nerve), 2 cc of saline are injected to know that the contraction of the muscle visualized is completely gone. At this point, a contrast material (Iohexol) up to 20 ml is injected. Note the figure showing the contrast extending from under the clavicle to the axillary sheath (Figure 24-11A, B, C, and D). Note also the swelling of the middle portion of the infraclavicular brachial plexus sheath from which the contrast is traversing toward musculocutaneous nerve

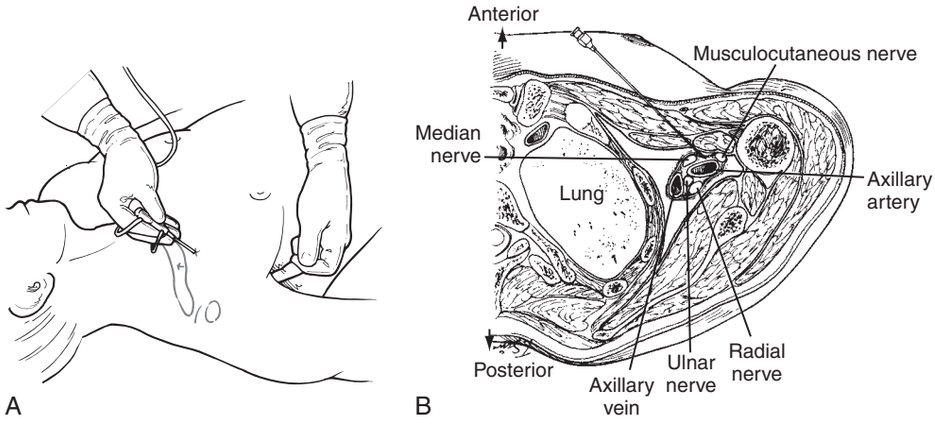


FIGURE 24–10

Infraclavicular approach to the brachial plexus. (A) Note the entry of a 22-gauge 3-1/2-inch needle 1 inch below the midclavicular point. It is directed laterally toward the axillary artery at a 45-degree angle to the skin with the patient's arm abducted to 90 degrees. (B) Transverse section of the axilla showing the relationship to the needle as it penetrates the pectoralis major and minor before entering the brachial plexus. Note the relationship of the neurovascular structures within the brachial plexus sheath. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, p. 349, with permission.)

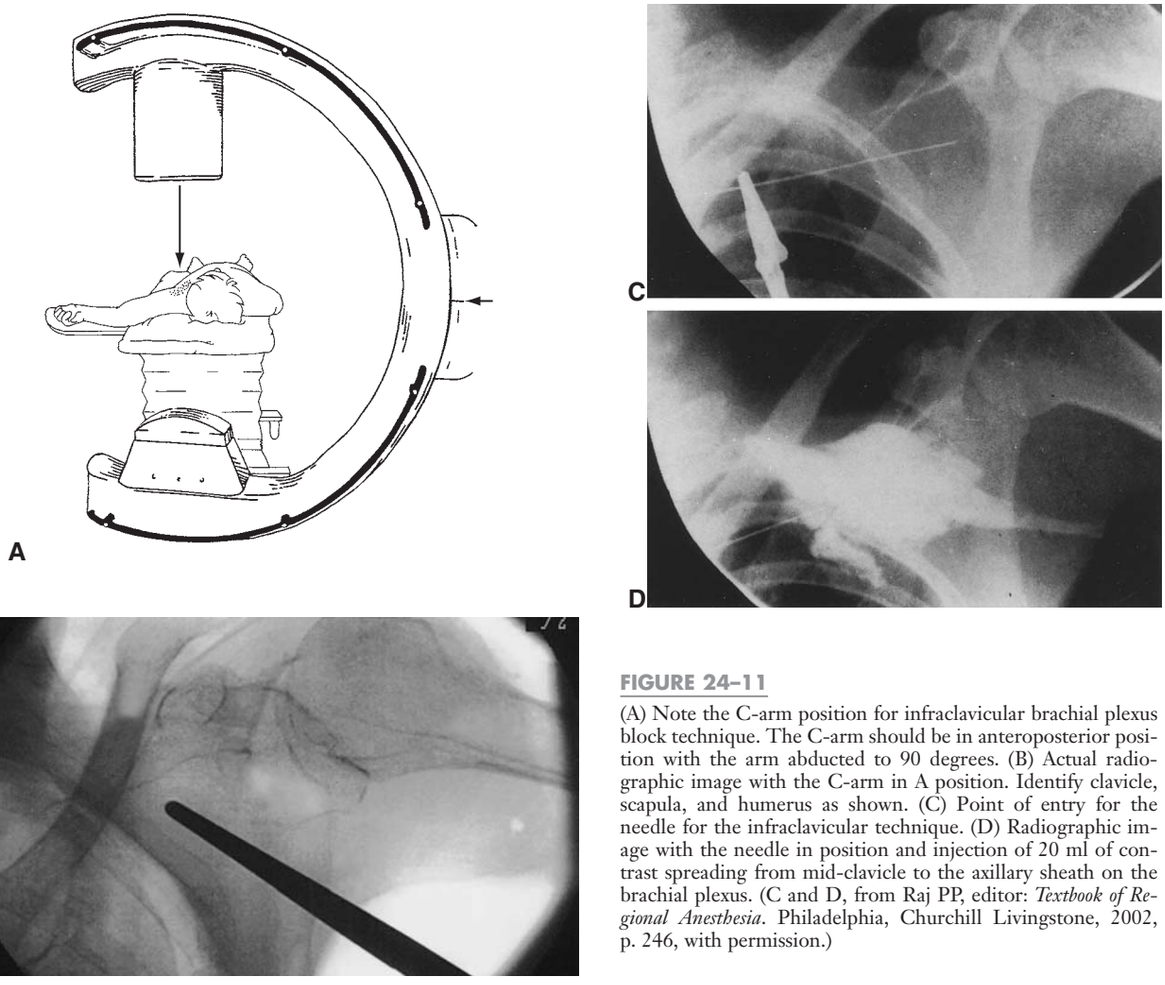


FIGURE 24–11

(A) Note the C-arm position for infraclavicular brachial plexus block technique. The C-arm should be in anteroposterior position with the arm abducted to 90 degrees. (B) Actual radiographic image with the C-arm in A position. Identify clavicle, scapula, and humerus as shown. (C) Point of entry for the needle for the infraclavicular technique. (D) Radiographic image with the needle in position and injection of 20 ml of contrast spreading from mid-clavicle to the axillary sheath on the brachial plexus. (C and D, from Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, p. 246, with permission.)

superiorly and second intercostal nerve inferiorly. Once the confirmation is obtained that the needle is correctly placed on the brachial plexus, up to 40 ml of local anesthetic are injected incrementally.[2,4,8]

AXILLARY BLOCK

The patient is positioned supine with the arm to be blocked abducted to 90 degrees, externally rotated, and flexed 90 degrees at the elbow. Abduction beyond 90 degrees can compress the brachial pulse against the humerus and lead to obliteration of the pulse (Figure 24-12). The landmarks used during the block follow:

- Pulse of axillary artery
- Coracobrachialis muscle
- Pectoralis major muscle

The axillary arterial pulse is palpated proximally and marked. The approximate location of the brachial plexus can also be determined by percutaneous nerve stimulation. After the skin is antiseptically prepared, local anesthetic is infiltrated subcutaneously at the needle insertion site. Two fingers are then used to palpate the gap between the axillary artery and the coracobrachialis muscle with pressure applied distally (minimizing the risk of inadvertent lateral or medial placement of the needle outside the sheath). The insulated needle is inserted at the lateral aspect of the pulse at a 45-degree angle and directed cephalad until median nerve stimulation is obtained (typically at a depth of 1–2 cm). Once muscular contraction of less than 0.5 mA is elicited, 40 ml of local anesthetic are injected incrementally. After injection, the needle is removed and constant digital pressure is maintained as the arm is adducted alongside the patient's body and continued for 1 minute following add-uction.[2,4,8]

CATHETER PLACEMENT FOR CONTINUOUS INFUSION

When there is an indication for continuous infusion of the brachial plexus in the axillary region, an intravenous catheter is used. As soon as the stimulation of the catheter determines the correct location, the stylette is removed and the catheter anchored well with a suture or steristrips. A clear plastic covering would be useful to monitor the catheter in later stages.

SUPRACLAVICULAR BLOCK

The patient is positioned supine with the head turned slightly to the contralateral side, arm at the side, and hand position on the abdomen. For ease, the anesthetist should stand next to the patient's head, opposite the side being operated on (Figure 24-13). The landmarks used during the block include the following:

- Sternal head of sternocleidomastoid muscle
- Clavicle head of sternocleidomastoid muscle
- Midclavicle

After the skin is antiseptically prepared, the needle is inserted superior to the midpoint of the clavicle in the backward-inward-downward direction. The needle should appear to be at right angles to all planes at this level of the neck, and it is not necessary to touch the first rib with the

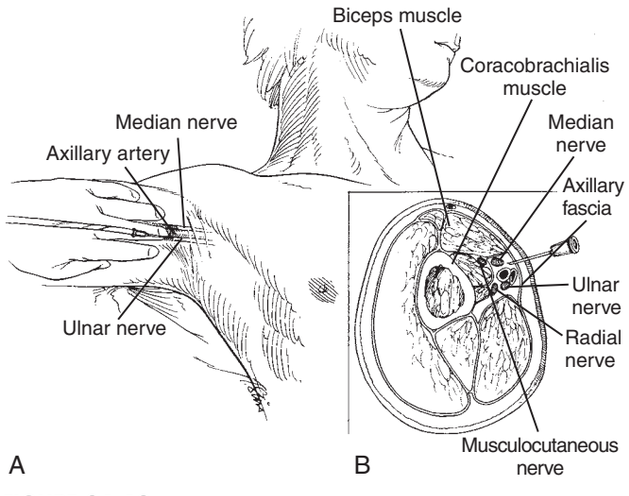


FIGURE 24–12

(A) Axillary approach to brachial plexus blockade. (B) The needle is in the neurovascular bundle close to the brachial artery. The musculocutaneous nerve lies in the coracobrachialis muscle, outside the brachial plexus sheath at this site. (From Raj PP, editor: *Pain Medicine: A Comprehensive Review*, 2nd ed. St. Louis, Mosby, 2003, p. 238, with permission.)

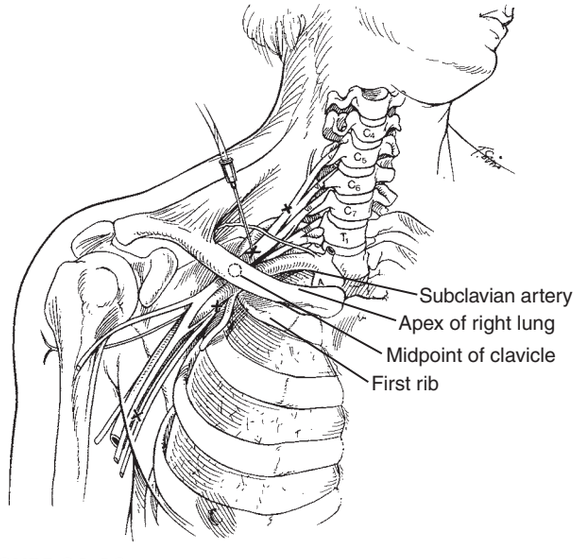


FIGURE 24–13

Supraclavicular approach to the brachial plexus. Note the insertion of the needle 1–2 cm superior to the midclavicular point in a backward-inward-downward direction toward the first rib. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, p. 347, with permission.)

needle. Stimulation of the forearm or hand is elicited. Once stimulation is obtained, negatively aspirate for air or blood, and then inject 1–3 ml of local anesthetic. If no systemic effects are detected after 5 minutes, the total calculated volume of local anesthetic (typically 40 ml) is injected incrementally.[2,3,11]

FLUOROSCOPIC TECHNIQUE

Lanz and Theiss[18] reported that the brachial plexus block near the first rib at the level of the trunks and divisions provided the most reliable efficacy. However, the use of this approach is limited by the increased risk of pneumothorax.[16,18] Furthermore, a successful block depends on achieving paresthesias, but this can be uncomfortable for the patient and even result in nerve injury. To address these problems, Theiss performed the supraclavicular using the contrast medium under fluoroscopic guidance. He advanced the needle near the first rib and posterior to the subclavian artery, which he believed to be in the interscalene space (Figures 24-14 and 24-15). The subclavian artery was identified by the groove where the first rib begins to curve posteriorly. During injection, the placement of the tip of the needle is corrected in accordance to the patient's anatomic characteristics, such as size of the first rib and its angle to the vertebra. Although significant complications are rare, inexperience in performing nerve blocks could result in the needle's improper advancement and possible infiltration of the lung or the subclavian artery.[16]

COMPUTED TOMOGRAPHY GUIDANCE

At times, the normal surface landmarks may be difficult to palpate during a brachial plexus block procedure, especially in patients who have short, thick necks or who have undergone surgery or radiation therapy. These conditions may result in an inability to properly localize the anatomic site for needle insertion and result in improper needle placement, thus resulting in inadequate pain control. The use of computed tomography (CT) can assist localizing the optimal site of needle insertion and to identify the extent of analgesic distribution. Mukherji et al.[19] described a technique for brachial plexus block guided by CT and reported initial results for regional pain management in patients with chronic pain referable to the brachial plexus and surface landmarks that could not be palpated (Figures 24-16 and 24-17). Although CT guidance is not commonly indicated, this technique should be considered for use with difficult patients.[16]

INTERSCALENE BLOCK

The patient is positioned supine or semi-sitting with the head turned to the opposite direction to the side to be blocked. The arm rests to the side and reaches toward the ipsilateral knee (Figure 24-18). The landmarks used during the block include:

- Sternal notch
- Clavicle
- Sternal head of sternocleidomastoid muscle
- Clavicular head of sternocleidomastoid muscle
- Mastoid process

The clavicle, external jugular vein, and the posterior border of the clavicular head of the sternocleidomastoid muscle are marked with a pen. The point of entry is close to the point where the external jugular vein crosses the sternomastoid. The palpating hand should be gently but firmly pressed between the anterior and middle scalene muscles, also known as the interscalene groove, to shorten distance from the skin to the brachial plexus. This is the point of needle insertion.[2,4]

The site is prepared with an antiseptic solution and subcutaneously injected with a local anesthetic. A stimulating needle is guided through insertion point and advanced perpendicular to the skin plane and slightly caudad; the needle should never be oriented cephalad. The slight caudad direction minimizes the chance of inadvertent spinal of epidural placement. Using an initial current of 1.5 mA, the needle is advanced until stimulation of the brachial plexus is elicited. Once stimulation is achieved (the contraction occurring preferably below the shoulder) at a current of less than 0.5 mA, 40 ml of local anesthetic is incrementally injected.[2,4] A new technique uses a catheter placement from the trapezius to the interscalene groove. The advantage of this technique is that the catheter can stay in the interscalene groove longer and not become dislodged in a short period.

COMPLICATIONS

AXILLARY BLOCK

Complications associated with the axillary brachial plexus block include axillary hematoma, neuropathy, infection, postblock ecchymosis, and pneumothorax. The proximity of the nerves to the axillary artery and other large vessels causes an increased risk for inadvertent vascular punctures or local anesthetic toxicity from intravascular absorption. Because paresthesias are elicited, the potential for postblock, persistent paresthesia exists.[3,4,9]

SUPRACLAVICULAR BLOCK

Complications associated with the supraclavicular brachial plexus block include neck hematomas, neuropathy, Horner's syndrome, intra-arterial injection, infection, and local anesthetic toxicity. The risk of pneumothorax is potentially higher than with other brachial plexus blocks and is associated with an overture of the clavicle–needle angle. Other complications can include phrenic paralysis, recurrent laryngeal nerve paralysis, and high spinal or epidural anesthesia.[3,11,20]

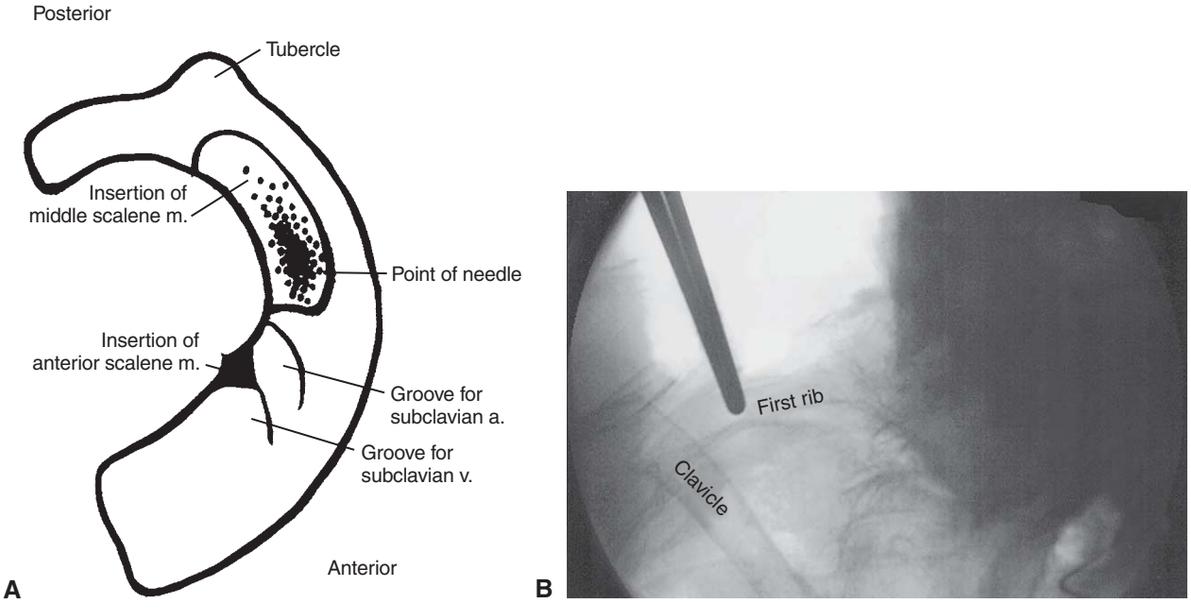


FIGURE 24–14

(A) The part of the first rib that the tip of the needle should touch, defined by plotting the points that the tip of needle touched in the first 80 successful blocks. a, artery; v, vein; m, muscle. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, p. 276, with permission.) (B) Position of the needle entry over the first rib for the supraclavicular technique.

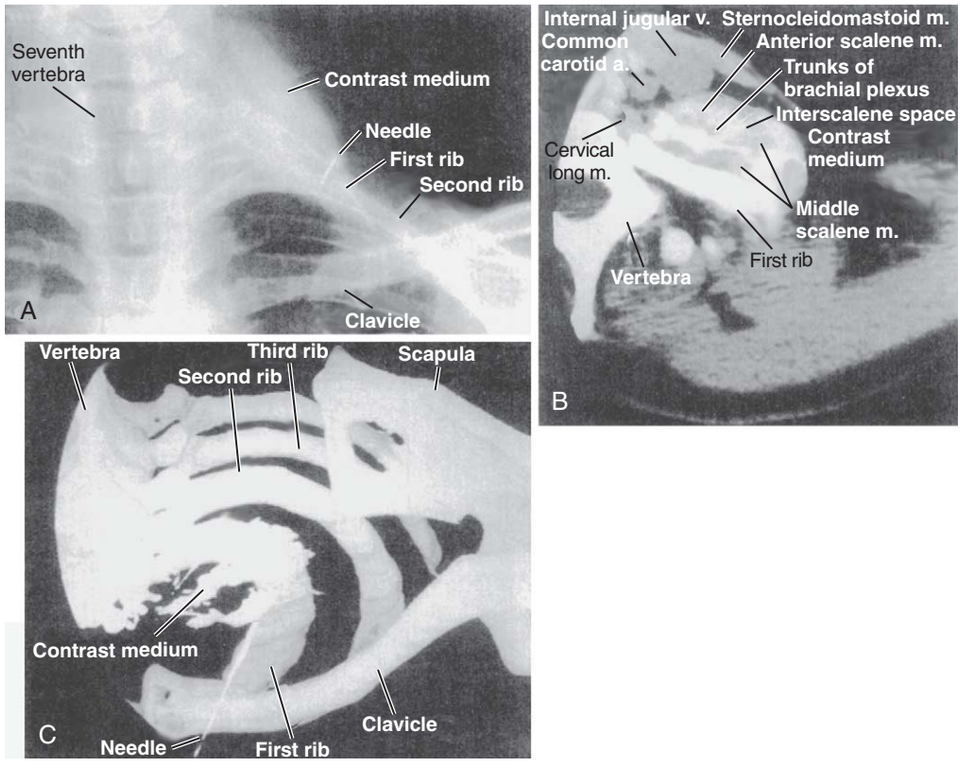


FIGURE 24–15

(A) Brachial plexus block for pain relief of recurrent Pancoast's tumor. Coronal nonenhanced T1-weighted image (repetition time, 600 milliseconds; echo time, 14 milliseconds) depicts a mass situated in the apex of the left lung, as well as obliteration of the apical fat and encasement of the left brachial plexus. On the contralateral side, note the normal appearance of the apical fat and brachial plexus. (B) Transverse CT scan obtained after injection of the solution of local anesthetic and contrast material shows the majority of the solution to be distributed between the anterior and the middle scalene muscles. (C) Transverse CT scan at the neurovascular bundle on the subclavian artery and vein, which indicates that the solution is to contact the brachial plexus. The patient experienced approximately 50% reduction in pain immediately after the injection. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, p. 277, with permission.)

INFRACLAVICULAR BLOCK

Complications associated with the infraclavicular brachial plexus block include hematoma, neuropathy infection, systemic toxicity, pneumothorax, and intravascular or intrathecal injection.[3,4,21]

INTERSCALENE BLOCK

Complications associated with interscalene brachial plexus block include infection, hematoma, vascular puncture, vertebral artery injection, Horner's syndrome, neuropathy, and local anesthetic toxicity. The phrenic nerve can be

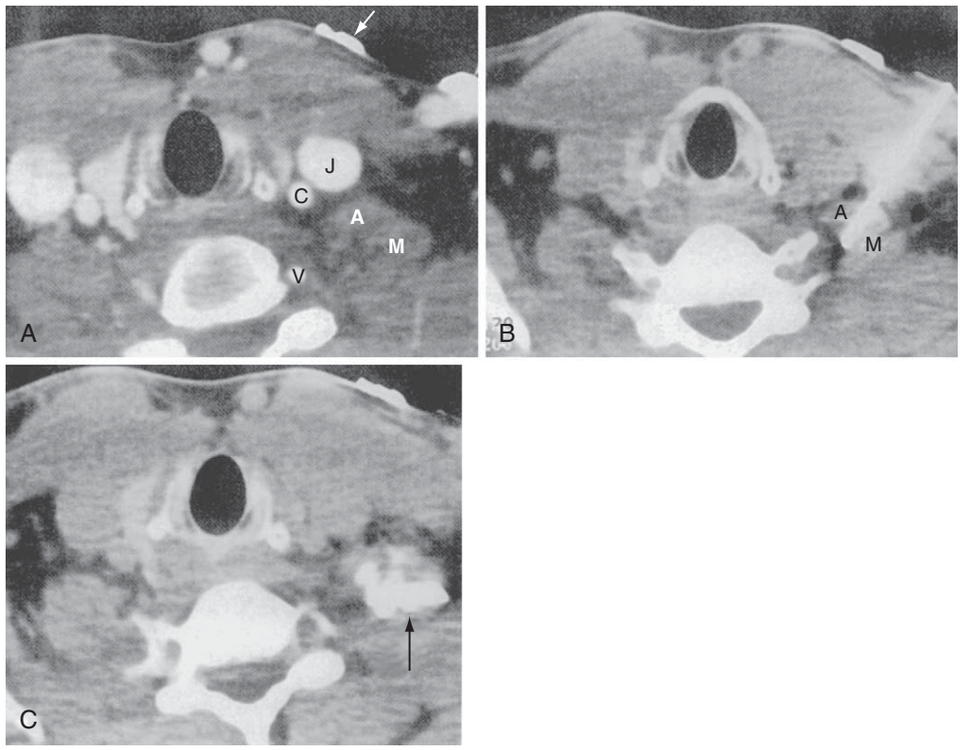


FIGURE 24–16

(A) brachial plexus block to treat a left C7 mononeuropathy. Transverse contrast-enhanced CT scan, obtained after the skin over the brachial plexus was marked with barium (arrows), was acquired to help identify the locations of the common carotid artery (C), internal jugular vein (J), and vertebral artery (V). (B) Transverse t scan demonstrates the tip of the needle inserted into the plane separating the anterior and middle scalene muscles. (C) Transverse CT scan helps confirm that the solution (2 ml) (arrow), which contains lidocaine and water-soluble contrast material and is injected through the needle, is located between the planes of the anterior and middle scalene muscles, and thereby must involve the supraclavicular brachial plexus. The patient experienced complete pain relief immediately after the injection. A, anterior scalene muscle; M, middle scalene muscle. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, p. 278, with permission.)

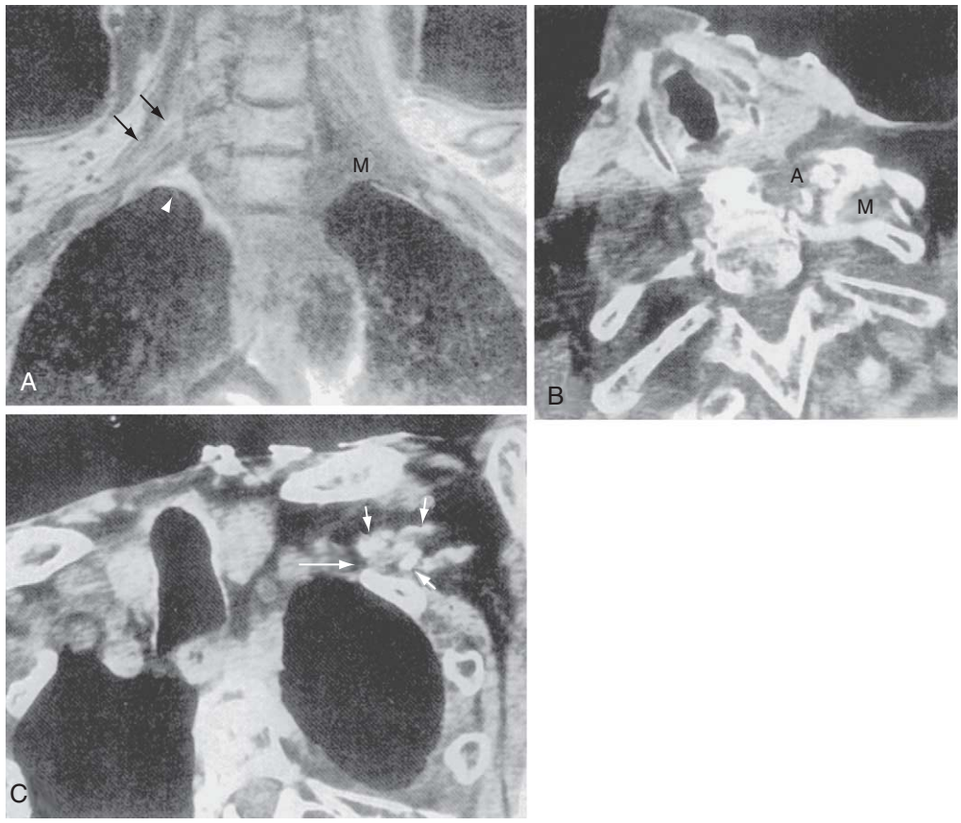


FIGURE 24–17

(A) Brachial plexus block for pain relief for recurrent Pancoast's tumor. Coronal nonenhanced T1-weighted image (repetition time, 600 milliseconds; echo time, 14 milliseconds) depicts a mass (M) situated in the apex of the left lung, as well as obliteration of the apical fat and encasement of the left brachial plexus. On the contralateral side, note the normal appearance of the apical fat (arrowhead) and brachial plexus (arrows). (B) Transverse CT scan obtained after injection of the solution of local anesthetic and contrast material shows the majority of the solution to be distributed between the anterior (A) and middle (M) scalene muscles. (C) Transverse CT scan at the thoracic inlet depicts contrast material (small arrows) extending along the neurovascular bundle (large arrow) of the subclavian artery and vein, which indicates that the solution is in contact with the brachial plexus. The patient experienced approximately 50% reduction in pain immediately after injection. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, p. 279, with permission.)

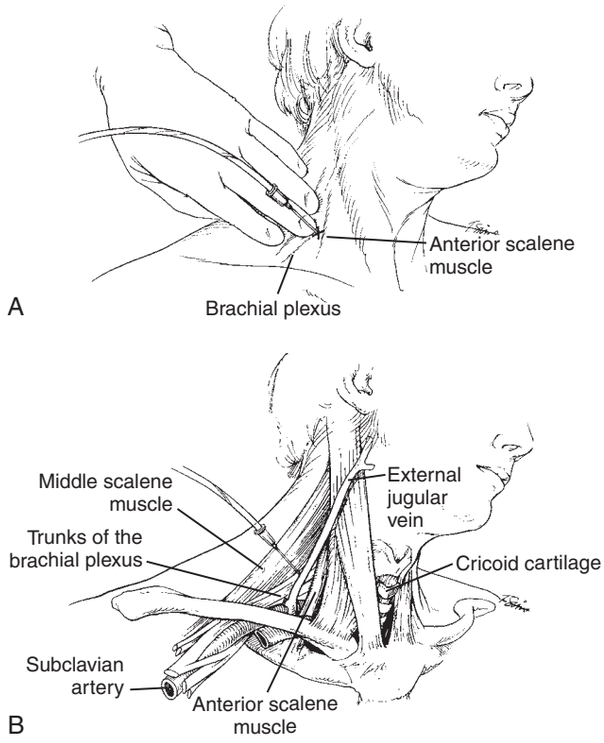


FIGURE 24–18

(A) Superficial landmarks, site of entry, and position of the needle for the interscalene approach to the brachial plexus. (B) The needle usually contacts the upper trunk. (From Raj PP, editor: *Pain Medicine: A Comprehensive Review*, 2nd ed. St. Louis, Mosby, 2003, p. 237, with permission.)

blocked frequently, as well as the blockade of recurrent laryngeal, vagus, and cervical sympathetic nerves. Brachial plexopathy, permanent spinal cord injury, total spinal anesthesia, and activation of the Bezold–Jarisch reflex have been reported.[2,4,11]

EFFICACY

The brachial plexus block is the most effective technique for providing anesthesia and postoperative analgesia to the upper extremity, be it for surgery or pain complication. Recent modifications to the brachial plexus block have further increased the safety and efficacy of this useful technique.

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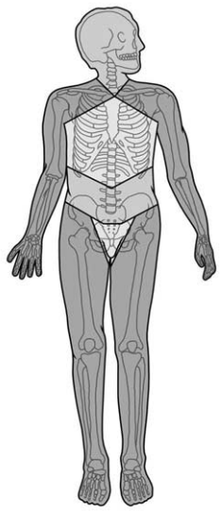
C H A P T E R



25 Joint Blocks of the Upper Extremity

STEVEN D. WALDMAN

HISTORY

As physicians gained experience with the techniques of regional anesthesia in the early 20th century, it was only logical that these injection techniques would be expanded to include therapeutic applications. One could speculate that it was in fact the ability to use regional anesthesia to anesthetize the upper extremity that led to an increased interest in the treatment of diseases of the joint. There are myriad anecdotal reports of physicians' attempts to treat joint pain by the intra-articular injection of a variety of substances including petroleum jelly, glycerin, lipodol, and formalin.

However, it was the isolation of Substance E by Kendall and Hench at the Mayo Clinic and subsequent clinical use in 1948 that paved the way for cortisone, the first truly effective drug for intra-articular administration.[1] It was only 3 years later that Hollander and colleagues[2] published their landmark article in the *Journal of the American Medical Association* demonstrating the efficacy on the intra-articular injection of cortisone. By the 1960s, the intra-articular administration of corticosteroids became a widely accepted treatment modality that remains one of the most commonly used injection techniques in clinical practice.[3] The continued search of drugs that can treat the most common forms of joint pain and functional disability is ongoing with the addition of hyaluronic acid derivatives enjoying a modicum of clinical favor at the time of this writing.

THE SHOULDER

ANATOMY

The rounded head of the humerus articulates with the pear-shaped glenoid fossa of the scapula.[4] The articular surface is covered with hyaline cartilage, which is susceptible to arthritis. The rim of the glenoid fossa is comprised of a fibrocartilaginous layer called the glenoid labrum, which is susceptible to trauma should the humerus be subluxed or dislocated (Figure 25-1). The joint is surrounded by a relatively lax capsule, which allows the wide range of motion of the shoulder joint at the expense of decreased joint stability. The joint capsule is lined with a synovial membrane that attaches to the articular cartilage. This membrane gives rise to synovial tendon sheaths and bursae that are subject to inflammation. The shoulder joint is innervated by the axillary and suprascapular nerves.

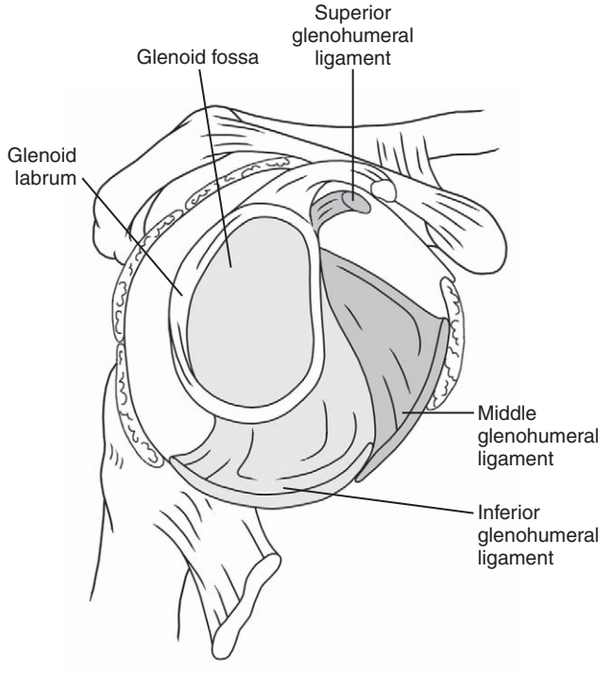


FIGURE 25-1
Anatomy of the glenoid fossa and labrum.

The major ligaments of the shoulder joint are the glenohumeral ligaments in front of the capsule; the transverse humeral ligament between the humeral tuberosities; and the coracohumeral ligament, which stretches from the coracoid process to the greater tuberosity of the humerus (Figure 25-2). Along with the accessory ligaments of the shoulder, these major ligaments provide strength to the shoulder joint. The strength of the shoulder joint is also dependent on short muscles that surround the joint: the subscapularis, the supraspinatus, the infraspinatus, and the teres minor. These muscles and their attaching tendons are susceptible to trauma and to wear and tear from overuse and misuse.

INDICATIONS

The shoulder joint is susceptible to the development of arthritis from a variety of conditions, which have in common the ability to damage the joint cartilage. Osteoarthritis of the joint is the most common form of arthritis that results in shoulder joint pain.[5] However, rheumatoid arthritis, post-traumatic arthritis and rotator cuff tear arthropathy are also common causes of shoulder pain secondary to arthritis[5] (Figure 25-3). Less common causes of arthritis-induced shoulder pain include the collagen vascular diseases, infection, villonodular synovitis, and Lyme disease. Acute infectious arthritis will usually be accompanied by significant systemic symptoms including fever and malaise and should be easily recognized by the astute clinician and treated appropriately with culture and antibiotics, rather than injection therapy. The collagen vascular diseases will generally present as a polyarthropathy rather than a monoarthropathy limited to the shoulder joint, although shoulder pain secondary to collagen vascular disease responds exceedingly well to the intra-articular injection technique described below.

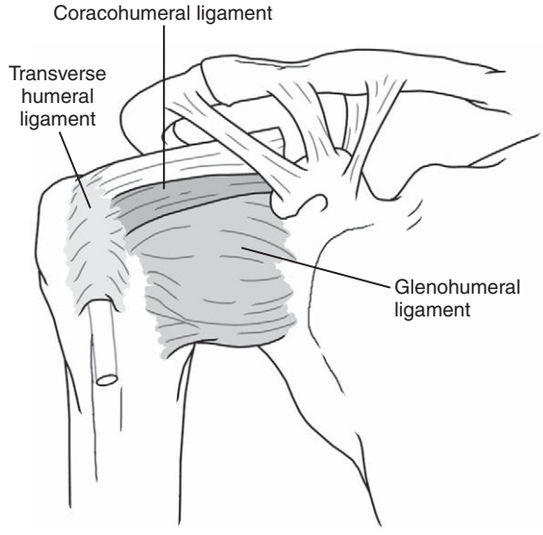


FIGURE 25-2
The ligaments of the shoulder joint.

The majority of patients presenting with shoulder pain secondary to osteoarthritis, rotator cuff arthropathy, and post-traumatic arthritis pain will present with the complaint of pain, which is localized around the shoulder and upper arm. Activity makes the pain worse, with rest and heat providing some relief. The pain is constant and characterized as aching in nature. The pain may interfere with sleep. Some patients will complain of a grating or popping sensation with use of the joint, and crepitus may be present on physical exam.

In addition to the above mentioned pain, patients suffering from arthritis of the shoulder joint will often experience a gradual decrease in functional ability with decreasing shoulder range of motion, making simple everyday tasks such as hair combing, fastening a brassiere, or reaching overhead quite difficult. With continued disuse, muscle wasting may occur and a frozen shoulder may develop.

Plain radiographs are indicated in all patients who present with shoulder pain. Based on the patient's clinical presentation, additional testing, including complete blood count, sedimentation rate and antinuclear antibody testing, may be indicated. Magnetic resonance imaging (MRI) scan of the shoulder is indicated if the rotator cuff tear is suspected.

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represent a relative contraindication to the performance of intra-articular injection of the shoulder, although the risk of hemarthrosis following intra-articular injection even in those patients with therapeutic INR levels is very low.[4,6] Local infection involving the area of the shoulder is also a contraindication to the performance of intra-articular injection of the shoulder.

EQUIPMENT

- Peripheral nerve block tray
- 22-gauge, 1-1/2-inch needle
- 18-gauge, 1-1/2-inch needle
- 12-ml sterile syringe

DRUGS

- 1% preservative-free lidocaine (for diagnostic or prognostic block)
- 0.25% preservative-free bupivacaine (for therapeutic block)
- Depot methylprednisolone (for therapeutic block)
- Water-soluble contrast suitable for intra-articular injection

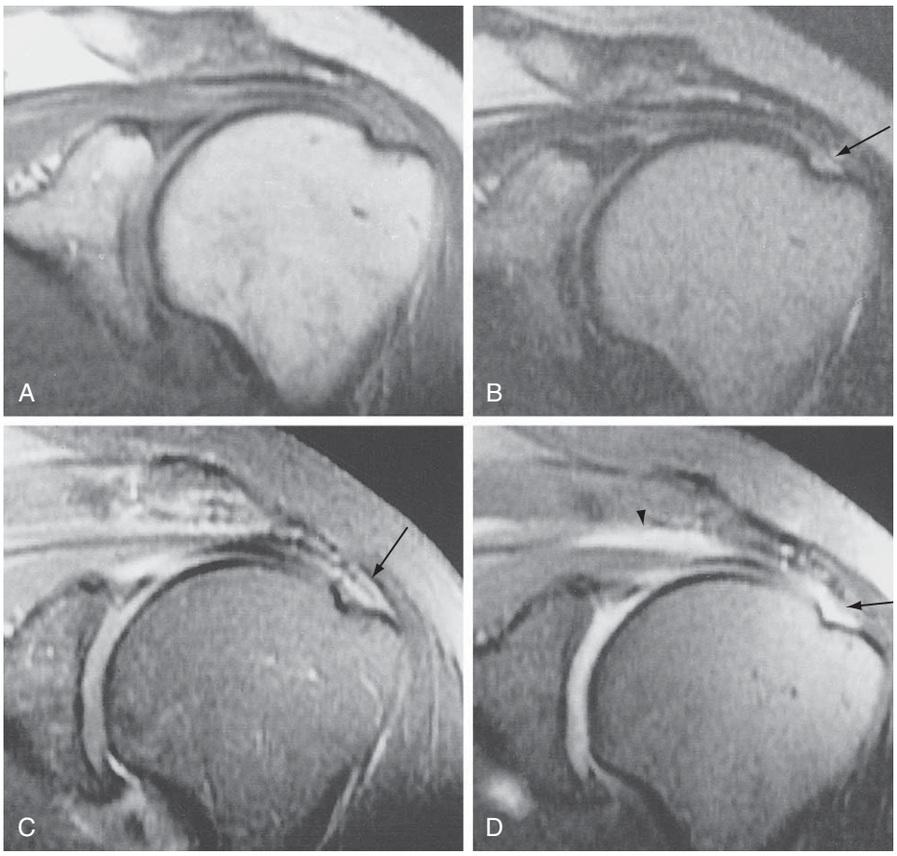


FIGURE 25-3

Full-thickness rotator cuff tears: MR imaging with and without intravenous gadolinium administration and MR arthrography. Coronal oblique intermediate-weighted (TR/TE, 2000/20) (A) and T2-weighted (TR/TE, 2000/80). (B) Spin-echo MR images reveal altered signal intensity in the distal portion of the supraspinatus tendon with high signal intensity in this region (arrow). The findings are consistent with a small full-thickness tendon tear. A coronal oblique T1-weighted (TR/TE, 600/20), spin-echo MR image obtained with fat suppression and immediately after intravenous administration of a gadolinium compound. (C) Enhancement of signal intensity in the region of the tendon tear (arrow) and in several areas of the glenohumeral joint itself. A coronal oblique T1-weighted (TR/TE, 600/15) spin-echo MR image obtained with fat suppression and after intra-articular injection of a gadolinium compound (D) confirms a full-thickness tear of the supraspinatus tendon with the contrast agent of high signal intensity located in the tendinous gap (arrow) and in the subacromial bursa (arrowhead), as well as within the joint. MR, magnetic resonance. (From Resnick D: *Diagnosis of Bone and Joint Disorders*, 4th ed. Philadelphia, Saunders, 2002, p. 3100.)

PROCEDURE

The goals of this injection technique are explained to the patient. The patient is placed in the supine position, and proper preparation with antiseptic solution of the skin overlying the shoulder, subacromial region, and joint space is carried out. A sterile syringe containing the 2.0 ml of 0.25% preservative-free bupivacaine and 40 mg of methylprednisolone is attached to a 1.5-inch, 22-gauge needle using strict aseptic technique. With strict aseptic technique, the midpoint of the acromion is identified and at a point approximately 1 inch below the midpoint, the shoulder joint space is identified. The needle is then carefully advanced through the skin and subcutaneous tissues through the joint capsule into the joint (Figure 25-4). If bone is encountered, the needle is withdrawn into the subcutaneous tissues and redirected superiorly and slightly more medial. After entering the joint space, the contents of the syringe are gently injected. There should be little resistance to injection. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced slightly into the joint space until the injection proceeds without significant resistance. If there is any question that the needle tip is in fact intra-articular, a small amount of water-soluble contrast suitable for intra-articular injection may be added to the injectate (Figure 25-5). The needle is

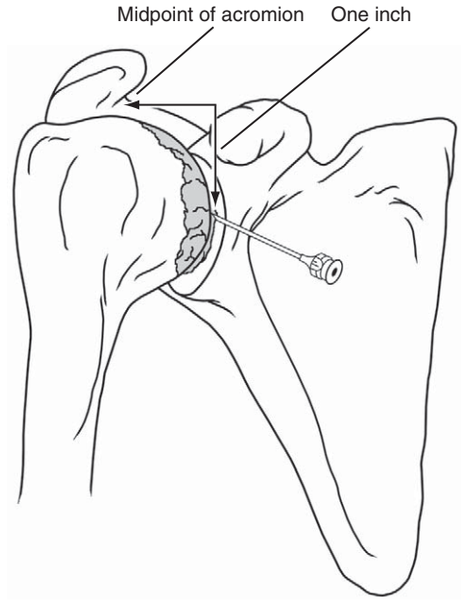


FIGURE 25-4

Needle placement for intra-articular injection of the shoulder joint.

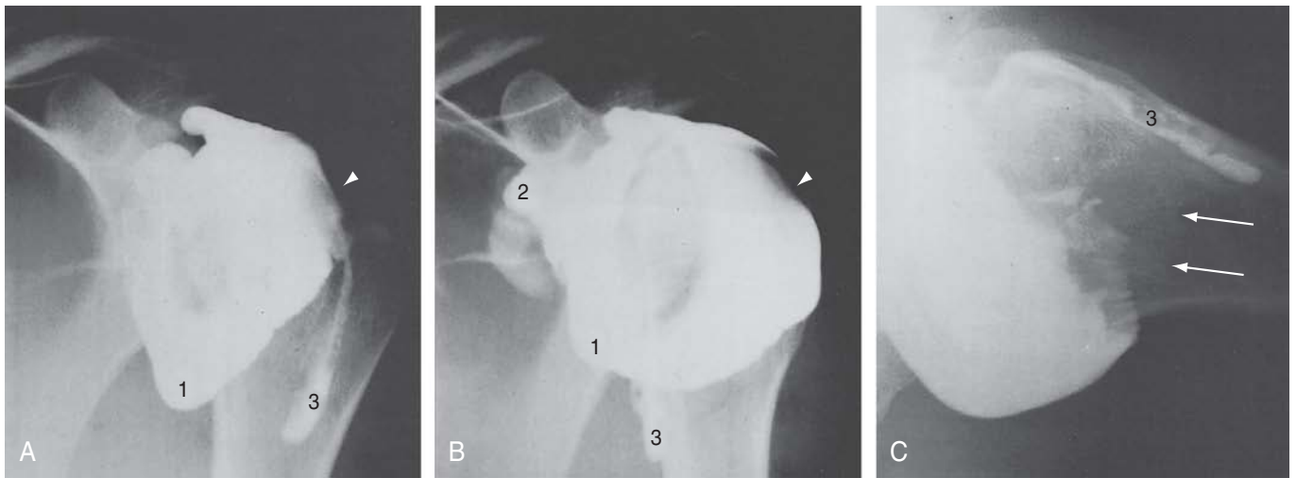


FIGURE 25-5

Glenohumeral joint arthrography. (A) Normal arthrogram: external rotation. Visualized structures include the axillary pouch (1) and bicipital tendon sheath (3). Note that the subscapular recess is not well seen and contrast material ends abruptly laterally at the anatomic neck of the humerus (arrowhead). (B) Normal arthrogram: internal rotation. Observe the prominent subscapular recess (2), axillary pouch (1), and bicipital tendon sheath (3). The articular cartilage of the humeral head is easily visible (arrowhead). Minimal extravasation of contrast material has occurred in the axilla near the injection site. (C) Normal arthrogram: axillary view. Observe the bicipital tendon (3) and the absence of contrast material over the surgical neck of the humerus (arrows). (From Resnick D: *Diagnosis of Bone and Joint Disorders*, 4th ed. Philadelphia, Saunders, 2002, p. 231.)

then removed, and a sterile pressure dressing and ice pack are placed at the injection site.

COMPLICATIONS

The major complication of intra-articular injection of the shoulder is infection.[4] This complication should be exceedingly rare if strict aseptic technique is adhered to.[7] Approximately 25% of patients will complain of a transient increase in pain following intra-articular injection of the shoulder joint and should be warned of such.

CLINICAL PEARLS

This injection technique is extremely effective in the treatment of pain secondary to the above mentioned causes of arthritis of the shoulder joint. Coexistent bursitis and tendonitis may also contribute to shoulder pain and may require additional treatment with more localized injection of local anesthetic and depot steroid. This technique is a safe procedure if careful attention is paid to the clinically relevant anatomy in the areas to be injected. Care must be taken to use sterile technique to avoid infection, as well as the use of universal precautions to avoid risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is placed on the injection site immediately following injection. The use of physical modalities including local heat, as well as gentle range of motion exercises, should be introduced several days after the patient undergoes this injection technique for shoulder pain. Vigorous exercises should be avoided as they will exacerbate the patient's symptomatology. Simple analgesics and nonsteroidal anti-inflammatory agents may be used concurrently with this injection technique.

THE ELBOW

ANATOMY

The elbow joint is a synovial hinge-type joint that serves as the articulation between the humerus, radius, and ulna[4] (Figure 25-6). The joint's primary function is to position the wrist to optimize hand function. The joint allows flexion and extension at the elbow, as well as pronation and supination of the forearm. The joint is lined with synovium, and the resultant synovial space allows intra-articular injection. The entire joint is covered by a dense capsule that thickens medially to form the ulnar collateral ligament and medially to form the radial collateral ligaments (Figure 25-7). These dense ligaments coupled with the elbow joint's deep bony socket makes this joint extremely stable and relatively resistant to subluxation and dislocation. The anterior and posterior joint capsule is less dense and may become distended if there is a joint effusion. The olecranon bursa lies in the posterior aspect of the elbow joint and may become inflamed as a result of direct trauma or overuse of the joint. Bursae susceptible to the development of bursitis also exist between the insertion of the biceps and the head of the radius, as well as in the antecubital and cubital area.

The elbow joint is innervated primarily by the musculocutaneous and radial nerves with the ulnar and median nerves providing varying degrees of innervation. At the

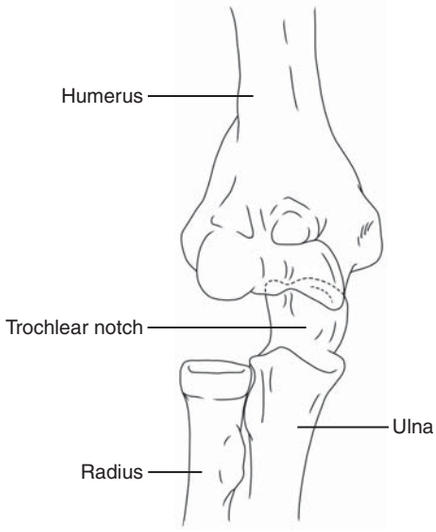


FIGURE 25-6
Bony anatomy of the elbow.

middle of the upper arm, the ulnar nerve courses medially to pass between the olecranon process and medial epicondyle of the humerus. The nerve is susceptible to entrapment and trauma at this point. At the elbow, the median nerve lies just medial to the brachial artery and is occasionally damaged during brachial artery cannulation for blood gases.

INDICATIONS

The elbow joint is susceptible to the development of arthritis from a variety of conditions, which have in common the ability to damage the joint cartilage. Osteoarthritis of the joint is the most common form of arthritis that results in elbow joint pain.[5] However, rheumatoid arthritis, post-traumatic arthritis, and psoriatic arthritis are also common causes of elbow pain secondary to arthritis. Less common causes of arthritis-induced elbow pain include the collagen vascular diseases, infection, and Lyme disease. Acute infectious arthritis will usually be accompanied by significant systemic symptoms including fever and malaise and should be easily recognized by the astute clinician and treated appropriately with culture and antibiotics, rather than injection therapy. The collagen vascular diseases will generally present as a polyarthropathy rather than a monoarthropathy limited to the elbow joint, although elbow pain secondary to collagen vascular disease responds exceedingly well to the intra-articular injection technique described below.

The majority of patients presenting with elbow pain secondary to osteoarthritis and post-traumatic arthritis pain will present with the complaint of pain that is localized around the elbow and forearm. Activity makes the pain worse, with rest and heat providing some relief. The pain is constant and characterized as aching in nature. The pain may interfere with sleep. Some patients will complain of a grating or popping sensation with use of the joint and crepitus may be present on physical exam.

In addition to the above mentioned pain, patients suffering from arthritis of the elbow joint will often experience a gradual decrease in functional ability with decreasing elbow range of motion, making simple everyday tasks such as using a computer keyboard, holding a coffee cup, or turning a doorknob overhead quite difficult. With continued disuse, muscle wasting may occur and an adhesive capsulitis with subsequent ankylosis may develop.

Plain radiographs are indicated in all patients who present with elbow pain. Based on the patient's clinical presentation, additional testing including complete blood count, sedimentation rate, and antinuclear antibody testing may be indicated. MRI scan of the elbow is indicated if joint instability is suspected.

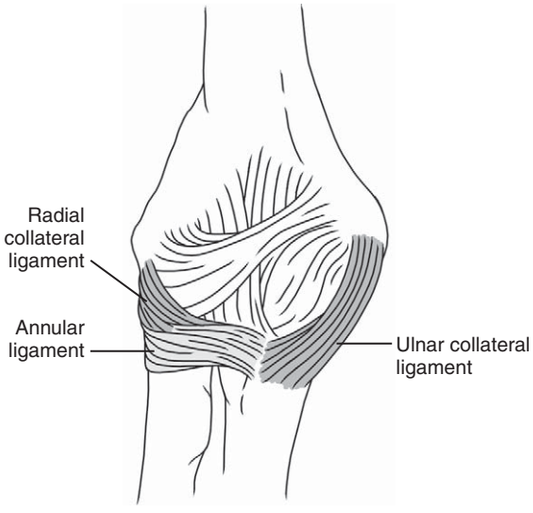


FIGURE 25-7
Joint capsule and ligaments of the elbow.

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represent a relative contraindication to the performance of intra-articular injection of the elbow, although the risk of hemarthrosis following intra-articular injection even in those patients with therapeutic INR levels is very low.[6] Local infection involving the area of the elbow is also a contraindication to the performance of intra-articular injection of the elbow.

EQUIPMENT

- Peripheral nerve block tray
- 22-gauge, 1-1/2-inch needle
- 18-gauge, 1-1/2-inch needle
- 12-ml sterile syringe

DRUGS

- 1% preservative-free lidocaine (for diagnostic or prognostic block)
- 0.25% preservative-free bupivacaine (for therapeutic block)
- Depot methylprednisolone (for therapeutic block)
- Water-soluble contrast suitable for intra-articular injection

PROCEDURE

The patient is placed in a supine position with the arm fully adducted at the patient's side and the elbow slightly flexed with the dorsum of the hand resting on a folded towel.[4] A total of 5 ml of local anesthetic and 40 mg of methylprednisolone are drawn up in a 12-ml sterile syringe.

After sterile preparation of skin overlying the posterolateral aspect of the joint, the head of the radius is identified. Just superior to the head of the radius is an indentation, which represents the space between the radial head and humerus. Using strict aseptic technique, a 1-inch, 25-gauge needle is inserted just above the superior aspect of the head of the radius through the skin, subcutaneous tissues, and joint capsule into the joint (Figure 25-8). If bone is encountered, the needle is withdrawn into the subcutaneous tissues and redirected superiorly. After entering the joint space, the contents of the syringe are gently injected. There should be little resistance to injection. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced slightly into the joint space until the injection proceeds without significant resistance. If there is any question that the needle tip is in fact intra-articular, a small amount of water-soluble contrast suitable for intra-articular injection may be added to the injectate (Figure 25-9). The needle is then removed, and a sterile pressure dressing and ice pack are placed at the injection site.

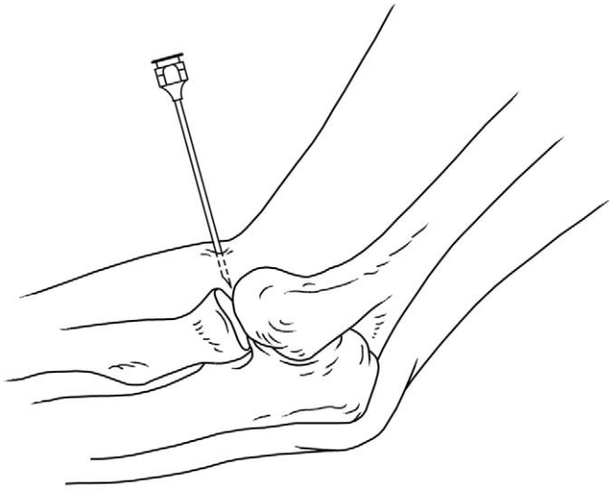


FIGURE 25-8
Needle placement for intra-articular injection of the elbow joint.

COMPLICATIONS

The major complication of intra-articular injection of the elbow is infection.[4] This complication should be exceedingly rare if strict aseptic technique is adhered to. As mentioned above, the ulnar nerve is especially susceptible to damage at the elbow. Approximately 25% of patients will complain of a transient increase in pain following intra-articular injection of the elbow joint and should be warned of such.

HELPFUL HINTS

This injection technique is extremely effective in the treatment of pain secondary to the above mentioned causes of arthritis of the elbow joint. Coexistent bursitis and tendonitis may also contribute to elbow pain and may require additional treatment with more localized injection of local anesthetic and depot steroid. This technique is a safe procedure if careful attention is paid to the clinically relevant anatomy in the areas to be injected. Care must be taken to use sterile technique to avoid infection, as well as the use of universal precautions to avoid risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is placed on the injection site immediately following injection. The use of physical modalities including local heat, as well as gentle range of motion exercises, should be introduced several days after the patient undergoes this injection technique for elbow pain. Vigorous exercises should be avoided as they will exacerbate the patient's symptomatology. Simple analgesics and nonsteroidal anti-inflammatory agents may be used concurrently with this injection technique.

THE WRIST

ANATOMY

The wrist joint is a biaxial ellipsoid-type joint that serves as the articulation between the distal end of the radius and the articular disc above and the scaphoid, lunate, and triquetrial bones below[4] (Figure 25-10). The joint's primary function is to optimize hand function. The joint allows flexion and extension, as well as abduction, adduction, and circumduction. The joint is lined with synovium, and the resultant synovial space allows intra-articular injection, although the septum within the synovial space may limit the flow of injectate. The entire joint is covered by a dense capsule that is attached above to the distal ends of the radius and ulna and below to the proximal row of metacarpal bones. The anterior and posterior

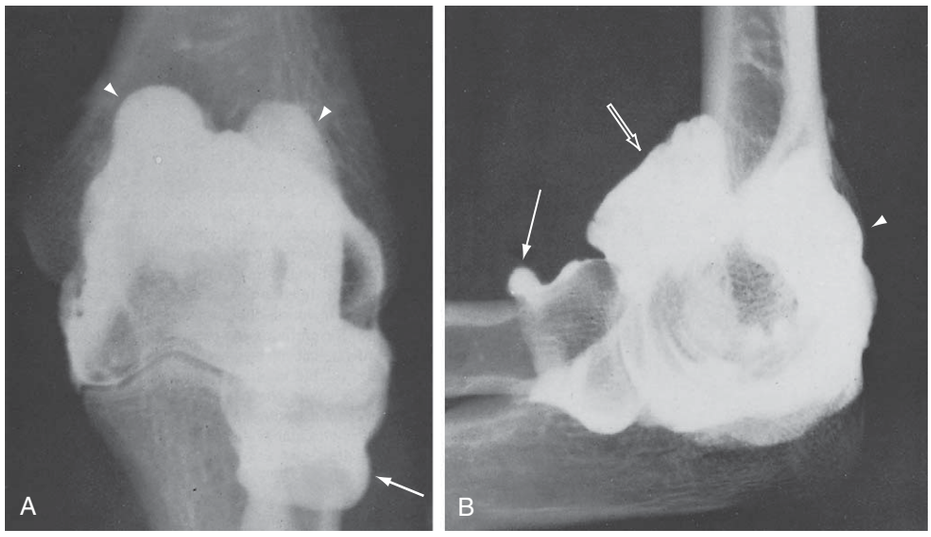


FIGURE 25-9
Elbow arthrography: normal arthrogram. (A) Anteroposterior radiograph. Observe the thin layer of contrast material between the humerus and ulna, the proximal extension of material in front of the humerus resembling the ears of a rabbit (arrowheads), and the periradial, or annular, recess (arrow). (B) Lateral radiograph. Note the periradial, or annular, recess (arrow), the coronoid, or anterior, recess (open arrow), and the olecranon, or posterior, recess (arrowhead). (From Resnick D: *Diagnosis of Bone and Joint Disorders*, 4th ed. Philadelphia, Saunders, 2002, p. 226.)

joint is strengthened by the anterior and posterior ligaments, with the medial and lateral ligaments strengthening the medial and lateral joint, respectively. The wrist joint may also become inflamed as a result of direct trauma or overuse of the joint.

The wrist joint is innervated primarily by the deep branch of the ulnar nerve, as well as the anterior and posterior interosseous nerves. Anteriorly, the wrist is bounded by the flexor tendons and the median and ulnar nerve. Posteriorly, the wrist is bounded by the extensor tendons. Laterally, the radial artery can be found. Medial to the joint runs the dorsal branch of the ulnar nerve, which is frequently damaged when the distal ulna is fractured.

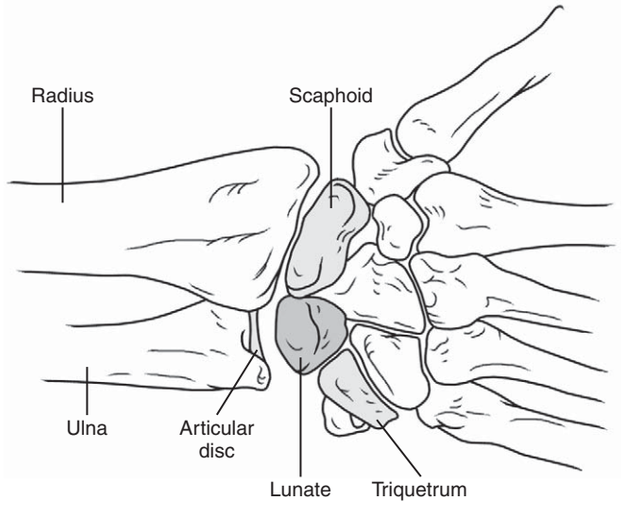


FIGURE 25-10
Bony anatomy of the wrist.

INDICATIONS

The wrist joint is susceptible to the development of arthritis from a variety of conditions, which have in common the ability to damage the joint cartilage. Osteoarthritis of the joint is the most common form of arthritis that results in wrist joint pain.[5] However, rheumatoid arthritis, post-traumatic arthritis, and psoriatic arthritis are also common causes of wrist pain secondary to arthritis. Less common causes of arthritis-induced wrist pain include the collagen vascular diseases, infection, and Lyme disease. Acute infectious arthritis will usually be accompanied by significant systemic symptoms including fever and malaise and should be easily recognized by the astute clinician and treated appropriately with culture and antibiotics, rather than injection therapy. The collagen vascular diseases will generally present as a polyarthropathy rather than a monoarthropathy limited to the wrist joint, although wrist pain secondary to collagen vascular disease responds exceedingly well to the intra-articular injection technique described below.

The majority of patients presenting with wrist pain secondary to osteoarthritis and post-traumatic arthritis pain will present with the complaint of pain, which is localized around the wrist and hand. Activity makes the pain worse, with rest and heat providing some relief. The pain is constant and characterized as aching in nature. The pain may interfere with sleep. Some patients will complain of a grating or popping sensation with use of the joint and crepitus may be present on physical exam.

In addition to the abovementioned pain, patients suffering from arthritis of the wrist joint will often experience a gradual decrease in functional ability with decreasing wrist range of motion making simple everyday tasks such as using a computer keyboard, holding a coffee cup, or turning a doorknob overhead quite difficult. With continued

disuse, muscle wasting may occur and an adhesive capsulitis with subsequent ankylosis may develop.

Plain radiographs are indicated in all patients who present with wrist pain. Based on the patient's clinical presentation, additional testing including complete blood count, sedimentation rate, and antinuclear antibody testing may be indicated. MRI scan of the wrist is indicated if joint instability is suspected.

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represent a contraindication to the performance of intra-articular injection of the wrist, although the risk of hemarthrosis following intra-articular injection, even in those patients with therapeutic INR levels, is very low.[6] Local infection involving the area of the wrist is also a contraindication to the performance of intra-articular injection of the wrist.

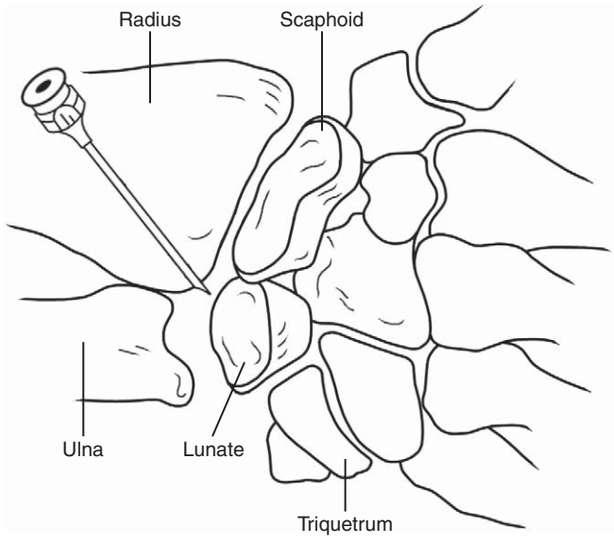


FIGURE 25-11
Needle placement for intra-articular injection of the wrist.

EQUIPMENT

- Peripheral nerve block tray
- 22-gauge, 1-1/2-inch needle
- 18-gauge, 1-1/2-inch needle
- 12-ml sterile syringe

DRUGS

- 1% preservative-free lidocaine (for diagnostic or prognostic block)
- 0.25% preservative-free bupivacaine (for therapeutic block)
- Depot methylprednisolone (for therapeutic block)
- Water-soluble contrast suitable for intra-articular injection

PROCEDURE

The patient is placed in a supine position with the arm fully adducted at the patient's side, and the elbow slightly flexed with the palm of the hand resting on a folded towel. A total of 1.5 ml of local anesthetic and 40 mg of methylprednisolone is drawn up in a 5-ml sterile syringe.

After sterile preparation of skin overlying the dorsal joint, the midcarpus proximal to the indentation of the capitate bone is identified. Just proximal to the capitate bone is an indentation, which allows easy access to the wrist joint. Using strict aseptic technique, a 1-1/2-inch, 25-gauge needle is inserted in the center of the midcarpal indentation through the skin, subcutaneous tissues, and joint capsule into the joint (Figure 25-11). If bone is encountered, the needle is withdrawn into the subcutaneous tissues and redirected superiorly. After entering the joint space, the contents of the syringe are gently injected. There should be little resistance to injection. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced slightly into the joint space until the injection proceeds without significant resistance. If there is any question that the needle tip is in fact intra-articular, a small amount of water-soluble contrast suitable for intra-articular injection may be added to the injectate (Figure 25-12). The needle is then removed, and a sterile pressure dressing and ice pack are placed at the injection site.

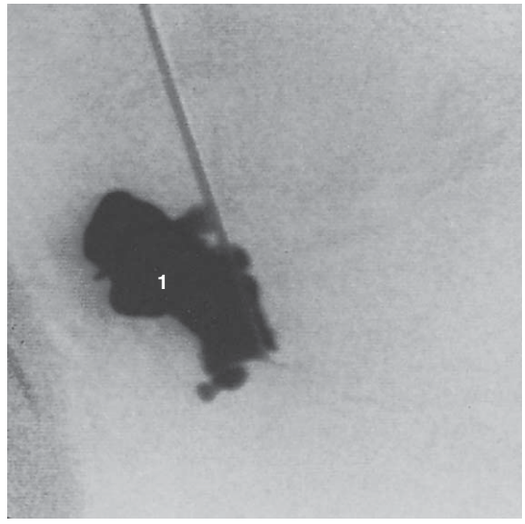


FIGURE 25-12
Wrist arthrography: digital technique—cadaver. Subtraction image is shown after the injection of 0.5 ml of the contrast agent into the radiocarpal joint. Slight irregularity of the contrast agent is seen in the radial aspect of the radiocarpal joint. (From Resnick D: *Diagnosis of Bone and Joint Disorders*, 4th ed. Philadelphia, Saunders, 2002, p. 201.)

COMPLICATIONS

The major complication of intra-articular injection of the wrist is infection.[4] This complication should be exceedingly rare if strict aseptic technique is adhered to.[7] As mentioned above, the ulnar nerve is especially susceptible to damage at the wrist. Approximately 25% of patients will complain of a transient increase in pain following intra-articular injection of the wrist joint and should be warned of such.

CLINICAL PEARLS

This injection technique is extremely effective in the treatment of pain secondary to the abovementioned causes of arthritis of the wrist joint. Coexistent bursitis and tendonitis may also contribute to wrist pain and may require additional treatment with more localized injection of local anesthetic and depot steroid. This technique is a safe procedure if careful attention is paid to the clinically relevant anatomy in the areas to be injected. Care must be taken to use sterile technique to avoid infection, as well as the use of universal precautions to avoid risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is placed on the injection site immediately following injection. The use of physical modalities including local heat, as well as gentle range of motion exercises, should be introduced several days after the patient undergoes this injection technique for wrist pain. Vigorous exercises should be avoided as they will exacerbate the patient's symptomatology. Simple analgesics and nonsteroidal anti-inflammatory agents may be used concurrently with this injection technique.

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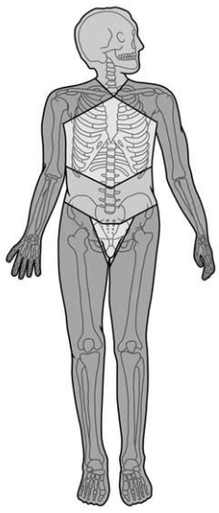
C H A P T E R



26

Somatic Blocks of the Lower Extremity

STEVEN D. WALDMAN, LEE ANN PARADISE, AND P. PRITHVI RAJ

HISTORY

The history of the use of regional anesthesia for blockade of the nerves of the lower extremity closely follows the evolution of regional anesthesia. From Koller's use of cocaine as a topical anesthetic in the summer of 1884 to the recent development of modern long-acting amide-type local anesthetics such as bupivacaine, regional anesthesia for lower extremity somatic nerve block has played an important role in the development of lower extremity surgery. In fact, shortly after Halsted and Hall introduced the concept of the nerve block in December of 1884, Stillman reported the repair of a congenital foot abnormality under tibial nerve block performed by Halstead. This landmark surgery was performed on April 30, 1885, and it forever changed the widespread belief that "etherization" with all of its attendant risks was the only option to avoid the pain of surgery. The ensuing years witnessed an explosion in research on the use of nerve block to provide anesthesia for lower extremity surgery. Descriptions of new techniques abounded, as did reports of techniques to prolong the rather evanescent anesthetic effects of cocaine, including Braun's landmark description of the addition of epinephrine to cocaine to produce a "chemical tourniquet" to prolong the duration of cocaine. One must surmise that this search for better ways to render the lower extremity insensate would have continued unabated but for the rapid acceptance of spinal anesthesia by Corning in the years following its introduction in 1885. By 1900, spinal anesthesia had gained wide acceptance and had supplanted general anesthesia and regional blocks as the preferred form of anesthesia for the lower extremity due to its ease of performance and reliability. Many surgical and later anesthesia training programs dropped regional nerve blocks of the lower extremity from their curricula in favor of spinal anesthesia. Mirroring the waning interest in regional anesthesia in general in both the operating room and obstetrical arenas, it was the introduction of the safer amide local anesthetics and in particular the introduction of the longer-acting amide-type drugs such as bupivacaine that led to a renewed interest in regional blockade of the somatic nerves of the lower extremity, not for surgical anesthesia, but for use in postoperative pain relief. Today, the blocks described below represent the standard of care for postoperative pain relief in most busy surgical centers and hospitals.

LUMBAR PLEXUS NERVE BLOCK: WINNIE 3-IN-1 TECHNIQUE

ANATOMY

The lumbar plexus lies within the substance of the psoas muscle. The plexus is made up of the ventral roots of the first four lumbar nerves, and in some patients, a contribution from the 12th thoracic nerve (Figure 26-1). The nerves lie in front of the transverse processes of their respective vertebrae; as they course inferolaterally, they divide into a number of peripheral nerves.[1] The ilioinguinal and iliohypogastric nerves are branches of the L1 nerves with an occasional contribution of fibers from T12. The genitofemoral nerve is made up of fibers from L1 and L2. The lateral femoral cutaneous nerve is derived from fibers of L2 and L3. The obturator nerve receives fibers from L2-L4, and the femoral nerve is made up of fibers from L2-L4. The pain management specialist should be aware of the considerable interpatient variability in terms of the actual spinal nerves that provide fibers to make up these peripheral branches. This variability means that differential neural blockade on an anatomic basis must be interpreted with caution. Because these nerves pass anteriorly beneath the inguinal ligament, they are accessible to blockade via this technique.

The rationale behind lumbar plexus block using the Winnie 3-in-1 technique is to block the three principle nerves that compose the lumbar plexus as they lie enclosed

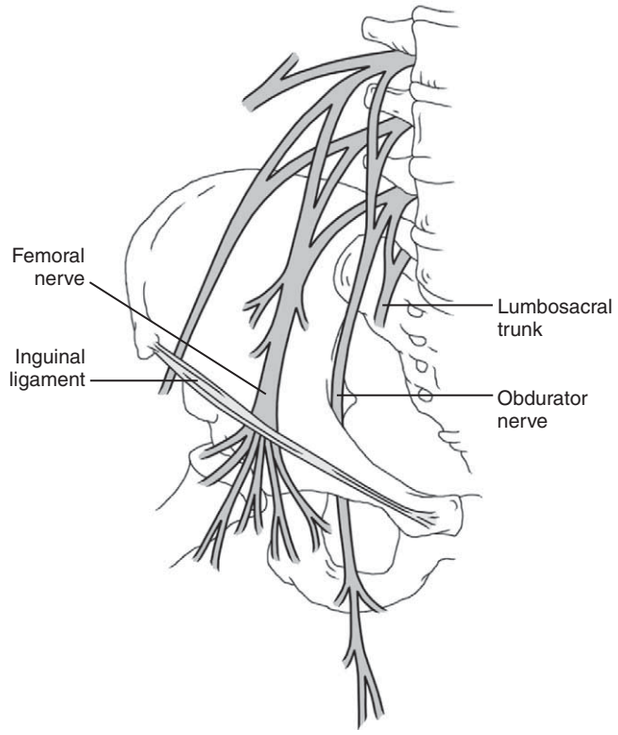


FIGURE 26–1

This drawing shows the formation of the lumbar plexus and its course from the plexus to its branches, distal to the inguinal ligament. Note the course of the femoral nerve and the obturator nerve as it exits from the pelvis to the groin.

by the fascial plane between the quadratus lumborum, the iliacus muscle, and the psoas major muscle.[1] Solutions injected in this fascial plane flow cranially to bathe the lateral femoral cutaneous nerve, the femoral nerve, and the obturator nerve as they pass below the inguinal ligament.

INDICATIONS

The Winnie 3-in-1 approach to lumbar plexus block has the advantage over the psoas compartment technique in that it is amenable to continuous infusion of local anesthetic by placement of either an 18-gauge intravenous catheter or an over-the-wire central venous catheter into the fascial plane. Lumbar plexus nerve block via the Winnie 3-in-1 technique is used primarily for surgical anesthesia of the lower extremity. It is occasionally used in the area of pain management when treating pain secondary to inflammatory conditions of the lumbar plexus or when tumor has invaded the tissues subserved by the lumbar plexus or the plexus itself. Lumbar plexus nerve block via the Winnie 3-in-1 technique with local anesthetic is occasionally used diagnostically during differential neural blockade on an anatomic basis in the evaluation of lower extremity and groin pain. If destruction of the lumbar plexus is being considered, this technique is useful as a prognostic indicator of the degree of motor and sensory impairment that the patient may experience.

Lumbar plexus nerve block via the Winnie 3-in-1 technique with local anesthetic may be used to palliate acute pain emergencies, including groin and lower extremity trauma or fracture, acute herpes zoster, and cancer pain, while waiting for pharmacologic, surgical, and antiblastic therapies to become effective. Lumbar plexus nerve block via the Winnie 3-in-1 technique with local anesthetic and steroid is also useful in the treatment of lumbar plexitis secondary to virus or diabetes. For most surgical and pain management applications, epidural or subarachnoid block is a better alternative, although one should expect fewer cardiovascular changes with lumbar plexus block compared with epidural or subarachnoid techniques. Destruction of the lumbar plexus is indicated for the palliation of cancer pain, including invasive tumors of the lumbar plexus and the tissues that the plexus innervates. More selective techniques such as radiofrequency lesioning of specific lumbar paravertebral nerve roots may cause less morbidity than lumbar plexus neurolysis.

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represents a strong contraindication to the performance of lumbar plexus block. Local infection involving the area of the lumbar plexus is also a contraindication to the performance of lumbar plexus block.

EQUIPMENT

- Peripheral nerve block tray
- 22-gauge, 1-1/2-inch needle
- 25-gauge, 1-1/2-inch needle
- 18-gauge, 1-1/2-inch needle
- 12-ml sterile syringe

DRUGS

- 1% preservative-free lidocaine (for diagnostic or prognostic block)
- 0.25% preservative-free bupivacaine (for therapeutic block)
- Depot methylprednisolone (for therapeutic block)
- 6.5% aqueous phenol (for chemical neurolytic block)

PROCEDURE

The patient is placed in the supine position. The inguinal ligament and the femoral artery on the side to be blocked are identified. At a point just lateral to the femoral artery and just below the inguinal ligament, the skin is prepared with antiseptic solution. A 22-gauge, 1-1/2-inch needle is slowly advanced in a slightly caudad direction until a paresthesia in the distribution of the femoral nerve is elicited (Figure 26-2).

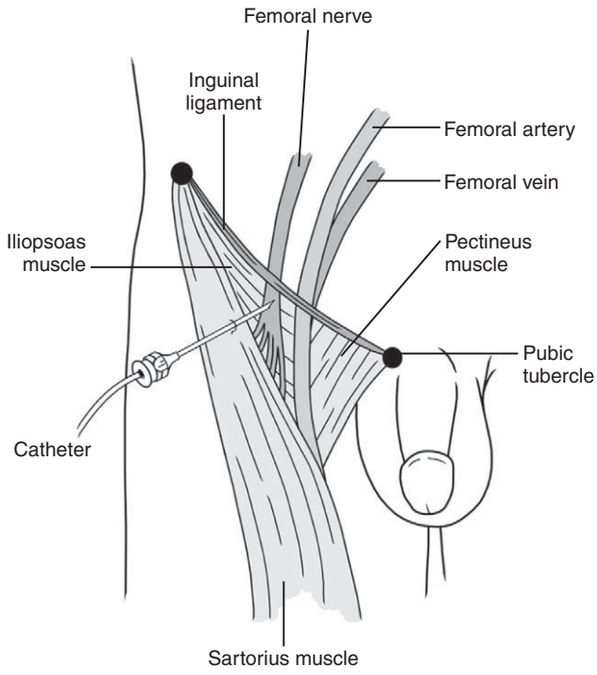


FIGURE 26–2

This drawing shows the site of catheter placement on the femoral nerve just inferior to the inguinal ligament. Note the femoral artery, which runs medial to the femoral nerve.

The patient should be warned of such and instructed to say "there!" immediately when perceiving the paresthesia. If there is no persistent paresthesia in the distribution of the femoral nerve and careful aspiration reveals no blood or cerebrospinal fluid, 25–30 ml of 1.0% preservative-free lidocaine is slowly injected in incremental doses, with care taken to observe the patient for signs of local anesthetic toxicity. Water-soluble contrast medium may be added to the local anesthetic to confirm appropriate needle placement. Pressure should be applied below the needle to force the solution to flow cranially along the fascial plane rather than distally into the leg. If the pain has an inflammatory component, the local anesthetic is combined with 80 mg of methylprednisolone and is injected in incremental doses. Subsequent daily nerve blocks are carried out in a similar manner, substituting 40 mg of methylprednisolone for the initial 80-mg dose. As mentioned earlier, an intravenous catheter can be placed into the fascial sheath to allow continuous infusion of local anesthetic.

COMPLICATIONS

The proximity to the femoral artery and vein makes the possibility of local anesthetic toxicity real. Persistent paresthesia secondary to trauma to the femoral nerve has rarely been reported after this technique. Although uncommon, infection remains an ever-present possibility, especially in the immunocompromised cancer patient. Early detection of infection is crucial to avoid potentially life-threatening sequelae. Post-block groin and back pain, as well as ecchymosis and hematoma of the groin, occur often enough that the patient should be warned of such prior to beginning lumbar plexus block using the Winnie 3-in-1 technique.

CLINICAL PEARLS

Lumbar plexus nerve block via the Winnie 3-in-1 technique is a simple technique for performing lumbar plexus block. It has the advantage over the psoas compartment approach in that it allows easy catheter placement for continuous infusions of local anesthetic. Unfortunately, most of the things that can be done with lumbar plexus block can be done more easily with epidural or spinal techniques, which may be more acceptable to the surgeon and pain specialist alike. Neurolytic block with small quantities of phenol in glycerin or with absolute alcohol has been shown to provide long-term relief for patients suffering from cancer-related pain in whom more conservative treatments have been ineffectual. As mentioned earlier, the proximity of the femoral artery and vein makes careful attention to technique mandatory.

The pain specialist should carefully examine the patient prior to performing lumbar plexus block using the Winnie 3-in-1 technique to identify pre-existing neural compromise that might subsequently be erroneously attributed to the block.

OBTURATOR NERVE BLOCK

ANATOMY

The obturator nerve provides the majority of innervation to the hip joint. It is derived from the posterior divisions of the L2, L3, and L4 nerves.[1] The nerve leaves the medial border psoas muscle and courses inferiorly to pass the pelvis, where it joins the obturator vessels to travel via the obturator canal to enter the thigh (Figure 26-3). The nerve then divides into anterior and posterior branches. The anterior branch supplies an articular branch to provide sensory innervation to the hip joint, motor branches to the superficial hip adductors, and a cutaneous branch to the medial aspect of the distal thigh (Figure 26-4). The posterior branch provides motor innervation to the deep hip adductors and an articular branch to the posterior knee joint.

INDICATIONS

Obturator nerve block is useful in the evaluation and management of hip pain and spasm of the hip adductors thought to be subserved by the obturator nerve. The technique is also useful to provide surgical anesthesia for the lower extremity when combined with lateral femoral

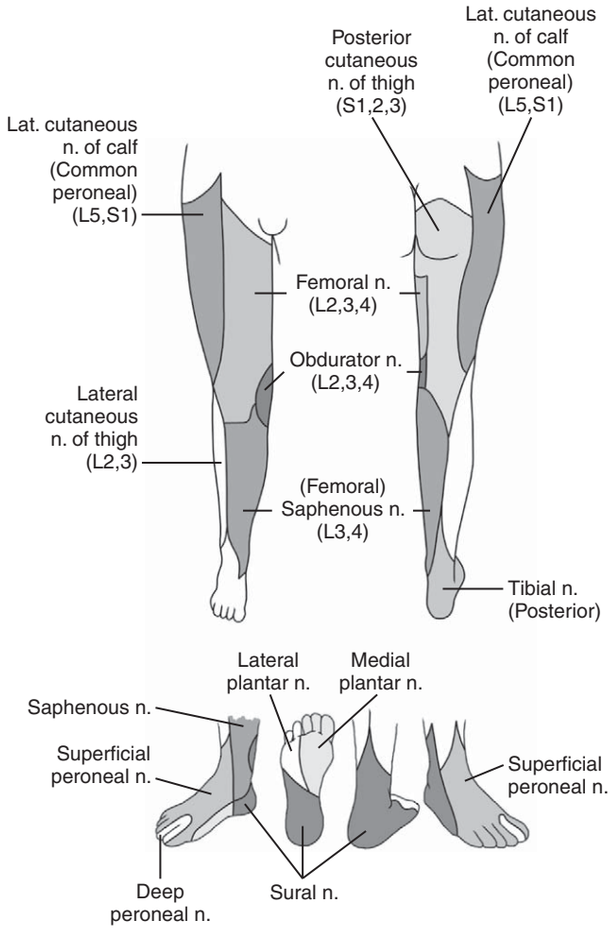


FIGURE 26–3

In this figure the dermatomal pattern of innervation of the femoral nerve, obturator nerve, lateral cutaneous nerve, and posterior cutaneous nerve of the thigh are shown. Note the roots from which they are formed.

cutaneous, femoral, and sciatic nerve block. Obturator nerve block with local anesthetic can be used as a diagnostic tool during differential neural blockade on an anatomic basis in the evaluation of hip pain. If destruction of the obturator nerve is being considered, this technique is useful as a prognostic indicator of the degree of motor and sensory impairment that the patient may experience. Obturator nerve block with local anesthetic may be used to palliate acute pain emergencies, including postoperative pain relief, while waiting for pharmacologic methods to become effective. Obturator nerve block with local anesthetic is also useful in the management of hip adductor spasm, which may make perineal care or urinary catheterization difficult. Obturator nerve block with local anesthetic and steroid is also useful in the treatment of persistent hip pain when the pain is thought to be secondary to inflammation or entrapment of the obturator nerve. Destruction of the obturator nerve is occasionally indicated for the palliation of persistent hip pain after

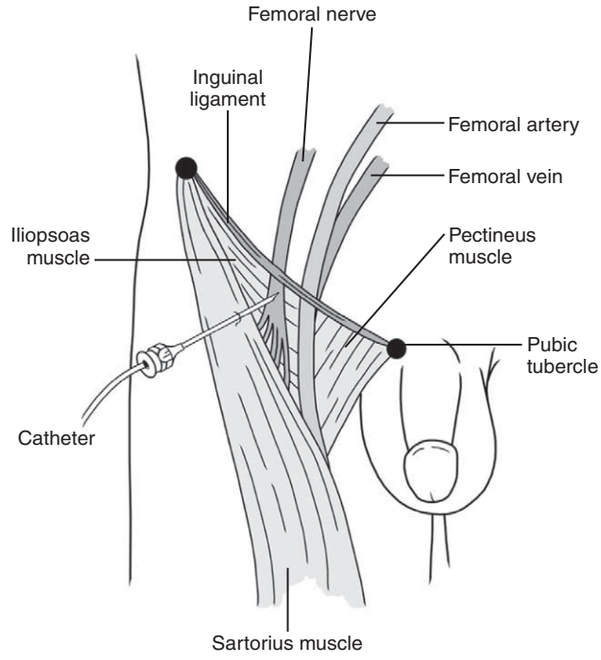


FIGURE 26–4

Step one: a 22-gauge, 3-1/2 inch needle is slowly advanced perpendicular to the skin until the needle is felt to impinge on the superior pubic ramus. Note the branches of the obturator nerve as they exit from the obturator foramen.

trauma to the hip that is mediated by the obturator nerve.

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represents a strong contraindication to the performance of obturator nerve block. Local infection involving the area of the obturator nerve block is also a contraindication to the performance of obturator nerve block.

EQUIPMENT

- Peripheral nerve block tray
- 22-gauge, 3-1/2-inch styletted needle
- 25-gauge, 1-1/2-inch needle
- 18-gauge, 1-1/2-inch needle
- 12-ml sterile syringe

DRUGS

- 1% preservative-free lidocaine (for diagnostic or prognostic block)
- 0.25% preservative-free bupivacaine (for therapeutic block)
- Depot methylprednisolone (for therapeutic block)
- 6.5% aqueous phenol (for chemical neurolytic block)

PROCEDURE

The patient is placed in the supine position with the legs slightly abducted. The pubic tubercle on the involved side is identified by palpation. A point 1 inch lateral and 1 inch inferior to the pubic tubercle is then identified and prepared with antiseptic solution. A 22-gauge, 3-1/2-inch needle is then slowly advanced perpendicular to the skin until the needle is felt to impinge on the superior pubic ramus (see Figure 26-4). The depth of bony contact is noted, and the needle is withdrawn and redirected laterally and slightly inferiorly (Figure 26-5). The needle is advanced approximately 3/4–1 inch deeper to place the needle tip in the obturator canal. A paresthesia in the distribution of the obturator nerve may be elicited. After careful aspiration, 10–15 ml of 1.0% preservative-free lidocaine are injected. Water-soluble contrast medium may be added to the local anesthetic to confirm appropriate needle placement. Care must be taken not to place the needle in the obturator artery or vein.

If the pain has an inflammatory component, the local anesthetic is combined with 80 mg of methylprednisolone and is injected in incremental doses. Subsequent daily nerve blocks are carried out in a similar manner, substituting 40 mg of methylprednisolone for the initial 80-mg dose. After injection of the solution, pressure is applied to the injection site to decrease the incidence of postblock ecchymosis and hematoma formation.

COMPLICATIONS

The main side effect of obturator nerve block is postblock ecchymosis and hematoma. Because of proximity to the obturator artery and vein, intravascular injection remains an ever-present possibility. As mentioned earlier, pressure should be maintained on the injection site postblock to avoid ecchymosis and hematoma formation. Infection in the area of the obturator nerve represents a contraindication to obturator nerve block.

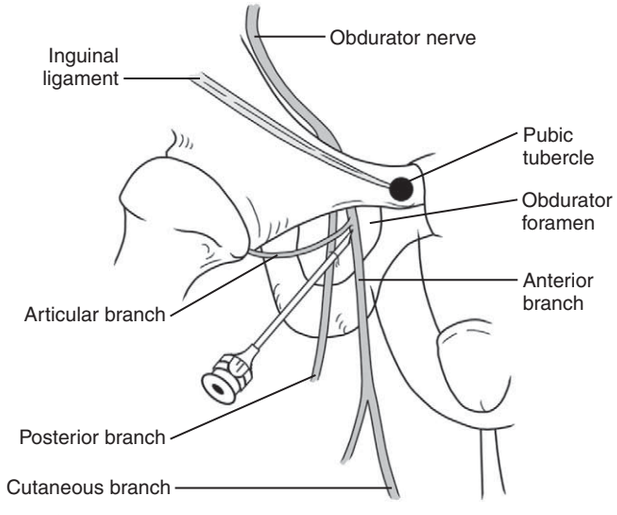


FIGURE 26–5

Step two: the depth of bony contact is noted, and the needle is withdrawn and redirected laterally and slightly inferiorly to touch the obturator nerve as it enters the obturator foramen.

CLINICAL PEARLS

Obturator nerve block is a simple technique that can produce dramatic relief for patients suffering from the previously mentioned pain complaints. Neurolytic block with small quantities of phenol in glycerin or by cryoneurolysis or radiofrequency lesioning has been shown to provide long-term relief for patients suffering from chronic pain secondary to trauma or tumor involving the hip joint in whom more conservative treatments have been ineffectual. Destruction of the obturator nerve is also useful in the palliation of hip adductor spasm after spinal cord injury or stroke that limits the ability to provide perineal care or allow sexual intercourse or urinary catheterization. Botulinum toxin may have an application for this indication.

If a patient presents with pain that is thought to be mediated via the obturator nerve and obturator nerve blocks are ineffectual, a diagnosis of lesions more proximal in the lumbar plexus or L2-L3-L4 radiculopathy should be considered. Such patients often respond to epidural steroid blocks. Electromyography and magnetic resonance imaging of the lumbar plexus are indicated in this patient population to help rule out other causes of hip pain, including malignancy invading the lumbar plexus or epidural or vertebral metastatic disease at L2-L3-L4. Plain radiographs of the hip should also be obtained to rule out local pathology.

FEMORAL NERVE BLOCK

ANATOMY

The femoral nerve innervates the anterior portion of the thigh and medial calf. The femoral nerve is derived from the posterior branches of the L2, L3, and L4 nerve roots.[1] The roots fuse together in the psoas muscle and descend laterally between the psoas and iliacus muscles to enter the iliac fossa. The femoral nerve gives off motor fibers to the iliac muscle and then passes beneath the inguinal ligament to enter the thigh (Figure 26-6). The femoral nerve is just lateral to the femoral artery as it passes beneath the inguinal ligament and is enclosed with the femoral artery and vein within the femoral sheath. The nerve gives off motor fibers to the sartorius, quadriceps femoris, and pectineus muscles. It also provides sensory fibers to the knee joint, as well as the skin overlying the anterior thigh (see Figure 26-3). The nerve is easily blocked as it passes through the femoral triangle.

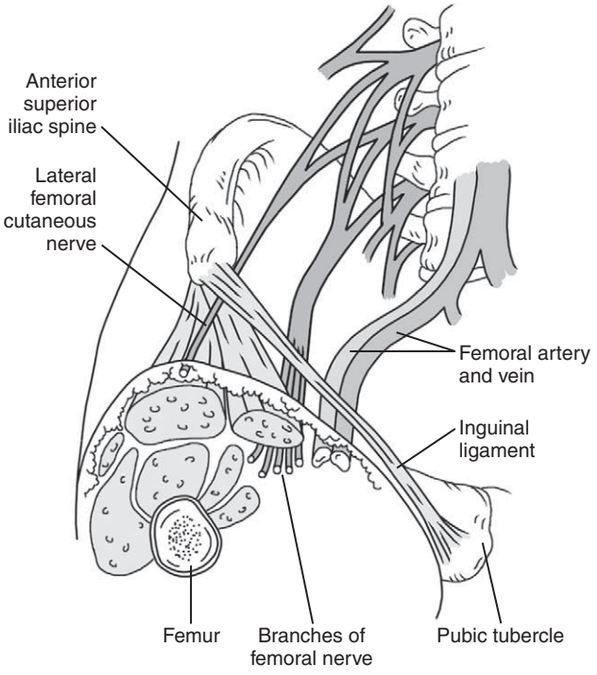


FIGURE 26–6

In this figure, the femoral nerve gives off motor fibers to the iliac muscle and then passes beneath the inguinal ligament to enter the thigh. The femoral nerve is just lateral to the femoral artery as it passes beneath the inguinal ligament and is enclosed with the femoral artery and vein within the femoral sheath.

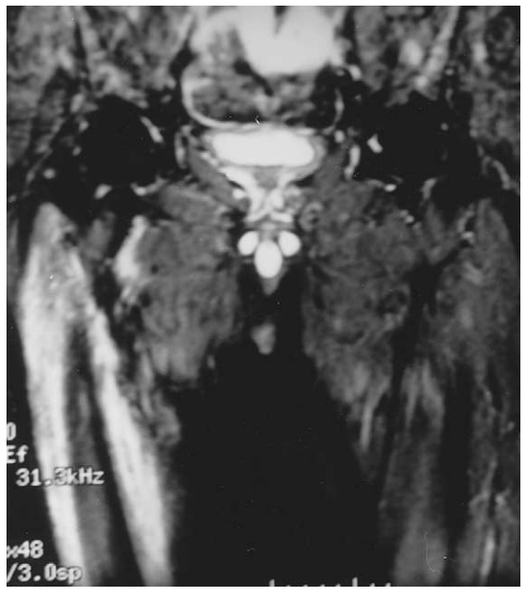


FIGURE 26–7

Involvement of the femoral nerve: injury. The right femoral nerve was injured during cardiac catheterization complicated by a retroperitoneal hematoma. A coronal STIR (TR/TE, 4000/22; inversion time, 165 milliseconds) magnetic resonance image demonstrates high signal intensity in portions of the quadriceps musculature (especially the vastus lateralis muscle) and in some of the adductor muscles as a result of denervation. (From Resnick D: *Diagnosis of Bone and Joint Disorders*, 4th ed. Philadelphia, Saunders, 2002, p. 3548.)

INDICATIONS

Femoral nerve block is useful in the evaluation and management of lower extremity pain thought to be subserved by the femoral nerve. The technique is also useful to provide surgical anesthesia for the lower extremity when combined with lateral femoral cutaneous, sciatic, and obturator nerve block or lumbar plexus block. It is used for this indication primarily in patients who would not tolerate the sympathetic changes induced by spinal or epidural anesthesia and who need lower extremity surgery. Femoral nerve block with local anesthetic can be used diagnostically during differential neural blockade on an anatomic basis in the evaluation of lower extremity pain. If destruction of the femoral nerve is being considered, this technique is useful as a prognostic indicator of the degree of motor and sensory impairment that the patient may experience. Femoral nerve block with local anesthetic may be used to palliate acute pain emergencies, including femoral neck and shaft fractures, and for postoperative pain relief while waiting for pharmacologic methods to become effective. Femoral nerve block with local anesthetic and steroid is occasionally used in the treatment of persistent lower extremity pain when the pain is thought to be secondary to inflammation or when entrapment of the femoral nerve as it passes under the inguinal ligament is suspected (Figure 26-7). Femoral nerve block with local anesthetic and steroid is also indicated in the palliation of pain and motor dysfunction associated with diabetic femoral neuropathy. Destruction of the femoral nerve is occasionally used in the palliation of persistent lower extremity pain secondary to invasive tumor that is mediated by the femoral nerve and has not responded to more conservative measures.

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represents a contraindication to the performance of a femoral nerve block. Local infection involving the area of the femoral nerve block is also a contraindication to the performance of femoral nerve block.

EQUIPMENT

- Peripheral nerve block tray
- 22-gauge, 3-1/2-inch styletted needle
- 25-gauge, 1-1/2-inch needle
- 18-gauge, 1-1/2-inch needle
- 12-ml sterile syringe

DRUGS

- 1% preservative-free lidocaine (for diagnostic or prognostic block)
- 0.25% preservative-free bupivacaine (for therapeutic block)

- Depot methylprednisolone (for therapeutic block)
- 6.5% aqueous phenol (for chemical neurolytic block)

PROCEDURE

The patient is placed in the supine position with the leg in neutral position. The femoral artery is identified just below the inguinal ligament by palpation. A point just lateral to the pulsations of the femoral artery and just inferior to the inguinal ligament is then identified and prepared with antiseptic solution. A 25-gauge, 1-1/2-inch needle is then advanced at this point slowly with a cephalad trajectory until a paresthesia in the distribution of the femoral nerve is elicited (Figure 26-8). The patient should be warned to expect such and should be told to say "there!" immediately when perceiving the paresthesia. Paresthesia usually is elicited at a depth of 1/2–3/4 inch. If paresthesia is not elicited, the needle is withdrawn and redirected slightly more medially until paresthesia is obtained. Once paresthesia in the distribution of the femoral nerve is elicited, the needle is withdrawn 1 mm and the patient is observed to be sure he or she is not experiencing any persistent paresthesia. If no persistent paresthesia is present and after careful aspiration, 15–18 ml of 1.0% preservative-free lidocaine are slowly injected. Water-soluble contrast medium may be added to the local anesthetic to confirm appropriate needle placement. Care must be taken not to advance the needle into the substance of the nerve during the injection and inject solution intraneurally.

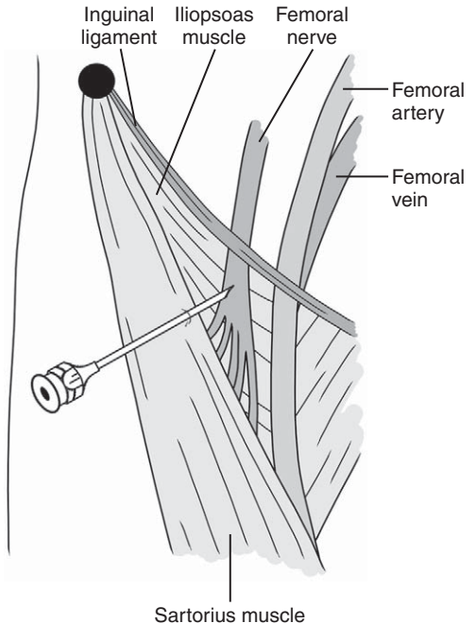


FIGURE 26–8

25-gauge, 1-1/2-inch needle is advanced at this point slowly with a cephalad trajectory until a paresthesia in the distribution of the femoral nerve is elicited.

If the pain has an inflammatory component, the local anesthetic is combined with 80 mg of methylprednisolone and is injected in incremental doses. Subsequent daily nerve blocks are performed in a similar manner, substituting 40 mg of methylprednisolone for the initial 80-mg dose. After injection of the solution, pressure is applied to the injection site to decrease the incidence of postblock ecchymosis and hematoma formation.

COMPLICATIONS

The main side effect of femoral nerve block is postblock ecchymosis and hematoma. As mentioned earlier, pressure should be maintained on the injection site post block to avoid ecchymosis and hematoma formation. Because paresthesia is elicited with this technique, needle-induced trauma to the femoral nerve remains a possibility. By advancing the needle slowly and then withdrawing the needle slightly away from the nerve, needle-induced trauma to the femoral nerve can be avoided.

CLINICAL PEARLS

Femoral nerve block is a simple technique that can produce dramatic relief for patients suffering from the mentioned pain complaints. This technique is especially useful in the emergency department to provide rapid relief for those patients suffering from fractures of the femoral neck and shaft. Careful preblock neurologic assessment is important to avoid later attribution of pre-existing neurologic deficits to the femoral nerve block. These assessments are especially important in patients who have sustained trauma to the pelvis or lower extremity or who suffer from diabetic femoral neuropathy in whom femoral nerve blocks are being used for acute pain control.

It should be remembered that the most common cause of pain radiating into the lower extremity is a herniated lumbar disc or nerve impingement secondary to degenerative arthritis of the spine, not disorders involving the femoral nerve per se. Electromyography and magnetic resonance imaging of the lumbar spine, combined with the clinical history and physical examination, help sort out the etiology of femoral pain.

SAPHENOUS NERVE BLOCK AT THE KNEE

ANATOMY

The saphenous nerve is the largest sensory branch of the femoral nerve. The saphenous nerve provides sensory innervation to the medial malleolus, the medial calf, and a portion of the medial arch of the foot[1] (see Figure 26-3). The saphenous nerve is derived primarily from the fibers of the L3 and L4 nerve roots. The nerve travels along with the femoral artery through Hunter's canal and moves

superficially as it approaches the knee. It passes over the medial condyle of the femur, splitting into terminal sensory branches (Figure 26-9). The saphenous nerve is subject to trauma or compression anywhere along its course. The nerve is frequently traumatized during vein harvest procedures for coronary artery bypass grafting procedures. The saphenous nerve is also subject to compression as it passes over the medial condyle of the femur.

INDICATIONS

Saphenous nerve block at the knee is useful in the evaluation and management of distal lower extremity pain thought to be subserved by the saphenous nerve. The technique is also useful to provide surgical anesthesia for the distal lower extremity when combined with tibial and common peroneal nerve block or lumbar plexus block. It is used for this indication primarily in patients who would not tolerate the sympathetic changes induced by spinal or epidural anesthesia who need distal lower extremity surgery, such as debridement or distal amputation. Saphenous nerve block at the knee with local anesthetic can be used diagnostically during differential neural blockade on an anatomic basis in the evaluation of lower extremity pain. If destruction of the saphenous nerve is being considered, this technique is useful as a prognostic indicator of the degree of motor and sensory impairment that the patient may experience. Saphenous nerve block at the knee with local anesthetic may be used to palliate acute pain emergencies, including distal lower extremity fractures and postoperative pain relief, when combined with the previously mentioned blocks while waiting for pharmacologic methods to become effective. Saphenous nerve block at the knee with local anesthetic and steroid is occasionally used in the treatment of persistent distal lower extremity pain when the pain is thought to be secondary to inflammation or when entrapment of the saphenous nerve as it passes through Hunter's canal is suspected. Saphenous nerve block at the knee with local anesthetic and steroid is also indicated in the palliation of pain and motor dysfunction associated with diabetic neuropathy. Destruction of the saphenous nerve is occasionally used in the palliation of persistent lower extremity pain secondary to invasive tumor that is mediated by the saphenous nerve and has not responded to more conservative measures.

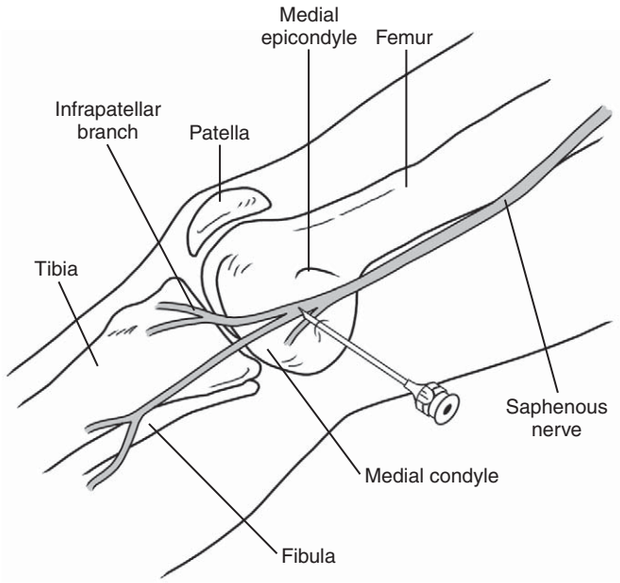


FIGURE 26–9

Course of the saphenous nerve is shown as it passes over the medial condyle of the femur, splitting into terminal sensory branches. Note the site of injection at the medial aspect of the knee joint.

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represents a contraindication to the performance of a saphenous nerve block. Local infection involving the area of the saphenous nerve block is also a contraindication to the performance of saphenous nerve block.

EQUIPMENT

- Peripheral nerve block tray
- 25-gauge, 1/2-inch needle
- 18-gauge, 1-1/2-inch needle
- 12-ml sterile syringe

DRUGS

- 1% preservative-free lidocaine (for diagnostic or prognostic block)
- 0.25% preservative-free bupivacaine (for therapeutic block)
- Depot methylprednisolone (for therapeutic block)
- 6.5% aqueous phenol (for chemical neurolytic block)

PROCEDURE

The patient is placed in the lateral position with the leg slightly flexed. The medial condyle of the femur is palpated. A point just in front of the posterior edge of the medial condyle is then identified and prepared with antiseptic solution. A 25-gauge, 1/2-inch needle is then slowly advanced through this point toward the medial condyle of the femur until paresthesia is elicited in the distribution of the saphenous nerve (see Figure 26-9). The patient should be warned to expect a paresthesia and should be told to say "there!" immediately when perceiving the paresthesia. Paresthesia usually is elicited at a depth of 1/4–1/2 inch. If a paresthesia is not elicited, the needle is withdrawn and

redirected slightly more anteriorly until a paresthesia is obtained. Once paresthesia is elicited in the distribution of the saphenous nerve, the needle is withdrawn 1 mm and the patient is observed to rule out any persistent paresthesia. If no persistent paresthesia is present and after careful aspiration, 5 ml of 1.0% preservative-free lidocaine are slowly injected. Water-soluble contrast medium may be added to the local anesthetic to confirm appropriate needle placement. Care must be taken not to advance the needle into the substance of the nerve during the injection and inject solution intraneurally.

If the pain has an inflammatory component, the local anesthetic is combined with 80 mg of methylprednisolone and is injected in incremental doses. Subsequent daily nerve blocks are carried out in a similar manner, substituting 40 mg of methylprednisolone for the initial 80-mg dose. After injection of the solution, pressure is applied to the injection site to decrease the incidence of postblock ecchymosis and hematoma formation.

COMPLICATIONS

The main side effect of saphenous nerve block at the knee is postblock ecchymosis and hematoma because the nerve is close to the greater saphenous artery. As mentioned earlier, pressure should be maintained on the injection site post block to avoid ecchymosis and hematoma formation. Because this technique elicits a paresthesia, needle-induced trauma to the saphenous nerve remains possible. By advancing the needle slowly and withdrawing the needle slightly away from the nerve prior to injection, one can avoid needle-induced trauma to the saphenous nerve.

CLINICAL PEARLS

Saphenous nerve block at the knee is a simple technique that can produce dramatic relief for patients suffering from the previously mentioned pain complaints. Careful preblock neurologic assessment is important to avoid the later attribution of pre-existing neurologic deficits to the saphenous nerve block at the knee. These assessments are especially important in patients who have sustained trauma to the distal femur, patients who have undergone vascular procedures on the lower extremity, or patients who suffer from diabetic neuropathy in whom saphenous nerve block at the knees is being used for acute pain control. Compressive neuropathy of the saphenous nerve at the knee sometimes occurs in musicians who play the cello. This painful syndrome is called viol paresthesia.

It should be remembered that the most common cause of pain radiating into the lower extremity is herniated lumbar disc or nerve impingement secondary to degenerative arthritis of the spine, not disorders involving the saphenous nerve per se. Other pain syndromes that may be confused with saphenous nerve entrapment include lesions either above the origin of the saphenous nerve, such as lesions of the femoral nerve, or lesions of the saphenous nerve at the ankle. Electromyography and magnetic resonance imaging of the lumbar spine, combined with the clinical history and physical examination, help to sort out the etiology of distal lower pain.

SCIATIC NERVE BLOCK

HISTORY

Labat wrote the first description of the posterior approach to the sciatic nerve in 1923.[2] The lateral approach was described by Molesworth[3] in 1944 and developed by Lchiyanagi[4] in 1959. The anterior approach was described by Beck in 1963.[5] The supine approach was described by Raj and associates[6] in 1975.

ANATOMY

The sciatic nerve (L4-L5, S1-S3), the largest nerve in the body, measures 1.5–2 cm in width and 0.3–0.9 cm in diameter as it leaves the pelvis and passes through a tunnel between the greater trochanter and the ischial tuberosity. The greater sciatic nerve then passes posterior to the gemmules, obturator internus, and quadriceps femoris muscles and anterior to the gluteus maximus muscles (Figure 26-10).

The posterior femoral cutaneous branch (S1-S3) innervates the posterior aspect of the thigh. Blood vessels accompanying the sciatic nerve at this point are the sciatic artery, a branch of the inferior gluteal artery, and the inferior gluteal veins.

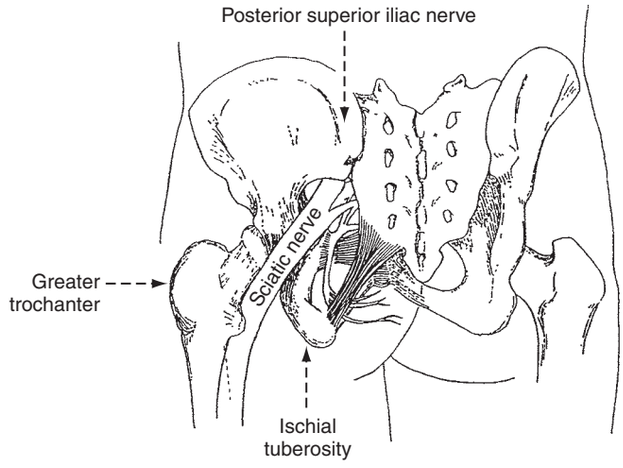


FIGURE 26–10

Shows the relationship of the sciatic nerve as it traverses via the greater sciatic notch toward the lesser trochanter.

INDICATIONS

Surgery

A sciatic nerve block is used to manage the pain associated with lower limb surgery. However, when lower limb surgery is to be performed, a sciatic nerve block is rarely sufficient as a sole anesthetic. Therefore, a sciatic nerve block is usually used in combination with a femoral nerve block, which anesthetizes the entire lower limb. For surgery of the ankle and foot, a sciatic nerve block in conjunction with a saphenous nerve block is indicated. The saphenous nerve is a branch of the femoral nerve. For surgery and analgesia of the knee, the sciatic nerve block should be used in combination with blocks of the femoral, obturator, and lateral femoral cutaneous nerves.

Continuous Infusions

The pain associated with complex regional pain syndrome type I or II, vascular insufficiency, or unilateral leg edema (of many causes) is often managed with lumbar epidural catheters. There are, however, risks associated with the long-term placement of epidural catheters. Therefore, using a new technique of catheter placement on the sciatic nerve can provide an alternative technique for pain relief. It can eliminate the risk of epidural abscess, hematoma formation, and catheter erosion of the dura. The affected limb can be separately treated without numbing or weakening the contralateral limb.

CONTRAINDICATIONS

Contraindications for sciatic nerve blocks include anticoagulant therapy, septicemia, local infection, recent injury at the site of injection to the nerve, inability of the patient to lie in a prone position, and distorted anatomy.

EQUIPMENT

Local Nerve Block
25-gauge needle to raise the skin wheal
21-gauge, 4- to 6-inch insulated needle
22-gauge, 9-cm, B-bevel needle (some clinicians prefer to use a 22-gauge, 10-cm, B-bevel needle when performing the posterior [Labat] sciatic block)[7]
3/4-inch infiltration needle
3-ml syringe
10-ml syringe
20-ml syringe
Intravenous T-piece extension
Continuous Infusion
B-D Longdwel catheter over 18-gauge, 6- to 8-inch needle
16-gauge RK epidural needle
24-cm Tun-L-XL epidural catheter or peripheral nerve stimulation (PN-STM) catheter
Aids to Procedure
Nerve stimulator with appropriate clips and wires

DRUGS

- 1.5% lidocaine
- 2% lidocaine
- 0.5% bupivacaine/ropivacaine
- Steroids

PREPARATION OF PATIENT

Physical Examination

Physical assessment should include a superficial examination for local infection and distorted anatomy. It should also include a neurological examination for documentation of abnormalities or changes. It is also important to assess the patient's ability to lie prone.

Preoperative Medication

For preoperative medication, use the standard recommendations for conscious sedation by the American Society of Anesthesiologists.

PROCEDURE

Posterior (Labat) Sciatic Block

As discussed earlier, Labat first published his description of the posterior approach in 1923 when he placed the patient in the Sims position, then located the posterior superior iliac spine and the greater trochanter. A line was drawn between the two. A perpendicular line was dropped at the midpoint of the first line; the point of entry was at a distance of 2.5–3.8 cm inferior to this, located on the line drawn from the sacral hiatus to the greater trochanter. Here a modified technique is described that places the patient in the prone position (Figure 26-11). Figure 26-12 shows radiographic landmarks in the posterior view.

After skin preparation and infiltration, a 22-gauge, 9-cm needle is inserted perpendicular to the skin at the chosen landmark. After passing through the piriformis muscle, the sciatic nerve is contacted (3.8 cm deep). The latter extends at this point toward the leg from the greater sciatic notch. With the nerve stimulator, muscle stimulation of the foot is obtained as dorsiflexion or plantiflexion is noted (Figure 26-13).

Catheter Placement

The patient is placed in a prone position (see Figure 26-11). The gluteal region ipsilateral to the affected side is sterilized and draped. Landmarks are located by fluoroscopy. These landmarks are the posterior superior iliac spine, the greater trochanter, and the ischial tuberosity[8,9] (see Figure 26-12). A

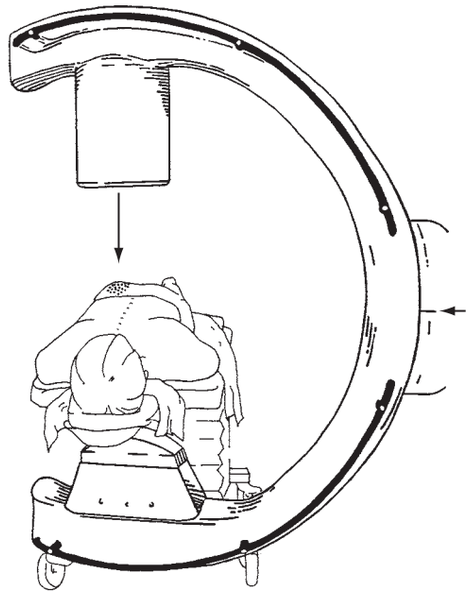


FIGURE 26–11

Patient is placed prone with the C-arm over the ipsilateral buttock for the sciatic nerve block.

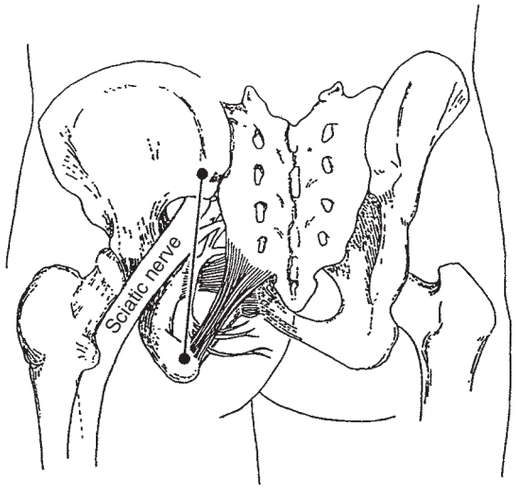


FIGURE 26–12

The drawing shows the landmarks to be identified by fluoroscopy. A, posterior superior iliac spine; B, greater trochanter; C, ischial tuberosity.

line is drawn connecting the posterior iliac spine and the greater trochanter. The midpoint is identified and a perpendicular line is drawn in a caudal direction. A second line is drawn from the greater trochanter to the ischial tuberosity. This line is divided into three parts. A third line is drawn vertically from the medial third mark upward to intersect the other line. The point of entry is where the two lines meet (Figure 26-14). A skin wheal is raised at the site with a 25-gauge needle. A larger needle (16–18 gauge) can pierce the skin. A 16-gauge, 7-inch blunt needle is introduced perpendicular, approximately 1 cm through the skin to reach the

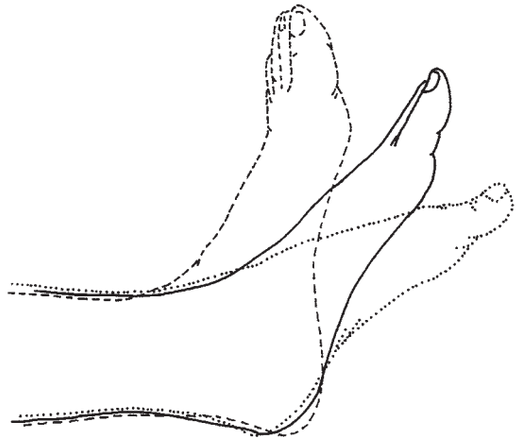


FIGURE 26–13

Plantar flexion and dorsiflexion of the foot occurring with each impulse of a nerve stimulator indicate that the needle is on the sciatic nerve.

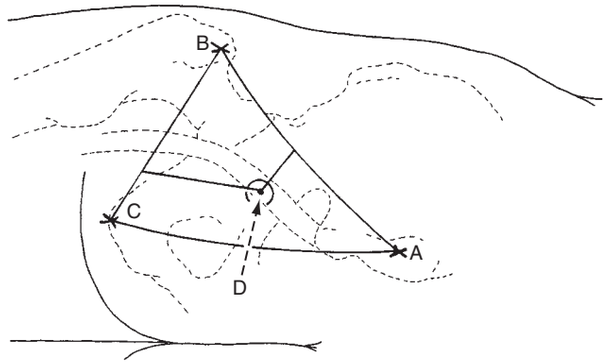


FIGURE 26–14

Surface landmarks and entry point of the needle. A, posterior superior iliac spine; B, greater trochanter; C, ischial tuberosity, and D, insertion site.

piriformis muscle. A 22-gauge needle is inserted subcutaneously and attached to a positive lead from the Medtronic test stimulator, which should be set to deliver 6 to 8 V at one impulse/sec. If a peripheral nerve stimulator is used, the current should be adjusted from 3 to 0.5 mA at 1 impulse/sec. The needle is slowly advanced anteriorly until the piriformis muscle, which is identified by contrast solution, is twitching. The needle is further advanced until the piriformis muscle stimulation stops and foot twitching (dorsiflexion) is observed in the affected limb. A stimulating catheter is then inserted through the needle. The negative lead of the stimulator is attached to the distal connect wire of the catheter. The catheter is passed to the level of the lesser trochanter for foot movement. The needle is then removed, and the catheter is attached to the hub connector. Placement can be confirmed with 3 ml of contrast dye introduced via the catheter (Figures 26-15 and 26-16). Another 3 ml of 0.2% ropivacaine may be injected, and stimulation of the sciatic nerve should cease.[8] Through an attached bacteriostatic filter, 15–30 ml of 2% lidocaine or 2.9% ropivacaine are, injected

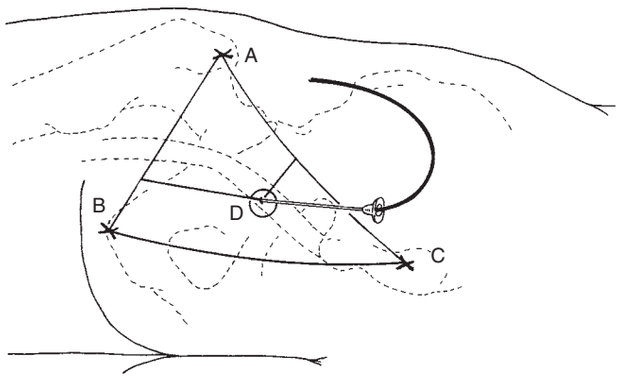


FIGURE 26–15

Surface view of the catheter after placement. A. posterior superior iliac spine. B. Greater trochanter. C. Ischial tuberosity. D. Point of entry is the skin.

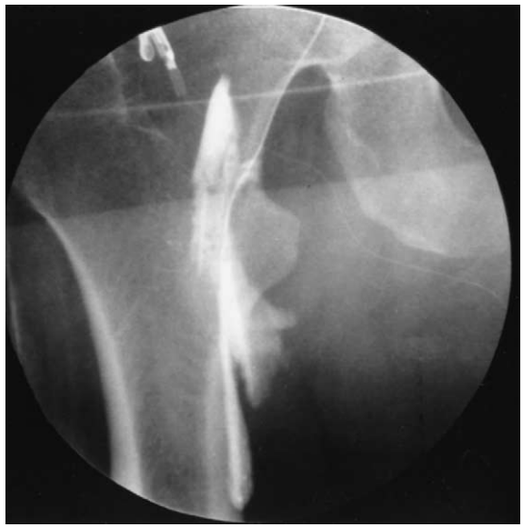


FIGURE 26–16

Fluoroscopic image of the catheter with contrast solution following the sciatic nerve sheath. (From Waldman SD, editor: *Interventional Pain Management*, 2nd ed. Philadelphia, Saunders, 2001, p. 431.)

in divided doses for immediate pain relief and nerve blockade. The constant infusion of 0.1% ropivacaine with fentanyl (5 μg/ml) may range from 4 to 10 ml/hr. Occasional bolus doses may be required and may be delivered by the patient through the pump with a bolus of 5 ml and a 30-minute lockout.[9] The catheter may be connected to a drug infusion balloon for outpatient care through home health services. The balloon delivers 4 ml/hr of the drug to the patient for 24 hours. (The volume of the balloon reservoir is 100 ml.)

Confirmation of Block

Motor and sensory pinprick loss in the lower extremity in the area of the sciatic nerve distribution, sparing the medial aspect of the leg, indicates that the block has been achieved. Sympathetic block of the leg and foot also confirms the sciatic nerve block.

SUPINE (RAJ) SCIATIC BLOCK

This approach is especially useful when treating morbidly obese patients or patients who have an abnormal posterior anatomy because the sciatic nerve is more superficial with this approach than with the other gluteal approaches. In addition, when more proximal approaches have failed, it is also useful as a "rescue" block.[9]

The patient is placed supine. The operative extremity is maximally flexed at the hip and flexed 90 degrees at the knee. This maneuver renders the sciatic nerve more superficial by reducing the redundant tissue in the buttocks and thinning the gluteus maximus muscles. The greater trochanter and ischial tuberosity are identified and subsequently marked. The midpoint of a line joining the trochanter and tuberosity is identified at the level of the gluteal crease.[9] Searching for contractions of the peroneal (toes up) or tibial (toes down) nerves, a 21-gauge, 4- to 6-inch needle is inserted perpendicular to the skin and directed cephalad. If no stimulation is recognized after the needle is inserted, the needle is re-inserted at the same point but directed 1 cm laterally or medially until the appropriate contractions occur. Once the sciatic nerve is successfully stimulated, local anesthetic is injected incrementally (Figure 26-17).

ANTERIOR SCIATIC BLOCK

With the patient placed in a supine position, a line is drawn between the anterior superior iliac spine and the pubic tubercle. Starting more caudally on the leg at the greater trochanter, a second line is drawn parallel to the first line. Then the first line is divided into three equal sections. At the junction of the medial and middle sections, a perpendicular line is drawn caudally until it intersects with the more caudal line, thus indicating the insertion point (Figure 26-18).[7] After skin preparation and local anesthetic infiltration, a 21-gauge, 4- to 6-inch needle is inserted until it contacts the medial aspect of the femur. Some clinicians prefer to use a 22-gauge, 12-cm needle.[7] The depth of the needle is noted, and then the needle is withdrawn to the depth of the skin and redirected in a more perpendicular direction to bypass the femur 5 cm beyond the depth at which the femur was first encountered.[10] Twenty to 30 ml of local anesthetic is injected incrementally after stimulation is successfully confirmed by dorsal or plantar flexion of the foot.

In 2003, Romanoff and associates[7] pointed out that "once the femur is contacted, lateral rotation of the hip may bring the neurovascular bundle more in line with the stimulating needle, and this maneuver should be entertained if there is difficulty locating the nerve."

LATERAL SCIATIC BLOCK

The patient is placed in the supine position with his or her hip in a neutral position. As described by Romanoff et al.,[7] "a point 3 cm distal to the point of maximal lateral

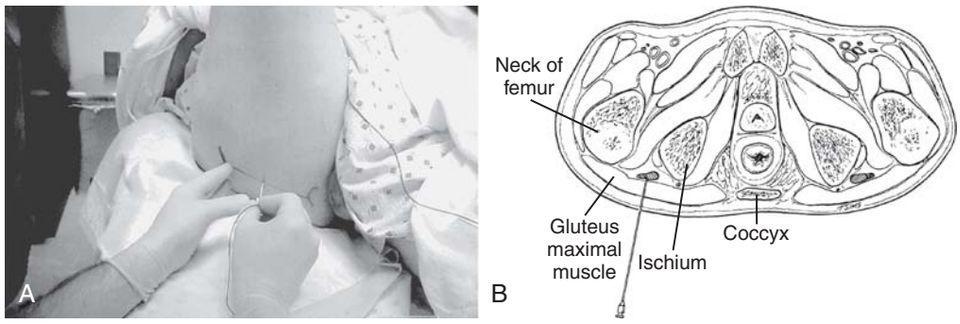


FIGURE 26–17

(A) Supine (Raj) sciatic block. (B) Cross-sections at the gluteal region show the depth of the sciatic nerve from the supine sciatic approach. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, p. 369.)

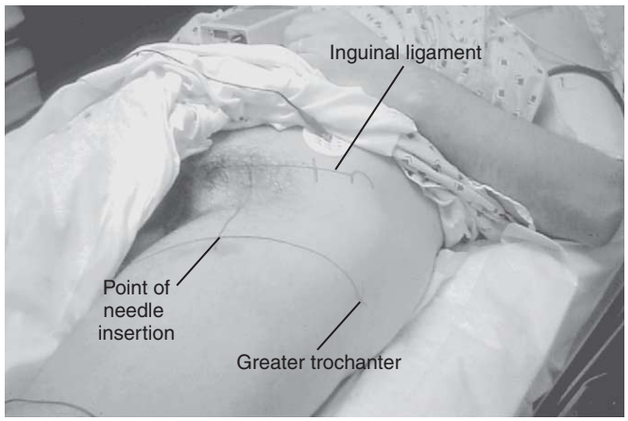


FIGURE 26–18

Sciatic nerve block: anterior approach. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, p. 369.)

prominence of the trochanter is identified along the posterior profile of the femur. After skin preparation and local anesthetic infiltration, a 21-gauge, 4- to 6-inch needle is inserted through a wheal perpendicular to the skin until bone is encountered. The needle is reintroduced to the skin and angulated approximately 20 degrees posteriorly to slide under the femur approximately 5 cm deeper than the depth at which the femur was originally contacted." Twenty to 30 ml of local anesthetic are injected incrementally after stimulation is successfully confirmed by dorsal or plantar flexion of the foot.

COMPLICATIONS

No significant complications secondary to the sciatic nerve block have been documented. However, there is a risk of nerve injury from the injections. This risk can be minimized by using a nerve stimulator and by advancing the needle slowly. It is unwise to forcefully administer local anesthetics when an abnormally high pressure of injection is noted. The risk of local anesthetic toxicity can be minimized by using test doses and incremental injections.

CLINICAL PEARLS

If intense contractions of the hamstrings occur when performing the posterior (Labat) sciatic block, the needle has been placed too far medially and should be reintroduced at a point that is 1 cm more laterally.

EFFICACY

Continuous regional anesthesia, whether central or peripheral, is safe and efficacious. The infusions may use local anesthetics, opioids, or a combination of the two. These infusions are performed when prolonged anesthesia is required for moderate to severe acute, chronic, or cancer pain. Comparing the four approaches, the anterior approach is associated with the highest failure rate, which might be due to its technical complexity and/or the clinician's attempt to perform it without a nerve stimulator.

COMMON PERONEAL NERVE BLOCK AT THE KNEE

ANATOMY

The common peroneal nerve is one of the two major continuations of the sciatic nerve, the other being the tibial nerve. The common peroneal nerve provides sensory innervation to the inferior portion of the knee joint and the posterior and lateral skin of the upper calf (see Figure 26-3). The common peroneal nerve is derived from the posterior branches of the L4, the L5, and the S1 and S2 nerve roots.[1] The nerve splits from the sciatic nerve at the superior margin of the popliteal fossa and descends laterally behind the head of the fibula (Figure 26-19). The common peroneal nerve is subject to compression at this point by such circumstances as improperly applied casts and tourniquets. The nerve is also subject to compression as it continues its lateral course, winding around the fibula through the fibular tunnel, which is made up of the posterior border of the tendinous insertion of the peroneus longus muscle and the fibula itself. Just distal to the fibular tunnel the nerve divides into its two terminal branches, the superficial and the deep

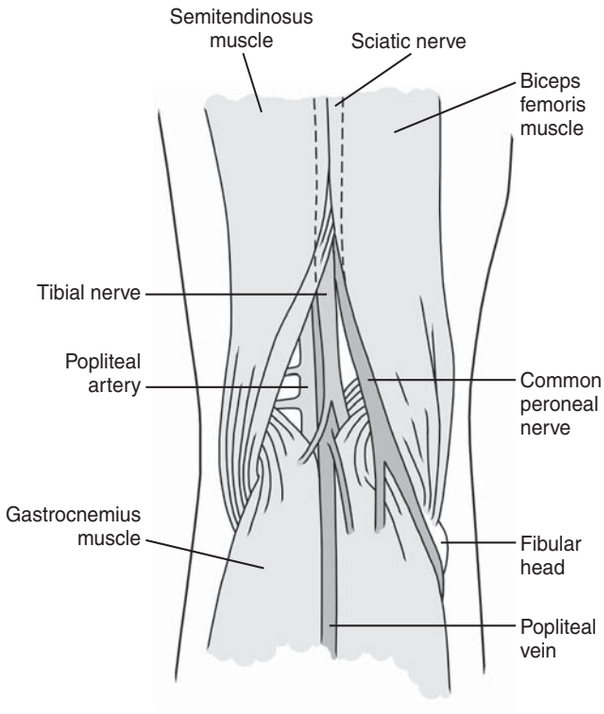


FIGURE 26–19

Common peroneal nerve is derived from the posterior branches of the L4, the L5, and the S1 and S2 nerve roots. The nerve splits from the sciatic nerve at the superior margin of the popliteal fossa and descends laterally behind the head of the fibula.

peroneal nerves. Each of these branches is subject to trauma and may be blocked individually as a diagnostic and therapeutic maneuver.

INDICATIONS

Common peroneal nerve block is useful in the evaluation and management of distal lower extremity pain thought to be subserved by the common peroneal nerve. The technique is also useful to provide surgical anesthesia for the distal lower extremity when combined with tibial and saphenous nerve block or lumbar plexus block. It is used for this indication primarily in patients who would not tolerate the sympathetic changes induced by spinal or epidural anesthesia who need distal lower extremity surgery, such as debridement or distal amputation. Common peroneal nerve block with local anesthetic can be used as a diagnostic tool when performing differential neural blockade on an anatomic basis in the evaluation of lower extremity pain. If destruction of the common peroneal nerve is being considered, this technique is useful as a prognostic indicator of the degree of motor and sensory impairment that the patient may experience. Common peroneal nerve block with local anesthetic may be used to palliate acute pain emergencies, including distal lower extremity fractures and postoperative pain relief, when combined with the previously mentioned blocks while waiting for pharmacologic methods to become effective. Common peroneal nerve block with local anesthetic and steroid is occasionally used in the treatment of persistent distal lower extremity pain when the pain is thought to be secondary to inflammation or when entrapment of the common peroneal nerve as it passes the head of the fibula is suspected (Figure 26-20). Common peroneal nerve block with local anesthetic and steroid is also indicated in the palliation of pain and motor dysfunction associated with diabetic neuropathy. Destruction of the common peroneal nerve is occasionally used in the palliation of persistent lower extremity pain secondary to invasive tumor that is mediated by the common peroneal nerve and has not responded to more conservative measures.

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represents a contraindication to the performance of a common peroneal nerve block. Local infection involving the area of the common peroneal nerve block is also a contra-

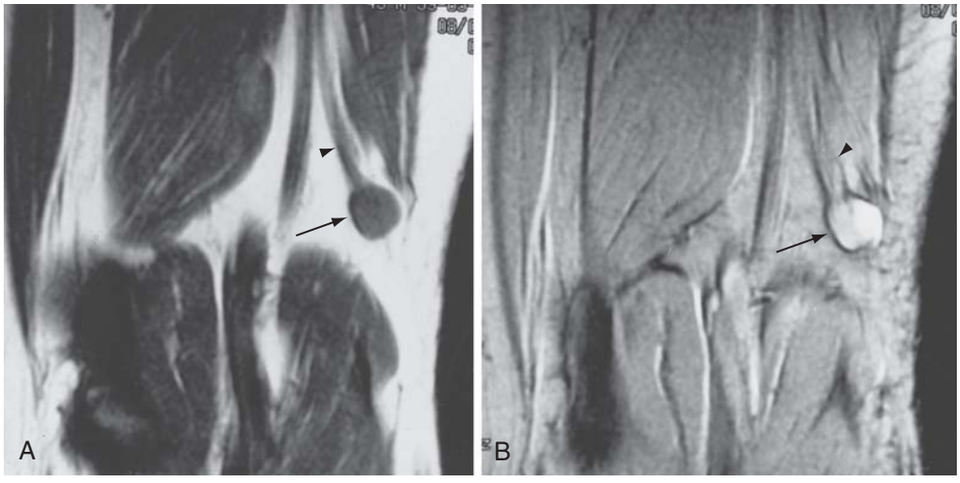


FIGURE 26–20

Involvement of the common peroneal nerve: neuroma. A neuroma (arrows) of the common peroneal nerve (arrowheads) is well seen on a coronal T1-weighted (TR/TE, 813/16), spin-echo MR image (A) and a coronal MPGR (TR/TE, 600/15; flip angle, 30 degrees) MR image (B). MR, magnetic resonance. (From Resnick D: *Diagnosis of Bone and Joint Disorders*, 4th ed. Philadelphia, Saunders, 2002, p. 3545.)

indication to the performance of common peroneal nerve block.

EQUIPMENT

- Peripheral nerve block tray
- 25-gauge, 1/2-inch needle
- 18-gauge, 1-1/2-inch needle
- 12–ml sterile syringe

DRUGS

- 1% preservative-free lidocaine (for diagnostic or prognostic block)
- 0.25% preservative-free bupivacaine (for therapeutic block)
- Depot methylprednisolone (for therapeutic block)
- 6.5% aqueous phenol (for chemical neurolytic block)

PROCEDURE

The patient is placed in the lateral position with the leg slightly flexed. The head of the fibula and the junction of fibular head and neck are palpated. A point just below the fibular head is then identified and prepared with antiseptic solution. A 25-gauge, 1/2-inch needle is then slowly advanced through this point toward the neck of the fibula until a paresthesia is elicited in the distribution of the common peroneal nerve (Figure 26-21). The patient should be warned to expect a paresthesia and should be told to say "there!" immediately when perceiving the paresthesia. Paresthesia usually is elicited at a depth of 1/4–1/2 inch. If a paresthesia is not elicited, the needle is withdrawn and redirected slightly more posteriorly until a paresthesia is obtained. Once a paresthesia is elicited in the distribution of the common peroneal nerve, the needle is withdrawn 1 mm and the patient is observed to rule out any persistent paresthesia. If no persistent paresthesia is present and after careful aspiration, 5 ml of 1.0% preservative-free lidocaine are slowly injected. Water-soluble contrast medium may be added to the local anesthetic to confirm appropriate needle placement. Care must be taken not to advance the needle into the substance of the nerve during the injection and inject solution intraneurally.

If the pain has an inflammatory component, the local anesthetic is combined with 80 mg of methylprednisolone and is injected in incremental doses. Subsequent daily nerve blocks are carried out in a similar manner, substituting 40 mg of methylprednisolone for the initial 80-mg dose. After injection of the solution, pressure is applied to the injection site to decrease the incidence of postblock ecchymosis and hematoma formation.

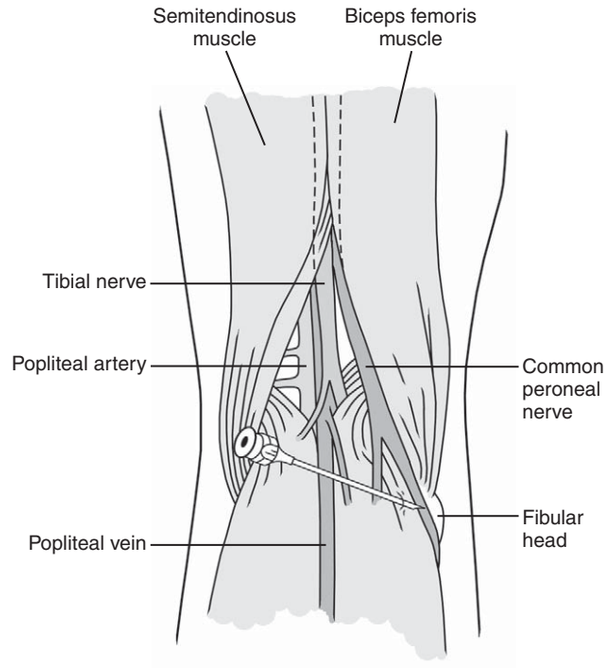


FIGURE 26–21

25-gauge, 1/2-inch needle is slowly advanced through this point toward the neck of the fibula until a paresthesia is elicited in the distribution of the common peroneal nerve.

COMPLICATIONS

The main side effect of common peroneal nerve block is postblock ecchymosis and hematoma. As mentioned earlier, pressure should be maintained on the injection site post block to avoid ecchymosis and hematoma formation. Because this technique elicits a paresthesia, needle-induced trauma to the common peroneal nerve remains possible. By advancing the needle slowly and withdrawing the needle slightly away from the nerve prior to injection, one can avoid needle-induced trauma to the common peroneal nerve.

CLINICAL PEARLS

Common peroneal nerve block is a simple technique that can produce dramatic relief for patients suffering from the previously mentioned pain complaints. Careful preblock neurologic assessment is important to avoid the later attribution of pre-existing neurologic deficits to the common peroneal nerve block. These assessments are especially important in patients who have sustained trauma to the proximal fibula and in patients suffering from diabetic neuropathy in whom common peroneal nerve blocks are being used for acute pain control.

It should be remembered that the most common cause of pain radiating into the lower extremity is herniated lumbar disc or nerve impingement secondary to degenerative arthritis of the spine, not disorders involving

the common peroneal nerve per se. Other pain syndromes that may be confused with common peroneal nerve entrapment include lesions either above the origin of the common peroneal nerve, such as lesions of the sciatic nerve, or lesions below the bifurcation of the common peroneal nerve, such as anterior tarsal tunnel syndrome. Electromyography and magnetic resonance imaging of the lumbar spine, combined with the clinical history and physical examination, help to sort out the etiology of distal lower extremity pain.

TIBIAL NERVE BLOCK AT THE KNEE

ANATOMY

The tibial nerve is one of the two major continuations of the sciatic nerve, the other being the common peroneal nerve. The tibial nerve provides sensory innervation to the posterior portion of the calf, the heel, and the medial plantar surface[1] (see Figure 26-3). The tibial nerve splits from the sciatic nerve at the superior margin of the popliteal fossa and descends in a slightly medial course through the popliteal fossa (Figure 26-22). The tibial nerve block at the knee lies just beneath the popliteal fascia and is readily accessible for neural blockade. The tibial nerve continues its downward course, running between the two heads of the gastrocnemius muscle, passing deep to the soleus muscle. The nerve courses medially between the Achilles tendon and the medial malleolus, where it divides into the medial and lateral plantar nerves, providing sensory innervation to the heel and medial plantar surface. The tibial nerve is occasionally subject to compression at this point and is known as posterior tarsal tunnel syndrome.

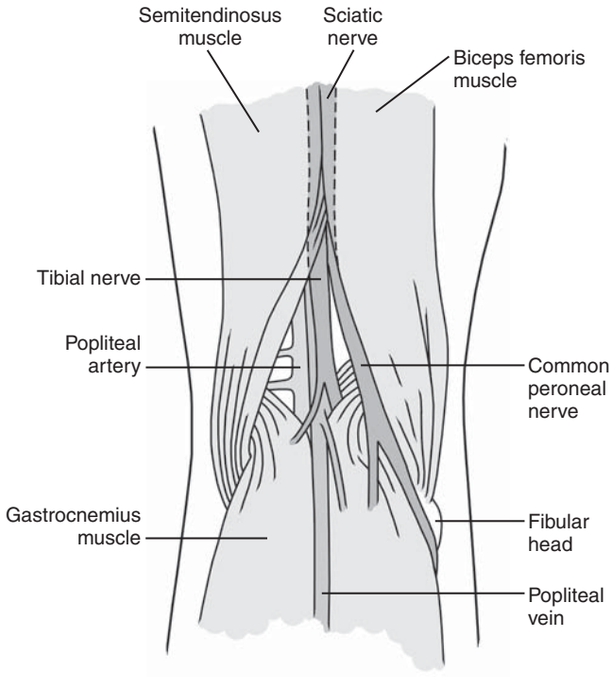


FIGURE 26–22

Tibial nerve splits from the sciatic nerve at the superior margin of the popliteal fossa and descends in a slightly medial course through the popliteal fossa.

INDICATIONS

Tibial nerve block at the knee is useful in the evaluation and management of foot and ankle pain thought to be subserved by the tibial nerve. The technique is also useful to provide surgical anesthesia for the distal lower extremity when combined with common peroneal and saphenous nerve block or lumbar plexus block. It is used for this indication primarily in patients who would not tolerate the sympathetic changes induced by spinal or epidural anesthesia who need distal lower extremity surgery, such as debridement or distal amputation. Tibial nerve block at the knee with local anesthetic can be used as a diagnostic tool during differential neural blockade on an anatomic basis in the evaluation of lower extremity pain. If destruction of the tibial nerve is being considered, this technique is useful as a prognostic indicator of the degree of motor and sensory impairment that the patient may experience. Tibial nerve block at the knee with local anesthetic may be used to palliate acute pain emergencies, including ankle and foot fractures and postoperative pain relief, when combined with the mentioned blocks while waiting for pharmacologic methods to become effective. Tibial nerve block at the knee with local anesthetic and steroid is occasionally used in the treatment of persistent ankle and foot pain when the pain is thought to be secondary to inflammation or when entrapment of the tibial nerve at the popliteal fossa is suspected (Figure 26-23). Tibial nerve block at the knee with local anesthetic and steroid is also indicated in the palliation of pain and motor dysfunction associated with diabetic neuropathy. Destruction of the tibial nerve block at the knee is occasionally used in the palliation of persistent lower extremity pain secondary to invasive tumor that is mediated by the tibial nerve and has not responded to more conservative measures.

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represents a contraindication to the performance of a tibial nerve block. Local infection involving the area of the tibial nerve block is also a contraindication to the performance of tibial nerve block.

EQUIPMENT

- Peripheral nerve block tray
- 25-gauge, 1/2-inch needle
- 18-gauge, 1-1/2-inch needle
- 12-ml sterile syringe

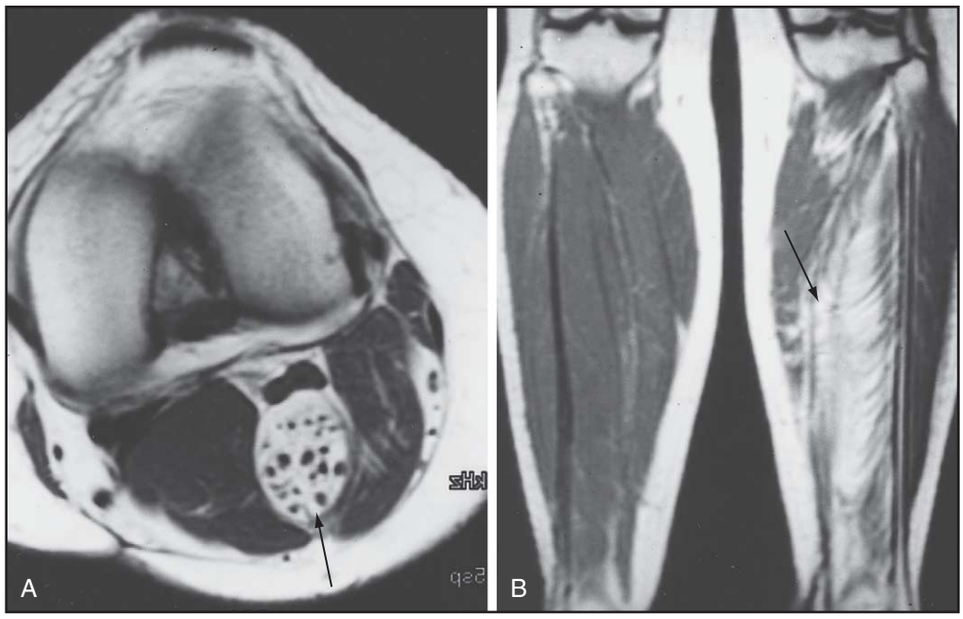


FIGURE 26–23

Involvement of tibial nerve: fibrolipomatous hamartoma. In this 38-year-old man, a transverse T1-weighted (TR/TE, 600/12), spin-echo MR image (A) shows a mass (arrow) between the medial and lateral heads of the gastrocnemius muscle. Its pattern of signal intensity, consisting of a background of high signal intensity with focal regions of low signal intensity, is typical of a fibrolipomatous hamartoma. In a coronal T1-weighted (TR/TE, 633/12), spin-echo MR image (B), fatty infiltration of the tibialis posterior muscle is seen (arrow), which could relate to denervation or fat proliferation in the territory of innervation of the tibial nerve, a well-known occurrence with this lesion. MR, magnetic resonance. (From Resnick D: *Diagnosis of Bone and Joint Disorders*, 4th ed. Philadelphia, Saunders, 2002, p. 3547.)

DRUGS

- 1% preservative-free lidocaine (for diagnostic or prognostic block)
- 0.25% preservative-free bupivacaine (for therapeutic block)
- Depot methylprednisolone (for therapeutic block)
- 6.5% aqueous phenol (for chemical neurolytic block)

PROCEDURE

The patient is placed in the prone position with the leg slightly flexed. The skin crease of the knee and margins of the semitendinous and biceps femoris muscles in the upper popliteal fossa are palpated. The margins of these muscles can be more easily identified by having the patient flex his or her leg under resistance. An imaginary triangle is envisioned with the apex being the convergence of these two muscles and the base being the skin crease of the knee (Figure 26-24). At a point in the center of this imaginary apex, the skin is prepared with antiseptic solution. A 25-gauge, 1-1/2-inch needle is then slowly advanced perpendicular to the skin through this point toward the tibial nerve until a paresthesia is elicited in the distribution of the tibial nerve. The patient should be warned to expect a paresthesia and should be told to say "there!" immediately when perceiving the paresthesia. Paresthesia usually is elicited at a depth of 1/2–3/4 inch. If a paresthesia is not elicited, the needle is withdrawn and redirected slightly more medially until paresthesia is obtained. Once a paresthesia is elicited in the distribution of the tibial nerve, the needle is withdrawn 1 mm and the patient is observed to rule out any persistent paresthesia. If no persistent paresthesia is present and after careful aspiration, 8 ml of 1.0% preservative-free lidocaine are slowly injected. Water-soluble contrast medium may be added to the local anesthetic to confirm appropriate needle placement. Care must be taken not to advance the needle

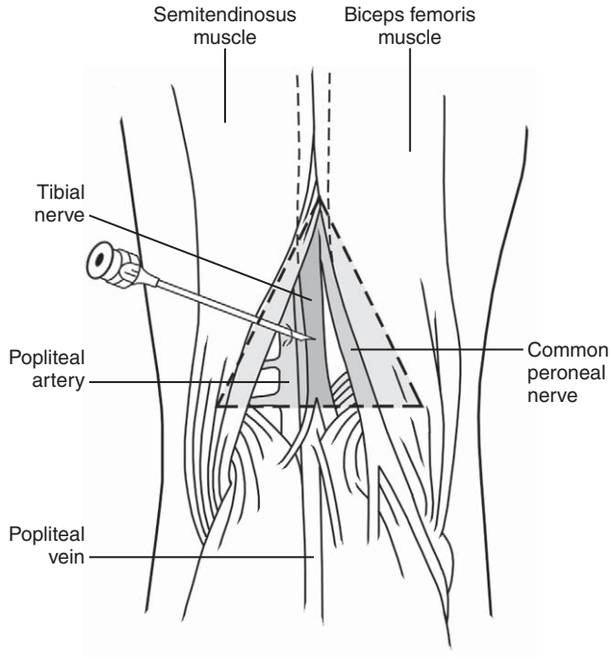


FIGURE 26-24

Triangle is drawn with the apex being the convergence of semitendinosus and biceps femoris muscles and the base being the skin crease of the knee. a 25-gauge, 1-1/2-inch needle is then slowly advanced perpendicular to the skin through this point toward the tibial nerve until a paresthesia is elicited in the distribution of the tibial nerve.

into the substance of the nerve during the injection and inject solution intraneurally. Given the proximity to the common peroneal nerve, this nerve may also be blocked when performing tibial nerve block at the knee.

If the pain has an inflammatory component, the local anesthetic is combined with 80 mg of methylprednisolone and is injected in incremental doses. Subsequent daily nerve blocks are carried out in a similar manner, substituting 40 mg of methylprednisolone for the initial 80-mg dose. After injection of the solution, pressure is applied to the injection site to decrease the incidence of postblock ecchymosis and hematoma formation.

COMPLICATIONS

The main side effect of tibial nerve block at the knee is postblock ecchymosis and hematoma. As mentioned earlier, pressure should be maintained on the injection site post block to avoid ecchymosis and hematoma formation. Because this technique elicits paresthesia, needle-induced trauma to the tibial nerve remains possible. By advancing the needle slowly and withdrawing the needle slightly away from the nerve prior to injection, one can avoid needle-induced trauma to the tibial nerve.

CLINICAL PEARLS

Tibial nerve block at the knee is a simple technique that can produce dramatic relief for patients suffering from the previously mentioned pain complaints. Careful preblock neurologic assessment is important to avoid the later attribution of pre-existing neurologic deficits to the tibial nerve block. These assessments are especially important in patients who have sustained trauma to the foot or ankle or in those patients suffering from diabetic neuropathy in whom tibial nerve block at the knee is being used for acute pain control.

It should be remembered that the most common cause of pain radiating into the lower extremity is herniated lumbar disc or nerve impingement secondary to degenerative arthritis of the spine, not disorders involving the tibial nerve per se. Other pain syndromes that may be confused with tibial nerve entrapment include lesions either above the origin of the tibial nerve, such as lesions of the sciatic nerve, or lesions below the division of the tibial nerve, such as posterior tarsal tunnel syndrome. Electromyography and magnetic resonance imaging of the lumbar spine, combined with the clinical history and physical examination, help to sort out the etiology of distal lower pain.

TIBIAL NERVE BLOCK AT THE ANKLE

ANATOMY

The tibial nerve is one of the two major continuations of the sciatic nerve, the other being the common peroneal nerve. The tibial nerve provides sensory innervation to the posterior portion of the calf, the heel, and the medial plantar surface (see Figure 26-3). The tibial nerve splits from the sciatic nerve at the superior margin of the popliteal fossa and descends in a slightly medial course through the popliteal fossa. The tibial nerve block at the ankle lies just beneath the popliteal fascia and is readily accessible for neural blockade. The tibial nerve continues its downward course, running between the two heads of the gastrocnemius muscle, passing deep to the soleus muscle. The nerve courses medially between the Achilles tendon and the medial malleolus, where it divides into the medial and lateral plantar nerves, providing sensory innervation to the heel and medial plantar surface. The tibial nerve is subject to compression at this point, which is known as posterior tarsal tunnel syndrome.

INDICATIONS

Tibial nerve block at the ankle is useful in the evaluation and management of foot and ankle pain thought to be subserved by the tibial nerve. The technique is also useful to provide surgical anesthesia for the ankle and foot when combined with common peroneal and saphenous nerve block or lumbar plexus block. It is used for this indication primarily in patients who would not tolerate the sympathetic changes induced by spinal or epidural anesthesia who need distal lower extremity surgery, such as debridement or distal amputation. Tibial nerve block at the ankle with local anesthetic can be used as a diagnostic tool during differential neural blockade on an anatomic basis in the evaluation of lower extremity pain. If destruction of the tibial nerve is being considered, this technique is useful as a prognostic indicator of the degree of motor and sensory impairment. Tibial nerve block at the ankle with local anesthetic may be used to palliate acute pain emergencies, including ankle and foot fractures and postoperative pain relief, when combined with the mentioned blocks while waiting for pharmacologic methods to become effective. Tibial nerve block at the ankle with local anesthetic and steroid is occasionally used in the treatment of persistent ankle and foot pain when the pain is thought to be secondary to inflammation or when entrapment of the tibial nerve at the posterior tarsal tunnel is suspected (Figure 26-25). Tibial nerve block at the ankle with local anesthetic and steroid is also indicated in the palliation of pain and motor dysfunction associated with diabetic neuropathy. Destruction of the tibial nerve block at the ankle is occasionally used in the palliation of persistent lower extremity pain secondary to invasive tumor that is mediated by the distal tibial nerve and has not responded to more conservative measures.

CONTRAINDICATIONS

The presence of anticoagulants and/or coagulopathy represents a contraindication to the performance of a tibial nerve block. Local infection involving the area of the tibial

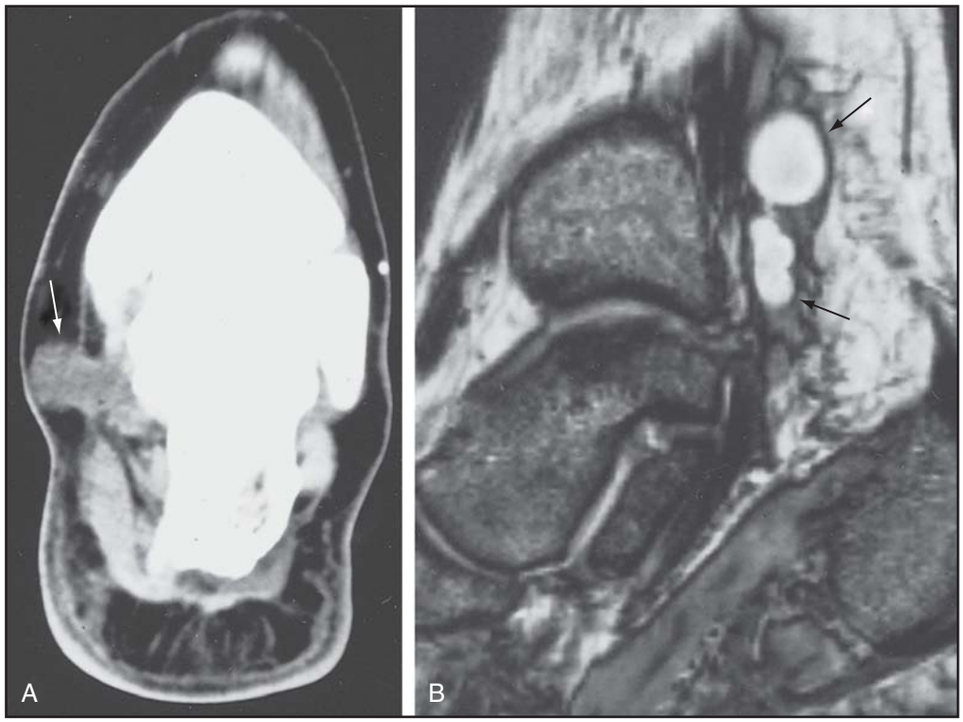


FIGURE 26–25

Entrapment of the tibial nerve: tarsal tunnel syndrome—ganglion. A ganglion (arrows) occurring on the medial and posterior portions of the ankle in this 48-year-old man is shown on a soft tissue image of a coronal CT scan (A) and on a sagittal gradient recalled acquisition in the steady state (GRASS) image obtained with volumetric acquisition (TR/TE, 30/12; flip angle, 40 degrees) (B). (From Resnick D: *Diagnosis of Bone and Joint Disorders*, 4th ed. Philadelphia, Saunders, 2002, p. 3540.)

nerve block is also a contraindication to the performance of tibial nerve block.

EQUIPMENT

- Peripheral nerve block tray
- 25-gauge, 1-1/2-inch needle
- 18-gauge, 1-1/2-inch needle
- 12-ml sterile syringe

DRUGS

- 1% preservative-free lidocaine (for diagnostic or prognostic block)
- 0.25% preservative-free bupivacaine (for therapeutic block)
- Depot methylprednisolone (for therapeutic block)
- 6.5% aqueous phenol (for chemical neurolytic block)

PROCEDURE

The patient is placed in the lateral position with the affected leg in the dependent position and slightly flexed. The posterior tibial artery at this level is then palpated. The area between the medial malleolus and the Achilles tendon is identified and prepared with antiseptic solutions. A 25-gauge, 1-1/2-inch needle is inserted at this level and directed anteriorly toward the pulsations of the posterior tibial artery. If the arterial pulsations cannot be identified, the needle is directed toward the posterior, superior border of the medial malleolus. The needle is then advanced slowly toward the tibial nerve, which lies in the posterior groove of the medial malleolus, until a paresthesia is elicited in the distribution of the tibial nerve (Figure 26-26). The patient should be warned to expect a paresthesia and should be told to say "there!" immediately when perceiving the paresthesia. Paresthesia is usually elicited after the needle is advanced 1/2–3/4 inch. If a paresthesia is not elicited, the needle is withdrawn and redirected slightly more cephalad until a paresthesia is obtained. Once a paresthesia is elicited in the distribution of the tibial nerve, the needle is withdrawn 1 mm and the patient is observed to rule out any persistent paresthesia. If no persistent paresthesia is present and after careful aspiration, 6 ml of 1.0% preservative-free lidocaine are slowly injected. Water-soluble contrast medium may be added to the local anesthetic to confirm appropriate needle placement. Care must be taken not to advance the needle into the substance of the nerve during the injection and inject solution intraneurally.

If the pain has an inflammatory component, the local anesthetic is combined with 80 mg of methylprednisolone and injected in incremental doses. Subsequent daily nerve blocks are carried out in a similar manner, substituting 40 mg of methylprednisolone for the initial 80-mg dose. After injection of the solution, pressure is applied to the injection site to decrease the incidence of postblock ecchymosis and hematoma formation.

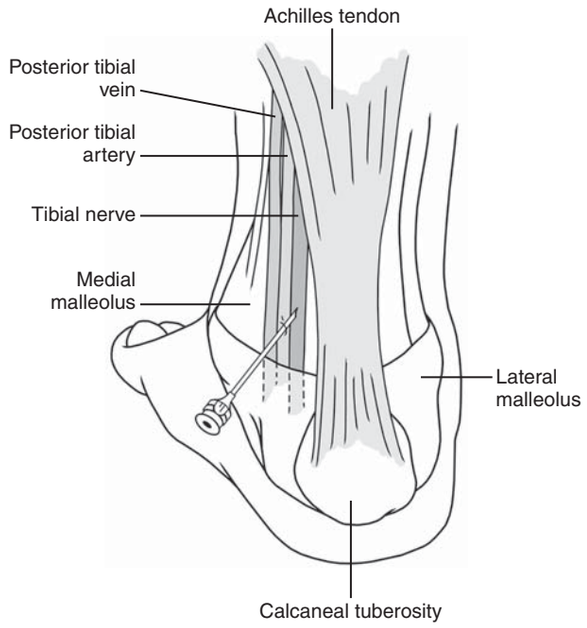


FIGURE 26–26

Needle is advanced slowly toward the tibial nerve, which lies in the posterior groove of the medial malleolus, until a paresthesia is elicited in the distribution of the tibial nerve.

SIDE EFFECTS AND COMPLICATIONS

The main side effect of tibial nerve block at the ankle is postblock ecchymosis and hematoma. As mentioned earlier, pressure should be maintained on the injection site post block to avoid ecchymosis and hematoma formation. Because this technique elicits paresthesia, needle-induced trauma to the tibial nerve remains possible. By advancing the needle slowly and withdrawing the needle slightly away from the nerve prior to injection, one can avoid needle-induced trauma to the tibial nerve. This technique can safely be performed in the presence of anticoagulation by using a 25- or 27-gauge needle, albeit at increased risk of hematoma, if the clinical situation dictates a favorable risk-to-benefit ratio.

CLINICAL PEARLS

Tibial nerve block at the ankle is a simple technique that can produce dramatic relief for patients suffering from the previously mentioned pain complaints. Careful preblock neurologic assessment is important to avoid the later attribution of pre-existing neurologic deficits to the tibial nerve block. These assessments are especially important in patients who have sustained trauma to the foot or ankle or in patients suffering from diabetic neuropathy in whom tibial nerve block at the ankle is being used for acute pain control.

Posterior tarsal tunnel syndrome presents as pain in the plantar surface of the foot. It frequently occurs after ankle fractures and dislocations or thrombophlebitis or tenosynovitis in the region. The pain is worse at night and frequently awakens the patient from sleep. The pain is burning and has the same unpleasant dysesthetic quality associated with its analogue, carpal tunnel syndrome.

It should be remembered that the most common cause of pain radiating into the lower extremity is herniated lumbar disc or nerve impingement secondary to degenerative arthritis of the spine, not disorders involving the tibial nerve per se. Other pain syndromes that may be confused with tibial nerve entrapment include lesions either above the origin of the tibial nerve, such as lesions of the sciatic nerve, or distal lesions of the tibial nerve, such as posterior tarsal tunnel syndrome. Electromyography and magnetic resonance imaging of the lumbar spine, combined with the clinical history and physical examination, help to sort out the etiology of distal lower pain.

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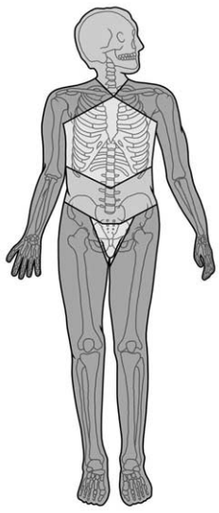
C H A P T E R



27

Joint Blocks of the Lower Extremity

LEE ANN PARADISE, MAURIZIO CHIRIVA INTERNATI, AND P. PRITHVI RAJ

HISTORY

Use of systemic corticosteroids for the treatment of symptomatic arthritis began after the discovery and synthesis of cortisone in the 1940s.[1] Hollander published the results of a large series of patients in 1951 who were treated with corticosteroid joint injections. Presently, intra-articular injections have played an important role in the diagnosis and management of acute and chronic joint pain.

ANATOMY

HIP JOINT

The hip joint is an enarthrodial or ball-and-socket joint, which is formed by the reception of the head of the femur into the cup-shaped cavity of the acetabulum. The articular cartilage on the head of the femur, thicker at the center than at the circumference, covers the entire surface with the exception of the fovea capitis femoris, to which the ligamentum teres is attached. The ligaments of the joint follow:

- Articular capsule
- Pubocapsular
- Iliofemoral
- Ligamentum teres femoris
- Ischiocapsular
- Glenoidal labrum
- Transverse acetabular

ARTICULAR CAPSULE OF HIP JOINT

The articular capsule is strong and dense. Above, it is attached to the margin of the acetabulum 5–6 mm beyond the glenoidal labrum; but in front, it is attached to the outer margin of the labrum, and, opposite to the notch where the margin of the cavity is deficient, it is connected to the transverse ligament, and by a few fibers to the edge of the obturator foramen. It surrounds the neck of the femur and is attached, in front, to the intertrochanteric line. The capsule is much thicker at the upper and forepart of the joint, where the greatest amount of resistance is required. Behind and below, it is thin and loose (Figures 27-1 and 27-2).

KNEE JOINT

The knee joint was formerly described as a ginglymus or hinge joint, but it is really much more complicated in character. It must be regarded as consisting of three articulations in one: two condyloid joints (one between each condyle of the femur and the corresponding meniscus and condyle of

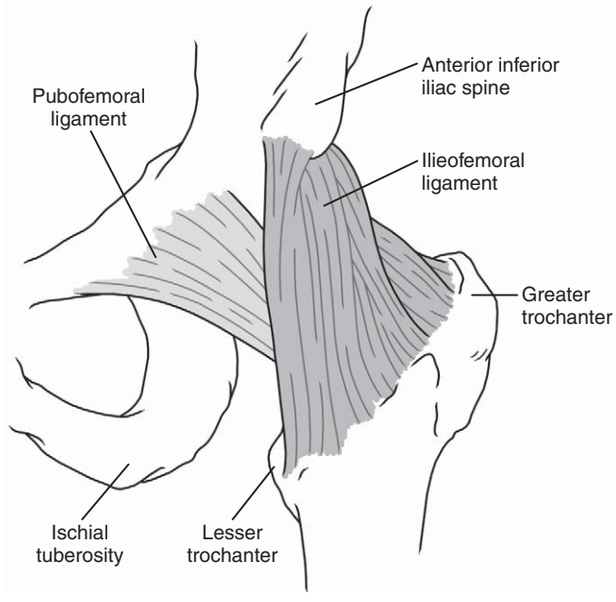


FIGURE 27-1
Drawing shows the attachments of the articular capsule (anterior view).

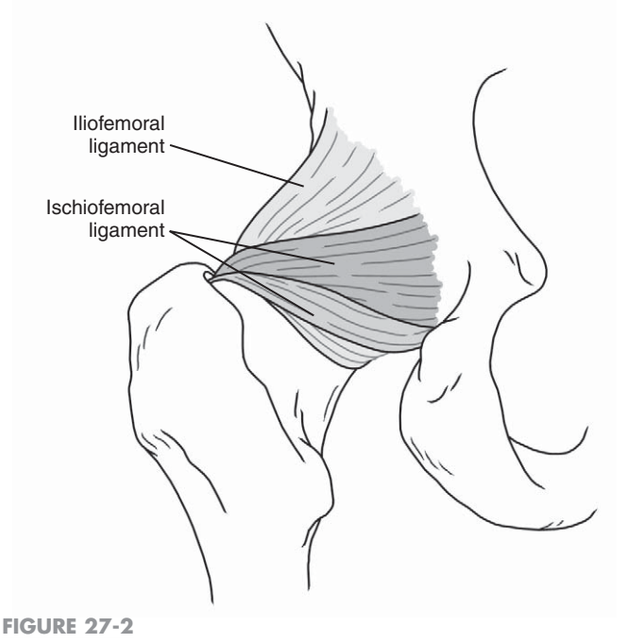


FIGURE 27-2
Drawing shows the attachments of the articular capsule (posterior view).

the tibia) and a third between the patella and the femur, partly arthrodial, but not completely so, since the articular surfaces are not mutually adapted to each other, so that the movement is not a simple gliding one. This view of the construction of the knee joint receives confirmation from the study of the articulation in some of the lower mammals, where, corresponding to these three subdivisions, three synovial cavities are sometimes found, either entirely distinct or only connected together by small communications. This view is further rendered probable by the existence in the middle of the joint of the two cruciate ligaments, which must be regarded as the collateral ligaments of the medial and lateral joints. The existence of the patellar fold of the synovial membrane would further indicate separation of the synovial cavity into two minor sacs, one corresponding to the lateral and the other to the medial joint. The bones are connected by the following ligaments:

- Articular capsule
- Anterior cruciate
- Ligamentum patella
- Posterior cruciate
- Oblique popliteal
- Medial and lateral menisci
- Tibial collateral
- Transverse
- Fibular collateral
- Coronary

ARTICULAR CAPSULE OF KNEE JOINT

The articular capsule consists of a thin, but strong, fibrous membrane that is strengthened in almost its entire extent by bands inseparably connected with it. Above and in front, beneath the tendon of the quadriceps femoris, it is represented only by the synovial membrane. Its chief strengthening bands are derived from the fascia lata and from the tendons surrounding the joint. In front, expansions from the vasti and from the fascia lata and its iliotibial band fill in the intervals between the anterior and collateral ligaments, constituting the medial and lateral patellar retinacula. Behind the capsule are vertical fibers that arise from the condyles and from the sides of the intercondyloid fossa of the femur; the posterior part of the capsule is therefore situated on the sides of and in front of the cruciate ligaments, which are thus excluded from the joint cavity (Figures 27-3 through 27-6). Behind the cruciate ligaments is the oblique popliteal ligament, which is augmented by fibers derived from the tendon of the semimembranosus muscle. Laterally, a prolongation from the iliotibial band fills in the interval between the oblique popliteal and the fibular collateral ligaments and partly covers the latter. Medially, expansions from the sartorius and semimembranosus pass upward to the tibial collateral ligament and strengthen the capsule (Figure 27-7).

ANKLE JOINT

The ankle joint is a ginglymus, or hinge joint. The structures entering into its formation are the lower end of the tibia and its malleolus, the malleolus of the fibula, and the transverse ligament, which together form a mortise

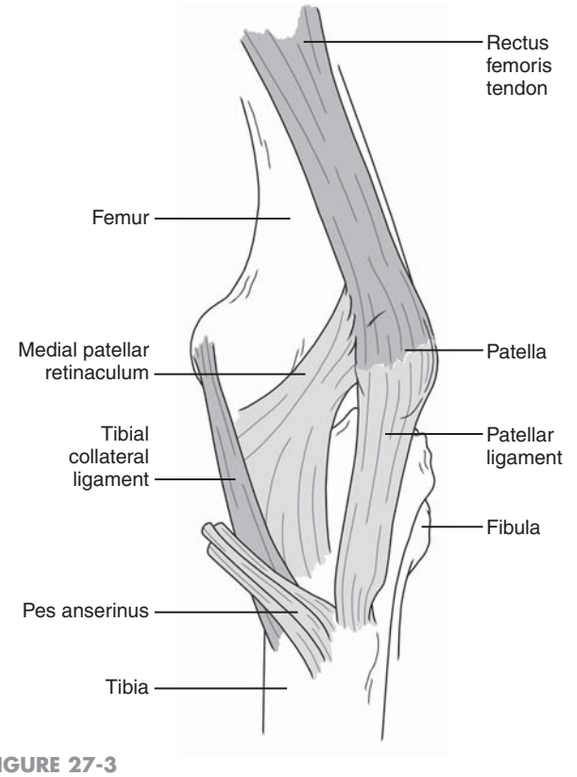


FIGURE 27-3
Right knee joint (anterior view).

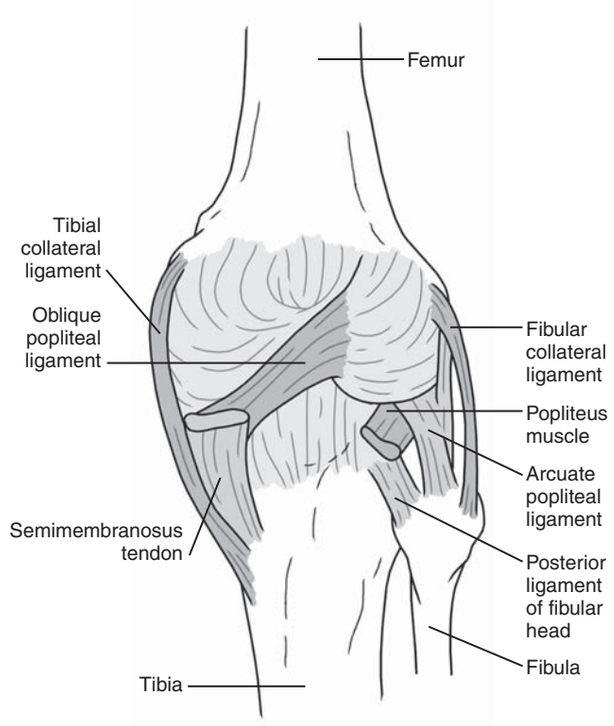


FIGURE 27-4
Right knee joint (posterior view).

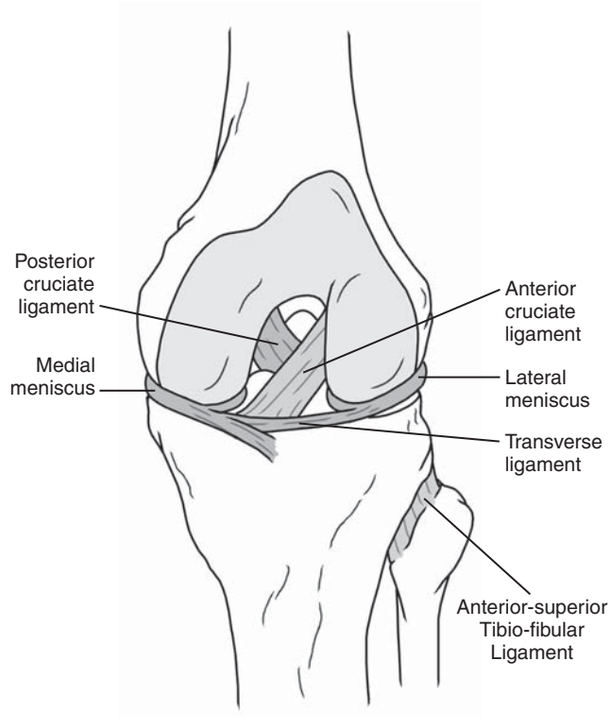


FIGURE 27-5
Right knee joint, from the front, showing interior ligaments.

for the reception of the upper convex surface of the talus and its medial and lateral facets. The bones are connected by the following ligaments:

- Articular capsule
- Anterior talofibular
- Deltoid
- Posterior talofibular
- Calcaneofibular

ARTICULAR CAPSULE OF ANKLE JOINT

The articular capsule surrounds the joints and is attached, above, to the borders of the articular surfaces of the tibia and malleoli, and below, to the talus around its upper articular surface. The anterior part of the capsule (anterior ligament) is a broad, thin, membranous layer, attached, above, to the anterior margin of the lower end of the tibia and below, to the talus, in front of its superior articular surface. It is in relation, in front, to the extensor tendons of the toes, the tendons of the tibialis anterior and peroneus tertius, and the anterior tibial vessels and deep peroneal nerve. The posterior part of the capsule (posterior ligament) is very thin and consists principally of transverse fibers. Above, it is attached to the margin of the articular surface of the tibia and, below, to the talus behind its superior articular facet. Laterally, it is somewhat thickened and is attached to the hollow on the medial surface of the lateral malleolus (Figure 27-8).

The tendons, vessels, and nerves in connection with the joint are, in front, from the medial side, the tibialis anterior, extensor hallucis proprius, anterior tibial vessels, deep peroneal nerve, extensor digitorum longus, and peroneus tertius; behind, from the medial side, the tibialis posterior, flexor digitorum longus, posterior tibial vessels, tibial nerve, flexor hallucis longus; and, in the groove behind the fibular malleolus, the tendons of the peroneus longus and brevis. The arteries supplying the joint are derived from the malleolar branches of the anterior tibial and the peroneal. The nerves are derived from the deep peroneal and tibial.

INDICATIONS

Pain in the hip, knee, and ankle joints can have multiple causes, such as inflammation, infection, arthritis, trauma, and cancer. Joint pain associated with primary or metastatic tumors can be challenging to treat. Tumor invasion affecting the bones and soft tissues of the lower extremity may cause regional musculoskeletal pain. Invasion of the spinal cord, lumbar and sacral plexus, and peripheral nerves may have neuropathic origins.[2,3] Circulatory occlusions may cause ischemic pain. When neuroablative procedures are used to treat cancer pain, the benefits of pain relief must be weighed against the risks of losing motor function and sensation.[2]

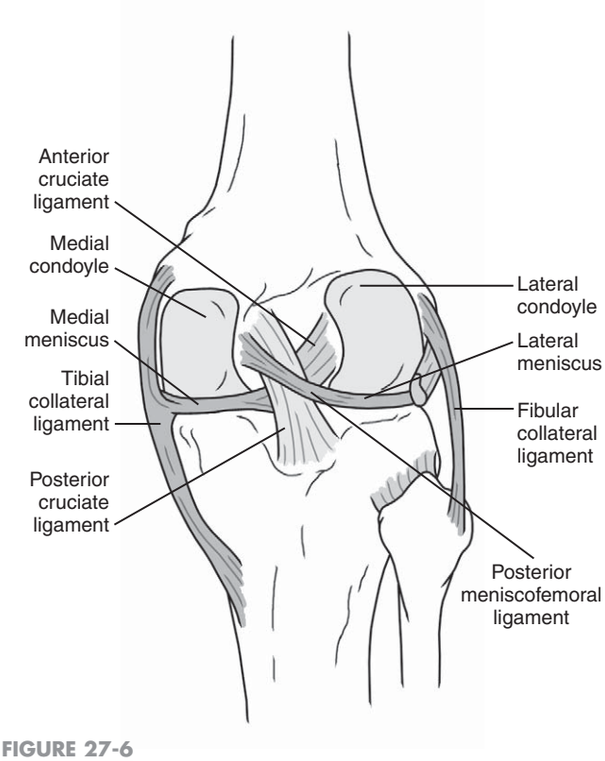


FIGURE 27-6
Left knee joint from behind, showing interior ligaments.

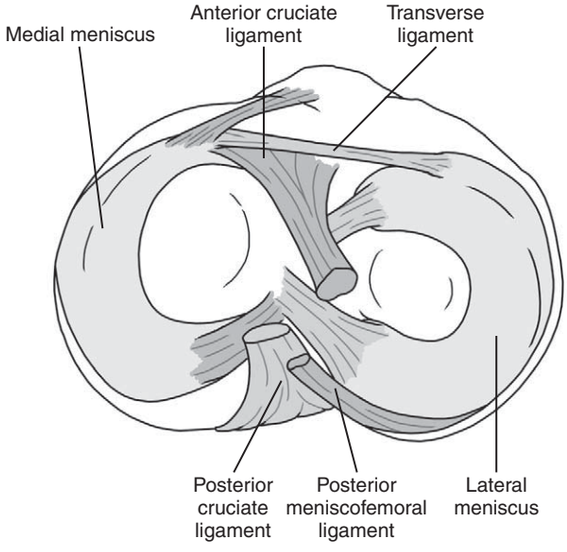


FIGURE 27-7
Head of right tibia seen from above, showing menisci and attachments of ligaments.

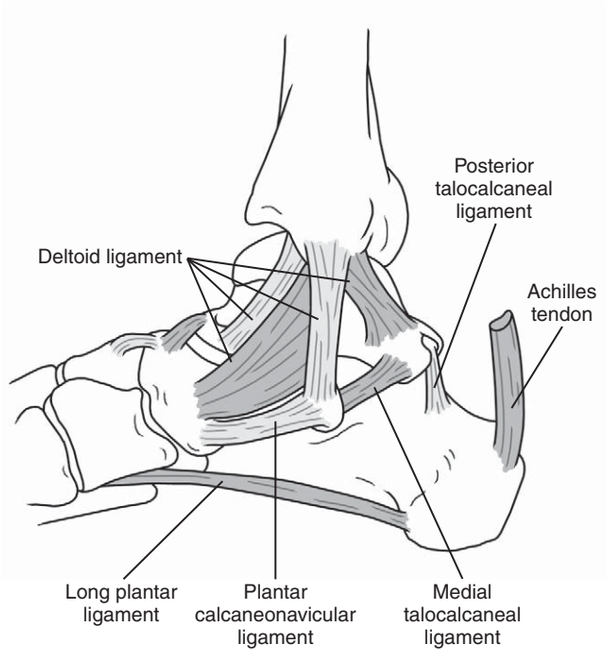


FIGURE 27-8
Ligaments of the medial aspect of the foot.

The first step is to diagnose the source of the pain. If the joint is injected with a local anesthetic and the pain does not go away, the source of the problem is probably somewhere other than the joint. If the pain immediately ceases, cortisone may be added before the needle is removed in order to reduce inflammation, which may be causing the pain. Because cortisone is long-lasting and can be slow releasing, it tends to provide effective pain relief. Although it may take several days to reduce the inflammation, the pain-relieving effects of injecting cortisone can last for weeks or even months. In some cases, it may be beneficial to add morphine or fentanyl to the cortisone for greater pain relief. However, this is usually reserved for very serious cases.

HIP INJECTION

Trochanteric bursitis, which is usually associated with trauma or pressure to the area, is a condition commonly treated with therapeutic injections. Frequently associated factors are leg-length abnormalities, obesity, rheumatoid arthritis, and osteoarthritis.[4,5] Sometimes the problem can be caused by friction from a tight iliotibial band, which is commonly associated with runners. Palpation of the tenderness and swelling in the region of the bursa confirms the diagnosis.[4] It is preferable to treat trochanteric bursitis early with a corticosteroid injection because it has been shown to be effective with satisfactory duration of effect.[4,6]

KNEE INJECTION

Unexpected effusion, possible septic arthritis, and pain relief are possible indications for aspiration.[7] Advanced osteoarthritis and other noninfectious inflammatory conditions, such as gout, are indications for corticosteroid injections.[7,8] Also, knee joint pain associated with advanced osteoarthritis can be treated with viscosupplementation therapy. One

commonly used viscosupplementation preparation is Hylan G-F 20 (Synvisc), which comes with prefilled syringes. Viscosupplementation and corticosteroid therapies are not used concurrently.[4]

ANKLE INJECTION

Older patients, as well as athletes with a history of trauma to the ankle, can develop arthritis requiring corticosteroid joint injections. Other possible indications for ankle joint injections besides osteoarthritis and rheumatoid arthritis are crystalloid deposition disease and synovitis.[9] Patients requiring an ankle injection typically complain of pain and difficulty walking. Upon examination, the patient's ankle may be tender to the touch and appear swollen. The patient's range of motion is often affected. The need for an ankle injection can be supported using radiographs. To confirm arthropathies, such as crystalloid deposition disease and Lyme arthritis, aspiration can be useful.[9] If an infection is suspected, an aspiration must be performed; however, a corticosteroid injection is absolutely contraindicated if an infection is present.[9]

EQUIPMENT

- Needles and syringes
- 22- or 25-gauge, 1-1/2-inch needle (5–10 ml syringe) (Use a 25-gauge, 1-1/2-inch needle [10-ml syringe] for ankle joint injections.)
- 18-, 20- or 22-gauge, 1-1/2-inch needle (30–60 ml syringe for aspiration, 10 ml syringe for injection) (Use an 18-gauge, 1-1/2-inch needle [30–60 ml syringe] for ankle joint aspirations.)

DRUGS

Anesthetic

3–5 ml of 1% lidocaine (Xylocaine) or 0.25–0.5% bupivacaine (Marcaine)

5–7 ml of 1% lidocaine or 0.25–0.5% bupivacaine

Corticosteroid

1 ml betamethasone sodium phosphate and acetate (Celestone Soluspan) or 1 ml methylprednisolone (Depo-Medrol), 40 mg per ml

2–3 ml betamethasone sodium phosphate and acetate or 2–3 ml methylprednisolone, 40 mg per ml

1 ml of Celestone or 1 ml of Solumedrol

PROCEDURES

HIP JOINT INJECTION

To perform the technique described by Cardone and Tallia,[4] the patient is placed in a lateral recumbent position with the affected side up. To encourage patient comfort and stabilization, the hip is flexed 30–50 degrees and the knee is flexed 60–90 degrees. By palpating the femur from the mid-shaft proximally until the area of the bony protrusion is reached, the greater trochanter is identified. The most significant point of tenderness or swelling is the best place for the injection. A 22- or 25-gauge, 1-1/2-inch needle is inserted perpendicular to the skin. A longer needle may be required if the patient is obese. The next step is to insert the needle directly to the bone and then withdraw it approximately 2–3 mm before giving the injection. This technique is helpful for trochanteric bursitis (Figures 27-9 through 27-11).

When giving a hip joint injection, the needle needs to slip over the greater trochanter toward the head of the femur. It can be injected 1–2 cm deeper than the greater trochanter in the capsule of the hip joint or when it touches the neck of the femur. If there is negative aspiration of fluids, then 2–3 cc of local anesthetic with or without corticosteroid can be injected.

KNEE JOINT INJECTION

Placed in a supine position, the patient's knee is slightly flexed with a rolled towel or pillow in the popliteal space. The medial, lateral, and superior borders of the patella need to be identified. Because there are a variety of different techniques for aspirating or injecting the knee joint, the physician's preference is often based on the merits of the technique, as well as the condition of the patient. To use the lateral approach, which is most commonly used, lines are drawn along the lateral and proximal borders of

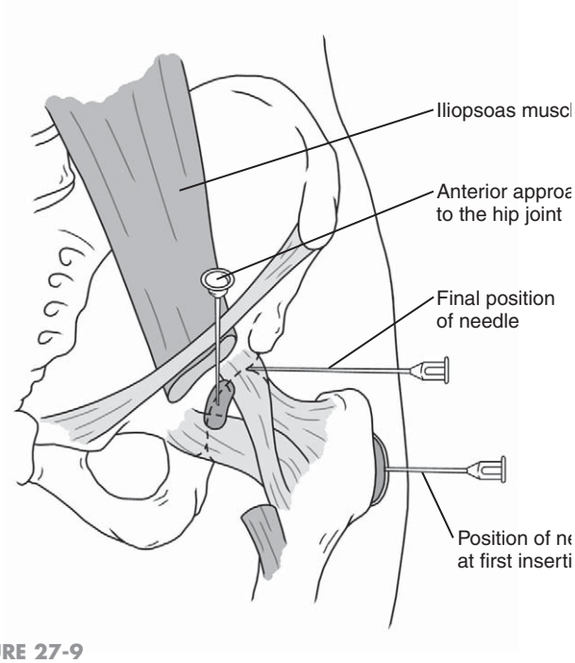


FIGURE 27-9
Drawing of injection sites of the hip joint.

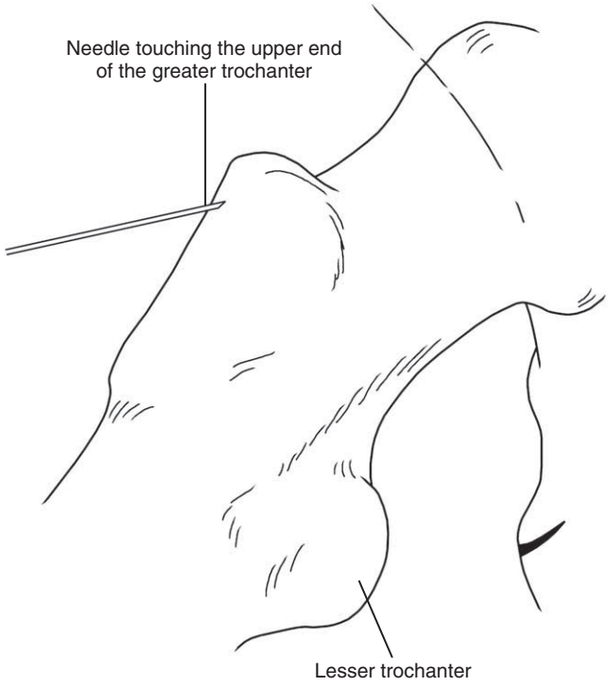


FIGURE 27-10
Drawing shows needle touching the greater trochanter.

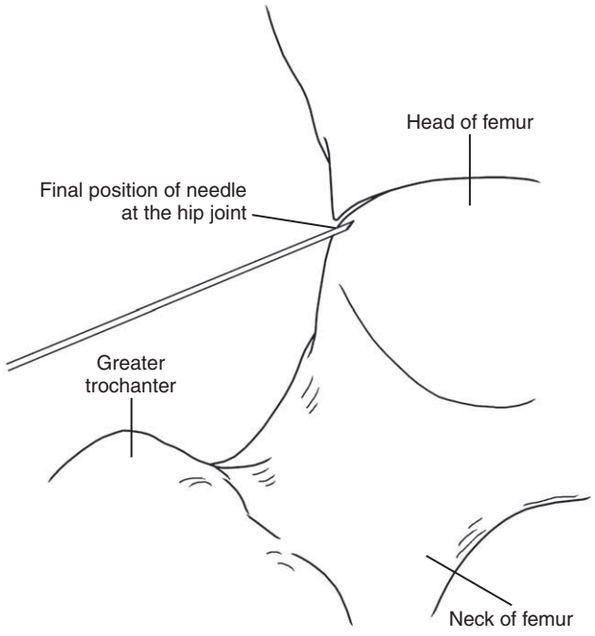


FIGURE 27-11
Drawing showing needle slipping off the greater trochanter and touching the hip joint.

the patella and the needle is inserted into the soft tissue between the patella and the femur near the point where the lines intersect.[4] The needle is then directed at a 45-degree angle toward the medial side of the joint. Before the injection or aspiration, the use of lidocaine is recommended. The flow of the injection should be even and not met with any resistance. To use the medial approach, the needle should be inserted at the medial side of the knee under the middle of the patella and directed toward the opposite patellar midpole.[4] To use the anterior approach, the knee is flexed approximately 60–90 degrees and the needle is inserted just medial to the patellar tendon and parallel to the tibial plateau.[4] Physicians tend to prefer this approach because the joint is easier to access when the patient has advanced osteoarthritis; however, there is a greater risk of meniscal damage with this approach than with the other approaches[4] (Figures 27-12 through 27-15).

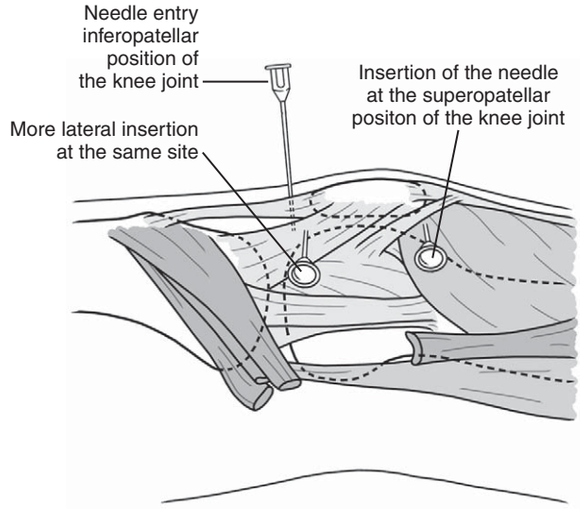


FIGURE 27-12
Multiple sites of injection of the knee joint.

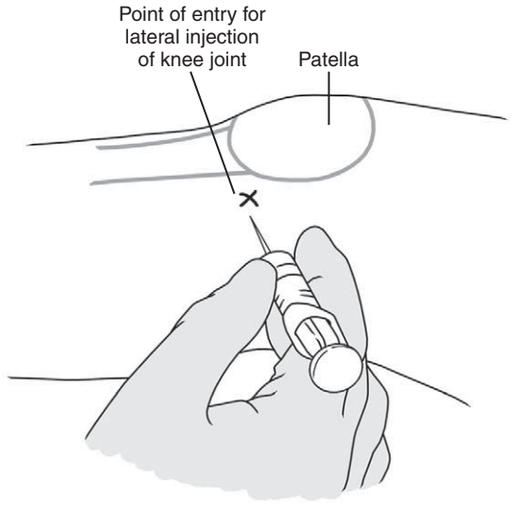


FIGURE 27-13
Drawing illustrates the technique of injecting lateral to the patella.

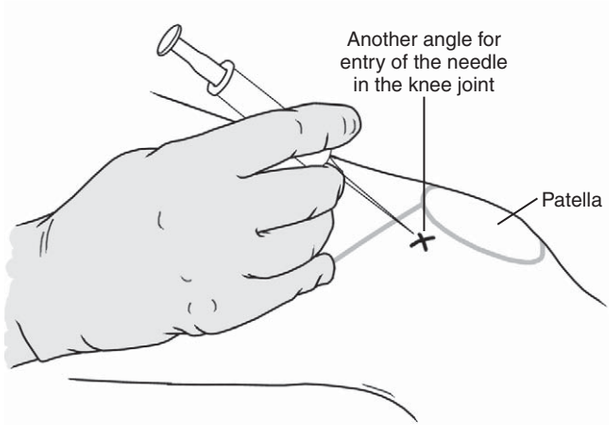


FIGURE 27-14
Drawing shows the point of injection into the knee joint from below.

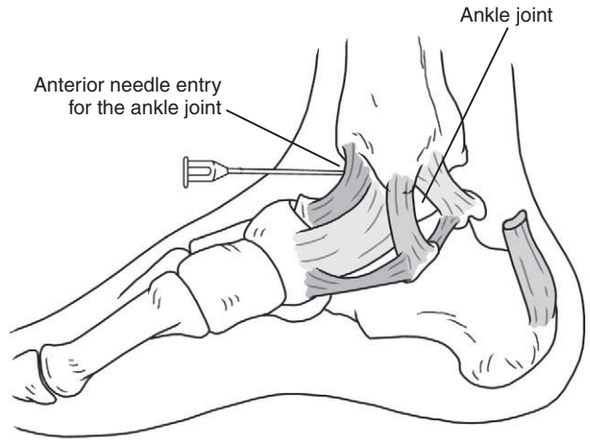


FIGURE 27-15
Drawing shows the attachments of the ankle joint through which the needle passes anteriorly.

ANKLE JOINT INJECTION

After the patient is placed in a supine position, the physician locates the space between the anterior border of the medial malleolus and the medial border of the tibialis anterior tendon, which is then palpated for the articulation of the talus and tibia. After sterilizing the skin, the needle is inserted and directed posterolaterally. Upon entering the joint space, reduced resistance will be felt. When it is necessary to aspirate the area before an injection, the needle is held with a hemostat while the syringe is changed[9] (Figures 27-16 and 27-17).

FOLLOW-UP CARE

After joint injections, the patient should be monitored for at least 30 minutes to see if any adverse reactions occur. Patients should be encouraged to avoid any strenuous activity for several days after the injection. Patients should also be instructed against the application of heat.[4]

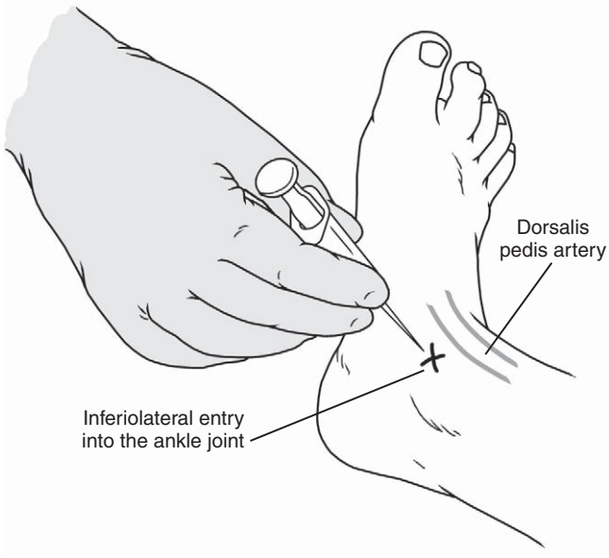


FIGURE 27-16
Technique of injecting the medial aspect of the ankle joint below the medial malleolus.

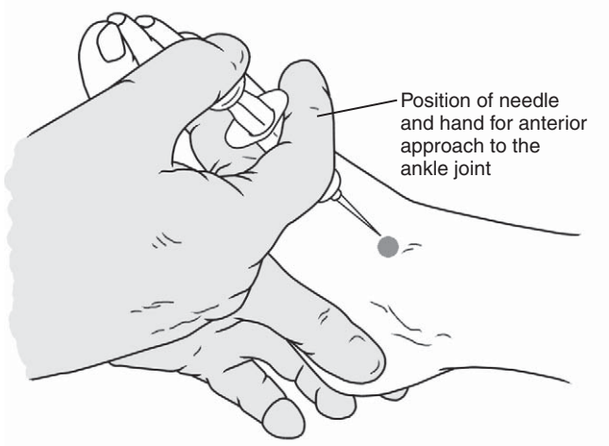


FIGURE 27-17
Technique of injecting the ankle joint from the anterior.

OTHER CONSIDERATIONS

TUMOR IMMUNOLOGY AND CANCER-RELATED BONE PAIN

Recent advances in understanding the fundamental mechanisms associated with the regulation of the immune response are helping us to understand more about tumor growth and the pain relating, for example, to diseases that affect bones, such as multiple myeloma. The understanding of the immunological aspects of tumor expansion is

leading to the development of new strategies to stimulate the immune system to mount more effective responses to tumors and reduce the pain associated with them in the bone site. There are few therapies in patients with bone disease such as multiple myeloma. We do know that the density of the bone is implicated and the pain associated is very high, but there aren't many effective methods to mitigate the pain and still treat the patients effectively. For example, this is especially true in young patients with acute lymphoblastic leukemia (ALL) or non-Hodgkin's lymphoma.

There is a need for additional therapies of bone marrow edema (BME) and aseptic osteonecrosis (AON) in both pediatric oncology patients and in adult patients, such as those with multiple myeloma. Research should focus on the exploration of both conventional and alternative medicine therapies for pain relief. For example, one alternative medicine therapy that has been employed with some success to treat AON is hyperbaric oxygenation (HBO). HBO is the medical use of oxygen in amounts higher than atmospheric pressure.

In order to treat pain associated with cancer that affects the bone, it is important to understand the immunology of cancer. It is also critical to understand why the tumor is growing and not only what nerves are being affected, but how to treat the associated pain. Metastatic tumors are the most common form of skeletal malignancy.

The skeleton is the most common organ to be affected by metastatic cancer.[10] Bone pain is the most common complication of metastatic bone disease, resulting from structural damage, periosteal irritation, and nerve entrapment. Studies suggest that pain caused by bone metastasis may also be related to the rate of bone resorption.[10] Hypercalcemia occurs in 5–10% of all patients with advanced cancer and is extremely common in patients with multiple myeloma.

Patients with skeletal pain caused by metastatic disease often have a known primary tumor. However, in a small number of patients, especially those over 40 years of age, the pain associated with a metastatic lesion may be the first sign that a malignancy exists.[11] Most metastatic tumors are found in the bone site, after the lungs and liver. A systematic approach to pain management that includes analgesics, counseling, and activity modification can benefit some patients with metastatic disease. Unremitting pain, pathologic fracture, hypercalcemia, and neurologic deficits may result from a delayed diagnosis and treatment.[11] In an interesting animal model study reported by Peters et al.[12] in 2005, the mechanisms leading to chronic pain associated with cancer were explored. The authors found that treatment with gabapentin did attenuate continued and movement-evoked cancer-related pain. Neither tumor growth nor tumor-induced bone destruction occurred in sensory neurons or the spinal cord. The author's findings suggest that tumor-derived, inflammatory, and neuropathic mechanisms are simultaneously affecting the chronic pain state.

More studies need to be conducted to understand the mechanisms that generate chronic pain in patients with metastatic cancer and why bone cancer can be refractory to treatment with opioids. Periodic episodes of spontaneous breakthrough pain can be especially difficult to treat as the doses of opioids required to control this type of pain are often at such high levels that the patient experiences numerous unwanted side effects, such as sedation and constipation. For patients who develop tumors in the bone, 40% will occur in the lower extremities, specifically in the hip and knee (Figure 27-18). For example, multiple myeloma is a malignancy of the bone marrow and is always a systemic disease. Spread of the disease to multiple bony areas is inevitable.

The reason why we have so many primary and secondary tumors is due to the lack of immunosurveillance and is based on the tumor escape mechanism induced from the tumor sites capable to evade the immune system's down-regulation of the antigen's surface. Furthermore, secretion of suppressed cytokines, such as transforming growth factor-beta (TGF-β) and interleukin 10 (IL-10), and T-cell anergy (due to the lack of costimulation), as well as the expression of fas ligand, will induce the capacity for the tumor to grow without any impediment.[13–16]

The major histocompatibility complex (MHC) class I antigens play a key role in a variety of immunological processes, including recognition, thereby functioning as target structures recognized by MCH class I restricted antigen-specific cytotoxic T lymphocytes (CTL). Cancer takes advantage of the regulatory role of the cytokines to

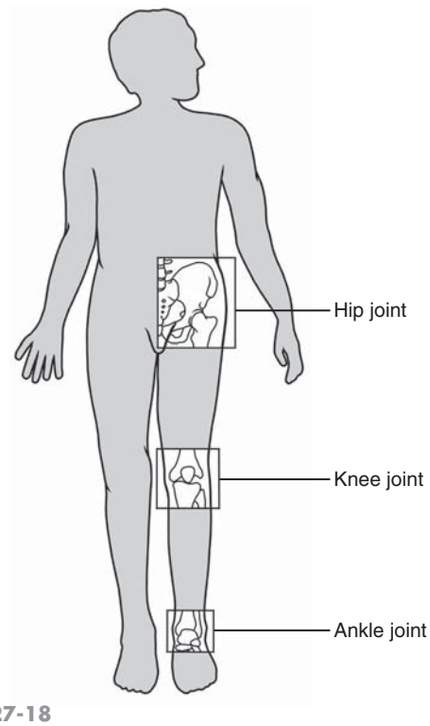


FIGURE 27-18
Site of tumors seen most commonly in the lower extremities.

down-regulate appropriate immune responses targeted at destroying cancer cells. They do this by secreting immunosuppressive cytokines that induce generalized and specific inhibition of the immune responses. This is due, in large part, to the immunosuppressive cytokine's release from the tumor cells. Immunosuppressive cytokines secreted by cancer cells include TGF-β and in IL-10.[17–20]

Secretion of TGF-β has been found in several cancer types involving the bone. TGF-β is one of the most potent immunosuppressive cytokines yet characterized. It is capable of affecting the proliferation, activation, and differentiation of cells participating in both the innate and acquired immune response.[21,22]

COMPLICATIONS

Rare complications include infection, hematoma, and fracture of the bone (metastatic state).

EFFICACY

Even though the injections have been useful for immediate transient relief of pain with a local anesthetic, there are not adequate data to suggest that they have been helpful for long-term chronic pain. The injections are best used for symptomatic pain relief.

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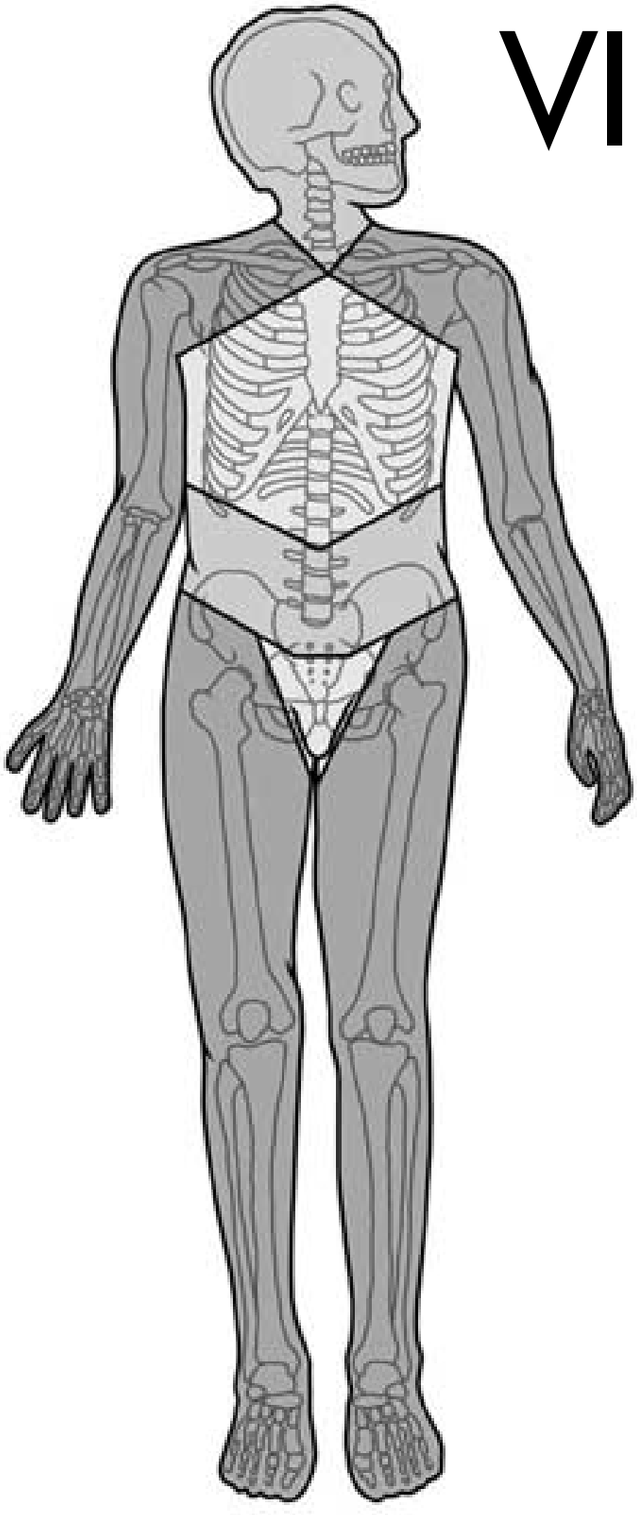
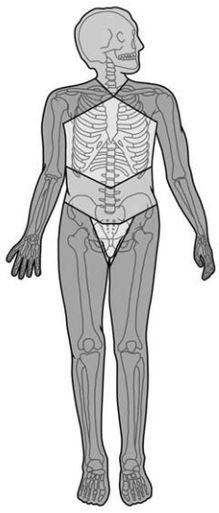
SECTION

VII



Advanced Techniques

C H A P T E R



28

Percutaneous Stimulation Systems

PETER S. STAATS

HISTORY

Neuromodulation involves the chemical or electrical modulation of the central or peripheral nervous system to modify diseases and symptoms of diseases, including pain. The ancient physician, Scribonius Largus, is the first practitioner credited with the use of electricity in the management of pain. He noted when certain fish, capable of an electric discharge, were applied to the painful areas, they had medical powers and could "carry off the pains of headaches and arthritites." The Leyden jar in 1745 made it easier for physicians to selectively apply electricity in the treatment in a variety of maladies. In the 1800s it rapidly spread as a viable treatment. In the 20th century, with the advent of more modern medicine, electrical stimulation fell out of favor. However in 1965, with the publication of the gate control theory, there was a renewed interest in the use of electrical stimulation in the treatment of pain. In addition, functional electrical stimulation has been focused on the use of electrical stimulation to improve hearing, vision, functional rehabilitation, and wound healing, among many other medical applications.

Today neurostimulation for the treatment of pain is commonly used with peripheral field stimulation, peripheral nerve stimulation, dorsal root stimulation, spinal cord stimulation, deep brain stimulation, and motor cortex stimulation. Techniques of implantation of deep brain stimulation and motor cortex stimulation are beyond the scope of this chapter.

Spinal cord stimulation (SCS) for control of pain was first introduced in 1967 by Shealy and colleagues.[1] Melzack and Wall recognized that peripheral nociceptive information is transmitted to the spinal cord in small-diameter, unmyelinated C fibers and lightly myelinated A delta fibers 1a. These fibers terminate at the substantia gelatinosa of the dorsal horn and are then transmitted cephalad via the spinal cord. Other sensory input, such as touch or vibration, is transmitted via large myelinated A beta fibers. The basic premise of Melzack and Walls theory was that reception of large-fiber information such as touch or vibration would turn off, or close the gate, or turn on small-fiber information or pain. The effect of this gate closure, these authors theorized, would be analgesia.

Because these authors believed that electrical stimulation is effective only at the dorsal horns of the spinal cord, they called this stimulation modality dorsal column stimulation (DCS). Since it is now known that inhibition of nociception can occur with electrical stimulation almost anywhere in the spinal cord, DCS has been supplanted in the literature by SCS, the more general but accurate term.

Studies supporting segmental antidromic inhibition of spinothalamic projection cells by electrically stimulating the dorsal columns soon appeared.[2] Foreman and coworkers investigated the effects of DSC on spinothalamic tract cells in anesthetized monkeys.[2] Dorsal column stimuli were applied to midthoracic or cervical levels of the spinal cord while responses of spinothalamic cells to von Frey hair activation of the sural nerves were examined. These authors found that DSC depressed the activity of spinothalamic tract cells for about 150 milliseconds and that the best points for stimulation producing inhibition were over the ipsilateral dorsal columns. Responses to electrical stimulation of peripheral nerves and mechanical stimulation of cutaneous nociceptors were similarly depressed by DCS. Lesioning the dorsal columns eliminated this depression of activity by DCS stimulation below the lesion. Lesioning the lateral columns in this model had no effects. Likewise, Handwerker and associates[3] and Feldman,[4] in studies from single dorsal horn neurons in anesthetized cats, found that the discharges of class 2 cells in the dorsal horn that respond to both noxious radiant heat stimulation and input from low-threshold cutaneous mechanoreceptors were suppressed by electrical stimulation of cutaneous, myelinated, afferent nerve fibers. The mechanisms of SCS are summarized in Table 28-1.

TABLE 28-1 Spinal Cord Stimulation: Mechanisms of Action

Segmental, antidromic activation restricted to A beta afferents, with a diameter of 10.7 μm (gate control theory).
Blocking of transmission in the spinothalamic tract.
Supraspinal pain inhibition.
Activation of central inhibitory mechanisms influencing sympathetic efferent neurons
Activation of putative neurotransmitters or neuromodulators.
A maximum of four to five fibers (10.7 μm) may be recruited in each dermatome near the discomfort threshold.
Paresthesia and pain relief in a dermatome may be affected by the stimulation of a single large A beta fiber.
The depth of stimulation may be increased twofold to threefold when stimulation is applied optimally (a narrow bi/triple or a transverse tripole).
The A beta fibers (12 μm) recruited when stimulation is applied in the dorsal epidural space.
Anodal exaltation and propagation are unlikely to occur with spinal cord stimulation.

In this chapter, the techniques of placement of suboccipital, cervical, thoracicolumbar, and sacral electrodes are described in the section on specific augmentation procedures.

INDICATIONS

There are a variety of indications or disease states in which electrical stimulation is commonly used in the management of pain. Determining which approach is optimal for any given patient may depend on many factors, and the current thinking is evolving. When conventional spinal cord stimulation is ineffective or impractical, it is possible to stimulate other areas of the nervous system. Indications are summarized as follows:

Failed back surgery syndrome with or without radiculopathy (spinal cord stimulation with or without dorsal root stimulation; single- versus two-lead stimulation)
Peripheral vascular disease and associated ischemic pain (spinal cord stimulation)
Complex regional pain syndrome types I and II (spinal cord stimulation with or without dorsal root stimulation)
Other painful conditions
Nerve injury–related pains
Atypical facial pains (peripheral nerve stimulation, trigeminal C1-C2 stimulation, deep brain stimulation, and motor cortex stimulation)
Peripheral neuropathy (spinal cord stimulation, peripheral nerve stimulation, deep brain stimulation, and motor cortex stimulation)
Phantom limb pain (peripheral nerve stimulation, trigeminal C1-C2 stimulation, deep brain stimulation, and motor cortex stimulation)
Postherpetic neuralgia (spinal cord stimulation, peripheral nerve stimulation, deep brain stimulation, and motor cortex stimulation)
Deafferentation pain (peripheral nerve stimulation, trigeminal C1-C2 stimulation, deep brain stimulation, and motor cortex stimulation)
Abdominal pain (spinal cord stimulation, peripheral nerve, and peripheral field stimulation are commonly used)
Pelvic pain (sacral nerve stimulation [multiple approaches], dorsal column stimulation)
Axial pain (spinal cord stimulation, peripheral field stimulation)
Spinal stenosis (spinal cord stimulation)
Vascular pain (spinal cord stimulation)
Cardiovascular (angina) pain
Peripheral vascular diseases
Motor disorders
Cerebral palsy
Multiple sclerosis

CONTRAINDICATIONS

Absolute
Sepsis
Coagulopathy
Previous surgery or trauma that obliterates the spinal canal
Local infection at implantation site
Relative
Physical and/or cognitive disability that interferes with proper usage and understanding of the device
Spinal bifida
Severe spinal stenosis
Psychological comorbidity that precludes success with permanent implant

EQUIPMENT

SPINAL CORD STIMULATION TRIAL PROCEDURES

- Trial electrode (ANS, Medtronic, Advanced bionics)
- Epidural needle
- 25-gauge infiltration needle
- 15–17-gauge needle
- 3-ml syringe
- 10-ml syringe
- Loss-of-resistance syringe
- Metal marker
- 2-0 nylon suture on computed tomography (CT) needle
- Needle driver
- Connecting cables to stimulator box
- Company-specific power source (stimulator box)

PERMANENT PERCUTANEOUS PLACEMENT OF ELECTRODES AND BATTERIES (PULSE GENERATOR OR RECEIVER)

Each vendor makes specific equipment. Internal pulse generators, rechargeable batteries, and external pulse generators (radiofrequency equipment) are available.

- Implantation accessories such as anchors, guide wires, stylette for electrodes, and protective sheaths
- Equipment for tunneling electrodes
- Connecting stimulation extension wire
- Implanted pulse generator or receiver
- 2-0 nylon/silk on CT needle
- 2-0 silk suture or rounded needle for purse-string closure
- Needle driver
- Forceps
- 3-0 vicryl
- 3-0 monocryl
- No. 10 blade scalpel
- Surgical kit for implantation or suturing

A company representative should be available for equipment-specific questions and concerns.

DRUGS

- 1% lidocaine with 1:200,000 or 1:400,000 epinephrine for skin infiltration
- Preservative-free normal saline
- Sterile water
- Triple-antibiotics to soak the implant
- Triple-antibiotic ointment for surface dressing
- Many physicians also premedicate with antibiotics to cover gram-positive cocci (typical skin flora)
- Sedatives for facilitating placement of the electrodes

OCCIPITAL NERVE STIMULATION

Initial reports of occipital nerve stimulation date back to the late 1970s when the electrode was actually applied to surgically exposed C2 and C3 nerves (i.e., greater and lesser occipital nerves).[6]

ANATOMY

The technique of occipital placement of electrodes is primarily subcutaneous at the C1-C2 level. Innervation of the region is by the medial branch of the C2 and C3 posterior primary rami; the lesser occipital nerve is supplied by the C3 posterior primary ramus. The greater occipital nerve exits the spinal canal between the posterior arches of C1 and C2 and then transverses the paraspinal (semispinalis and trapezius) muscles near the nuchal ridge of the occipital bone (Figure 28-1).

INDICATIONS

- Greater occipital neuralgia (common cause of headaches)
- Tension-type headaches in the occipital region
- Whiplash injuries causing irritation or compression of suboccipital nerves
- Migraine headaches/transform migraine
- Damage of the greater occipital nerve after halo pin placement for cervical spine or intracranial procedures[7]

CONTRAINDICATIONS

- Local infection
- Coagulopathies
- Cognitive deficit in a patient precluding implant

EQUIPMENT

- 25-gauge, 3/4-inch infiltration needle
- 18-gauge, 1-1/4-inch needle
- 15-gauge, 3-1/2-inch Tuohy epidural needle
- 1 or 2 electrode arrays, 4–8 contacts each

ELECTRODE PLACEMENT FOR TRIAL STIMULATION

- Trial electrode
- Trial screener

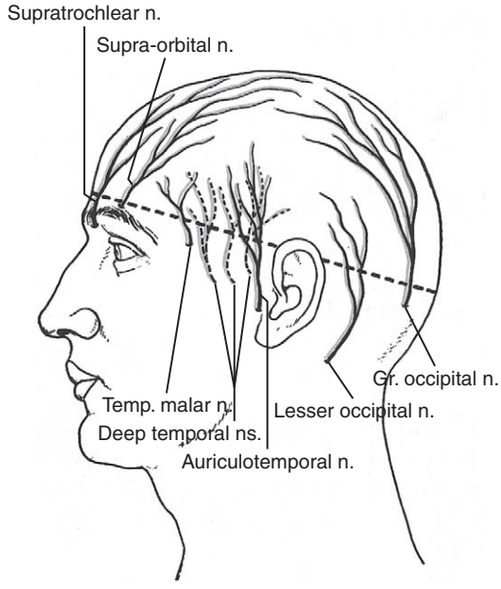


FIGURE 28-1
Innervation of the scalp and cranium.

ELECTRODE PLACEMENT FOR PERMANENT IMPLANTATION

- Connecting wires
- Tunneling equipment
- Power source (internal pulse generator, receiver, or rechargeable battery
- No. 10 blade scalpel
- Metzenbaum scissors
- Bovie

DRUGS

- 1.5% lidocaine or equivalent for infiltration

PREPARATION OF PATIENT

All patients should have previously had diagnostic and therapeutic local anesthetic blocks into the region of the affected occipital nerve with initial benefit. Prior to a permanent implant procedure more conservative measures should have failed, and patients should have demonstrated adequate analgesia with a temporary electrode array.

Laboratory Studies

Not all patients require preoperative laboratory work. Depending on the clinical situation it may be appropriate to obtain the following laboratory values:

- Complete blood count with platelets
- Prothrombin time, partial thromboplastin time
- Bleeding time or platelet function studies

Preoperative Medication and Monitoring

Follow the standard recommendations for preoperative medication and monitoring by the American Society of Anesthesiologists.

PROCEDURE

The prone position is commonly used, with the head in a horseshoe frame or other suitable device (Figure 28-2). However, other patients are positioned laterally to allow for electrode or extender wire tunneling medial to the scapula for flank, abdominal, or buttock receiver-generator pocketing (Figure 28-3). The supine position with the head turned to the opposite side allows for anterior tunneling to the subclavicular or abdominal regions. However, care must be taken to avoid proximity of the extender wire connector to the carotid artery and other vascular and neurologic structures. This is easily solved with a longer wire electrode-connector array.

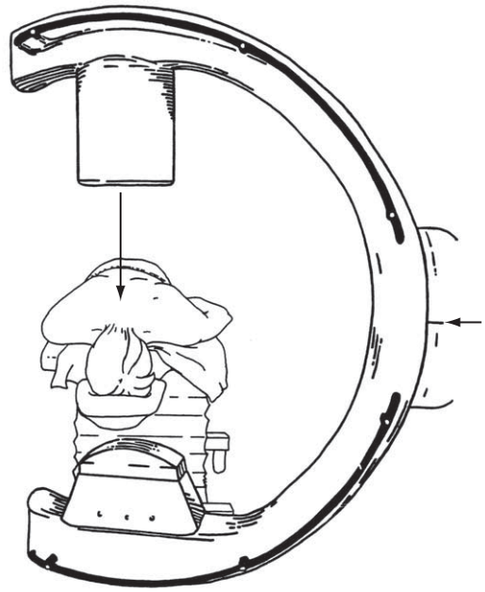


FIGURE 28-2
Position of the patient in the prone position with the C-arm for radiographic imaging during occipital stimulation.

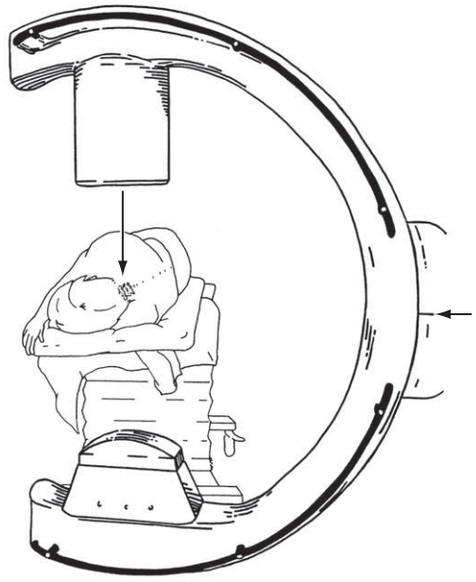


FIGURE 28-3
Position of the patient in a lateral position with the C-arm for radiographic imaging during occipital stimulation.

Technique of Needle Entry and Placement of Electrodes

Using local anesthesia, a 2-cm vertical skin incision is made at the level of C1 lamina. Physicians have used a lateral, as well as medial, approach tunneling horizontally across the suboccipital region. Other physicians have

placed the electrodes vertically along the path of the occipital nerve. The subcutaneous tissues are undermined with sharp scissors to accept a loop of wire electrode created after placement and tunneling to prevent electrode migration. A Tuohy needle is gently curved to conform to transverse cervical curvature (bevel inward) and without further dissection is passed transversely in the subcutaneous space at the level of C1 (Figure 28-4A). Single or dual quadripolar or octapolar electrodes may be passed from a midline incision to either affected side or alternatively placed to traverse the entire cervical curvature bilaterally from a single side. Rapid needle insertion usually obviates the need for even a short-acting general anesthetic. Following placement of the electrode through the Tuohy needle (Figure 28-4B), the needle is withdrawn and the electrode connected to an extender cable for intraoperative testing (Figure 28-4C). Stimulation is then applied using a temporary radiofrequency (RF) transmitter to various selected electrode combinations, enabling the patient to report stimulation location, intensity, overall sensation, and effect.

Stimulation

Most of the patients have reported immediate stimulation in the selected occipital nerve distribution with voltage settings usually below 2 V. A report of burning pain or muscle pulling should alert the interventionist that the electrode is probably placed either too close to the fascia or too far above or below the C1 level, and it should be repositioned more superficially in the subcutaneous space. Repeated needle passage for electrode placement should be avoided to reduce the risk of subcutaneous edema and/or hematoma formation, which can result in loss of stimulation.

The electrode is then sutured to the underlying fascia with the silicone fastener and 2-0 silk suture. A loop of electrode is also sutured in place to reduce the risk of electrode migration (Figure 28-4D). A short-acting general anesthetic is used to tunnel the electrodes or extender wire to the distal site for connection and implantation of the receiver-generator. Typical stimulator parameters include pulse widths of 40 to 240 microseconds, frequency of 60 to 130 Hz, and power of 0.5 to 2 V. Higher rates of up to 400 Hz with RF systems have also been beneficial. An example of dual suboccipital subcutaneous electrode placement is shown in Figure 28-5.

COMPLICATIONS

- Electrode fracture and/or displacement
- Infection
- Subcutaneous tension causing dehiscence
- Spinal cord injury with electrode placement
- Vascular and neural injury with tunneling

CLINICAL PEARLS

Infection can be decreased by careful cleansing of the whole neck. The risk of infection is due to bacterial population in the hairline. Bending the tunneling epidural needle to the contour of the neck facilitates placement of the electrode. Finally, to prevent migration, the distal top of the electrode can be sutured into place.

EFFICACY

Although still relatively new, the follow-up success appears to be relatively high with this technique.[8] In one study, 12 of 17 patients had greater than 50% reduction in pain at the last follow-up.

CERVICAL SPINAL CORD STIMULATION

ANATOMY

Anatomy of the cervical epidural space is described elsewhere and is not discussed here.

INDICATIONS

Cervical placement of the SCS is indicated for painful conditions of the neck, upper extremities, and upper torso for which cervical SCS is indicated. Typical diagnoses include the following:

- Complex regional pain syndrome
- Peripheral neuropathy of the upper extremity
- Brachial plexus injuries, including stretch injury, radiation burns, and traumatic injuries
- Somatic skeletal injuries (e.g., whiplash)
- Carpal tunnel syndrome
- Postherpetic neuralgia
- Scleroderma
- Failed surgical procedures in the neck
- Facial pain

CONTRAINDICATIONS

- Sepsis
- Coagulopathy
- Spinal stenosis

PATIENT SELECTION

To be considered for SCS procedure, the patient must meet the following general criteria:

- There is a demonstrable pathology and an objective basis for the pain complaint.
- Conservative therapies have failed.

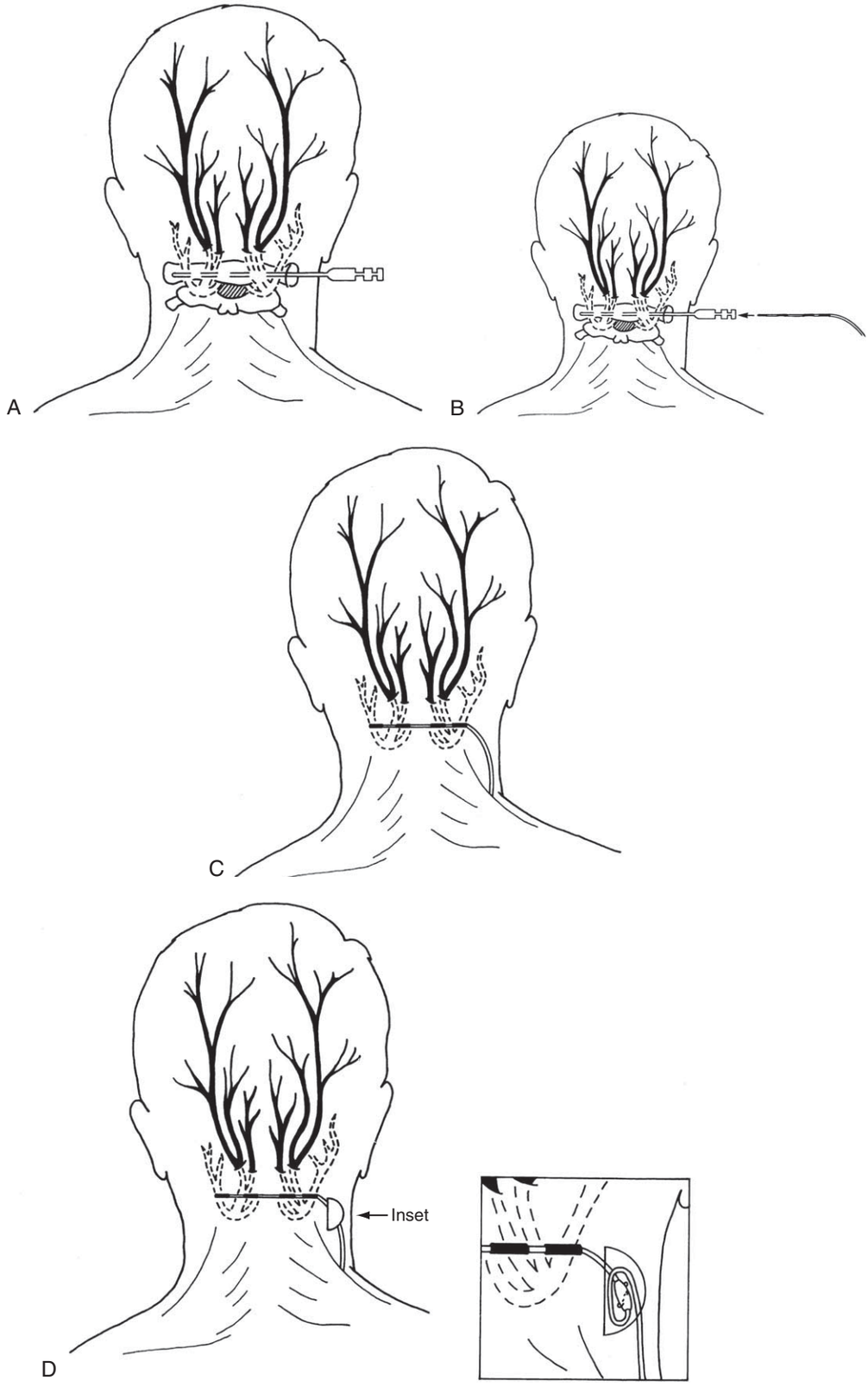


FIGURE 28-4

(A) Therapy placement at the C1 level during the procedure for occipital stimulation. (B) Electrode placement through the Tuohy needle. (C) Removal of Tuohy needle with the occipital electrode in place. (D) Lead anchored to fascia with an electrode loop to prevent migration.

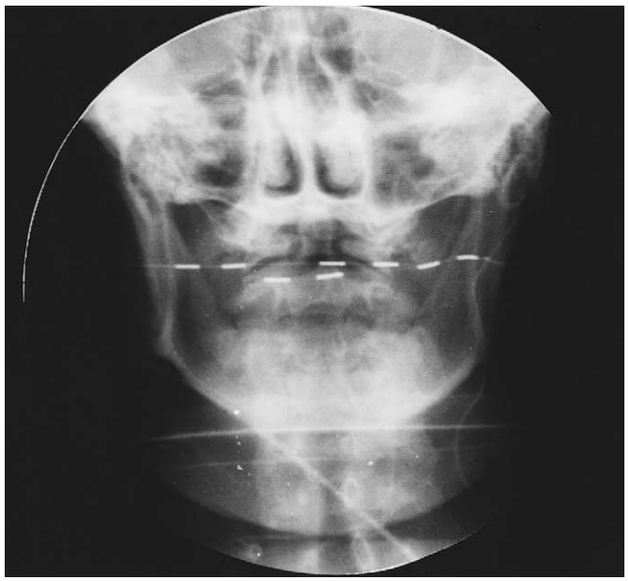


FIGURE 28-5
Two occipital electrodes in place for the patient with headaches.

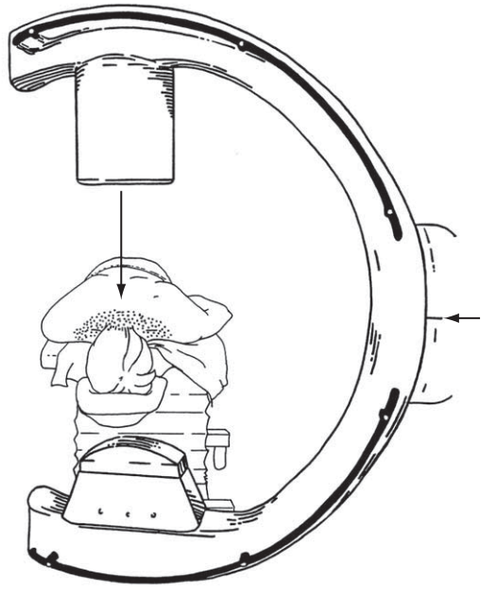


FIGURE 28-6
Position of the patient and the C-arm for cervical electrode placement.

- Further major surgical intervention is not indicated.
- No serious drug habituation problems exist.
- Psychiatric or psychological clearance has been obtained.
- The patient has primarily radiating extremity pain.
- Trial stimulation has been successful or will be done prior to permanent placement.

LABORATORY STUDIES

Not all patients require preoperative laboratory work. Depending on the clinical situation, it may be appropriate to obtain the following laboratory values:

- Complete blood count with platelets
- Prothrombin time, partial thromboplastin time
- Platelet function studies or bleeding times
- Urinanalysis, electrocardiogram, and chest radiograph when appropriate

PREOPERATIVE MEDICATION AND MONITORING

For preoperative medication and for monitoring, use the standard recommendations by the American Society of Anesthesiologists.

PROCEDURE

The prone position is commonly used (Figure 28-6) or lateral decubitus (see Figure 28-3).

Trial Placement of Percutaneous Cervical Spinal Cord Stimulation Electrode

As with all trial placements of SCS electrodes, an alert and communicative patient is essential to correct lead positioning. The patient should be made comfortable with local anesthetic infiltration at the insertion site. The patient should lie in the left lateral position. With the fluoroscopic C-arm in the anteroposterior (AP) view at the T1-T2 level, the spinous process and the patient's chest should be perfectly vertical or with a slight tilt forward. Prior to anesthetizing the skin, the most appropriate site of entry should be determined. At times it will be appropriate to enter the epidural space caudad to the T1-T2 level. For bilateral pain, a paramedian needle entry approach is still appropriate, with electrode placement at the midline. The direction of the shaft of the needle and the curved tip of the guide-wire influence where the electrode travels.

Using a paramedian approach with a shallow angle 1/2 inch off midline, the physician aims the needle at the target, moving toward the painful side. The use of an angled paramedian approach for the needle, as opposed to a midline approach, is a recent improvement in technique, which speeds lead placement considerably in the upper cervical area (Figure 28-7A).

The C-arm is then rotated to the lateral view to look down on the patient. The needle is advanced with the stylette in place to the ligamentum flavum area. Then, by rotating the C-arm back to the AP view, any necessary corrections can be made to get the needle to the target area (Figure 28-7B). The stylette is then removed, and a syringe is attached to the needle, which is then advanced into the epidural space using the "loss-of-bounce" technique.

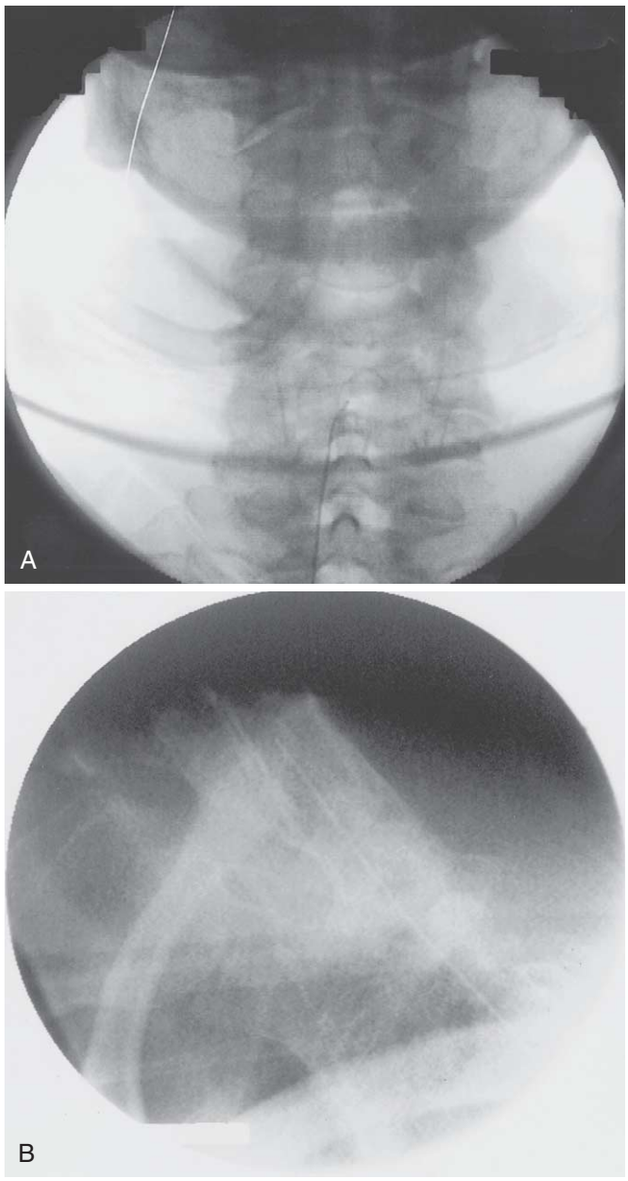


FIGURE 28-7
Anteroposterior (A) and lateral (B) views of cervical trial electrocatheter in place ready to be stimulated.

With the syringe filled with 4 ml of preservative-free saline or 2 ml of air, the physician bounces the plunger constantly with the right hand while advancing the needle with the left hand until there is a loss of bounce. Confirmation of entrance into the epidural space is frequently performed with easy placement of the guide wire into the dorsal epidural space. The physician then attempts to pass the electrode. If the electrode will not pass easily, it is probable that an insufficient portion of the needle has entered the epidural space. The needle advanced further or repositioned to allow the electrode to pass easily. The target area for the most distal electrode should be just off midline to the painful side, with placement depending on the pain pattern. If there is specific nerve root involvement, such as in cases of postherpetic neuralgia or intercostal neuritis, the electrode may be placed on the nerve root itself lateral to the spinal cord for entry zone stimulation. For facial pain, the electrode is frequently advanced to the C1-C2 segment.

Stimulation Testing

One can use a trial screening lead during the screening trial. Once the screening lead is positioned, it is connected to a temporary, external power source (screener). If the patient does not experience satisfactory paresthesia, one should reposition the lead and try again. When both the patient and physician are satisfied that the stimulation coverage is satisfactory, then complete the circuits using an external ground patch on the patient's abdomen and tape the screener to the patient's body. To verify the electrode position, AP and lateral radiographs should be obtained (Figure 28-8).

For the next 24 hours following lead placement, the patient wears a soft cervical collar and is instructed to remain flat while sleeping to reduce the chance of lead migration. Patients are sent home to test the stimulation with the screener for 4–5 days as they go about their normal activities. When patients are clear that they have had good relief or clear that they have not had good relief with the test stimulation, the sutures are cut and the lead is removed. If patients are not clear whether they have had 50% relief and they feel that they need more time, or if additional stimulation programming can improve the stimulation and pain control, it is reasonable to extend the trial if there are no signs of infection. Those who elect to have a permanent cervical SCS system implanted are scheduled for permanent implant at a later date.

PERMANENT IMPLANTATION OF CERVICAL SPINAL CORD STIMULATION ELECTRODE

Following a successful screening and month-long consideration period, if the patient elects to have a permanent SCS system implanted, one can use either a percutaneous electrode or a laminectomy lead. This largely depends on the comfort level of the implanting physicians, as well as the degree of any spinal stenosis that may be present, previous migration of an electrical array, power use, and pain with the use of electricity (in an attempt to avoid ligamentum flavum stimulation). When the electrode is in place, it is connected to the screener and paresthesia patterns are tested. When excellent stimulation coverage is achieved, the lead is anchored using company specific anchors. One is then ready for implantation of the pulse generator. For additional information, see Figures 28-14 to 28-18 in the thoracolumbar SCS implantation technique section.

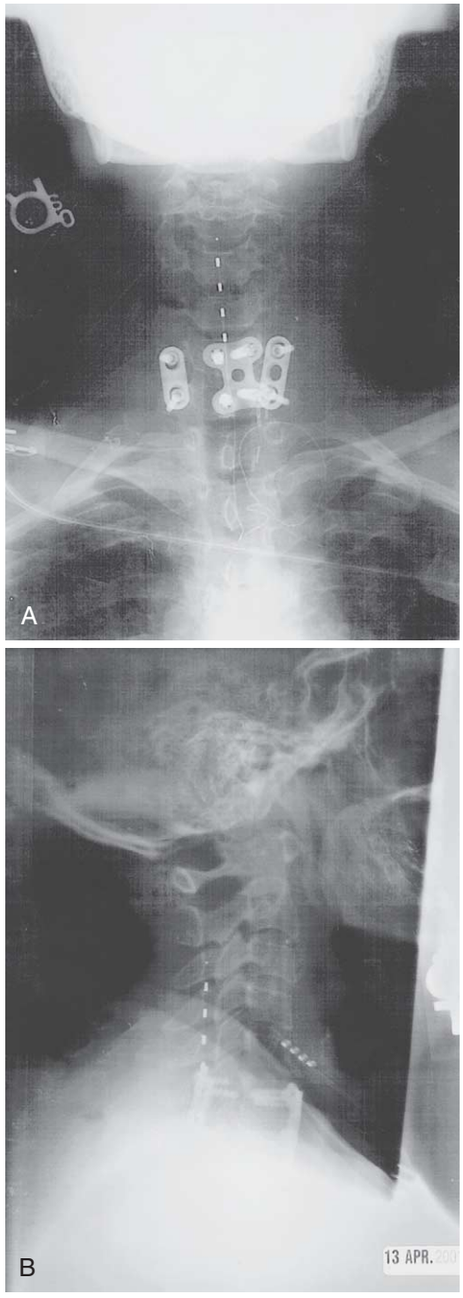


FIGURE 28-8

Anteroposterior (A) and lateral (B) views of quadripolar electrode in place in the cervical region.

OPTIMAL ELECTRODE PLACEMENT FOR VARIOUS PAIN SITES

Typically, good stimulation can be achieved using the following upper cervical placements for specific nerve involvement:

- C1-C3—facial pain
- C2-C3—upper neck
- C4-C5—radial nerve
- Just below C5—median nerve
- C6-C7—ulnar nerve

A percutaneous lead with a broad area of coverage can provide good stimulation for these areas with four- or eight-contact electrodes. When pain is bilateral, the lead can be placed at the midline or two separate electrode arrays (placed bilaterally on the spinal cord) can be used.

ANCHORING AND TUNNELING

Ideally, dissection of subcutaneous tissue using combined blunt and sharp dissection with Metzenbaum scissors is targeted at exposing the supraspinous ligament, which is shiny and striated, in contrast to the fat and subcutaneous tissue. A dry sponge is helpful in removing fatty tissue and exposing this target to fix the lead anchors. Failure to suture the anchors securely to the ligament and the lead to the anchor is the most common cause of lead migration. Traditional soft anchors have either a butterfly or a lead-through configuration. Tunneling rods of various configurations are supplied in the SCS system surgical kits. Depending on the type, they have different endpieces for tunneling and pulling through leads or extensions. With the patient in the prone position, many surgeons are placing the power sources in the posterosuperior aspect of the buttock.

POCKET FORMATION

Preparing the pocket for either an RF receiver or an implanted pulse generator is a relatively straightforward and simple procedure. After infiltration of the proposed incision with local anesthetic, an incision of appropriate length is made, the goal being to produce a pocket that is the right size for the device and for any extra lead or extension that may be coiled behind it.

All power sources should be placed no more than 1 cm (<1 inch) from the skin surface and implanted generators. Formation of these pockets can be accomplished mainly with blunt finger dissection, when necessary, by instrument dissection, or with the aid of bovie. General principles of tissue handling and hemostasis with electrocautery are standard. Pockets are best closed in two layers to prevent stress on the suture line from the implant. Integrity of the system should always be tested with the use of an impedance test before the patient leaves the operating room to detect easily correctable errors.

IMPLANTATION OF PULSE GENERATOR

There are two common placements for the pulse generator when the lead is placed in the upper cervical spine: the anterior chest wall, lower quadrant, same side as the pain, or just below the iliac crest away from the sacroiliac joint in the buttock. A pocket is made for the pulse generator.

A tunnel is made for the lead, and the lead and pulse generator are connected using an extension. One can keep the patient in the same position so that redraping is not necessary. It is important to use nonabsorbable sutures when suturing the pulse generator to the deep fascia to help prevent lead migration.

POSTIMPLANT PROCEDURE

Patients need to wear a soft cervical collar for the first 24 hours following surgery and are instructed to remain flat while sleeping to reduce the possibility of lead migration. During that time, the staff assesses them frequently to make any necessary adjustments in the stimulation. The patients are then discharged home with appropriate instructions for home care and a return visit in the outpatient clinic for wound inspection and, if necessary, for future removal and/or reprogramming of the SCS.

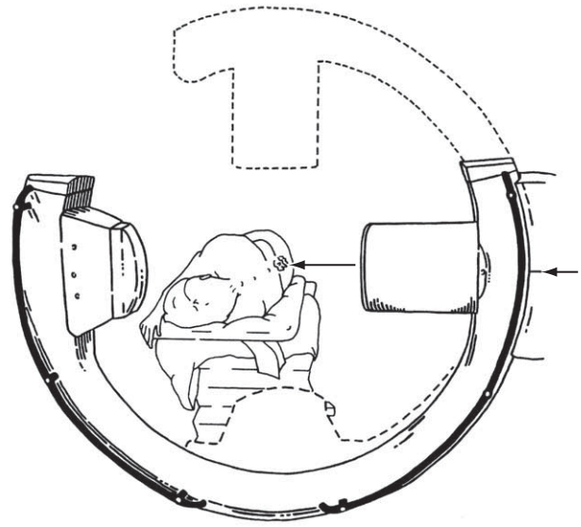


FIGURE 28-9

A drawing of the patient in the lateral position with the C-arm in anteroposterior and lateral positions for entry of Tuohy needle.

THORACOLUMBAR SPINAL CORD STIMULATION

ANATOMY

Anatomy of the epidural space is described in Chapter 13 and is not duplicated here.

INDICATIONS

In the United States, the primary indications for SCS are failed back surgery syndrome, sympathetically mediated pain, and sympathetically independent pain of complex regional pain syndrome.[9–13] In Europe, interest in SCS has been greatest in connection with treatment of chronic, intractable angina and pain and disability due to peripheral vascular disease.[13–22]

CONTRAINDICATIONS

The complications of thoracolumbar spinal cord stimulation are similar to other implantation procedures.

PROCEDURE

Once the trial for SCS is scheduled, the patient is seen in the clinic the day before surgery, at which time a thorough history and physical examination are performed and appropriate laboratory studies are obtained.

All patients should have undergone a thorough psychological assessment and clearance. On the morning of the procedure, the patient is admitted to outpatient surgery for the trial stimulation procedure and 24-hour observation. The patient is placed in the lateral position for electrode placement (Figure 28-9). The lateral position minimizes the risk of spinal cord damage caused by the patient moving back onto the needle suddenly.

Minimal intravenous sedation is given because it is important to be able to communicate with the patient while positioning the stimulating electrode. The initial dose of antibiotics is given prophylactically at this time. The patient is then prepared and draped in a sterile fashion, and local anesthetic is infiltrated at the site of entry.

A 15- or 16-gauge Tuohy needle to the epidural space at a point roughly approximately three segments below the target site using the three-dimensional technique: direction, depth, direction (Figure 28-10). An electrode array is placed through the needle to the epidural space and threaded to the appropriate level. A small bend in the electrode array just distal to the end facilitates steering of the electrode to a specific site. A trial-stimulating unit is connected to the proximal end of the electrode array to the power source. The primary goal during the testing period in the operating room is to evaluate the stimulation pattern obtained and confirm that there is a sensation of stimulation overlapping the painful area (Figure 28-11). Once adequate stimulation over the site of the patient's pain is achieved, the needle is removed under fluoroscopic visualization with care being taken to maintain the position of the electrode array (Figure 28-12). AP and lateral radiographs are frequently taken to document catheter position (Figure 28-13). The electrode is then secured to the skin with a suture or steristrip. The distal end of the catheter electrode is anchored with skin suture and dressing.

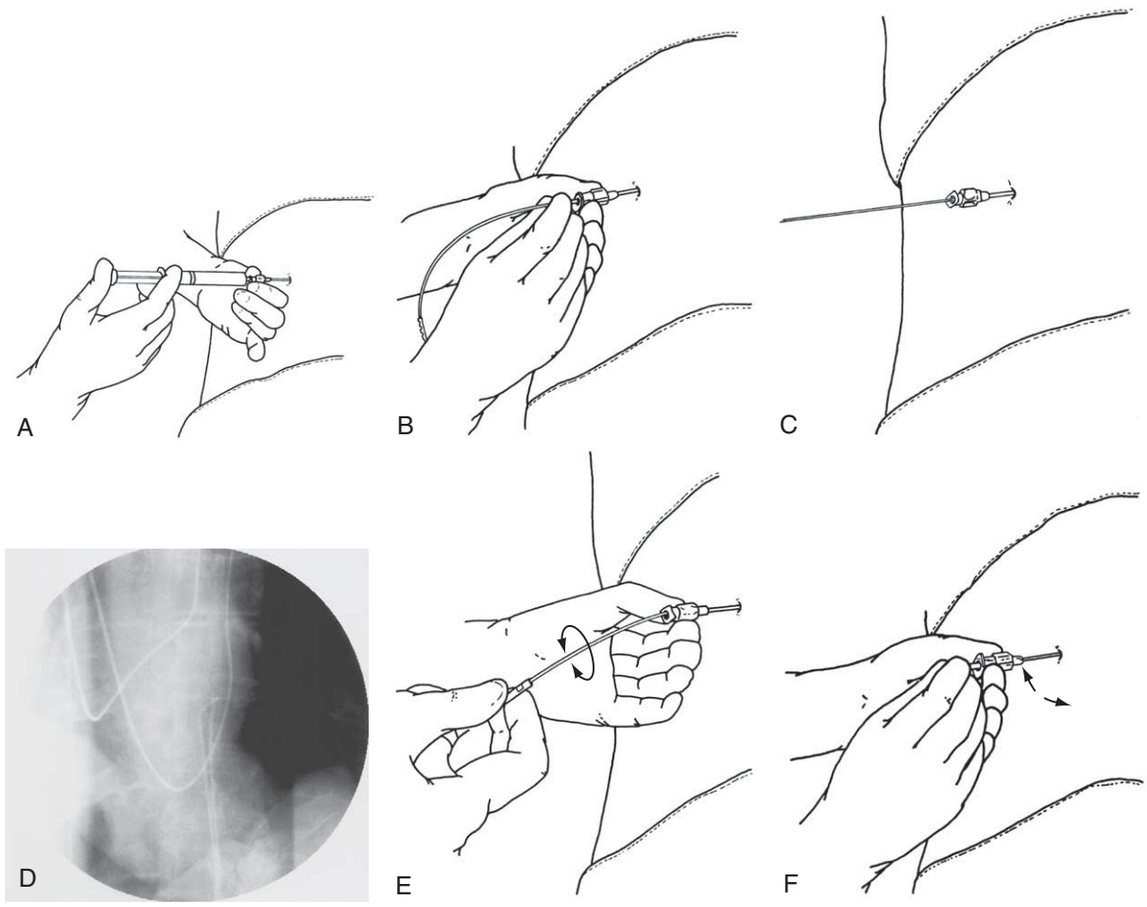


FIGURE 28-10

(A) Insertion of a Tuohy needle with the beveled edge facing cephalad. (B) Trial screening electrode inserted through the Tuohy needle. (C) If there is difficulty, a guidewire is inserted to facilitate the track. (D) Racz's electrocatheter in place in the thoracolumbar region for stimulation trial. E, The verification screening electrode is now connected to the stimulator screening box to test stimulation. F, Once the electrode is in good position, the Tuohy needle is cautiously removed without pushing the electrode any further.

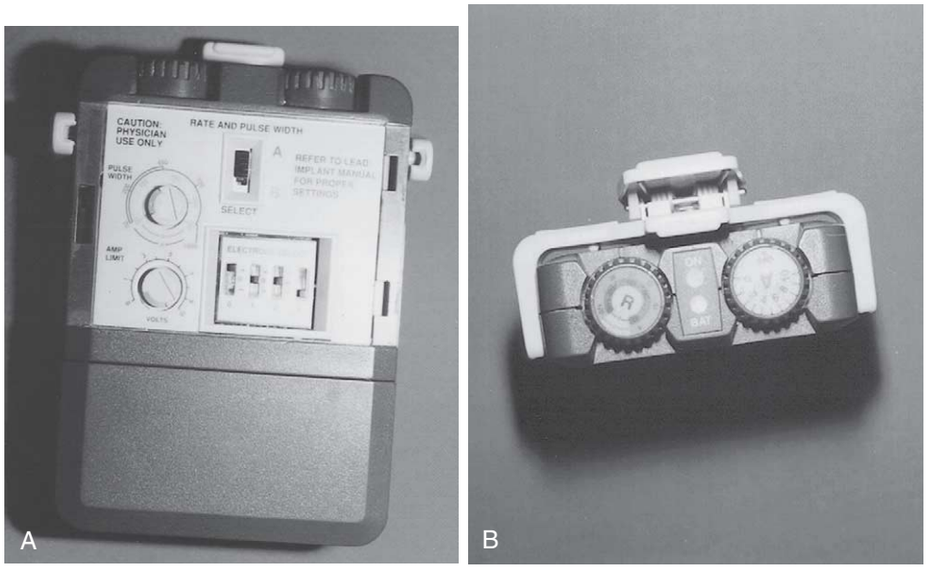


FIGURE 28-11

(A) The trial stimulator is used by the patient to evaluate the effect of stimulation for the next 5 days. (B) View from the top of the same screener.

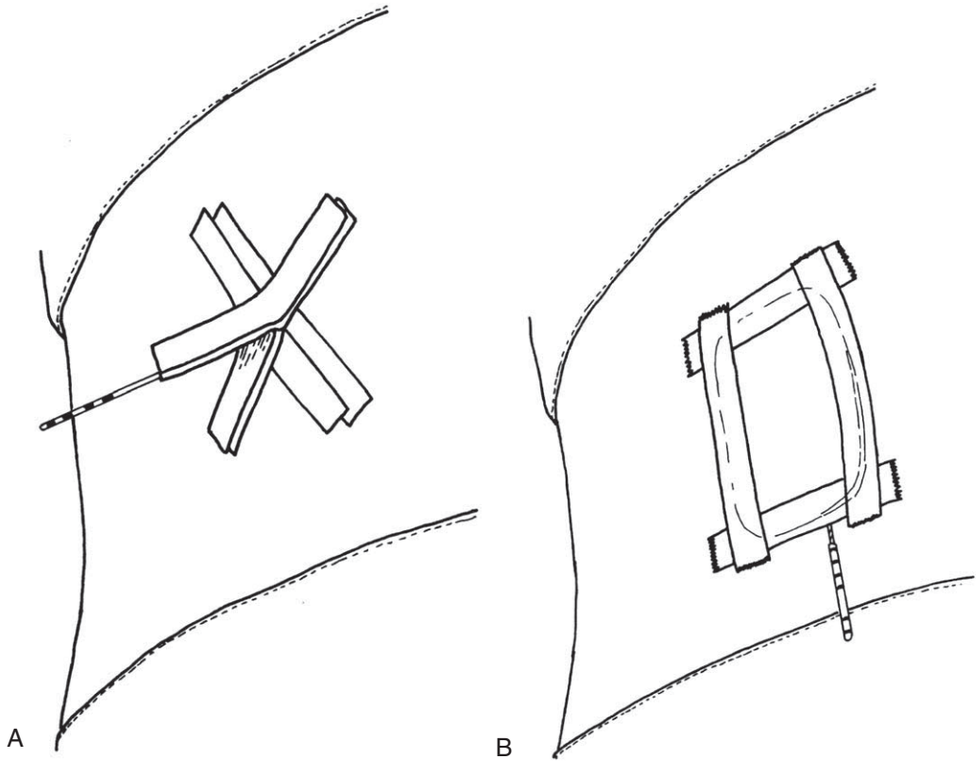


FIGURE 28-12
A and B, The distal end of the catheter electrode is permanently anchored with skin suture and dressing.

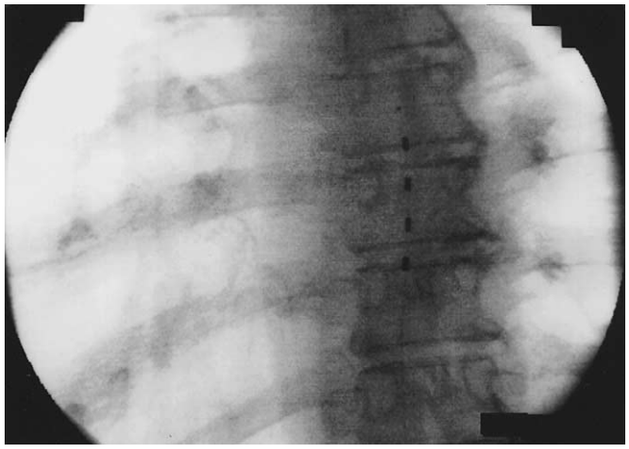


FIGURE 28-13
Medtronic single lead in place with four sites of stimulation.

PERMANENT PLACEMENT OF THORACOLUMBAR ELECTRODES AND PULSE GENERATOR

Once the type of electrode and style of system are chosen, the surgical technique for all implantation is similar. The practitioner must become familiar with the specific issues of each manufacturer's equipment and of the various models.

THORACOLUMBAR ELECTRODE PLACEMENT

For lead placement, the patient can lie in the lateral decubitus position, but increasingly the prone position is being used and a bolster placed under the thorax to promote adequate flexion of the spine and facilitate epidural lead placement. The patient is prepared and draped in the usual manner for surgery, with a strict aseptic technique. The site of entry of the epidural needle for patients with lower extremity, hip, or back pain can be variable but is frequently at the L1-L2 (Figure 28-14). Fluoroscopy is used to guide and confirm the needle of entry into the epidural space. Care must be taken to drape the fluoroscopy unit and to provide an extra side drape, to prevent contamination of the surgical field during cross-table views. Fluoroscopy helps guide placement of two leads, if needed, for bilateral pain distribution. A combination of 1% lidocaine and 0.5% bupivacaine with epinephrine is a useful mixture for both preoperative and postoperative analgesia. Once the needle is confirmed to be in the epidural space, a lateral view is taken to confirm that the electrode enters cephalad dorsally (Figures 28-15 and 28-16).

Two-stage initial lead placement or definitive placement of an SCS system after a successful trial proceeds with a midline or slightly paramedian skin incision after local anesthetic infiltration (Figure 28-17). Patients must be alert and responsive enough to report on stimulation coverage. Therefore, general anesthesia is contraindicated and appropriate

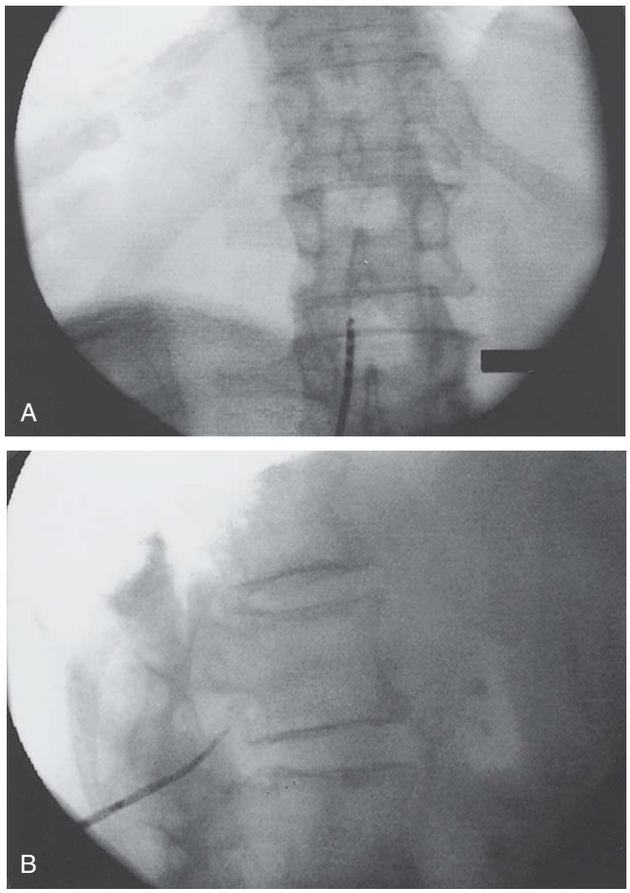


FIGURE 28-14

(A) Step 1. In the anteroposterior view, the site of entry of the Tuohy needle between L1-L2 interspace just lateral to the spinous process. (B) In the lateral view, the needle is confirmed to be in the epidural space after a positive "loss of bounce" technique.

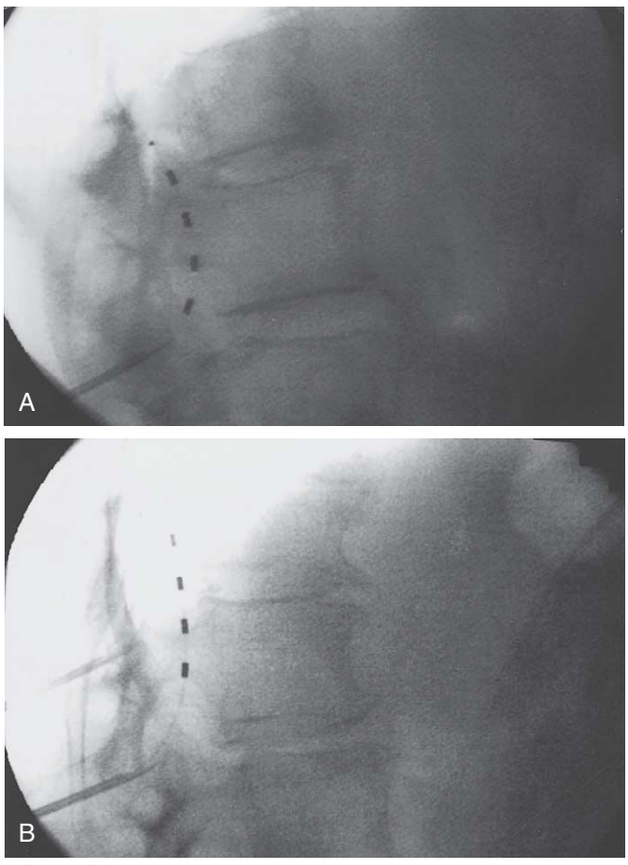


FIGURE 28-15

(A) When the electrode is inserted, the lateral view is important. The electrode has to remain posterior. In this radiograph the electrode has gone anteriorly and the tip is bounced back posteriorly. (B) In this radiograph the electrode is more posterior than in A.

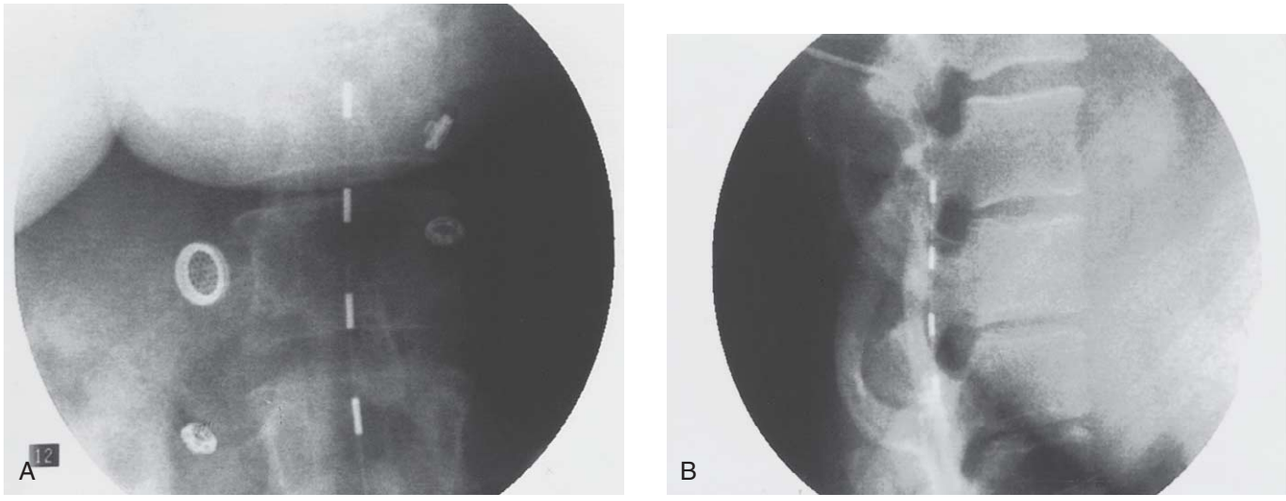


FIGURE 28-16

Anteroposterior (A) and lateral (B) views of correct quadripolar electrode placement.

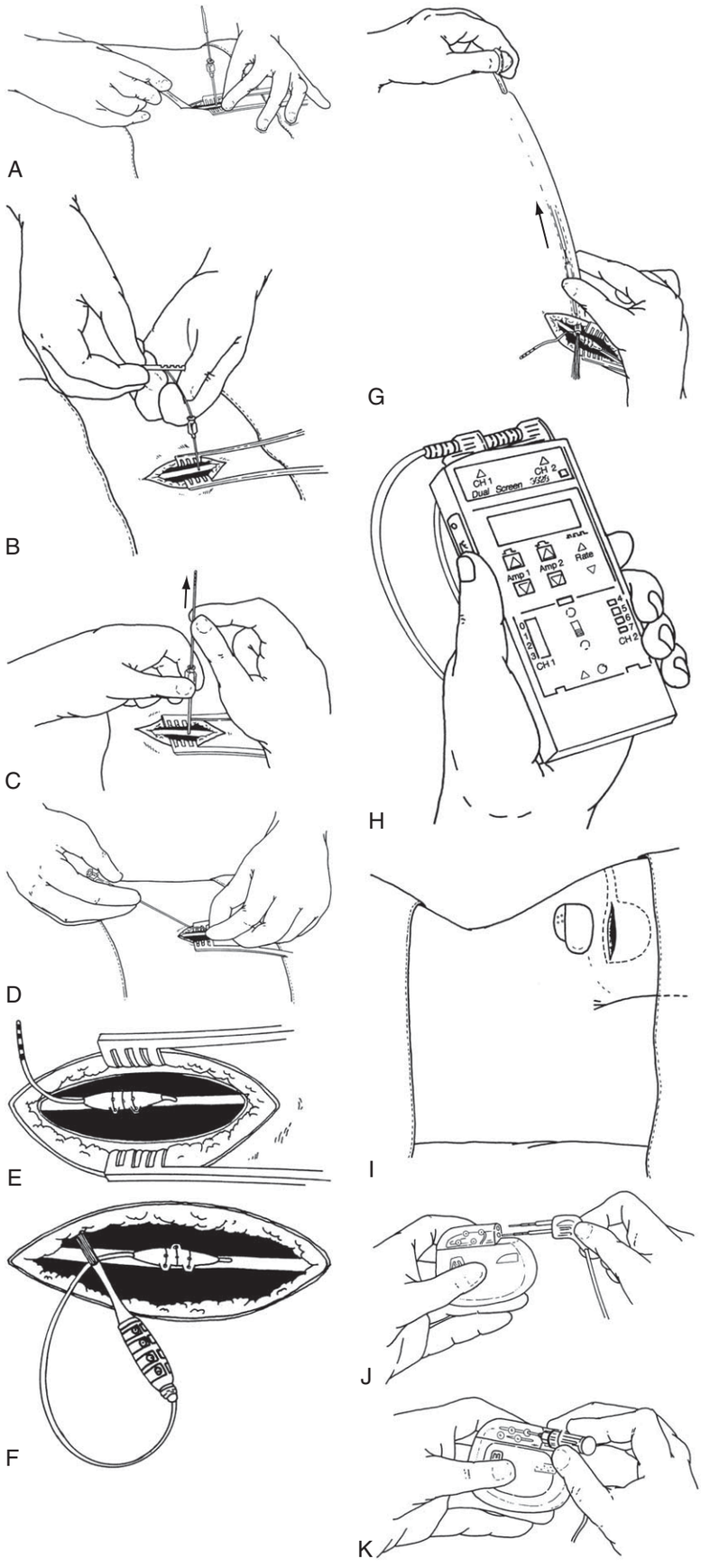


FIGURE 28-17

Placement of spinal cord stimulation system. (A) Making a 5- to 7-cm longitudinal incision. (B) Disconnecting the stylette from the lead. (C) Exposing the lead beyond the tip of the needle. (D) Slipping the Tuohy needle off the lead body. (E) Suturing the lead anchor. (F) Guiding the tool subcutaneously along the tunneling route and pulling the assembly through the passing straw. (G) Suturing the wide end of the extension connector. (H) Stimulator used after electrodes are in place to confirm the area of stimulation prior to starting the pocket. (I) Checking incision length. (J) Inserting the extension connector pins into the connector block. (K) Tightening each set's screw on the connector block.

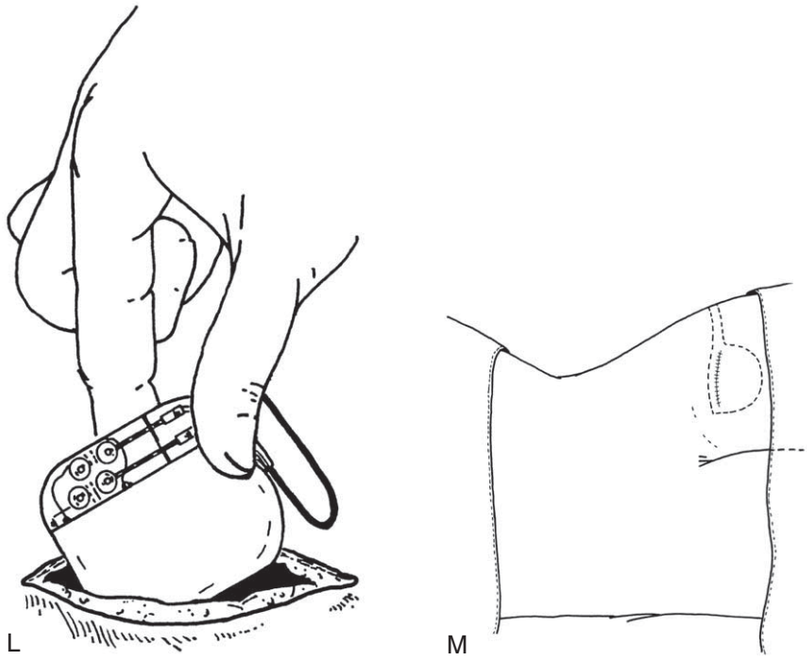


FIGURE 28-17

(L) Inserting the stimulator (implantable pulse generator [IPG]) into the pocket with the lettered side facing the skin. (M) IPG buried subcutaneously and the incision closed in a standard fashion.

and judicious sedation may be employed by the anesthesiologist. Entry into the epidural space, using the manufacturer's supplied modified Tuohy needle, is facilitated by a slightly paramedian approach. It is important to keep the angle of entry as shallow as possible, to more easily advance the lead cephalad. With a shallower angle, steering of the lead is easier because of the mechanical advantage it affords.

Fluoroscopy combined with the standard loss-of-resistance technique increases the chance of nontraumatic entry into the epidural space. Real-time imaging can often guide placement of the lead through resistance in the epidural space, along the way to final placement. A single lead should be placed slightly ipsilateral to the painful side and as close as possible to the physiologic midline for bilateral pain coverage. Coverage of the painful region with stimulation paresthesia determines the final lead placement. A dual lead may be necessary for better coverage in the same side or for bilateral coverage of the extremity, as well as for capturing axial low back pain. Various electrode placements are illustrated in Figure 28-18.

IMMEDIATE POSTIMPLANT CHANGES

The ideal stimulation pattern—and resulting pain relief—are often lost within the first few weeks after implantation. Periodic reprogramming then becomes essential. As the fibrous tissue invests the lead electrodes, resistance to delivery of the electrical impulses can increase. The result is the need to substantially increase the amplitude over time. This should be expected, and the patient made aware that it is a normal occurrence. This maturation process can often require reprogramming of the electrode array, pulse width, and frequency. The three-dimensional space surrounding the lead can be altered by the natural process of healing in a manner that renders the stimulator system ineffective, despite a successful trial.

Migration of the lead after maturation is much less likely, but it still can occur. "Electrical repositioning" of the electrode array recovers the optimal stimulation pattern. By varying the programs from time to time, accommodation can be avoided. Accommodation describes the phenomenon by which the body comes to "ignore" a steady, unvarying electrical stimulus over time. Patients who leave their stimulator systems on continuously may accommodate much more rapidly, causing the stimulation to become ineffective.

TISSUE CHANGES AFTER 4 WEEKS

Once the patient has passed the fourth postoperative week, the system can be said to have matured. The body has now formed a fibrous capsule around the various components of the implant, which is less likely to migrate or produce any of the complications mentioned in the previous section. Several potential difficulties still lie in wait for the unsuspecting physician implanter.

SACRAL NERVE STIMULATION

ANATOMY

The sacrum is a large, triangular bone situated below L5 (Figure 28-19). Its apex articulates with the coccyx. Its anterior surface is concave. Anteriorly, four transverse

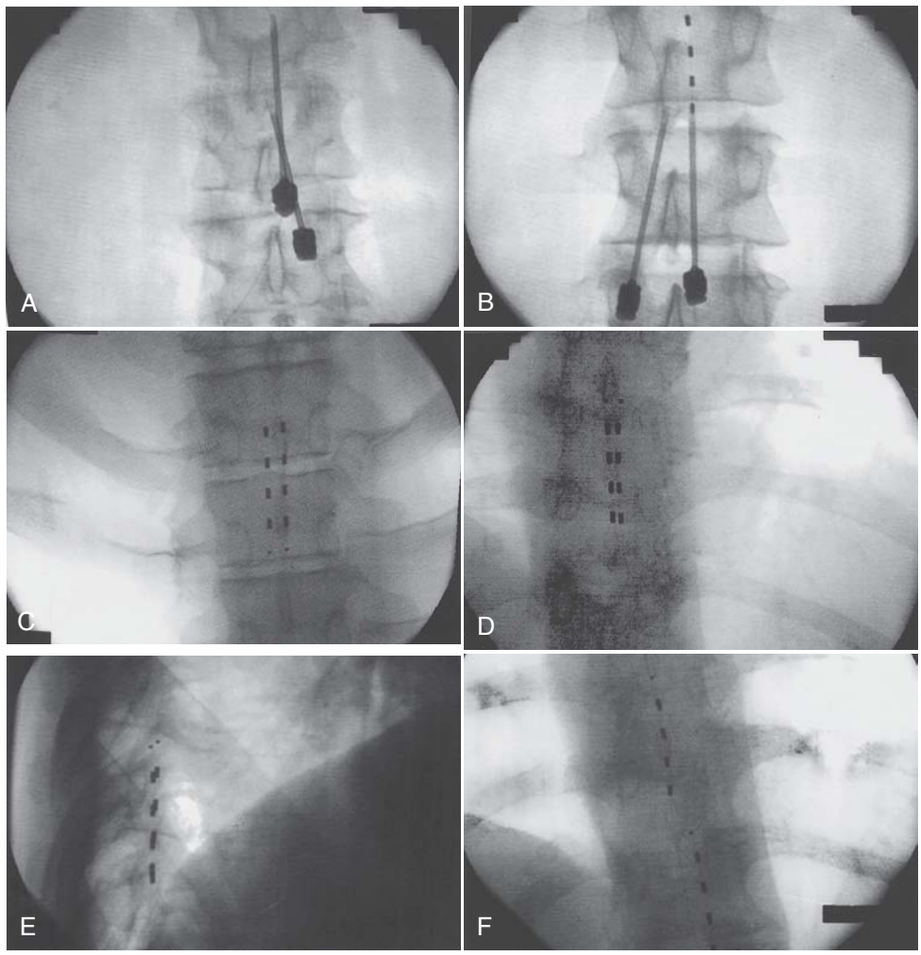


FIGURE 28-18

(A) When two electrodes are needed, the Tuohy needles could be entered at two different levels, as shown. (B) Another way of inserting Tuohy needle at the same level. (C) This radiograph shows dual leads off of midline. (D) Anteroposterior view in the radiograph shows dual leads close to the midline. (E) Lateral view of two leads in place. (F) When a larger area needs to be stimulated, the leads can be placed one over the other, as shown.

ridges cross its median part. The portions of the bone between the ridges are the bodies of the sacrum. There are four anterior sacral foramina through which the sacral nerves exit and lateral sacral arteries enter. The posterior surface of the sacrum is convex. There are rudimentary spinous processes from the first three or four sacral segments in the midline. The laminae unite to form the sacral groove. The sacral hiatus is formed by the failure of the laminae of S5 to unite posteriorly. The tubercles that represent remnants of the inferior articular processes are known as the sacral cornua; they are connected inferiorly to the coccygeal cornua. Laterally, one can identify four dorsal sacral foramina. They transmit the posterior divisions of the sacral nerves. The sacrum may have many variations. The bodies of S1 and S2 may fail to unite or the sacral canal may remain open throughout its length.

INDICATIONS

- Voiding disorders (urinary incontinence, urinary retention, voiding dysfunction)
- Chronic pelvic pain (interstitial cystitis, pudendal neuralgia, vulvodynia)

CONTRAINDICATIONS

- Local infection
- Any infection involving bladder or pelvis
- Coagulopathy

PREPARATION OF PATIENT

Examine the patient for superficial infection in the surgical area and for distorted anatomy that may affect performance of the procedure.

Laboratory Studies

- Complete blood count with platelets
- Prothrombin time, partial thromboplastin time
- Platelet function test or bleeding times
- Urinalysis
- Magnetic resonance imaging (optional) for canal size

Preoperative Medication

For preoperative medication, use the standard recommendations for conscious sedation by the American Society of Anesthesiologists.

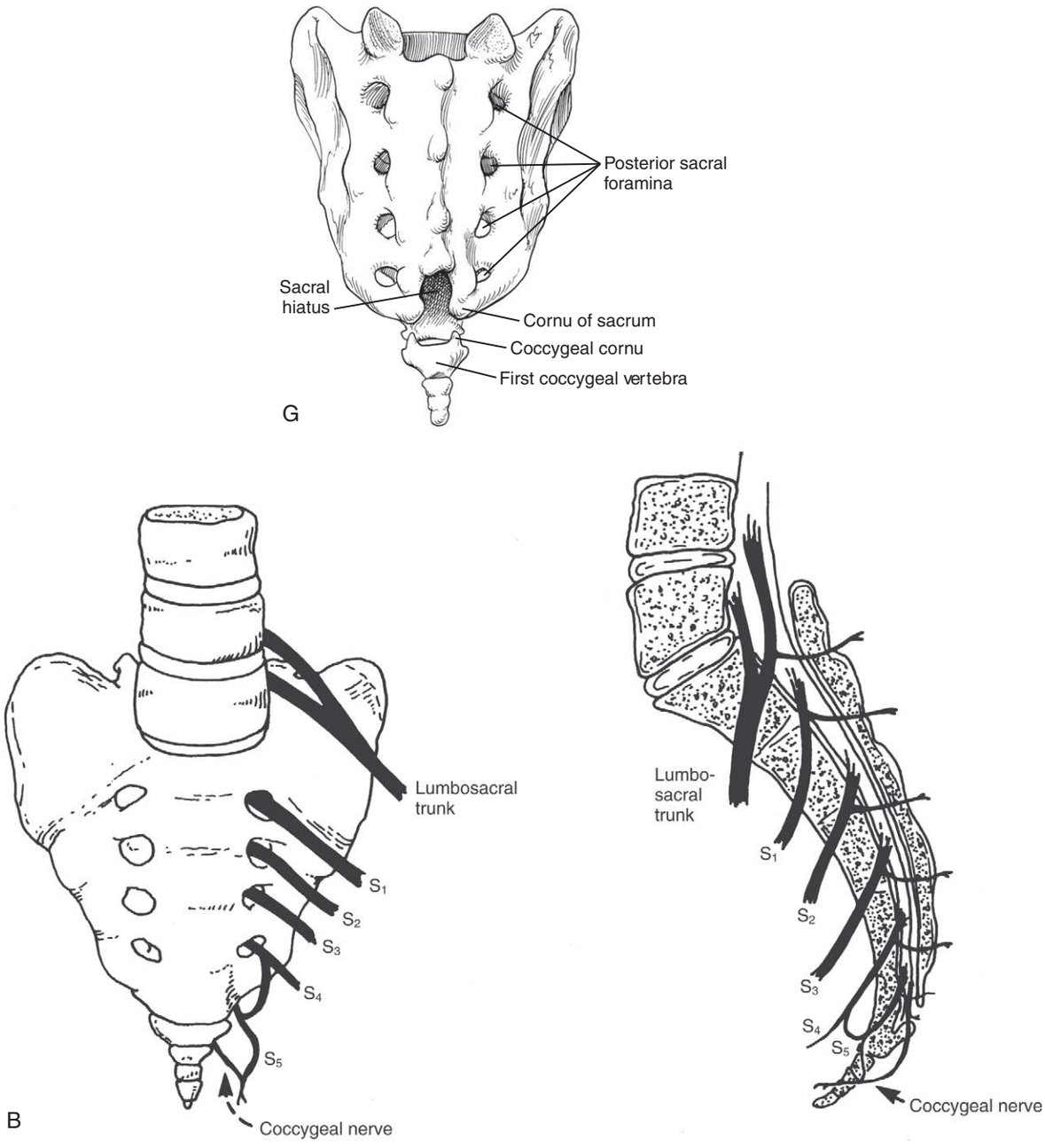


FIGURE 28-19

(A) Anatomy of the sacrum. (B) Anterior view of the sacrum with the anterior primary rami exiting. Note the presence of the coccygeal nerve. (C) Lateral view of the sacrum shows both the anterior and posterior primary rami of the sacral nerves exiting their respective foramina.

TRIAL SACRAL STIMULATION

Patients must undergo acute percutaneous electrical stimulation of the ventral ramus at the level of the third (S3), and possibly S2 and S4 sacral foramina, to establish functional integrity of the sacral nerves, to locate the nerves that can elicit beneficial responses and confirm that nerve stimulation elicits contractions of the appropriate muscle groups. The search for the S3 foramen can be done either with anatomic palpation or fluoroscopy (Figure 28-20).

If adequate responses are obtained during the acute testing, then test stimulation needs to be conducted for several days (not to exceed 7 days). Stimulation is achieved by replacing the stimulation needle with a temporary screening lead placed through the needle and connected to the same external screener that is used during the test phase. The amplitude of stimulation and "on/off" are controlled by the patient.

The patient controls the amplitude of the stimulation so that it is sensate but painless. Patients are informed that

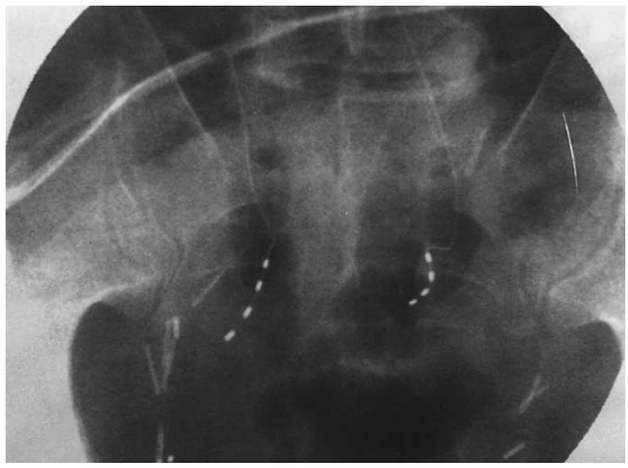


FIGURE 28-20

Anteroposterior view of electrodes through S3 foramina bilaterally for trial testing of the area of stimulation and analgesia.

the sensation can change according to their positioning (sitting, standing, lying) and movements. Continuous stimulation is used (day and night, 10 Hz, 210 milliseconds), and patients must be educated to manage power according to the severity of symptoms and feelings and to report all modifications of stimulation parameters on the voiding diary.

PERMANENT SACRAL STIMULATION

Sacral nerve root stimulation is performed under light sedation, using local infiltration. Patients are positioned prone on a radiolucent table. Electrodes are placed percutaneously in the epidural space under fluoroscopic guidance at the appropriate level as determined by patient paresthesia. The lumbar and sacral nerve roots are approached in a caudal direction (retrograde approach). In this approach, a Tuohy-type needle is inserted into the skin at a level superior rather than inferior to the interlaminar space and advanced in a caudal rather than a cranial direction. To reach sacral nerve roots, it is imperative to insert the needle in the same direction that the nerve root travels within the epidural nerve root sleeve. Therefore, the needle has to be placed in a paramedian fashion and advanced caudally into the epidural space. Once the epidural space is penetrated, the electrode will then follow the path of the needle and continue to advance toward the targeted nerve root. The experts in this technique (Quattrode or Octrode) recommend multielectrode systems. Initial positioning is according to the anatomically predicted locations appropriate for the patient's pain. The exact position of the physiologic target varies and can be determined only intraoperatively by communicating with the patient about the location of the perceived paresthesias as different neural targets are stimulated. The perceptual threshold is defined as the stimulation amplitude at which level the patient first perceives the paresthesias. The discomfort threshold is the amplitude at which the paresthesias become uncomfortable. In all patients, criteria for a successful trial generally includes greater than 50% reduction in pain level, reduced consumption of pain medications, and increased activities of daily living. If the patient had a successful trial, then permanent sacral electrodes are implanted.

COMPLICATIONS

- Epidural hematoma
- Spinal headache
- Infection
- Lead migration
- Seroma or stitch abscess
- Wound dehiscence

CLINICAL PEARLS

If the patient has had previous scar formation that makes it difficult to pass the electrode, the physician may put a curve at one end of the guidewire to use as a tool for opening the space. Alternatively, a tunneling catheter with a soft spring tip and a small syringe, such as the Tun-L-Kath, can be used to open the space using 1 ml of saline. When the space is opened, the lead can be advanced.

EFFICACY OF SPINAL CORD STIMULATION

There are numerous retrospective studies that tout the efficacy of SCS. These studies or reports usually lump together patients who have various pain syndromes of different kinds. The substance of many of these studies suggests that, for many such syndromes, efficacy is approximately 60% and relief lasts about 2 years. After 2 years, for whatever reasons, in some patients efficacy seems to fall off. SCS is effective not only for neuropathic pain of appendicular and axial origin but also for complex regional pain syndrome, peripheral vascular disease, and the pain of intractable angina.

The most comprehensive published study is the one by North and associates,[23] in which patients with up to 18 years follow-up were subjected to an extensive questionnaire.

If one considers only the most stringently analyzed series, good to excellent results are reported in 40–60% of the implanted patients.[23–27] In these published series, the percentage of patients with no or minimal results varied from 30–60%. Other less rigorous general series in the literature claim good to excellent results, ranging from 47–66%, and failure rates ranging between 20 and 39%.[28,29] Limiting success to only reports of good/excellent relief, however, would not do justice to an average of 20% of implanted patients who still use the stimulator with moderate pain relief. See Table 28-2 for studies of SCS by various groups.

TABLE 28-2 Most Relevant Published General Series on Spinal Cord Stimulation for Chronic Pain Management

Primary Author	Publication Year (Time Span of Implants)	Type of Study	Number of Implanted Patients	Average – Follow–up (Years)	Extended Trial Screening	RESULTS None/Poor	Fair	Good/Excellent
Barolat Group A[31,a]	1998 (1985–1992)	3	102	3.8	No	51	15	34[b]
Barolat Group B[31,c]	1998 (1985–1992)	3	80	3.8	Equivalent to yes	37.5	11.5	51[b]
Burchiel[32]	1996 (1990–1992)	Retrospective	70	1	Yes	22	43	35
Devulder[33]	1991 (1982–1990)	1	69	At least 2	Yes	30	11	59
Koeze[25]	1987	3	26	22	No	30	20	50
Kumar[28]	1991 (1980–1989)	n/a	94	3.4	Yes	44	n/a	56
Kupers[34]	1994	3	70	3.5	Yes	27	21	52
Ohnmeiss[26]	1996	Retrospective	40	2	No	30	44	26
Meglio[35]	1989 (1979–1986)							
Nielson[36]	1975 (1969–1973)	2	130	[d]	Yes	39	12	49
North[23]	1991 (1971–1990)	3	171	7	Yes	37	11	52
Racz[37]	1989 (1984–1986)	2	26	1.8	No	21	18	61
Ray[38]	1982 (1976–1982)	1	78	1.8	Yes	23	20	57
Siegfried[39]	1982 (1972–1980)	1	89	4	Yes	39	24	37
Simpson[29]	1991	2	60	2.3	24 Yes; 26 No	20	33	47
Spiegelmann[27]	1991 (1987–1989)	3	30	1	Yes	36	4	60

[a]All implanted patients.
[b]Patients who rated their pain relief at 50% or better during survey.
[c]Implanted patients, excluding the ones who never experienced any pain relief (equivalent to patients in published series with trial screening who passed the trial and underwent implantation).
[d]In 32% of patients, less than 1 year, in 45% of patients more than 1 but less than 2 years, and in 24% of patients more than 2 years.
Patients who rated their pain relief at the survey between 25% and 49%.
1, detailed data; 2, detailed data + methodology of data collection clearly specified; 3, detailed data + methodology of data collection clearly specified + survey by disinterested third party.

The psychological preparation of the patient for the procedure seemed to have a substantial impact on the results. The author believes that this is due in part to the elimination of patients who have frank psychopathology. In a larger percentage, however, the better results are to be explained by the fact that patients who underwent psychological counseling came to the surgical implantation better prepared mentally and with a more realistic approach. The author feels that administering psychological testing is just as important as providing extensive psychological support before, during, and following the implantation procedure. Therefore, continuing psychological screening as an integral part of SCS implantation program is appropriate.[30]

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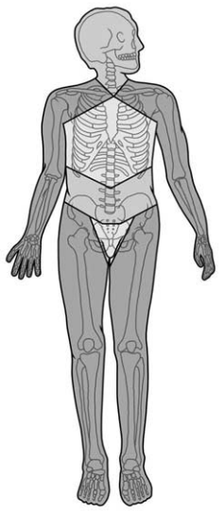
C H A P T E R



29 Intrathecal Drug Delivery Systems

PETER S. STAATS

HISTORY

In 1979, Wang and colleagues[1] reported that the use of morphine for cancer-related pain at doses of 0.5–1 mg resulted in excellent pain relief for 8–30 hours. Snyder demonstrated that the efficacy was largely due to a receptor of the morphine to an opiate receptor. Yaksh[2] documented the physiologic basis of the pain relief produced by the intraspinal administration of opioids was determined by the modulation of inhibitory mechanisms occurring at the level of the spinal cord.

Opioids produce a profound inhibition of the evoked discharge of spinal nociceptive neurons, resulting in a significant elevation of the pain threshold in animals.[3] At analgesic doses, spinal opioids, unlike local anesthetic agents, have no effect on the response to light touch, autonomic outflow, or voluntary motor function. The analgesic functions of intraspinally administered opioids are dose dependent and stereospecific. Opioids have a highly regular structure-activity relationship and are antagonized in a dose-dependent fashion by naloxone. This highly regular pharmacology suggests an effect mediated by receptors that are located in the spinal cord. Opiate-binding studies revealed high levels of binding in the substantia gelatinosa, where the bulk of the small primary afferent fibers terminate. The local action of morphine in the substantia gelatinosa inhibits the discharge of nociceptive neurons, thereby inhibiting the transmission of pain.[3,4]

Although percutaneous externalized epidural or even intrathecal catheter placement is feasible for short-term treatment, vulnerability to infection and economic considerations preclude serious considerations for long-term use (>3 months). It may be possible to decrease infection rate and prolong an externalized trial by tunneling the catheters for prolonged administration of analgesics.[5,6]

Coombs and coworkers[7] and Poletti and colleagues[8] initially described the use of an implanted reservoir that, on repeated compression, delivered a bolus of medication into the epidural space. Percutaneous injection of an implanted infusion port connected to a spinal catheter was also described.[9] Theoretical and practical objections to bolus dosing arose when primate studies indicated that tolerance to the opioids developed more rapidly when they were delivered in this manner.[10] These techniques also required sufficient personnel to obtain and administer the medication, primarily morphine, on an outpatient basis. Infusion ports could be connected to external pumps, which avoided the risk of rapid tolerance but resulted in the patient's discomfort and increased risk of infection.[11]

ANATOMY

Usual lumbar anatomy knowledge is required for intrathecal catheter placement. In addition, one must be aware of the anatomic structures related to performance of a chronic intrathecal infusion of sterile, preservative-free morphine sulfate or other commonly used analgesics. Whenever possible the physician should enter the intrathecal space below the level of the spinal cord ends (usually L1). However, there are times when accessing the intrathecal via posterior approach in the lumbar space may be impractical. For example, when patients have undergone a posterior fusion that limits access to the posterior epidural space in the lumbar spine, the physician may make the decision to enter the space in the thoracic spine, or when a high cervical catheter is planned it may not be possible to thread a catheter adequately. The physician must, however, recognize the increased risks of spinal cord injury with this technique.

The field is somewhat divided on the use of catheters placed above the end of the thoracic spine. One school of thought is that by using lipophylic agents, one can decrease total dosage analgesics and catheters placed in a more cephalad position for upper extremity and upper

thoracic pains. Another school of thought attempts to keep the catheter in the lumbar spine, mitigating against the risks of granuloma formation.

PHARMACOLOGY

The technical placement of an intrathecal catheter and pump is the easy portion of this procedure. Understanding the appropriate patient and indication for implantation, the method of performing a trial and pharmacologic agents to administer via the pump are much more challenging. While at this point there are only three drugs approved by the U.S. Food and Drug Administration for intrathecal drug delivery (morphine, baclofen, and ziconotide), a knowledge and implementation of other analgesics are necessary to optimize outcomes. Other drugs (such as hydromorphone, other lipophylic opioids, clonidine, and local anesthetics) appear to be safe and effective in treating pain when delivered into the intrathecal space. Guidelines outlining appropriate analgesics and doses should be consulted.[12] Chronic intrathecal infusion of baclofen injection is accomplished for severe spasticity from a variety of etiologies.

INDICATIONS AND CONTRAINDICATIONS

In general, chronic intraspinal infusion therapy using implantable drug administration systems has been reserved for patients whose condition is considered chronic, and have failed more conservative therapies. Patients must have either inadequate pain control or intolerable side effects on systemic opiates and adjuvant therapy.[13]

Patients with cancer-related pain can have excellent pain relief with intrathecal therapy, with studies demonstrating an improvement in pain control, side effects, and probably an improvement in life expectancy in patients randomized to receive intrathecal therapy over maximal medical management alone.[14] General guidelines for considering intrathecal therapy include an expected 3-month survival. This is based on data demonstrating an improvement in cost efficacy with the use of a totally implanted intrathecal device compared with an externalized infusion of analgesics.[15]

The indication for use of implantable drug administration systems then includes the treatment of chronic pain of both cancer-related and non–cancer-related varieties.

INCLUSION CRITERIA

Pain type and generator appropriate
Demonstrated opioid responsivity
No untreated psychopathology that might predispose to an unsuccessful outcome
Successful completion of a screening trial

EXCLUSION CRITERIA

Absolute
Aplastic anemia or other
Systemic infection
Known allergies to materials in implant
Known allergies to intended medications
Active intravenous drug abuse
Psychosis or dementia
Infection at proposed implantation site
Relative
Emaciated patient
Ongoing anticoagulation therapy
Active bleeding diathesis (would need correction prior to instrumentation of spine)
Child before fusion of epiphyses
Occult infection possible
Recovering drug addict
Opioid nonresponsivity (other drugs may be considered)
Lack of social or family support
Socioeconomic problems
Lack of access to medical care

EQUIPMENT

- Implantable intrathecal infusion pump
- Connecting tubing
- Intrathecal catheter (if separate)
- Appropriate surgical instruments for implantation

DRUGS

- Local anesthetics for infiltration
- Drug to be used for intrathecal infusion

PATIENT PREPARATION

Laboratory studies that should be obtained include complete blood count with platelets, prothrombin time, partial thromboplastin time, and platelet function studies and bleeding time. For preoperative medication, use the standard American Society of Anesthesiologists (ASA) recommendations for conscious sedation. Use the standard ASA-recommended monitoring protocol.

PROCEDURE

Implantation may take place with the patient under general or local anesthesia with monitoring. Local anesthesia is often preferred in an outpatient setting because it lends itself to rapid recovery after the procedure. When general anesthesia is chosen, the use of muscle relaxants is

frequently deferred until after the catheter is threaded into the intrathecal space. The steps are as follows:

1. Before the implantation, spend some time with the patient to decide on the side and location of the pump. About the only area amenable to the implantation of these generally large devices is the right or left lower quadrant of the abdomen. The anatomic constraints tend to be the iliac crest, the symphysis pubis, the ilioinguinal ligament, and the costal margin. These structures should not touch the pump when the patient is in the seated position. This task is easier with obese patients and can be difficult with cachectic cancer patients. On the other hand, suturing the pump in place in morbidly obese patients can be challenging. If the pump is not clearly anchored or secured, it is likely to rotate, running the risk of catheter malfunction or dislodgment.
2. After the mode of anesthesia is chosen, position the patient in the lateral decubitus position on the operating table with the side of implantation upward. Most physicians premedicate with intravenous antibiotics prophylactically. The back and abdomen should be steriley prepped and draped. At this stage, C-arm fluoroscopy is usually necessary to confirm access to the intrathecal space, oblique entry to the intrathecal space and eventual position of the tip of the catheter (Figure 29-1).
3. Position the instrument to permit an anteroposterior view, allowing easy lumbar puncture and identification of the catheter tip level (Figure 29-2).
4. Depending on body habitus, make a 5-cm incision in the skin down to the dorso-lumbar fascia in the lumbar spine. Through the exposed tissue, place a Tuohy needle into the intrathecal space using a paramedian approach (Figure 29-3). A gentle oblique angle optimizes flow and decreases the risk of catheter kink or fracture.
5. Document a good flow of cerebrospinal fluid (CSF), and clamp the catheter to the drape to prevent CSF loss.
6. Pack the incision with an antibiotic-soaked sponge.
7. If the existing catheter is to be used as the permanent delivery catheter, as in the screening technique, place the patient on the operating table in the decubitus position, with the implantation side upward and the exiting screening extension catheter downward.
8. Clamp the intrathecal catheter to prevent CSF loss. The rest of the implantation proceeds in the usual manner (Figures 29-4 and 29-5).
9. Turning attention to the lower quadrant of the abdomen, make a 10-cm incision down the underlying subcutaneous fat layer. Fashion a subcutaneous pocket large enough to admit the particular pump being used. Generally, if all four fingers can be admitted to the metacarpophalangeal joints in the pocket, it is large enough (Figure 29-6).
10. Undermine the upper side of the incision roughly the width of the pump or about 2.5 cm to allow closure without tension. The eccentric location of the pocket allows the pump to be placed in such a fashion that the refill port is clear of the incisional scar and easier to locate. An ideal pocket is one that allows the pump to be placed in such a fashion that the refill port is clear of the incisional scar and easier to locate. Another aim is to have a pocket that allows placement of the pump without struggle but is tight enough to aid in preventing pump rotation. The depth of the pocket below the skin is critical for programmable pumps. A depth greater than 2.5 cm may not allow reliable telemetry and will make pump refilling at a later date more difficult.
11. In fashioning the pocket, maintain meticulous hemostasis to avoid postoperative hematoma formation. At this point, pack the pocket with an

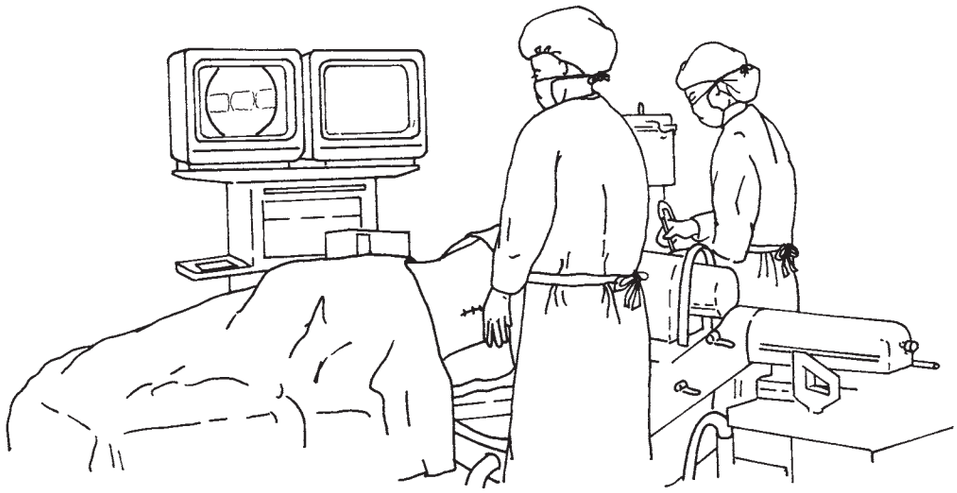


FIGURE 29–1
Preparing and draping the patient.

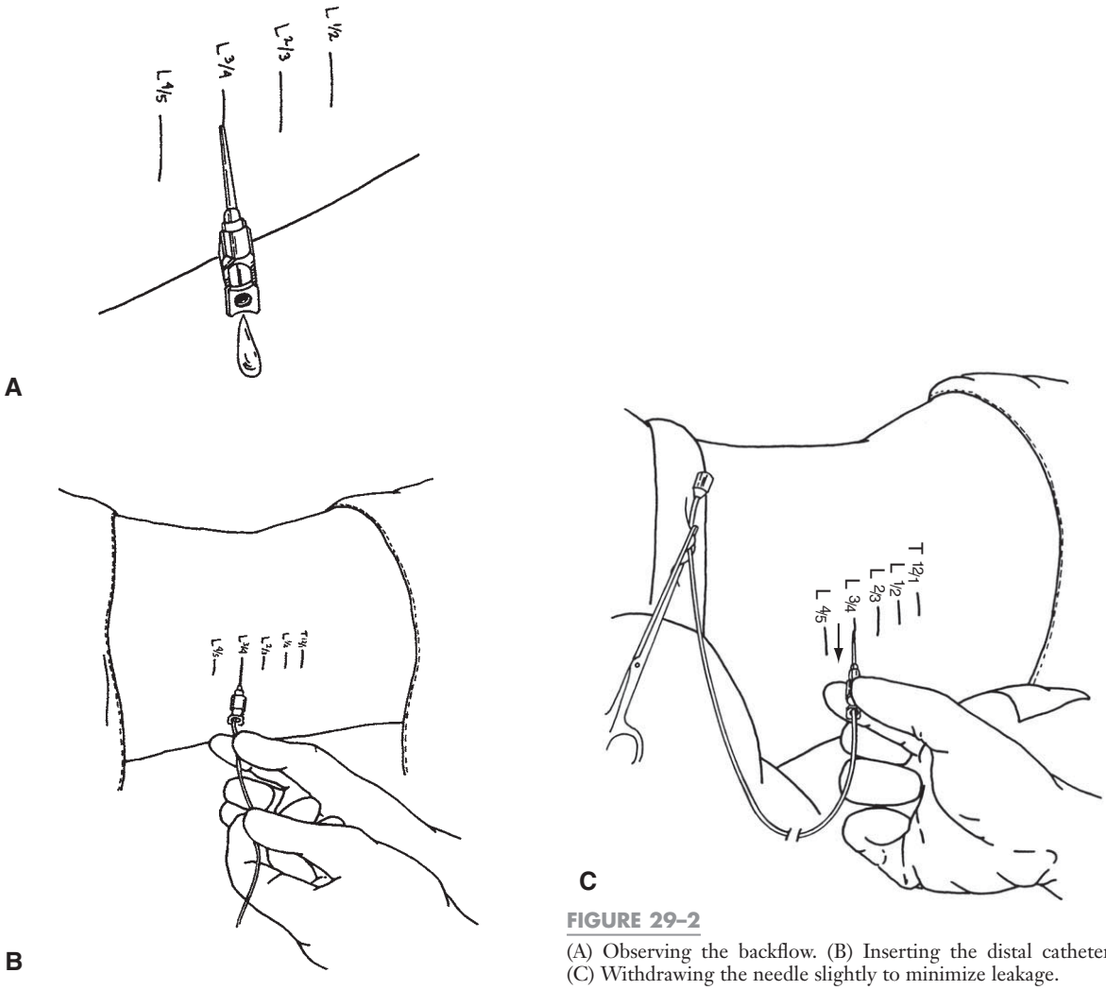


FIGURE 29–2
(A) Observing the backflow. (B) Inserting the distal catheter section. (C) Withdrawing the needle slightly to minimize leakage.

antibiotic-soaked sponge or irrigate the wounds with bibiotic solution.

12. Next, tunnel the catheter connecting the intrathecal catheter to the pump (the extension catheter) from the pump pocket to the back incision using a malleable tunneling device. Shunt tunneling tools may also be used, and the tunneling system provided with the programmable pump works well. Because most constant-flow-rate pumps come with the extension catheter connected to the pump at the factory, the catheter must be attached to the programmable pump (Figure 29-7).
13. Cover this construct with some type of anchoring device, which is secured to the connector with 2-0 nonabsorbable braided tie, and anchor the construct to the underlying muscle fascia in a figure-of-8 fashion. Do not skip the anchoring; without it, the intrathecal catheter will migrate, usually coiling itself under the skin (Figure 29-8).
14. Connect the extension catheter to the previously prepared programmable pump, and secure it to the pump with a 2-0 braided tie. Pumps with a previously attached catheter must be placed into the pocket at the time of catheter tunneling.
15. Place the programmable pump into the subcutaneous pocket. The SynchroMed pump in its polyester (Dacron) pouch may be placed without need for further suturing in nonobese patients. Pumps without this pouch may have anchoring loops manufactured around the pump circumference. Place a nonabsorbable stitch into a tissue that does not necrose rapidly, such as fat or muscle. Use at least two stitches to prevent rotation; three may be necessary to prevent flipping. A dermal or fascial stitch is usually required, and there is a risk that the anchor will be painful. If this technique is used, place the stitches into the pocket first, then through the pump suture loops. Place the pump

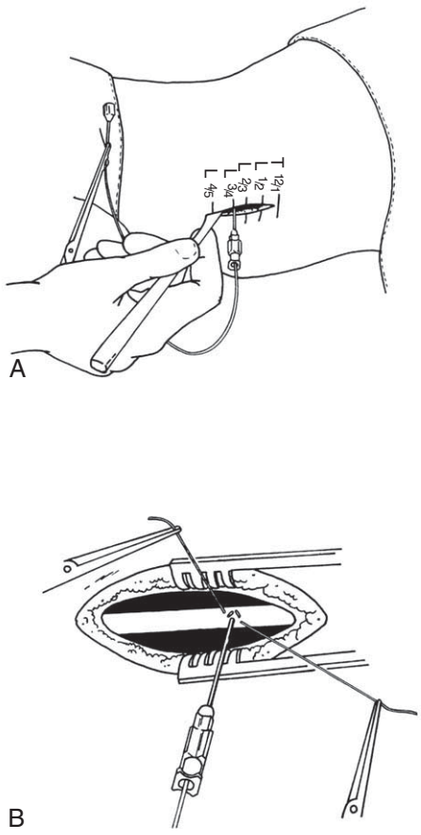


FIGURE 29–3

(A) Making a small vertical incision to expose the supraspinous ligament and dorsal lumbar fascia. (B) Placing pursestring sutures around the catheter.

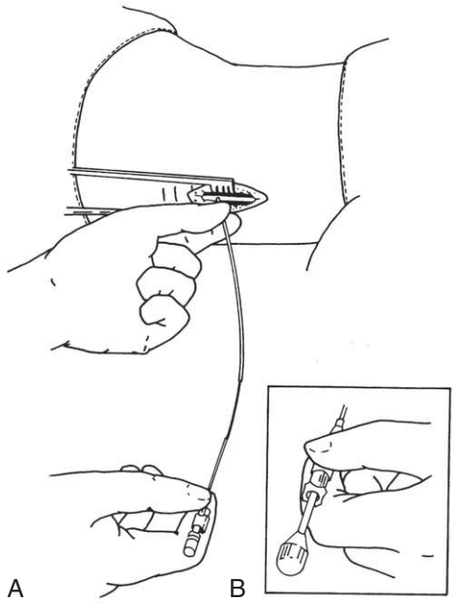


FIGURE 29–4

(A) Removing the introducer needle. (B) Withdrawing the needle and guidewire.

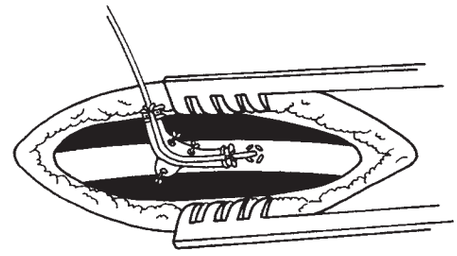


FIGURE 29–5

Inserting the catheter into an anchor and securing it with ligatures.

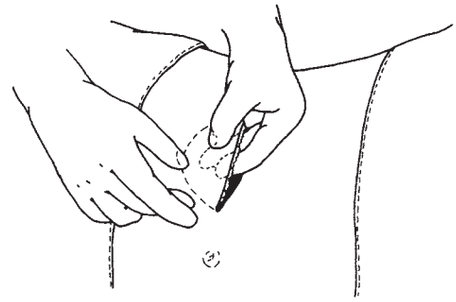


FIGURE 29–6

Forming a pump pocket.

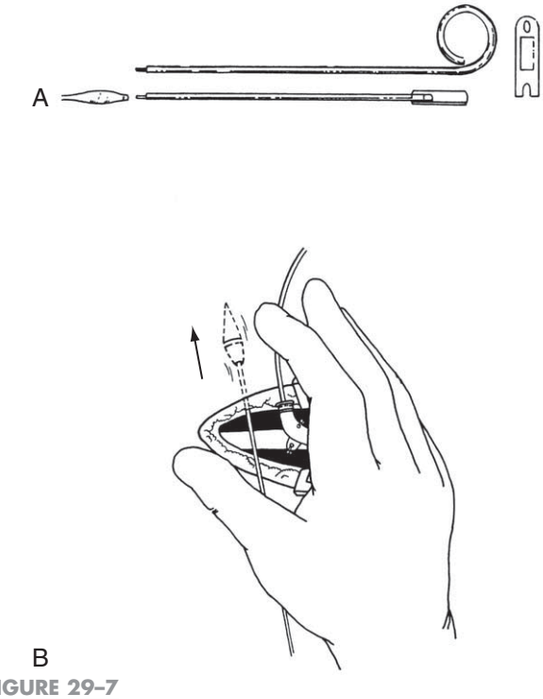


FIGURE 29–7

(A) Assembling the tunneling tool or preparing the catheter passer. (B) Creating a subcutaneous tunnel.

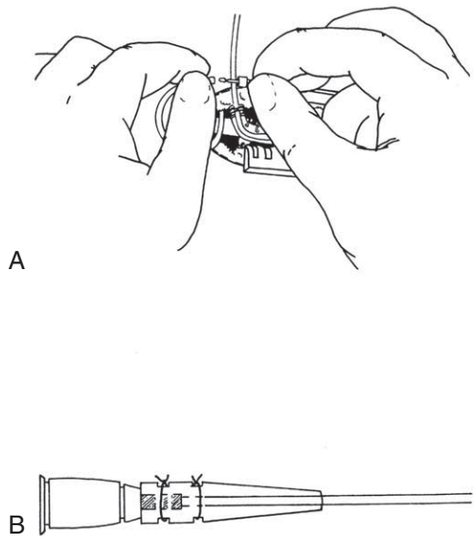


FIGURE 29–8

(A) Inserting a metal tubing connector into the proximal catheter section. (B) Suturing a sleeve onto the connector.

into the pocket, and tie the sutures. If the pocket is carefully fashioned, even a pump lacking a Dacron pouch may be placed without suturing, especially in thin patients (Figure 29-9A).

16. Carefully close the incisions. An interrupted, inverted layer of 2-0 absorbable suture in the abdomen and 3-0 absorbable suture in the back is sufficient. Then appose the skin edges with steristrips. If tension is a problem, use surgical staples to reinforce the closure (Figure 29-9B).
17. The intrathecal pump is programmed prior to connection to the intrathecal catheter with a continuous mode and single bolus. The bolus setting serves to clear the intrathecal tubing and infuse the drug to the tip of the catheter. By having the catheter filled with drug, potential drug-related adverse effects could be monitored in a hospital environment. This is done by bolusing the catheter with 0.4 ml of infusion drug over an hour (Figure 29-10).

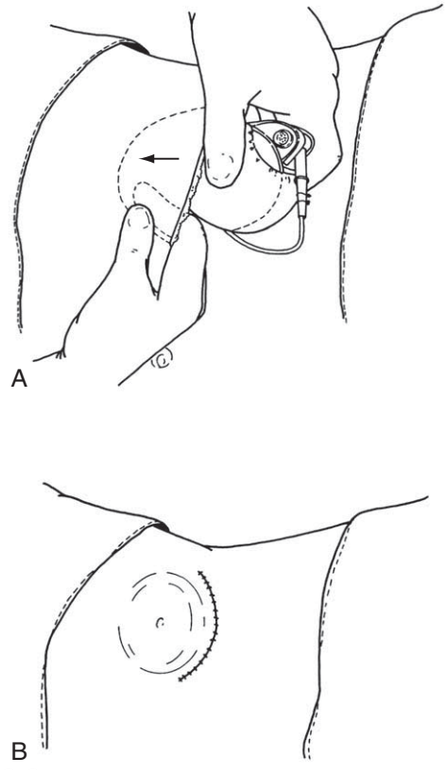


FIGURE 29–9

(A) Inserting a pump into the pocket. (B) Closing the pocket and spinal incisions.

COMPLICATIONS

SURGERY-RELATED COMPLICATIONS

In the perioperative period, bleeding with subsequent development of a pocket hematoma is perhaps the most troublesome and preventable problem. Meticulous attention to hemostasis during pump pocket formation prevents this situation. Prevention is aided by placing an abdominal binder, such as a 6-inch elastic wrap, around the abdomen and lightly compressing the fresh pump pocket for 24–48 hours. This compression dressing helps avoid accumulation of blood or fluid in the pocket.

The possibility of epidural and intrathecal hemorrhage with the obvious risk of neurologic injury is frequently mentioned. This complication, unfortunately, is likely to be unnoticeable at the time of catheter implantation. Preoperatively, care should be taken to discontinue nonsteroidal anti-inflammatory drugs and reverse any anticoagulation. Signs of a developing hematoma are usually a sudden increase in focal back pain associated with tenderness, progressing numbness, or weakness in the lower extremities, and loss of bowel or bladder control resulting in either retention (constipation) or incontinence. This clinical presentation warrants immediate imaging studies with magnetic resonance imaging (MRI) or computed tomography (CT) myelography and emergent neurosurgical intervention if there is neurologic deterioration.

With implantable devices, one of the most feared complications is that of wound infection. Prophylactic antibiotics have been controversial, but a consensus seems to have developed for using some preoperative antibiosis. One method is to use a cephalosporin intravenously 1 hour before surgery with subsequent antibiosis. Some clinics use daily prophylaxis while an externalized screening electrode trial is performed. Intraoperatively, antibiotic irrigation may be used. Attention on the part of surgical personnel to handling all parts with care and avoiding unnecessary contact with any, even prepared, skin may cut down on contamination.

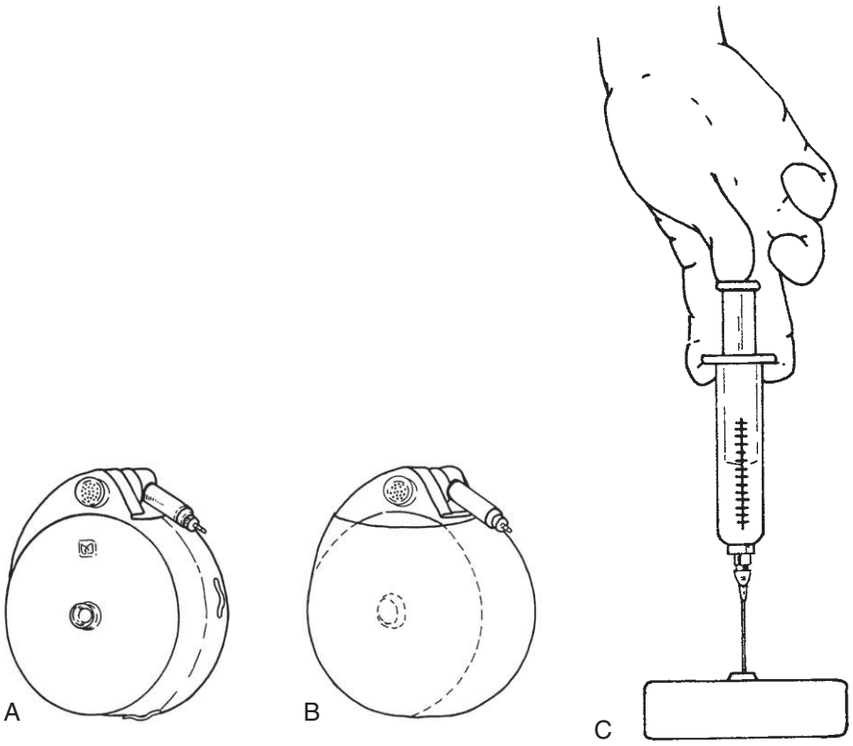


FIGURE 29–10
(A) Pump with suture loops and side-catheter access port. (B) Pump with mesh pouch. (C) Injecting prescribed fluid into the reservoir.

Although not all wound infections require removal of the device, general experience with foreign bodies implanted in the body, such as CSF shunts, spinal instrumentation, and prosthetic devices, indicates that all but superficial infections require system removal.[16] Implantable pumps contain an internal filter that guards against direct contamination resulting in meningitis. However, with infection tracking along the intrathecal catheter, either an epidural abscess or meningitis may result.

Neurologic injury is a definite possibility whenever the CSF space is entered. Needle placement, even when guided fluoroscopically, is essentially "blind" with respect to intraspinal neural structures. Potential injury to the nerve roots can occur and, to some extent, can be mitigated by performing the catheter placement using local anesthesia. The patient under local anesthesia will report a radiating electric shock-like or burning sensation in the distribution of the involved nerve root. The needle should be immediately withdrawn and placement at a different level should be considered.

With catheter placement, the spinal cord is at risk. Catheters that are spring wound or have stiffening wired internally must not be forced through the spinal canal because the tip may be buried in an intramedullary position. Penetration of the spinal cord often results in the production of dysesthesias or a burning, stinging pain below the lesion that is not nondermatomal and may not result in noticeable neurologic signs immediately. Intramedullary infusion of drug may result in progressive signs of a spinal cord lesion, and this should be immediately evaluated with MRI or CT myelography and dealt with appropriately by the neurosurgeon.

Cerebrospinal fluid leaks are a natural consequence of placing catheters in the subarachnoid space. The opening created in the dura mater by the introducing needle is larger than the entering catheter, predisposing to some potential leakage. The dura mater has a moderate amount of elasticity, which probably explains why the incidence of leaks is not higher. If the particular technique used seems to result in a relatively high incidence of spinal headache or CSF collection under the skin, a blood patch injecting 10 to 20 ml of autologous venous blood one level above the catheter entry point or at the entry point under fluoroscopic control (to avoid shearing the intrathecal catheter) may treat this problem effectively.

DEVICE-RELATED COMPLICATIONS

The most frequently reported complications with implantable pump systems involve some failure in the catheter system. Pump complications are quite rare. Early reports contained many catheter-related complications.[17,18] With the development of more thick-walled and reinforced catheters, new anchoring techniques, and paramedian approaches to placement, this problem seems to have decreased.[19]

Catheter tip obstruction can be a problem and may require revision of the catheter. This problem is usually

suspected when the expected and measured residual volumes vary by more than 20%. A complete evaluation of the catheter must be performed if obstruction, kinking, or separation is suspected. This evaluation is made more important by the increasing reports of sterile granulomatous masses forming at the tip of the catheter. These masses may cause obstruction but most commonly produce increasing pain and a progressive neurologic deficit.[20]

Evaluation of a catheter problem includes some type of imaging. Simple radiography with a soft tissue technique can demonstrate breakage or suggest a kink, migration, or disconnection from the extension or pump catheter. The evaluation of suspected obstruction related to an intraspinal problem or catheter leakage requires the use of the injection side port, if present. Injection of nonionic contrast material confirms obstruction and often shows the point of leakage. The risk with this technique is that delivery of a large bolus of medication directly into the subarachnoid space will lead to significant overdosage. When this procedure is performed, preparation for management of an overdose should be made. An attempt to aspirate the catheter should take place before injecting the contrast material to avoid this problem. In the absence of a side port, the evaluation of catheter problems is more difficult. A radioisotope may be injected into an emptied pump, and if the system is programmable, a bolus is programmed; if it is nonprogrammable, an appropriate time must elapse and the catheter is scanned.

Treatment of catheter problems usually requires removal and replacement of the catheter. Occasionally, a disconnected catheter may simply be reconnected, usually with local anesthesia. Demonstration of a granulomatous mass may require neurosurgical intervention to resect the lesion.

Pump-related complications common to nonprogrammable and programmable systems include overfilling of the pump, failure of the self-sealing septum at the refill port, and movement of the pump in the pocket. Overfilling can result in overpressurization with delivery of an unpredictable amount of drug, failure of the system, or activation of the reservoir valve preventing infusion with a programmable pump. Nonprogrammable pumps may show a slight decline in drug delivery as they approach their refill time. The decline is most likely due to a decrease in the pressure of gas against the bellows as the Freon reaches the maximum volume it has to occupy. This behavior should be anticipated and may require a slight shortening of the refill time if it is troublesome to the patient.

Programmable pumps have an additional set of potential problems because of the internal modules and mechanical components necessary with this type of device. Battery failure, pump rotor failure, and failure of the telemetry or electronic modules may occur. The battery lifetime of the pumps has been quite acceptable and is generally in the range of 3–5 years. Battery depletion requires surgical removal of the existing pump and replacement with a new pump. Pump rotor stalls may be confirmed by taking a radiograph of the pump showing the rotor, programming a bolus dose, and repeating the radiograph 15 minutes later. The pump rotor should have turned 90 degrees if the rotor is functioning. A stalled rotor requires pump replacement. Failure of the electronic or telemetry module results in inability of the pump to receive a change in programming. The pump will, however, continue to function as a nonprogrammable pump at its last prescription infusion rate. The decision to replace the pump is based on the need to make programming changes.

Movement of the pump in the pocket may result in dislodgment of the catheters (extension or intrathecal, or both). The pump may rotate in the pocket, resulting in a coiling of the catheter much like that of a fishing reel, or it may flip in the pocket, resulting in a progressive winding of the catheter. Revision of the pump and possibly the catheters may be necessary if catheter movement is occurring. A flipped pump is usually noticed by the patient but may be noted and verified in the clinic at the time of attempted refill. Revision of the pump is probably necessary and often requires anchoring the pump.

CLINICAL PEARLS

An important concern is the patient's current opioid use and how to manage it at the time of screening. Eliminating opioids before screening may cause unwarranted discomfort to the patient and may add to the expense of the trial.[21] A complete conversion from systemic opioid to intraspinal opioid may result in an abstinence syndrome. Therefore, a clinical protocol during the screening trial is necessary to prevent withdrawal effects. One such protocol, suggested by Krames,[19] involves converting 50% of the pretrial oral dose to an intrathecal equivalent dose and withdrawing the remaining oral dose by 20% per day, converting it to an equianalgesic intrathecal dose. The dose may then be increased to effecting intrathecally while systemic medication is decreased.

For the tunneled catheter period, the patient is usually kept in the hospital for a 3-day period, although some clinicians are beginning to use outpatient trials of 1 week or longer. The length of trial may be an important consideration. Presumably, the longer the trial proceeds, the less likely it is that a placebo response will account for the outcome. It is fair to say that most clinicians feel that a longer screening trial predisposes to a more successful outcome.

If the screening trial is successful, the patient generally reports at least 50% pain relief as measured by some standard self-reports to no intolerable side effects. The patient then proceeds to implantation of the chosen drug administration system.

EFFICACY

Although most patients with chronic non–cancer-related and cancer-related pain are adequately managed with oral analgesic medications, electrostimulation, or behavioral

techniques, studies indicate that only about half of the patients so treated with back pain or neuropathic pain achieve good reduction of pain, and a full 21% are unresponsive to opioid therapy.[22,23]

CANCER-RELATED PAIN

Early studies of cancer-related pain demonstrated that intrathecal administration of opioids was much more effective than other routes of administration.[9,24–28] The most common early use of intrathecal infusion of morphine was for cancer-related pain.

In a retrospective multicenter study of the use of intraspinal morphine for all types of pain, 32.7% of the patients analyzed had cancer-related pain.[25] The average length of treatment in the study was 14.6 months (range, 8–94 months). Patients with cancer-related pain were treated with higher initial doses and escalated to a stable level more rapidly than those with non–cancer-related pain. The most frequently used drug was morphine. In the population with cancer-related pain, 13.6% had somatic pain, 25.4% neuropathic pain, 16.9% visceral pain, and 44.1% a mixed pain presentation. The long-term stability of dosing in the population with cancer-related pain has also been documented elsewhere.[29]

Cancer pain of all types remains an excellent indication for the use of intrathecal opioids, especially with a programmable pump, which can aid in matching pain relief to progression of disease. It is probable that about 5–10% of the population with cancer pain are candidates for an implantable pump system according to the selection criteria noted previously.

NON–CANCER-RELATED PAIN

The use of intrathecal opioids for non–cancer-related pain has increased despite a lack of prospective studies. The most definitive data supporting such an increase in use are those provided by the survey of physicians in the United States by Paice and colleagues[25] for cancer-related pain and including non–cancer-related pain and by the retrospective study of Winkelmuller and Winkelmuller from Europe.[22]

In the American study, two-thirds of the patients had non–cancer-related pain. The most common condition was failed back syndrome (42.4%). Other pain syndromes treated included complex regional pain syndrome (5.6%), postherpetic neuralgia (5.1%), and peripheral nerve injury (3.7%). The most common screening technique was continuous epidural infusion (35.3%), followed by bolus intrathecal injection (33.7%). Psychological screening was used for 77.6%. Morphine was the most commonly infused drug (95.5%), but a wide variety of medications were used. Doses for neuropathic pain tended to be higher at 6 months than for somatic or visceral pain. A local anesthetic (bupivacaine) was used as an adjuvant to morphine in 19.8%. These patients had a linear increase in dose over time, eventually reaching stable levels by 1 year at 9.2 mg/24 hours. By physician report, 52.4% of the patients had excellent pain relief, 42.9% good relief, and 4.8% poor relief, testifying to the considerable efficacy of this technique.[26]

Specific outcome measures employed by Paice and colleagues[25] included activities of daily living (ADL), employment, percentage of pain relief, a global pain relief score incorporating intensity and pain medication changes, and activity levels. ADL were 82% improved. Patients with visceral pain showed the greatest improvement in ADL. Twenty-four of the patients with non–cancer-related pain returned to work.

In a long-term follow-up of 120 patients with non–cancer-related pain in Europe with a mean of 3.4 years (range, 0.5–5.7 years), 73 patients had mixed neuropathic and nociceptive pain because of multiple back surgeries, and 34 had conditions such as postherpetic neuralgia, stump and phantom limb pain, and various peripheral nerve injuries.[23] Six months after implantation, the average pain intensity score was 30.5. At the conclusion of follow-up, the score was 39.2. The best initial response was seen in the group with nociceptive pain, which had a 77% initial reduction in pain intensity and declined to 48% at last follow-up. Groups with deafferentation and neuropathic pain benefited from therapy and over the long term showed the best results, with 68 and 62% pain reduction as measured by visual analog scale, respectively.

Although these results for a population of patients unresponsive to more conventional methods are impressive, prospective studies comparing this therapy with alternative therapies would establish intrathecal infusion of medication as a treatment of choice in a more rigorous fashion. The current acceptance in clinical practice empirically validates the technique but also makes prospective and certainly randomized studies difficult to implement.

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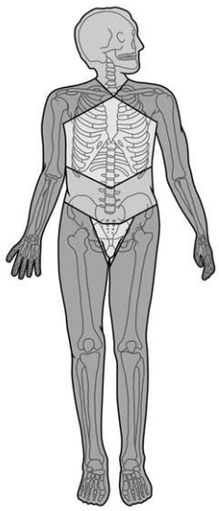
C H A P T E R



30 Epiduroscopy

JAMES E. HEAVNER, HEMMO BOSSCHER, AND SUSAN ANDERSON

HISTORY

Interest in viewing the contents of the bony vertebral canal using percutaneously placed devices has existed for a long time. As early as 1931, Burman[1] used arthroscopic equipment to examine the anatomy of vertebral columns removed from cadavers. Significant advancement of epiduroscopy toward clinical application occurred only after introduction of flexible fiberscopes capable of delivering high-quality images, especially with computer enhancement, and development of suitable light sources. This equipment has been used to view both the spinal epidural space as well as the spinal subarachnoid space.

Pursuit of the use of lumbosacral epiduroscopy for diagnostic and therapeutic purposes in pain management has increased rapidly beginning in the mid-1980s. The authors and others began exploring the use of lumbosacral epiduroscopy as an aid to lysis of epidural adhesions. The lysis procedure is based on evidence that adhesions in the epidural space are involved in the pathophysiology of low back pain and/or radiculopathy and prevent delivery of therapeutic agents to target sites. The procedure involves (1) definition of a filling defect on epidurography that corresponds to the spinal segment innervating the painful area; (2) insertion of a catheter into the defect and injection of normal saline and hyaluronidase to remove tissue (fibrosis) barriers to contrast flow; and (3) injection of therapeutic agents through the catheter to the target site. It was reasoned that a flexible endoscopic device with a deflectable tip and a working channel would facilitate catheter placement, provide mechanical in addition to hydraulic forces to break tissue barriers, and provide visual information that would aid in diagnosis and prognosis. Substantial clinical experience and published data demonstrated the safety and efficacy of epiduroscopy.

ANATOMY AND PHYSIOLOGY

As an epiduroscope is advanced through the sacral and lumbar bony vertebral canal, it transverses an anatomical area referred to as the epidural space. It generally is stated that the epidural space is a potential space. However, we know this area between the dura and the walls of the bony vertebral canal is a real space filled with, for example, fat, nerve roots, and fibrous tissue. Successful application of epiduroscopy requires unique knowledge of the anatomy of the contents of the bony vertebral canal.

This canal varies in shape and size, and the contents differ depending on region (Figure 30-1). The spinal cord, which is surrounded by the pia mater, cerebrospinal fluid, arachnoid mater, and dura mater (from closest to the cord outward), ends, in the adult at about L1 or L2. The spinal cord tapers at its end forming the conus medullaris and then the filum terminale. The filum terminale and lumbar, sacral, and coccygeal anterior and posterior nerve roots continue in the caudal sac. The caudal sac is filled with CSF and is bounded outwardly first by the arachnoid and then by the dura. Nerve roots traverse through the caudal sac for varying distances, depending on where they exit the spinal cord proximally and where they exit the intervertebral foramen distally. These nerves form a structure called the cauda equina. As the roots exit the caudal sac, they are covered by extensions of the meninges and travel between the lateral bounds of the bony vertebral canal and the dura for varying distances and at varying angles, depending on where they exit the canal. The distance becomes longer, and the angle less steep from the lumbar to the sacral region. The diameter of the nerves varies from spinal segment to spinal segment. The dorsal root ganglion is very near or in the intervertebral foramen, and the anterior and posterior nerve roots join to become the spinal nerve in the foramen. The caudal sac ends at approximately S2, and the filum terminale, fused with the filum of the dura,

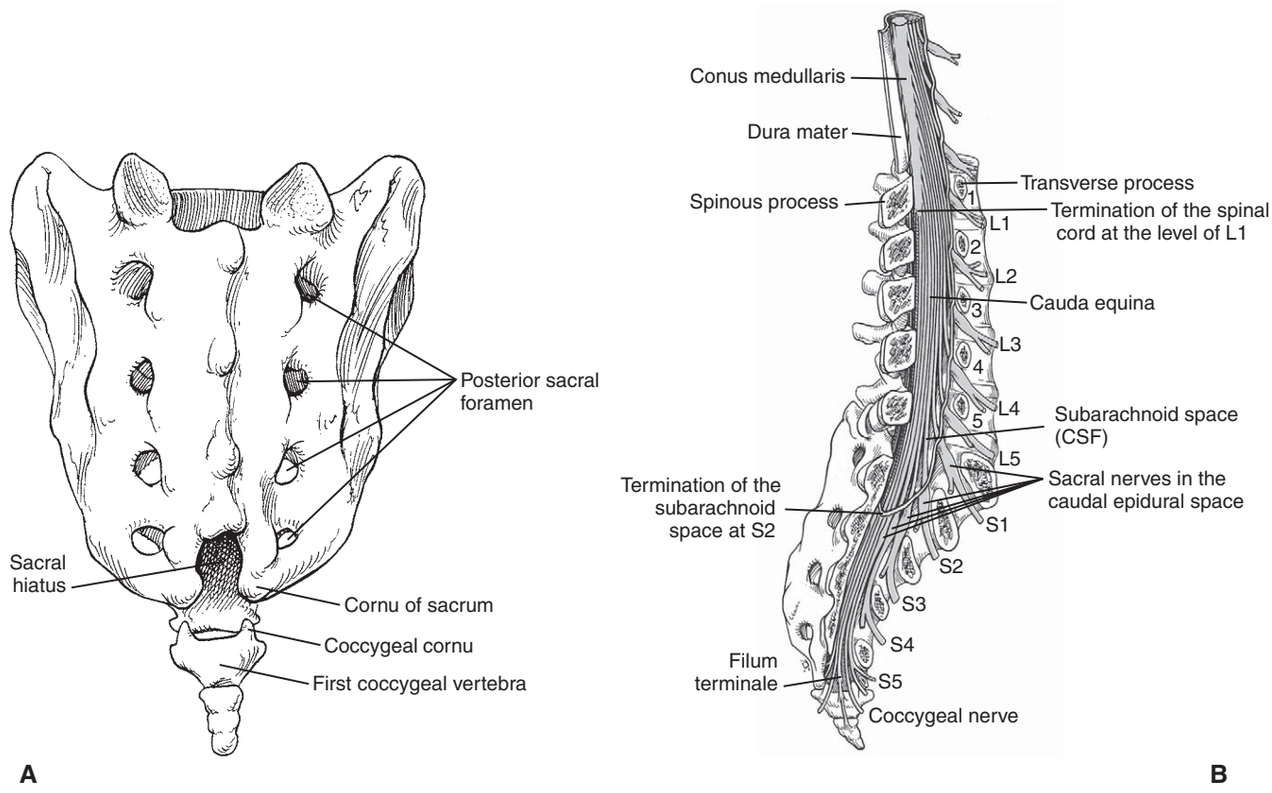


FIGURE 30–1

(A) Sacral hiatus and its anatomic relationships. This is the site where the epiduroscope is inserted. (B) The longitudinal section of the lumbosacral vertebral spine demonstrates the neural contents as they traverse toward their foramina.

continues caudally from there to the coccyx, where it blends with the periosteum.

Epidural fat normally is present in discrete pockets in the posterior and lateral epidural space.[2] Epidural veins are restricted to the anterior epidural space.[2–4] In the sacral canal, peridural fat with sparse small blood vessels running through it is usually encountered during epiduroscopy and the filum terminale may be seen. More cephalad in the sacral and lumbar canal, the dura surrounding the caudal sac may be viewed as a bluish-white structure. Nerve roots with an attached blood vessel may be seen through the dura and on the right and left sides of the canal. Occasional thin sheets of fibrous tissue may be seen in the fat or between fat deposits.

When the scope tip approaches an intervertebral foramen, nerve roots are more readily identified, as are larger blood vessels that traverse the foramen. In the posterior epidural space, the ligamentum flavum and less peridural fat usually are viewed. The plica mediana dorsalis, a connective tissue band in the dorsomedial epidural space attached at one end to the posterior dura and to the periosteum of the vertebral arch at the other, may or may not be seen. Careful examination of lateral intervertebral spaces may reveal articular surfaces and vertebral pedicles. Examination with epiduroscopy is usually limited to the posterior, posterolateral, lateral, and anterolateral epidural space.

INDICATIONS

General indications for epiduroscopy (spinal canal epiduroscopy) presented in a consensus paper by an international group of experts follow:

- Observation of pathology and anatomy
- Direct drug application
- Direct lysis of scarring (with medication, blunt dissection, laser and other instruments)
- Placement of catheter and electrode systems (epidural, subarachnoid)
- An adjunct to minimally invasive surgery

The most common indications for epiduroscopy are to examine the lumbosacral epidural space of patients with chronic radicular symptoms and/or low back pain to identify pathology and to administer therapy to the area(s) where there is pathology.

CONTRAINDICATIONS

Epiduroscopy should not be performed on patients for whom other diagnostic approaches are definitive and for whom other therapy, such as surgery, is clearly indicated.

Other contraindications include systemic infection or local infection at the epidural access site, uncontrolled drug abuse or dependency, uncontrolled major depression or psychiatric disorders, uncontrolled or acute medical illnesses, bleeding dysesthesia or abnormal laboratory values reflective of impaired blood clotting capability, pregnancy or lactation, and cerebrovascular disease or space-occupying lesions in the central nervous system.

EPIDUROSCOPY EQUIPMENT

- Epiduroscope with light source and video display. Epiduroscopes are available from Karl Storz, Myelotec and Equip. (The authors prefer and use the Karl Storz equipment, a video display system that allows simultaneous viewing of fluoroscopy and epiduroscopy images [Karl Storz Twin Video] [Figures 30-2 and 30-3].)
- Percutaneous introducer set and guide wire (supplied with Storz and Myelotec equipment. (The authors prefer to use a 9- or 10-French Super Arrow-Flex vascular access set from Arrow International.)
- 25-gauge, 3/4-inch, and 18-gauge infiltration needles
- Two 3-way stopcocks and IV set with extensions
- No. 10 blade scalpel
- Assorted syringes (5 ml, 10 ml, and 20 ml)
- 18-gauge Tuohy epidural needle
- 36-cm epidural (Tun-L) catheter with connector (optional)
- Two 2 × 2 split IV sponges
- Transparent surgical dressing
- Nerve stimulators (optional)

DRUGS

- 2% lidocaine for skin infiltration
- 0.25% bupivacaine or 0.2% ropivacaine
- Preservative-free normal saline

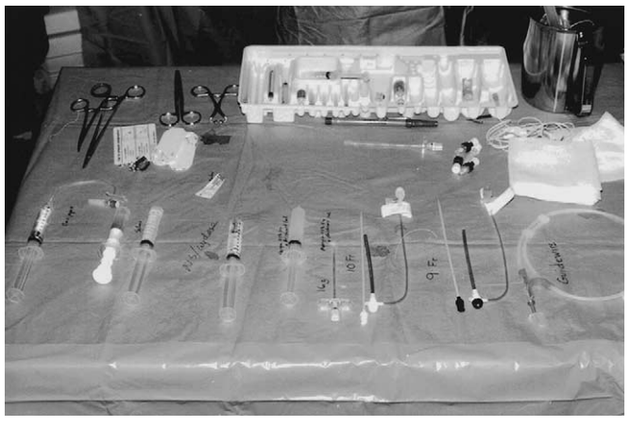


FIGURE 30–2
How a sterile table should be set up for flexible epiduroscopy.

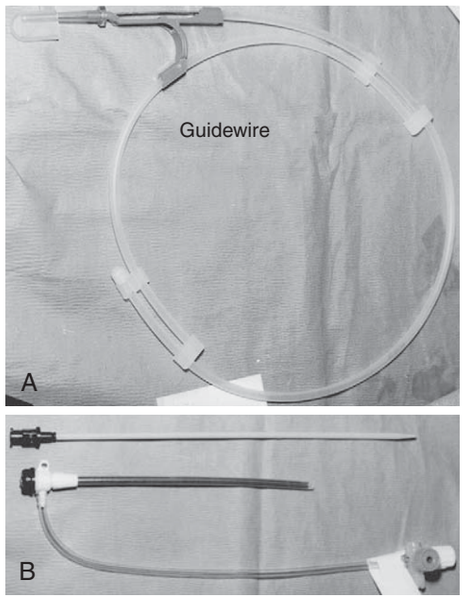


FIGURE 30–3
Guidewire (A) and dilator (B).

- 1500 units of hyaluronidase (Wydase)
- 10% hypertonic saline (optional)
- Steroid
- Iohexol (Omnipaque 240) radiographic contrast

PREPARATION OF PATIENT

Physical examination (including examination of the entry site for local infection and distorted anatomy), lumbosacral magnetic resonance imaging, urology evaluation (if necessary), and laboratory studies, including complete blood count with platelets, prothrombin time, partial thromboplastin time, platelet function studies and bleeding time, and urinalysis, are required.

For preoperative medication, use the standard recommendations for conscious sedation by the American Society of Anesthesiologists. Preprocedure sedatives, analgesics, and ancillary drugs are administered as needed in the surgical holding area. The patient is given 1 g of ceftriaxone (Rocephin) intravenously prior to the start of the case. (Ciprofloxin [Cipro], 400 mg, given 1 hour before epiduroscopy is started may be substituted if there is concern about allergy.)

PROCEDURE

LOCATION

At Texas Tech University Health Sciences Center/University Medical Center, epiduroscopy is performed in the operating room.

POSITION OF PATIENT

The patient is in the prone position (Figure 30-4). A pillow usually is placed under the abdomen to reduce lumbar lordosis. The epidural access site is prepared for sterile entry, and a full-body sterile surgical drape is placed over the patient, leaving only the access site exposed (Figure 30-5).

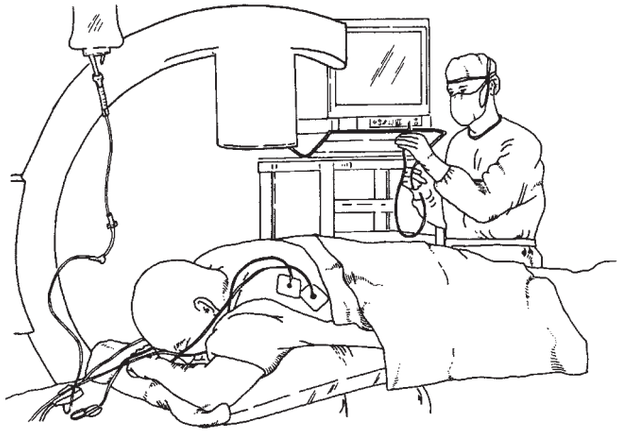


FIGURE 30–4

Patient is draped in a sterile manner in the prone position with all other connections in place.

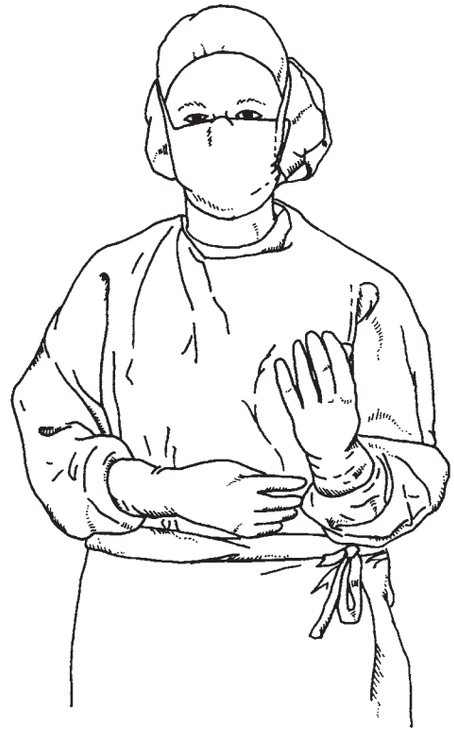


FIGURE 30–5

Physician appropriately draped and gloved.

ANESTHETIC CARE

The goals for anesthetic care for epiduroscopy are to make the patient comfortable and provide amnesia. Local anesthetic is infiltrated at the epidural access site, hypnotic is administered to obtund consciousness, systemic opioid is administered for pain control, and an amnesic is administered (e.g., propofol with ketamine, fentanyl, midazolam).

ACCESS TO EPIDURAL SPACE

Access to the epidural space for epiduroscopy occurs via the sacral hiatus using the Seldinger technique. A skin wheal is raised over the sacral hiatus using 1–2 ml of 1% lidocaine and a 25-gauge needle (Figure 30-6A). An 18-gauge needle is used to penetrate the skin. An 18-gauge, Tuohy epidural needle is then inserted through the puncture site and into the sacral hiatus (Figure 30-6B). This may be verified in both the posteroanterior and lateral fluoroscopic views (Figure 30-7). A guidewire is inserted through the needle and advanced to approximately the L5 or S1 level (Figure 30-8A). A small stab wound is made through the skin and underlying tissue and sacrococcygeal ligament with a No. 10 one-blade scalpel.

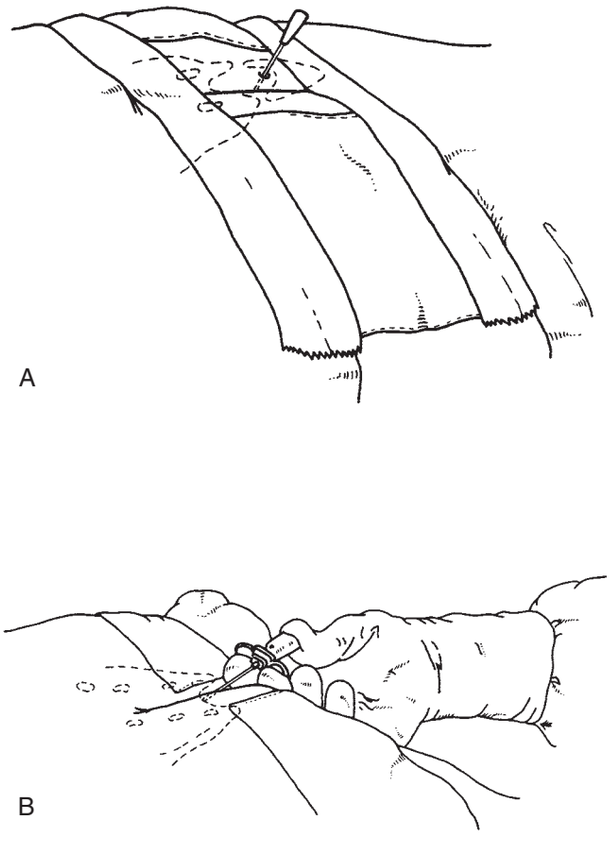


FIGURE 30–6

(A) Introduction of infiltration needle in the caudal region. (B) Insertion of epidural needle in caudal space.

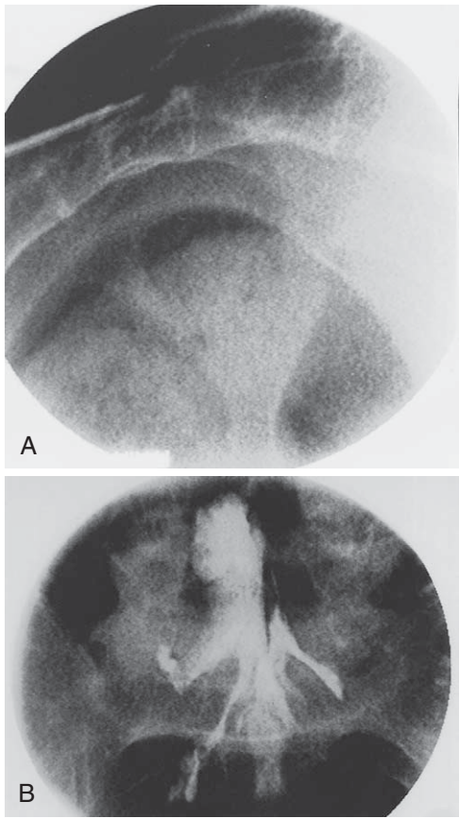


FIGURE 30–7

(A) Lateral view of the RK needle (arrow B) in the caudal canal (arrow A). (B) The radiographic dye spread in the caudal canal confirms the correct position of the needle.

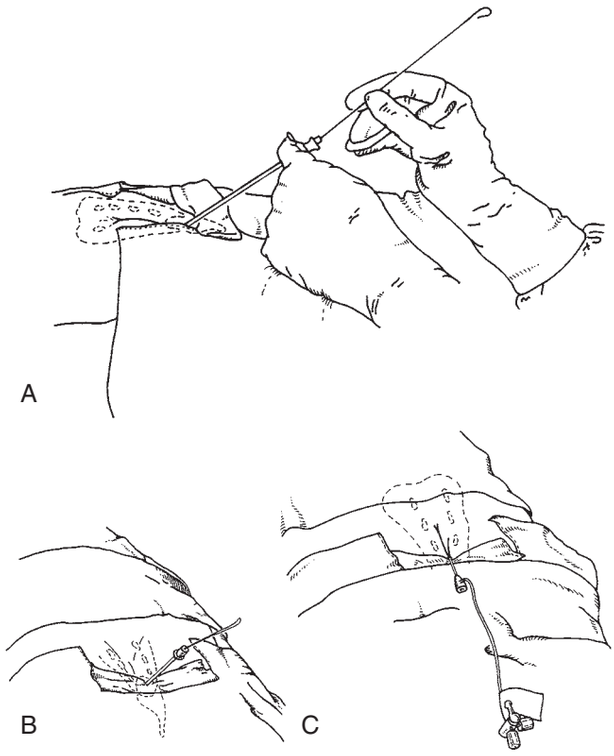


FIGURE 30–8

(A) Guidewire being inserted through the RK needle. (B) The RK needle is then removed, and a 9- or 10-French dilator catheter is inserted over the guidewire. (C) After the dilation, the guidewire is recovered and the introduction sheath is in place for the fiberscope to be introduced.

The epidural needle is removed and 10-French dilator is then inserted over the guidewire, through the incision into the sacral canal (Figure 30-8B). Then the dilator is removed, and the dilator with a sheath is placed over the guidewire into the space. It is important to check the guidewire for freedom of movement to avoid kinking it. It is also important to advance the dilator and sheath together until the sheath passes through the sacrococcygeal ligament. This can be confirmed both by feeling a "pop" when the sheath passes through the ligament, as well as by a lateral fluoroscopic view of the sacrum. The dilator and guidewire are removed, leaving the sheath in place (Figure 30-8C). Before the sheath is inserted, it is filled with saline and when in place, 5 ml of saline are injected through the injection port to expand the epidural space.

TECHNIQUE

The epiduroscope allows for three-dimensional direct visual observation. During epiduroscopy, equipment that displays on a monitor both the epiduroscopic image and the fluoroscopic image is used. Primary and secondary images can be changed. The authors usually have the epiduroscopy image as the primary one except when more detail is needed from the fluoroscopy image than can be obtained with it as a smaller secondary image. Examination of the epidural space may extend from the sacrum to as far cephalad as the posterior border of L2, depending on the patient's area of symptoms and the extent of abnormalities encountered. Skill is required to manipulate the epiduroscope through the bony vertebral canal and to direct the scope tip to areas of interest.

The epiduroscope is inserted through the sheath (Figure 30-9). Fluoroscopy is used to verify proper placement. During the epiduroscopy procedure, preservative-free 0.9% saline is injected through the working channel of the epiduroscope to expand the epidural space and to flush away tissue debris and any extravasalated blood to provide optimal viewing. How successful this is depends on many factors, including how fast fluid is infused, compliance of the contents of the space, presence of compartments, and how fast fluid exits through the intervertebral foramen into the paraspinal area. Care must be taken to use the minimal amount of saline. The total volume infused should generally not exceed 100 ml. In addition to monitoring the volume infused, epiduroscopy time should be monitored. At Texas Tech, epiduroscopy time usually is less than 30 minutes.

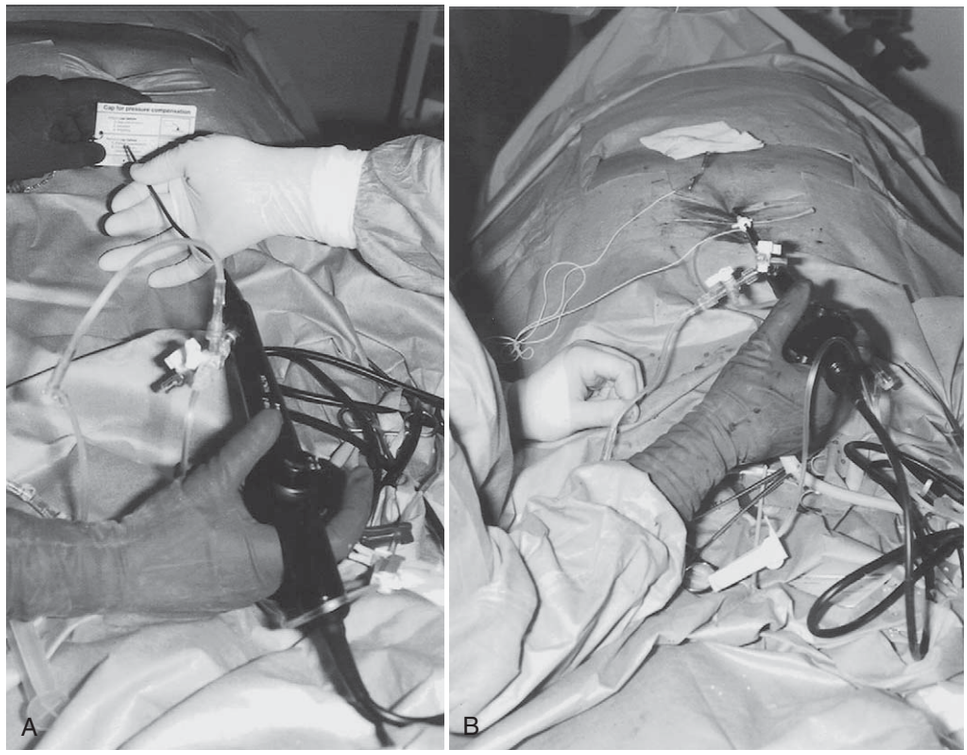


FIGURE 30–9
(A) Fiberscope focused for clear vision prior to introduction. (B) The fiberscope introduced in the sheath for direct vision of the caudal space.

If the patient has unilateral symptoms, the epidural space is examined first contralateral to the symptomatic side. This provides the general appearance of the "normal" epidural space for the patient. Next, the symptomatic side is examined with emphasis on viewing the area where the nerve or nerves that innervate the symptomatic side transverse the epidural space and pass through the intervertebral foramen. The authors note the presence or absence and the character of the following tissue: fat, blood vessels, and fibrous tissue, as well as the presence of abnormal tissue and of inflammation. The scope tip readily exits a normal intervertebral foramen. The goal is to find an area or areas of pathology that when touched by the tip of the epiduroscope produce arousal and/or pain in the painful area ascertained during the preprocedure evaluation.

Abnormalities observed include discrete or diffuse inflammation and diffuse or discrete fibrosis that ranges from mild (through which the scope easily passes) to a dense, solid mass through which the scope cannot be passed (Figure 30-10). Increased vascularity (small and/or larger vessels) and/or engorged, distended blood vessels may be seen. Fibrous scars may be avascular or have varying degrees of vascularity.

After the examination (diagnostic) phase of the procedure, we proceed with treatment, which includes breaking any existing fibrosis that was not lysed during the examination. The lysis is accomplished by using mechanical force delivered by moving the tip of the epiduroscope and by injecting hyaluronidase and normal saline through the working channel of the scope. Radiopaque contrast material is injected through the working channel to determine if there is a path for fluid to flow into the area of pathology associated with the patient's symptoms. Then local anesthetic and corticosteroid are injected through the scope's working channel to the target site. More than one area may be treated depending on clinical presentation of symptoms and epiduroscopy findings. After the treatment is finished, the epiduroscope and the sheath are removed. The site is covered with antibiotic ointment and occlusive dressing. The patient is observed until criteria for discharge from the hospital are met.

COMPLICATIONS

To date, no complications unique to epiduroscopy have been reported in the literature. Complications generally reported associated with accessing the epidural space, inserting needles and catheters, and injecting fluids have been reported or may occur. Included are infections, epidural hematoma, bowel or bladder dysfunction, headache, visual disturbances secondary to retinal hemorrhage, and dysesthesia and residual pain at the injection site. Entry into the subarachnoid or subdural space may occur. If not recognized, this may lead to

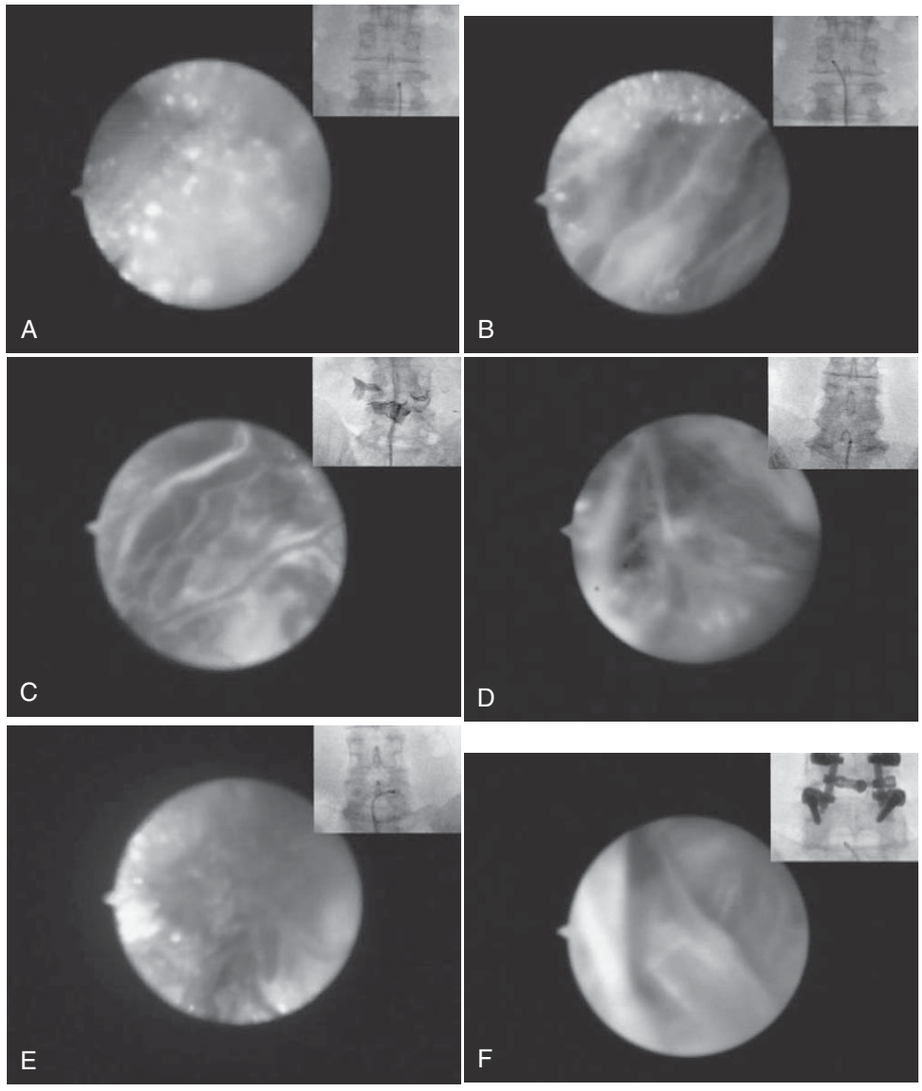


FIGURE 30–10

Fiberscopic views. (A) L5 right side: normal looking fat. (B) L4 left side: engorged blood vessels. (C) L4-L5 midline: grade 2 fibrosis with increased vascularity. (D) L5 left side: grade 3 fibrosis. (E) L5 right side: active inflammation with increased vascularity. (F) L5 left side: dense laminotomy scar.

complications resulting from injection of drugs or fluid volumes appropriate only for epidural administration.

CLINICAL PEARLS

In our experience, the most difficult technical aspects of epiduroscopy are placing the dilator and sheath and advancing the epiduroscope to the area(s) of interest. Anteroposterior and lateral fluoroscopic viewing should be used as needed to assure that the dilator and sheath follow the guidewire and are directed as straight as possible toward the sacral hiatus. The dilator and sheath must be advanced as a unit, and there must not be tissue between the stab wound and the path of the dilator and sheath. The authors usually use a 10F dilator and sheath, but if difficulty is encountered, a 9F dilator is used, followed by a switch to a 10F sheath or continuing with a 9F sheath. Access usually is much easier in females than in males. The authors rarely fail with females but fail in about 1 of 15 males.

Before inserting the epiduroscope, be sure it is in focus and color balanced. Establish a neutral or reference position for the scope to aid orientation and to establish up/down, right/left for scope tip manipulation, as well as for image interpretation. When advancing the scope, rotate it and deflect the tip so the scope follows the spinal canal. Identification of the correct spinal level is almost impossible without simultaneous fluoroscopy. The authors use fluoroscopy in the pulse mode. Total fluoroscopy time for a procedure is usually less than 1 minute.

Avoid introducing air into the injected fluid. This causes distortion of the epiduroscopic image and can be difficult to move out of the visual field.

Recognition of structures and pathology is challenging. It is absolutely essential to be familiar with the anatomy of the epidural space and other contents of the spinal canal, especially as viewed through an epiduroscope. Also essential is familiarity with the type and appearance of pathology that might be encountered.

EFFICACY

In a systematic review of spinal endoscopy, Chopra et al.[5] retrieved 112 articles, 8 of which were considered to be relevant reports of studies of spinal endoscopic adhesiolysis. Two randomized, double-blind evaluations, three prospective evaluations; three retrospective evaluations; and multiple case reports were available for review.

The randomized trials showed significant improvement in pain relief, as well as multiple other parameters including return to work at 3 months, 6 months, and 1 year. The prospective evaluations also showed improvement. Two retrospective evaluations included in the analysis showed positive short-term and long-term results.

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4. Gershater R, Holgate RC: Lumbar epidural venography in the diagnosis of disc herniations. *Am J Roentgenol* 126:992–1002, 1976.
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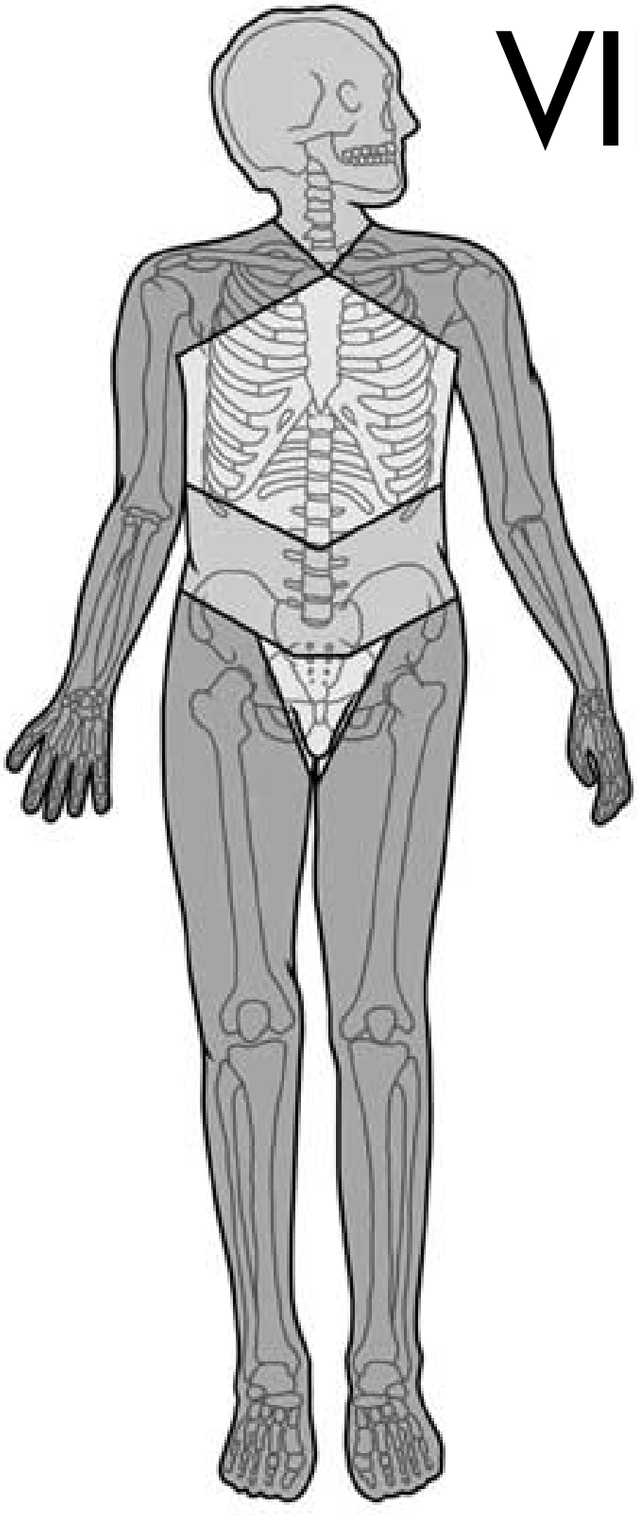
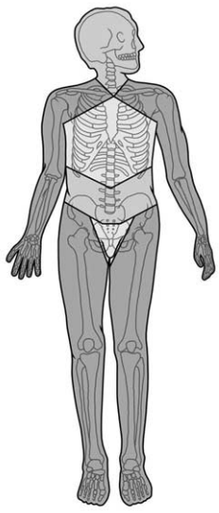
SECTION

VIII



EMERGING TECHNIQUES

C H A P T E R



31

Percutaneous Therapeutic Procedures for Disc Lesions

RICARDO RUIZ-LOPEZ AND CARMEN PICHOT

Minimally invasive spine surgery has evolved rapidly within the last two decades in an effort to decrease morbidities associated with open surgical procedures. The first description of an operative treatment of the spinal column was proposed by Paulus of Aegina in the 7th century.[1] Earlier, Hippocrates was credited through his seminal teachings and writings as the father of spine surgery.[2] During the 20th century, the evolution of minimally invasive techniques started in 1962 by Fang and Ong[3] with the publication of transoral decompression for irreducible atlantoaxial abnormalities. The microendoscopic discectomy (MED) was developed to minimize the tissue trauma seen with open procedures, enabling cervical and lumbar discectomy through a tubular retractor, with endoscopic observation.[4]

In 1910, Jacobaeus in Sweden is credited with performing the first thoracoscopic procedure.[5] In 1993, Mack et al.[6] and Rosenthal et al.[7] reported the technique of video-assisted thoracic surgery. Collis first demonstrated successful keyhole surgery for lumbar disc herniation through a speculum in the 1960s.[8] The introduction of the operating microscope for discectomy by Yasargil in 1967 and later by Williams encouraged smaller incisions for the standard posterior approach.[9] Since then, the advent of better endoscopes, high-resolution video-scopes, operative microscopes and high-quality imaging, and neuronavigation and robotics have set the stage for advances, changing the character of spinal surgery and leading to a completely new surgical field with the development of a subspecialization in minimally invasive spine surgery.[10]

INTERNAL DISC DISRUPTION

HISTORY

Internal disc disruption (IDD) was first described by Crock in 1970[11] and again in 1986.[12] It was then described as a "disruption" of the internal architecture of the disc without signs of disc protrusions or without positive signs for nerve root compression. In 1995, Schwarzer set out to test and further develop Crock's theory of IDD, and convincingly calculated the prevalence of IDD in patients with chronic low back pain.[13] The study also attempted to determine if traditional examination findings and/or specific patient symptoms could be predictive of the diagnosis of IDD. By following the strict criteria specified by the International Society for the Study of Pain in its taxonomy,[14] these investigators calculated the prevalence of IDD to be between 30 and 50% with a 95% confidence limit. They also concluded that neither traditional examination findings nor patient symptoms could predict whether a patient had IDD; thus, provocation discography remains the only way to confirm the diagnosis of IDD.

The theory of IDD as a source of chronic back pain is not without its critics. In 2003, Lee et al.[15] reviewed the research on IDD from 1985 through 2000, although the reviews were mostly on radial tears, and the high-intensity zone (HIZ). They studied the 13 papers on IDD and similar topics. There was not much agreement on what confirmed the diagnosis of IDD. There was some general agreement among groups on what constituted the diagnosis of IDD. Lower back pain patients reproduced on provocative discography concordant pain and a normal neurological examination. Other criteria for the diagnosis of IDD not universally agreed upon were the presence of an HIZ within the posterior outermost region of the disc on the T2-weighted magnetic resonance image (MRI), disc degeneration, and a history of trauma.

PATHOPHYSIOLOGY

The initial step in the IDD process is for the disc to first degenerate by losing water content and then become brittle (Figure 31-1). Second, tears open from the inside out of the disc when there is trauma to the back or neck.[16–21] On the T2-weighted MRI, a degenerated disc is seen in Figure 31-2. This disc is represented as a

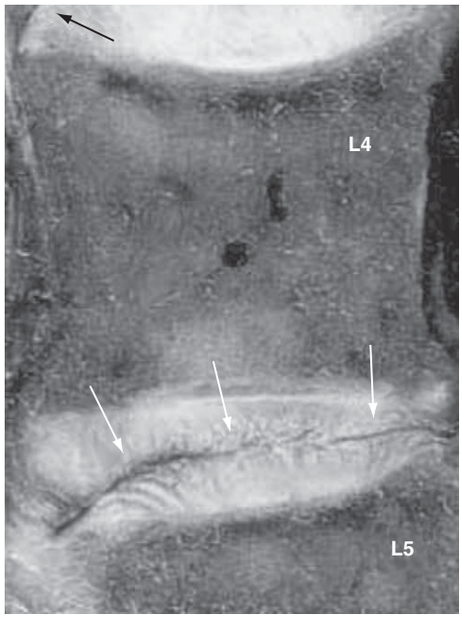


FIGURE 31–1

An annular tear in the disc is demonstrated in a human cadaver. The white arrows show the radial annular tear within the L4-L5 disc. (Courtesy of Douglas M. Gillard, DC, IDE, QME.)

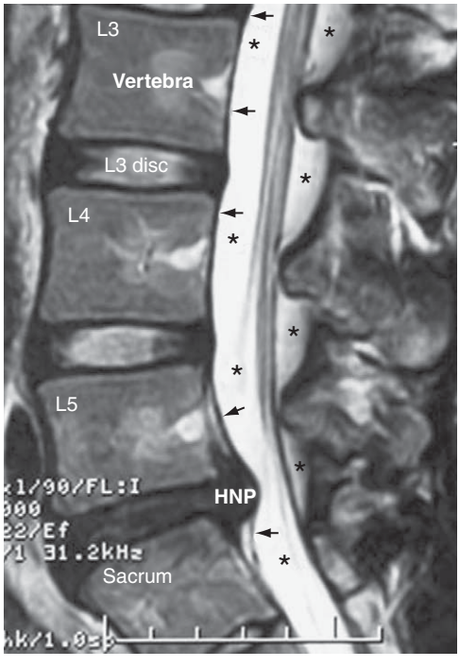


FIGURE 31–2

A proton density image of the sagittal view of the lumbar spine. Note that this image is in between the T2- and T1-weighted MRI images. This is the best image to determine whether a disc herniation has "extruded" through the posterior longitudinal ligament (PLL). Note the "blackness" (desiccation) of the L5 disc (disc between L5 and sacrum); this represents moderate to severe degenerative disc disease. (Courtesy of Douglas M. Gillard, DC, IDE, QME.)

grade IV radial annular tear (Figure 31-3). The disc changes appearance with further progression and a full-thickness radial annular tear (Figure 31-4). The tears allow nuclear material to irritate sensitive sinuvertebral nerve-endings. Full-thickness radial annular tears, however, are not the only annular sign of the degeneration process. The *concentric annular tears* and *rim lesions* are often present (Figure 31-5). This may lead to the nuclear material extruded from the disc and irritate the sinuvertebral nerve-endings.[16–20] Note in Figure 31-6 that the sinuvertebral nerve endings adjacent to the annular tear have become inflamed and are causing pain through both the sympathetics (gray ramus) and the same-level afferent nerve roots.

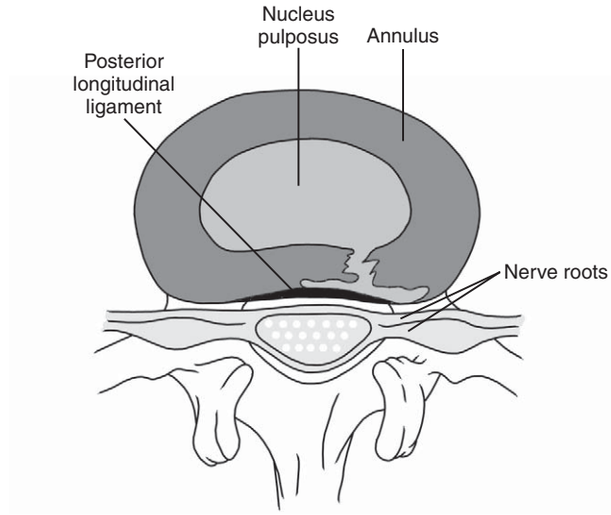


FIGURE 31–3

Grade 4 radial annular tear is shown at the 5 o'clock position of the disc.

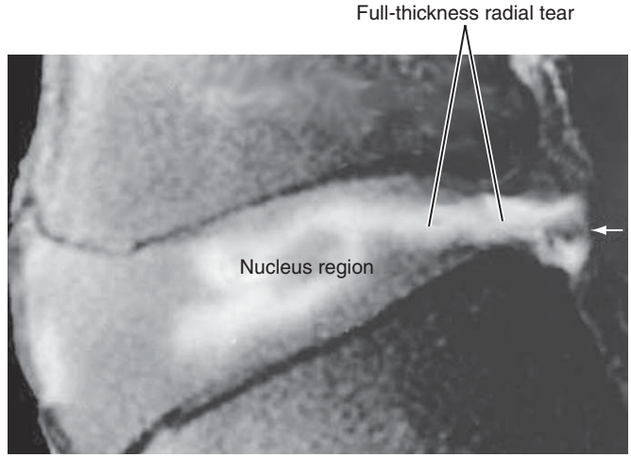


FIGURE 31–4

Full-thickness radial tear. (Courtesy of Douglas M. Gillard, DC, IDE, QME.)

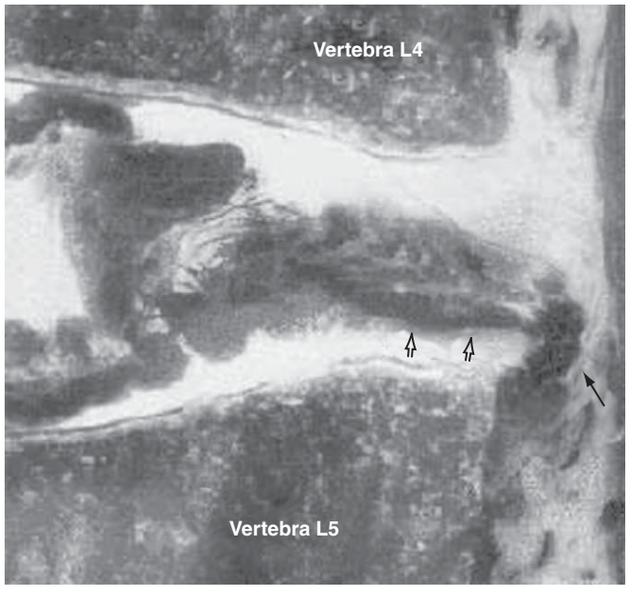


FIGURE 31–5

In this cadaver section, note the concentric annular tear and rim lesion (arrows). (Courtesy of Douglas M. Gillard, DC, IDE, QME.)

DALLAS DISCOGRAM CLASSIFICATION OF INTERNAL DISC DISRUPTION

The disc lesion can be described as contained or noncontained. The progression of disc pathology is classified as follows:

Grade 0: Normal nonleaking nucleus—all the contrast material stays within the nucleus after injection (Figure 31-7A).

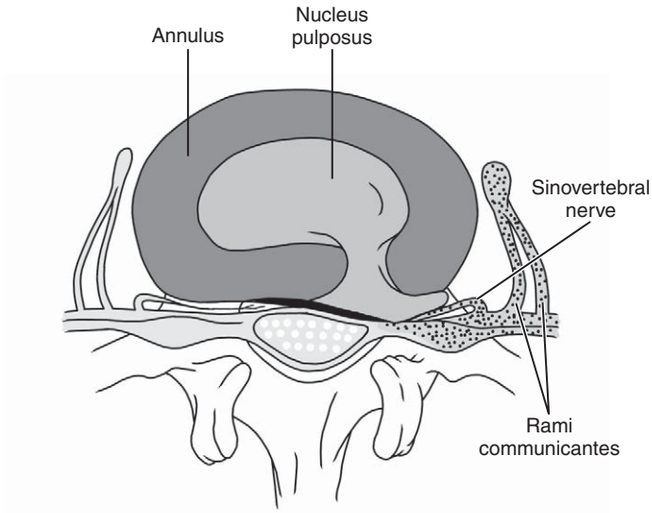


FIGURE 31–6

Nuclear material extrudes from the disc, which then irritates the sinuvertebral nerve endings. The pain can be caused through both the sympathetic (gray ramus) and the afferent nerve roots at that level.

Grade 1: Annular tearing confined to the inner region of the annulus fibrosis. This figure demonstrates the image of the disc on the axial computed tomography (CT) scan. Figure 31-7B shows the injection of a radio-opaque dye into the center of the disc. At about 5 o'clock position, a tear or fissure becomes visible. It extends from the nucleus radially into the inner one third of the annulus fibrosis. This fissure usually is not painful since there are no pain fibers in this region. This is described as a grade 1 radial annular tear, or grade 1 IDD.

Grade 2: In this condition, annular tears have completely disrupted the disc architecture but do not affect the outer contour of the annulus, which is exemplified by containment of the leak. This figure demonstrates the progression of the annular tear. The entire annulus is shown to be disrupted. Note that there is no leakage of dye from the disc, nor bulging or protrusion of the disc. This state of the disc is classified as a grade 2 IDD or grade 2 radial annular tear. There is no compressive effect on the nerve root. Many of these patients (grade 2 IDD) complain of lower back pain, which may travel into the lower limb and even past the knee into the lower leg and foot (Figure 31-7C).

Grade 3: In this situation, annular tears have now completely disrupted the annulus and posterior longitudinal ligament (PLL) and deformed the contour of the posterior disc. This figure demonstrates a full-thickness annular tear in which the annulus, outer annulus (Sharpey's fibers), and PLL have been completely ruptured. Contrast material is seen leaking out of the back of the disc into the epidural space (Figure 31-7D). The presence of a disc bulge and/or disc herniation is also included in this category. This condition is classified as grade 3 IDD or grade 3 radial annular tear. This more serious form of disc pathology produces the same incidence of patients having sciatica as the grade 2 IDD patients; it indicates that the nerve fibers in the posterior annulus are a strong trigger for the perception of sciatic pain in the lower limbs. Disc herniation can be described as contained or herniated (Table 31-1).

MODIFIED DALLAS DISCOGRAM CLASSIFICATION

The modified classification was finalized in the 1990s and is now the gold standard for the CT classification of annular tears.[22] This classification was modified by Bogduk et al.[23] in 1992, and then further modified by Schellhas et al. in 1996.[24]

Figure 31-8 demonstrates the five possible severities of the radial annular tear, as seen on an axial CT image. Grade 0 is a normal disc, where no contrast material in the center of the disc has leaked from the confines of the nucleus pulposus. A grade 1 tear has leaked contrast material but

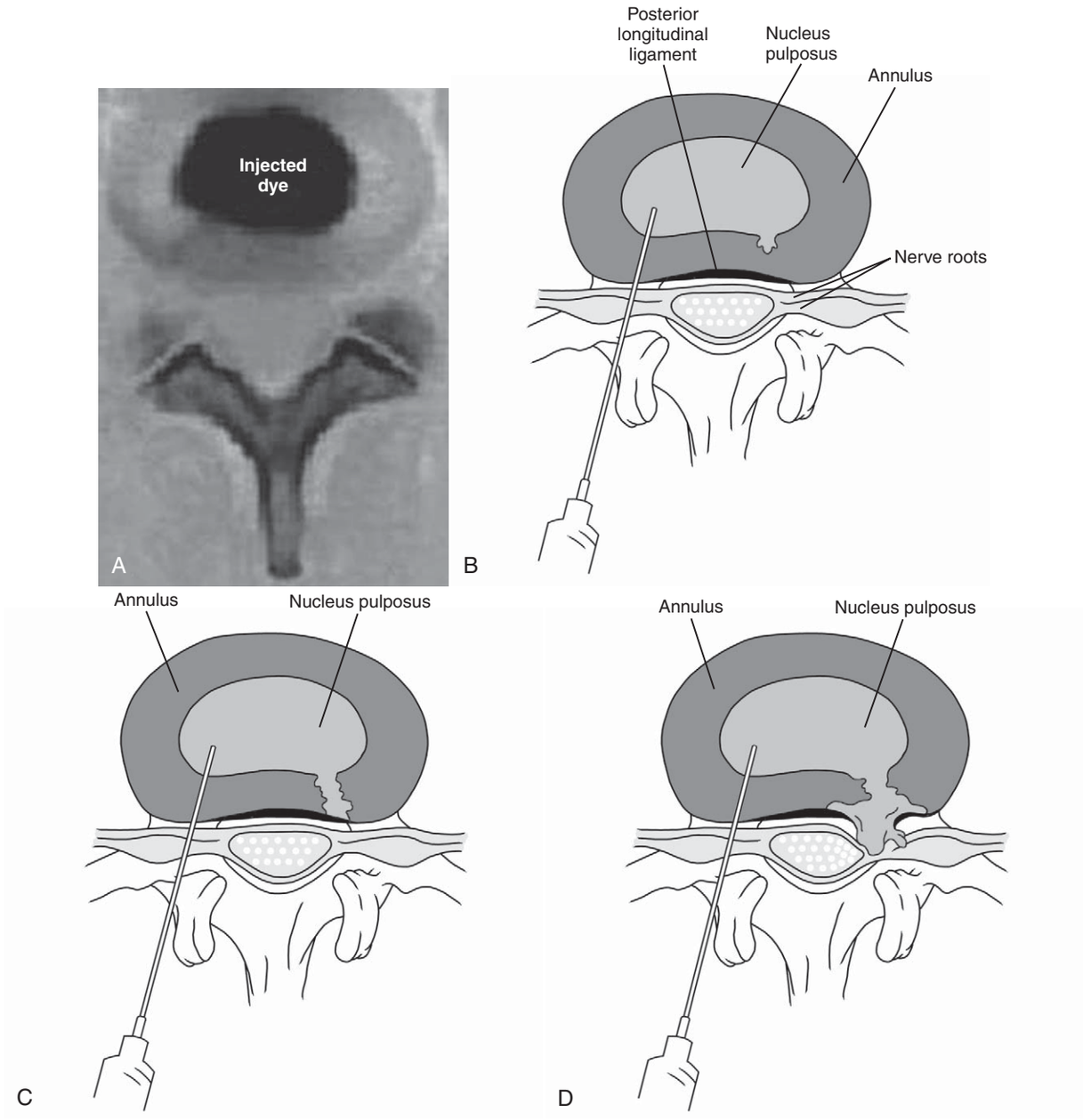


FIGURE 31–7

(A) This CT discography shows that the injected dye (black) does not leak out of the nucleus. This is considered a grade 0 normal disc. (Courtesy of Douglas M. Gillard, DC, IDE, QME.) (B) CT-scan axial view of the disc. The injection of a radio-opaque dye into the center of the disc demonstrates a tear at the 5 o'clock position of the disc. Note that the tear extends radially from the nucleus into the inner one third of the annulus fibrosis. This is described as grade 1 radial annular tear. (C) Progression of the annular tear: the entire annulus is shown to be disrupted. However, there is no leakage of the contrast solution from the disc. This is classified as grade 2 radial annular tear. (D) Complete disruption of the annulus and posterior longitudinal ligament. Contrast material is shown to be leaking out the back of the disc in the epidural space. This is classified as grade 3 radial annular tear. CT, computed tomography.

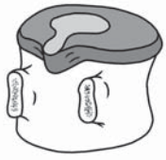
only into the inner one third of the annulus. The grade 2 tear has leaked contrast from the nucleus into the outer two thirds of the annulus. The grade 3 tear has leaked contrast completely through all three zones of the annulus. This tear is now believed to be painful since the outer third of the disc has many tiny nerve fibers that may be irritated. The grade 4 tear further describes the grade 3 tear, in that now the contrast has spread circumferentially around the disc, often resembling a ship's anchor. To qualify as a grade 4 tear, the circumferential spread must be greater than 30 degrees. Pathologically, this represents the merging of a full-thickness radial tear with a concentric annular tear. The grade 5 tear describes either a grade 3 or grade 4 radial tear that has completely ruptured the disc outer layers and is leaking contrast material from the disc into the epidural space. This type of tear is thought to have the ability to induce a severe inflam-

TABLE 31–1

DEGREES OF HERNIATION

Disc degeneration

Drying and thinning of the disc as a result of accelerated wear and tear

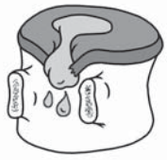


Prolapse

A disc that bulges out from its position between two vertebrae

Extrusion

A disc that bulges out from its position between two vertebrae



Sequestration

End of the extrusion phase where a portion of the nucleus pulposus is completely detached from the nucleus pulposus in the disc

matory reaction in posterior neural structures. In some patients, this inflammatory process is so severe that it causes a painful chemical radiculopathy and sciatica without the presence of nerve root compression.

PAIN MECHANISM

In 1979, Brodsky and Binder[25]characterized the mechanism for the provocation of pain with discography. Their findings included (1) stretching of the fibers of an abnormal annulus, (2) extravasation of extradurally irritating substances such as glycosaminoglycans, lactic acid and acidic media, (3) pressure on nerves posteriorly caused by bulging of the annulus, (4) hyperflexion of posterior joints on disc injection,[26] and (5) the presence of vascular granulation tissue, with pain caused by scar distension.[27] Another mechanism speculated was pain generators in the end plates that may be provoked by end-plate deflection.[26]

MODIFIED DALLAS DISCOGRAM

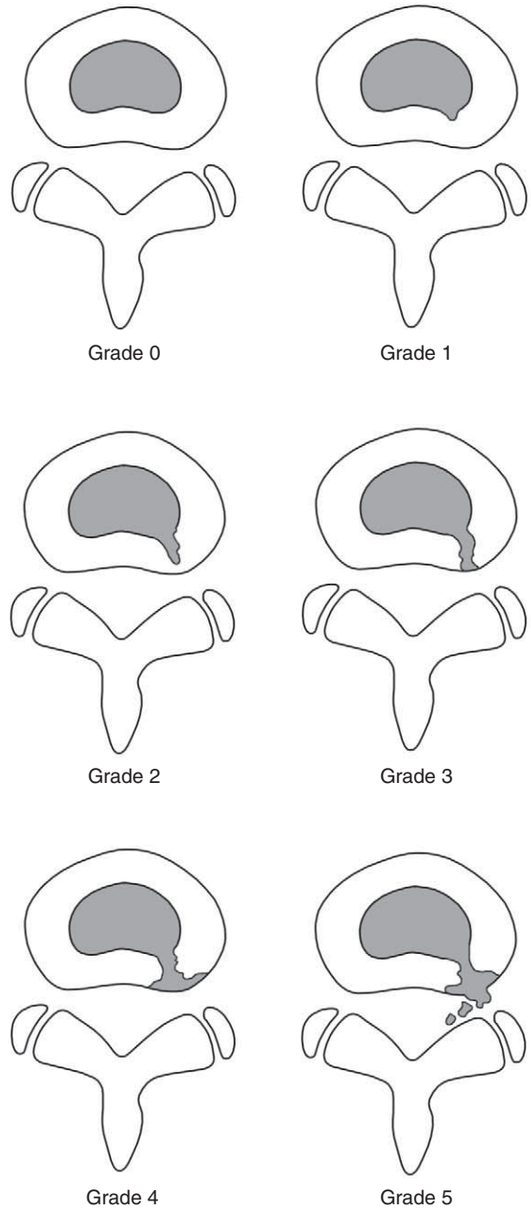


FIGURE 31–8

Five degrees of severity of the radial annular tear as seen on the axial computed tomography scan.

Ohnmeiss et al.[28] studied the typical patterns of pain referral from different degrees of discogram-confirmed posterior annular tears (i.e., internal disc disruption, IDD, radial fissures/tears). Surprisingly, they discovered that the disc does not have to be completely torn through (disrupted) for the patient to suffer lower limb pain. In fact, the discs did not even need to be completely ruptured or even bulging.

Grade 2 (outer annulus nonruptured and nonleaking) were found to reproduce lower limb pain on discography just as often as grade 3 discs (bulging, herniated, completely ruptured and leaking). About 60% of both types of IDD reproduced lower limb pains on provocative discography. This study supports the theory that nuclear material in the outer region of the posterior annulus may be a cause of sciatica on its own.

The pain pathways for discogenic pain are still very controversial.Traditionally, pain signals that originate in the nerve roots adjacent to the disc were thought to move from that root into the corresponding dorsal root ganglia (DRG) and then into the spinal cord. However, recent new research suggests that pain signals from the lower lumbar discs (L5 and L4) are detoured up the sympathetic nerves (gray ramus communicans) and into the upper lumbar DRGS—especially at the l2 level.[29–31] Clinically, it then would be possible for some patients with L4 and L5 disc problems to have L1 or L2 dermatomal pain (groin and anterior thigh pain).

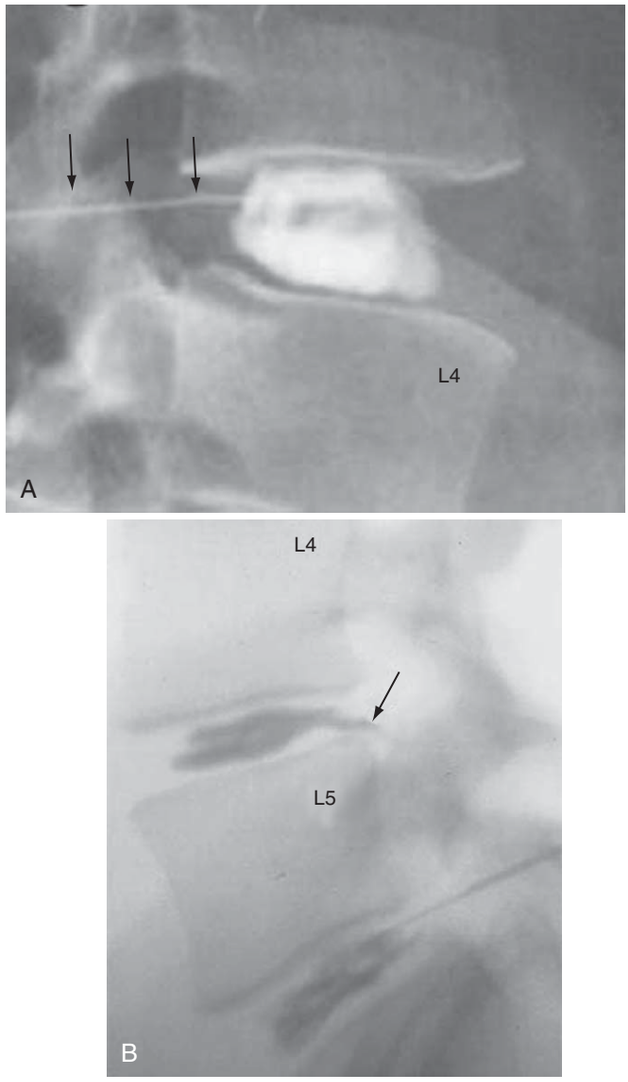


FIGURE 31–9

(A) Lateral fluoroscopic view of the lumbar spine. The needle is shown entering the L3-L4 disc space (arrows). The contrast material is shown in white. Note that the disc is contained within the disc space. (B) This is a fluoroscopic image (lateral view). The contrast material (black) is shown between the L4-L5 and L5-S1 discs. L5-S1 disc is shown to be normal. The L4-L5 disc shows leakage of the disc in the epidural space (black arrow). (Courtesy of Douglas M. Gillard, DC, IDE, QME.)

DIAGNOSIS OF INTERNAL DISC DISRUPTION

PROVOCATIVE DISCOGRAPHY

The gold standard in making the diagnosis of IDD is a very painful and invasive test called *provocation discography* with follow-up CT discogram. There are two components to provocation discography: the first is an attempt by the physician to provoke the patient to feel their usual pain (concordant pain) by pressurizing the disc with a contrast material. The second is a painless discogram in the adjacent discs (Figure 31-9).

In its taxonomy, the International Society for the Study of Pain[14] has adapted the following set of criteria for diagnosing IDD: (1) no visible disc herniations seen on MRI or CT; (2) during provocation discography injection of the suspected disc causes a recreation of patients' exact back and/or leg pain must occur[12,32]; (3) injection of the disc above or below the suspect disc must be nonpainful, as this acts as a control disc or normal disc; (4) and a grade 3 or 4 radial annular fissure must be demonstrated on CT discography.[13,33–35]

GADOLINIUM-DTPA ENHANCED MAGNETIC RESONANCE IMAGING

Although provocation discography with CT discography is the "gold standard" to make the diagnosis of symptomatic IDD, the procedure itself can damage the disc and spread the degenerative disc disease.[16–21] As an alternative, the use of gadolinium (contrast) enhancement may be considered. Gadolinium-DTPA, when injected into the vein during the MRI, will "light-up" the granulation tissue that forms within a healing/healed full-thickness annular disc tear (Figure 31-10).

The HIZ phenomenon also gives another clue that internal disc disruption might be involved in the patient's pain syndrome, although this T2-weighted MRI finding is highly controversial (Figure 31-11).

OPTIONS FOR TREATMENT OF DISC LESIONS

- Conservative management
- Chemonucleolysis
- Annuloplasty

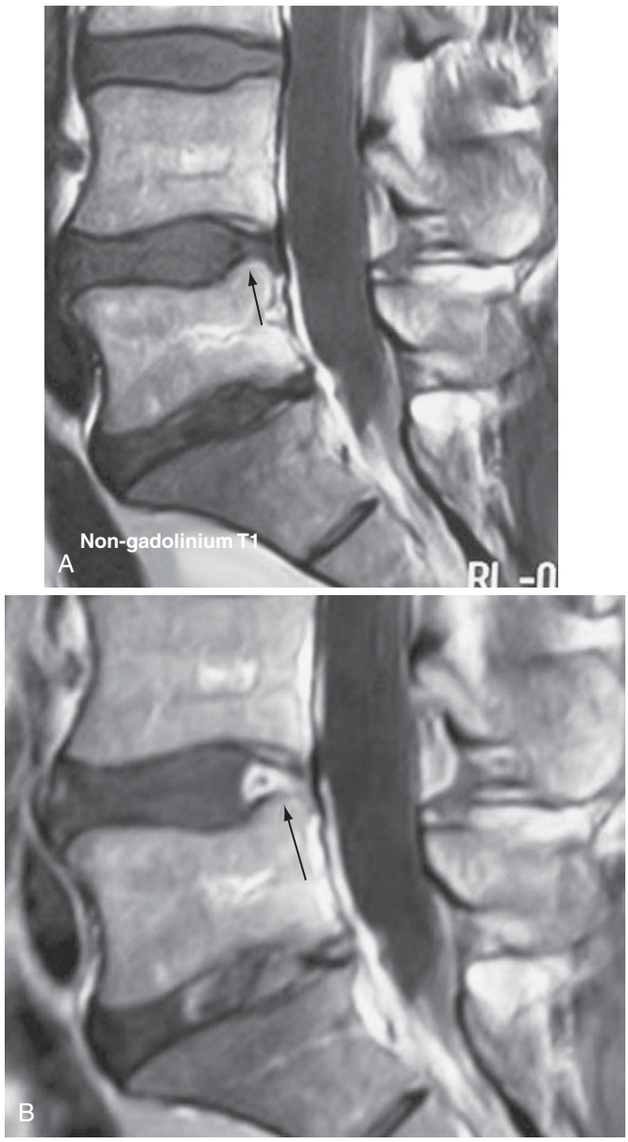


FIGURE 31–10

(A) T1-weighted MRI without gadolinium. Note the L4 disc shows no sign of posterior tearing (black arrow). (B) T1-weighted MRI after gadolinium. This image demonstrates the remains of the massive annular tear (red arrow). MRI, magnetic resonance image. (Courtesy of Douglas M. Gillard, DC, IDE, QME.)

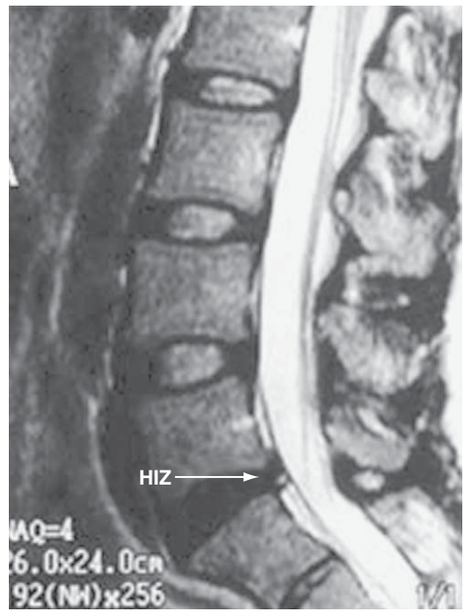


FIGURE 31–11

Pathological marker has been described as the HIZ viewed on magnetic resonance imaging scans using spin-echo gradient T2 imaging (arrow). There has been a significant correlation with the presence of HIZ in patients with symptomatic grade 3 annular fissures. The high intensity of the zone differentiates the material entrapped from that of herniated nuclear matrix is believed to indicate that the HIZ reflects the presence of inflammatory fluid. HIZ, high-intensity zone.

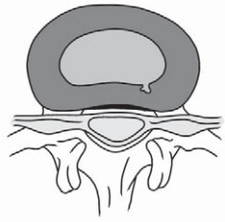
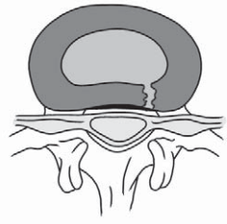
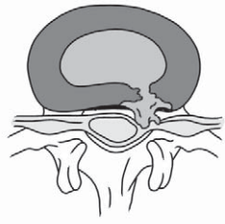
Intradiscal electrothermal therapy (IDET)
DiscTRODE™ procedure

- Percutaneous discectomy
  - Nucleoplasty
  - Dekompressor® procedure
  - Laser discectomy
- Endoscopic percutaneous discectomy (Table 31-2)

CONSERVATIVE MANAGEMENT

Ninety percent of all IDD patients will obtain satisfactory pain relief by conservative measures. However, for the conservative treatment to be effective, it may take many

TABLE 31–2

INDICATIONS		
	Axial Discogenic back pain	IDET
	Herniation with back and leg pain Herniation with radiculopathy	IDET/ Decompression
	Large extrusion or sequestration	Surgery

months. Conservative treatment often takes the form of oral analgesics, gentle traction, and nondynamic spinal stabilization treatments and exercise.

CHEMONUCLEOLYSIS

In the early 1940s, chymopapain was derived from the papaya fruit by Jansen and Balls, as reported by Jaikumar and colleagues.[36,37] By 1956, Thomas saw the potential for this enzymatic substance and began work on determining a use for this substance. He injected the chymopapain intravenously into the ears of rabbits and noted that the ears became floppy.[38] Thomas confirmed that the softening of the cartilaginous material in the ear was due to the chymopapain. Smith et al. picked up the torch and hypothesized that chymopapain could be used to treat chondroblastic tumors, which it did not; however, they did find that when injected into the intradiscal space of rabbits, the nucleus pulposus disappeared but left the annulus intact.[37,38] In 1963, Smith injected the first human patient with chymopapain to treat sciatica. Chymopapain works by depolymerizing the proteoglycan and glycoprotein molecules in the nucleus pulposus.[37] These large molecules are responsible for water retention and turgidity. When exposed to chymopapain, the water content within the disc plummets; shrinkage follows and causes a reduction in disc height and girth. The bulging disc therefore shrinks.

The patient is placed in either a lateral or a prone position. Under conscious sedation and guided by fluoroscopy, a 6-inch, 18-gauge needle is inserted posterolaterally and placed centrally within the disc. Discography, along with the pain provocation test, is performed for evaluation of the affected disc. Chymopapain is then injected into the nucleus pulposus in amounts ranging from 1000–4000 U. The number of units injected decreases if more than one disc is to be treated[37,38] (Figure 31-12).

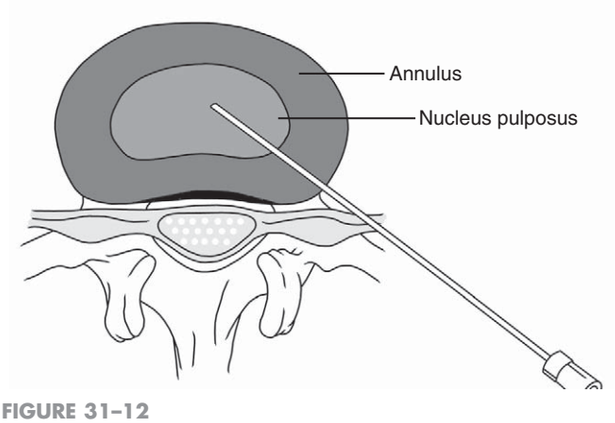


FIGURE 31–12
Introduction of chymopapain in the center of the nucleus pulposus of the disc.

Chymopapain has been in use for over 30 years. After approval by the U.S. Food and Drug Administration (FDA) in 1983, early complications were reported even though studies had shown a very safe record.[38] Anaphylaxis, reported in 1% of cases, proved to be the most severe complication.[38] It became clear that good patient selection, proper surgical training and technique, preoperative hypersensitivity testing, and antihistamine administration could greatly reduce the complication rate when using chymopapain. In fact, complications became almost nonexistent in the late 1980s and 1990s when utilizing the aforementioned criteria.

Indications for chymopapain include those patients who present with radicular pain as the chief complaint; confirmation of the disc herniation via MRI, CT, or myelogram; and patients having failed conservative treatment.[37–39] Kim et al.[40] found that patients with moderate to severe positive straight leg raise had a significantly higher success rate as compared to those with no or mild SLR pain. The younger the patient, the better the outcome. Younger patients had a success rate ranging from 82.3% for those in their 30s to 94.6% for those in their teens. Patients 50 years and older had only a 71% success rate.[40] Patients in whom the pain provocation test was positive had a 91.7% success rate compared to those who did not experience pain provocation at a 73.1% success rate.[40]

Chemonucleolysis has been fraught with controversy since FDA approval in 1982. Some wish to dismiss the procedure as dead due to complications in earlier use (and lack of manufacture and distribution within the United States since 1999), but there are others who state that with the proper inclusion criteria, preprocedure testing, and good technique, chemonucleolysis has a significant place in the minimally invasive category.[37,38,40]

ANNULOPLASTY

Intradiscal Electrothermal Therapy (Annuloplasty)

Intradiscal electrothermal therapy (IDET) was developed by Saal in 2000 as an alternative to fusion for patients with chronic discogenic low back pain.[41,42] There are several proposed causes for discogenic back pain. Some have stated that discogenic back pain is due to an internal disc disruption, most likely due to annular tears or fissures.[37] Others feel that the discogenic pain may be a result of degenerative disc disease.[39] Even the developers of the current IDET procedure state that the pathophysiology of discogenic pain is complex and hard to pin down into a single complete and true definition.[40] What is agreed upon is that the intervertebral disc, particularly the annulus, has nociceptive nerve receptors, which increase when the disc degenerates, or is injured or exposed to a variety of inflammatory substances. This increase in neuro pain receptors causes increased and unremitting low back pain.[41,43,44] The IDET was therefore developed to modify collagen—making it thicker and causing it to contract—and thus decreasing the body's ability to revascularize (Figures 31-13 to 31-16).

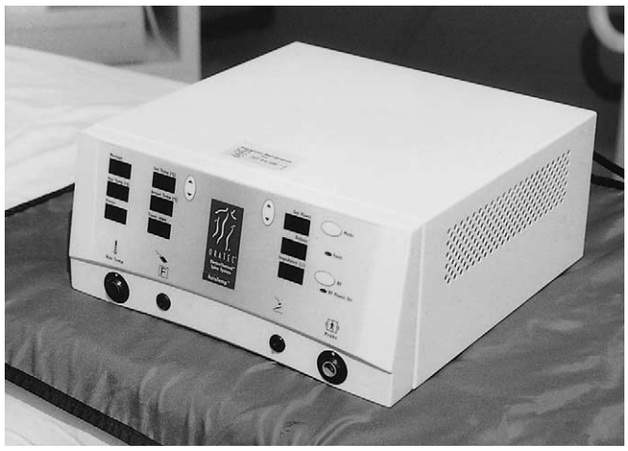


FIGURE 31–13
Oratec intradiscal electrothermal coagulation lesioning base unit.

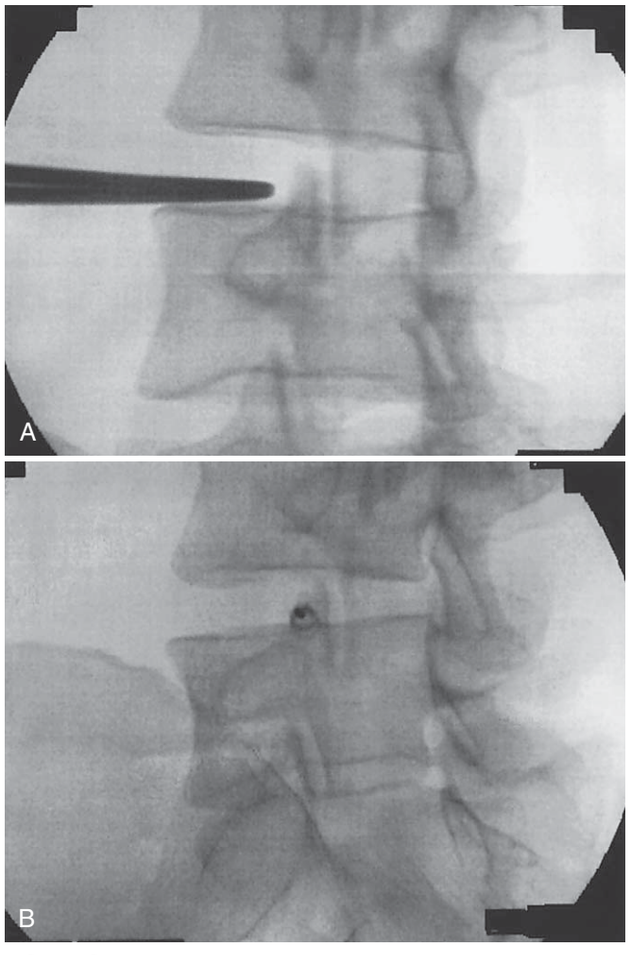


FIGURE 31–14
(A) Patient's position and the C-arm in the oblique position to visualize the disc and its endplates. Radiographic marker that identifies the entry point on the skin for approaching the disc. (B) "Tunnel" view of the introducer needle (arrow) just lateral to the superior pars articulares.

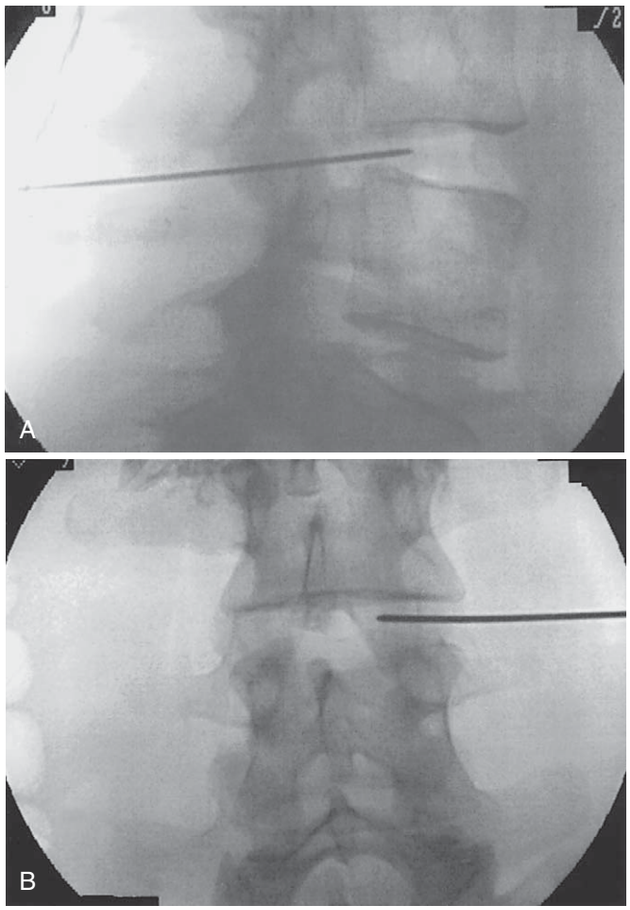


FIGURE 31–15
(A) Lateral view of the introducer needle in the transition zone between the annulus fibrosis and the nucleus pulposus. (B) Anteroposterior view of the introducer needle in the transition zone between the annulus fibrosis and the nucleus pulposus.

The procedure itself is performed with fluoroscopic guidance, while the patient is under conscious sedation lying prone. As with many intradiscal procedures, discography, along with the pain provocation test are used to evaluate the affected disc. A 17-gauge needle is inserted posterolaterally into the disc, generally from the patient's less painful side. A 30-cm catheter with a flexible 5–6-cm heating tip is threaded circumferentially into the disc through the nucleus pulposus to the pathologic area of the annulus. After fluoroscopic confirmation, the catheter tip is heated to 90°C over a 13-minute period. Once at 90°C, the temperature is maintained for an additional 4 minutes. The catheter and needle are then removed. The patient is then transferred and observed in recovery before being discharged home the same day.[40,41,43,44]

Indications for the IDET include long-term low back pain, failure of conservative therapy, normal neurologic exam, negative straight leg raise, MRI confirmation of no neural compressive lesion, and positive pain provocation test.[44] Exclusion criteria include inflammatory arthritis,

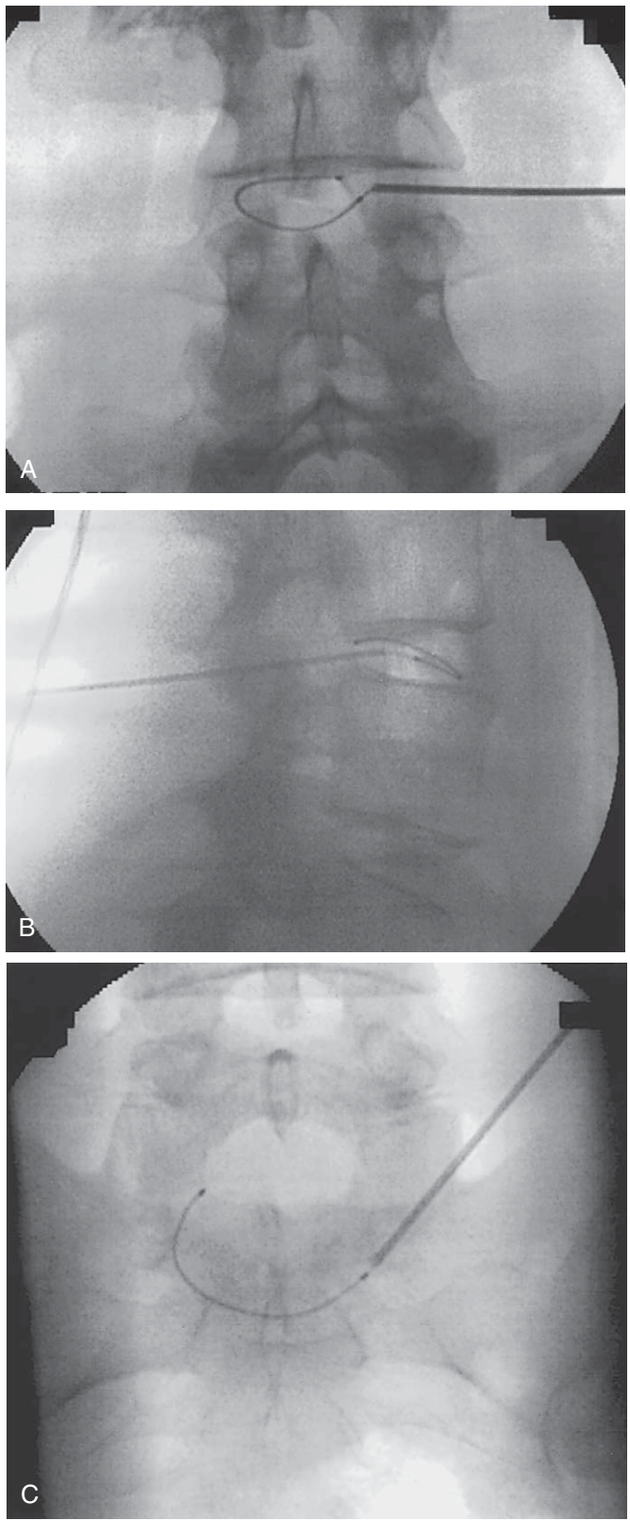


FIGURE 31–16

Anteroposterior (A), lateral (B), and cephalocaudal (C) fluoroscopic images showing the introduction and placement of spine catheter during the intradiscal electrothermal therapy procedure.

nonspinal conditions that mimic lumbar pain, and any medical or metabolic condition that would preclude proper follow-up.[41,44,45] Much debate still centers on this technique, as there have been limited independent studies and long-term follow-up of patients receiving IDET.[41,42,46] The collagen modification could also lead to a reduction in size of annular fissures and increase the stability of the disc itself.[40,43] IDET also thermocoagulates the nociceptors within the annular walls, thus destroying the ability to transmit nociceptive input.[43,44]

Complications include catheter breakage, nerve root injuries, post-IDET disc herniation, cauda equina syndrome, infection, epidural abscess, and spinal cord damage.

In summary, the evidence for IDET is moderate in managing chronic discogenic low back pain.

discTRODE™ Procedure

Radiofrequency (RF) annuloplasty is a minimally invasive method of delivering RF thermal energy to the disc to treat lower back pain. Valleylab's discTRODE™ RF catheter electrode system uses heat to coagulate and decompress disc material, providing effective pain relief. Ideal candidates for a discTRODE™ procedure are patients with chronic low back pain that has been determined to result from an internally disrupted disc.

The discTRODE™ procedure is typically performed on an outpatient basis in a clinical setting. Both a mild sedative and a local anesthetic may be used to reduce any discomfort that the patient might experience (Figure 31-17).

Using x-ray guidance, the physician will insert the discTRODE™ cannula into an intervertebral disc. The catheter electrode is passed through the cannula into the outer disc tissue. RF current flows through the electrode, heating the tissue directly adjacent to the active tip of the electrode to a specific treatment temperature. An additional external temperature monitor allows the physician to continuously observe temperature changes in surrounding tissue throughout the procedure.

Complications are similar to IDET with catheter breakage, nerve root injuries, discitis, disc herniation, cauda equina syndrome, infection, epidural abscess, and spinal cord damage.[47–49]

THERMAL DISCOPLASTY (NUCLEOPLASTY)

Thermal discoplasty or nucleoplasty™ (ArthroCare, Sunnyvale, CA) is another procedure that has broken into the minimally invasive field in the 21st century. The first nucleoplasty was performed in 2000.

Procedure combines disc removal and thermal coagulation to decompress a contained herniated disc (Figure 31-18). With the patient in a prone or lateral position under sedation, a posterolateral approach guided by fluoroscopy is made with a 17-gauge obturator stylette. A discogram may take place at this time to confirm location and for a positive provocation test. Taking care not to contact the anterior annulus, the

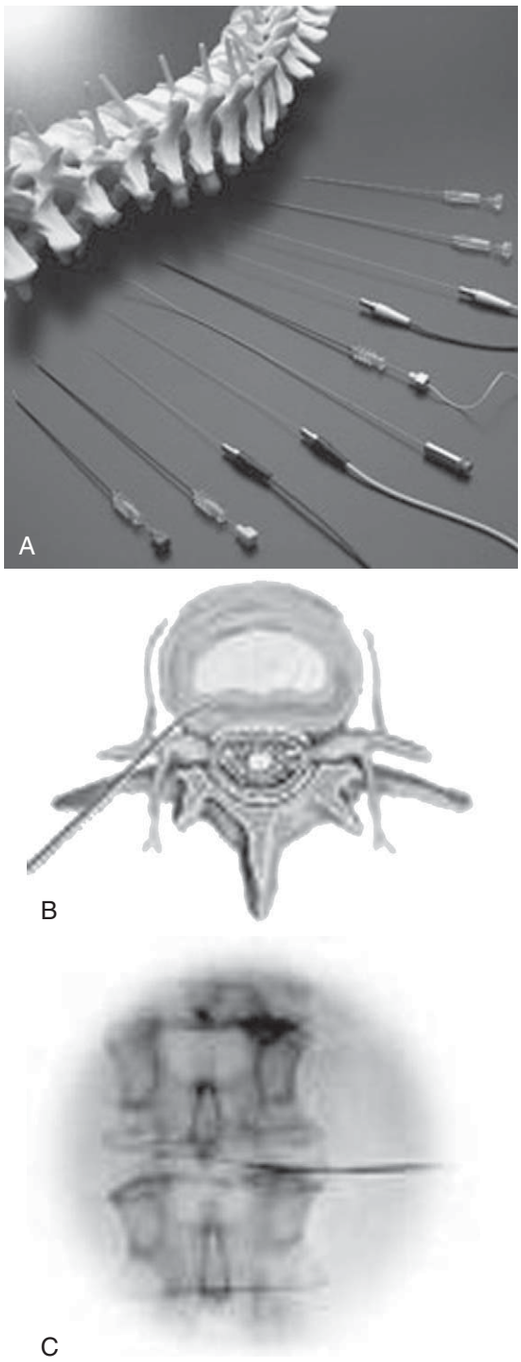


FIGURE 31–17

(A) This figure shows a discTRODE curved cannula introducer for radiofrequency and annuloplasty. (B) Line drawing of placement of the catheter in the disc at the specific site of radiofrequency annuloplasty (posteriorly). (C) Fluoroscopic view of the discTRODE in place in the posteroanterior view. (Courtesy of Valleylab, a division of Tyco Healthcare Group LP.)

nucleus pulposus is first ablated with RF waves as the wand is advanced, causing a molecular dissociation process converting tissue into gas, which is removed through the needle. As the wand is withdrawn, coagulation takes place thermally treating the channel, which leads to a denaturing of nerve fibers adjacent to the channel within the nucleus pulposus. This process is repeated up to six times within an individual disc.[50,51] The patient is then sent to recovery and later sent home the same day.

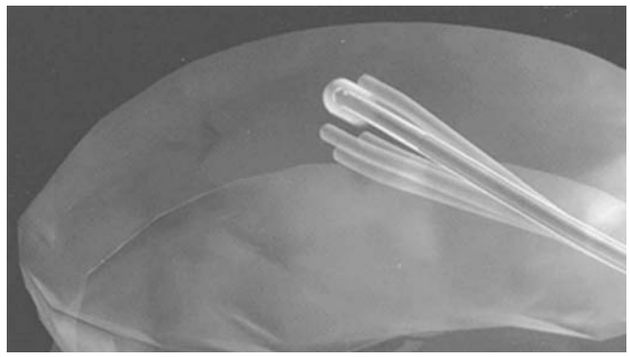


FIGURE 31–18

Nucleoplasty coagulation channels within the nucleus pulposus after ablation. (Courtesy of ArthroCare, Sunnyvale, CA.)

Indications for this procedure include low back pain with or without radiculopathy, MRI confirmation of contained herniated disc, and failed conservative therapy.[50] Patients who should be excluded from receiving this procedure include those with spinal stenosis, a loss of disc height of 50%, severe disc degeneration, or spinal fracture or tumor.[50]

PERCUTANEOUS DISCECTOMY

Percutaneous lumbar discectomies (PLDs) have been performed for over 30 years with overall results ranging from disappointing to good. The techniques and equipment used for percutaneous discectomy vary widely and have fallen in and out of favor. Hijikata et al.[52] first reported performing a percutaneous *nucleotomy* in 1975. The procedure included the use of 3–5-mm cannulas from the posterolateral approach, curettes, and time consuming manual removal of the nucleus pulposus with pituitary forceps. The theory was that the reduction of intradiscal pressure would reduce irritation of the nerve root and the nociceptive nerve receptors in the annulus.[53] The procedure remained limited in use until 1985, when Onik et al.[54] developed a new and smaller type of aspiration probe, which reduced risk of injury to the peripheral nerves and the annulus, facilitated easier removal of the nucleus pulposus with an all-in-one suction cutting device, and decreased surgery time.

Both procedures use a posterolateral approach to the affected disc on an outpatient basis. The automated procedure is performed while the patient is in a lateral decubitus or prone position. An 18-gauge hubless sheath with a central trocar is guided toward the affected disc. The trocar is removed, and a smaller 2.5-mm cannula with an inner blunt end sleeve is placed over the hubless sheath. Once correct placement is confirmed, the hubless sheath is removed, leaving the 2.5-mm cannula. A 2-mm saw is

threaded through the cannula, and a hole is cut into the annulus for the aspiration probe to be inserted. The aspiration probe is a sharpened cannula, fitted through an outer needle. Using suction to pull in disc material, the inner, sharpened cannula uses a slide-like cutting motion to slice the tissue, which is then aspirated, along with irrigation, through the inner cannula to a collection bottle.[10] The probe, which is pedal activated, is gently moved back and forth within the disc until no more disc material is aspirated and then the probe is rotated. When aspirated disc material decreases significantly, the probe is removed from the disc space, usually within 20–40 minutes.[55,56]

Dekompressor Procedure

Little changes have been made in automated discectomy until recently with innovations in automation. The newest entry is the Dekompressor® (Stryker Corp., Kalamazoo, MI), introduced in 2002. The Dekompressor® is a disposable, self-contained, battery-operated hand piece connected to a helical probe. The outer cannula measures 1.5 mm with an inner rotating probe. When activated, the probe rotates creating suction to pull milled nucleus pulposus from the disc up the cannula to a suction chamber at the base of the hand-held unit. Approximately 0.5–2 cc of nucleus pulposus is removed. This efficient removal of disc material decreases surgical procedure times to approximately 30 minutes, with the actual time of use for the probe not exceeding 10 minutes.[57] This procedure is done under fluoroscopic guidance (Figure 31-19). The Dekompressor® technique has yet to be studied in a controlled clinical trial, and results with this new automated technique are limited. Percutaneous discectomy has a success rate with multiple authors reporting 60–87% positive outcomes.[58]

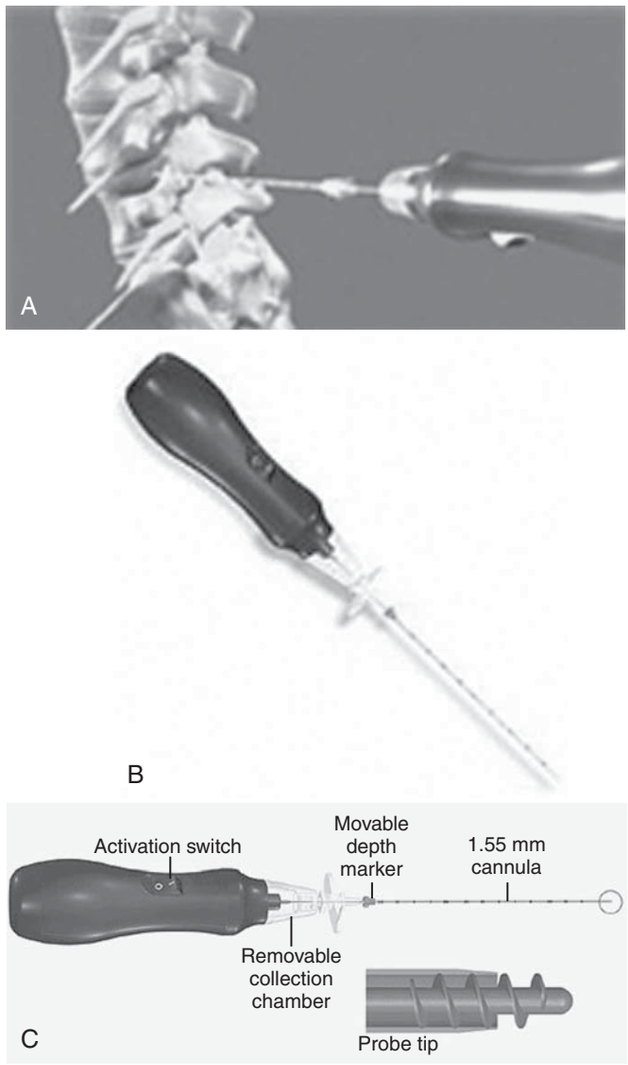


FIGURE 31–19

(A) This drawing shows the placement of the needle into the inflamed disc with the posterolateral approach in a model skeleton. (B) The Dekompressor is a disposable, battery-operated, self-contained unit connected to a helical probe. The outer cannula (shown here) measures 1.5 mm with an inner rotating probe. When activated, the probe rotates, creating suction to pull nucleus pulposus from the disc up the cannula to a suction chamber at the base of the hand-held unit. (C) Shows and describes the various parts of the Dekompressor unit in detail. (Courtesy of Stryker Corporation, Kalamazoo, MI.)

Laser Discectomy

Medical lasers have been used since the early 1960s. Jaikumar et al.[36] and Choy et al.[59] published their experiences with the use of a neodymium:yttrium-aluminum-garnet (Nd: YAG) laser on the lumbar spine for nucleolysis. There are several types of lasers in use for the lumbar spine, with the most common being the holmium:yttrium-aluminum-garnet (Ho:YAG) laser. The others are potassium-titanyl-phosphate (KTP) and the neodymium (Nd):YAG laser. The Ho:YAG laser is most commonly paired with the endoscope for disc ablation and removal capabilities.[60,61] This laser-assisted technique combines two effective but limited approaches (Figure 31-20).

As the affected tissues absorb the laser, light is converted to heat. At 100°C, tissue vaporizes and ablation takes place. As a small amount of nucleus pulposus is vaporized, intradiscal pressure decreases, allowing the disc to return to its normal state.[53,62] If any disc material needs to be removed, endoscopic tools can be used to do so.

The patient is placed in a prone or lateral position under conscious sedation. An 18-gauge, 7-inch needle is introduced just anterior to the superior articular process and superior to the transverse process via a triangular safe zone. Using fluoroscopy, the needle is placed 1 cm beyond the annulus into the nucleus pulposus just parallel to the disc axis, preferably halfway between the superior and inferior end plates.[62] If the procedure is endoscopically assisted, dilators are placed over the guide needle for visualization and the introduction of the endoscope. Irrigation with saline allows for better visualization of the spaces. Depending on the type, the laser is either fired as a pulse or continuously.[62] The Ho:YAG laser is pulse fired. Newer laser models offer side-firing capabilities. This advancement helps to provide more control of laser placement, provide better observation, and can help reduce the risk of

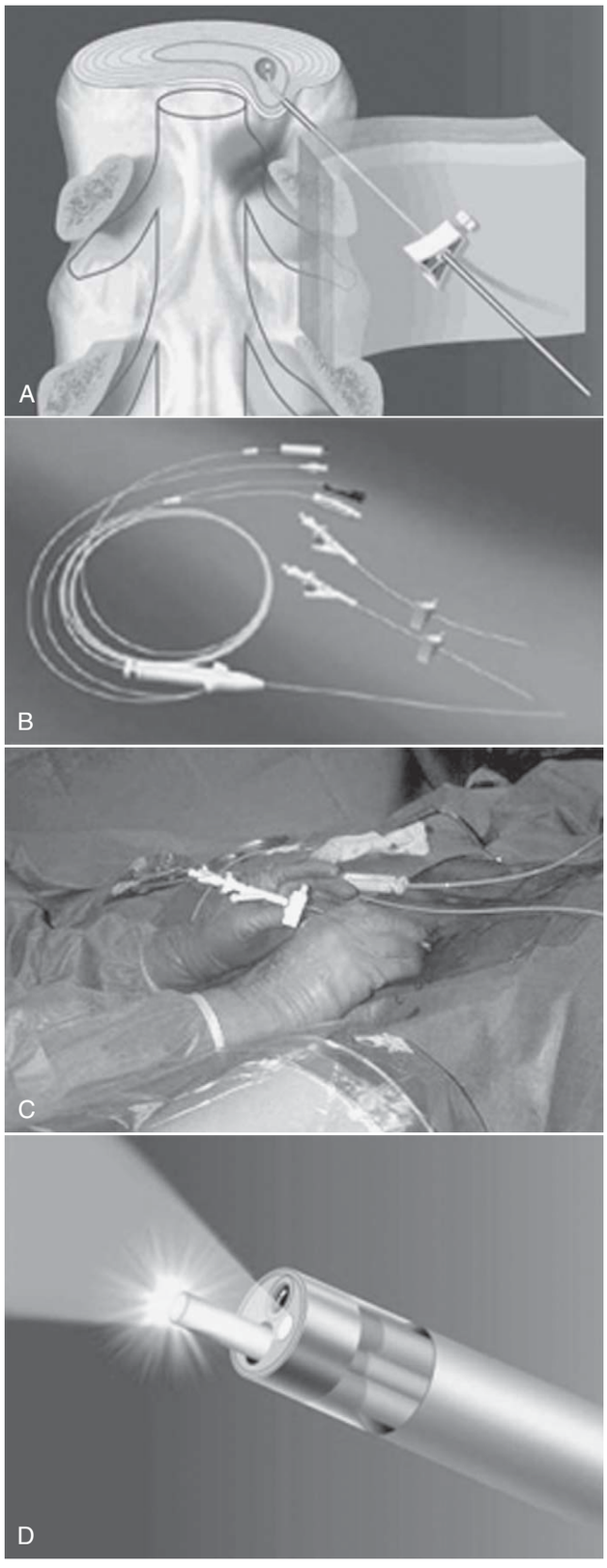


FIGURE 31-20

(A) This picture shows the technique of placing the LASE® cannula (18-gauge, 7-inch needle) into the affected disc. (B) Shows LASE unit prior to use. (C) This figure shows the LASE procedure being used for endoscopic percutaneous discectomy. (D) Light projection from the LASE device from the endoscope. (Courtesy of Clarus Medical, LLC.)

injury to several areas, especially those anterior to the spinal column.[37] Larger fragments, which are more difficult to remove through the endoscope, can be laser ablated. After firing the laser and adequate nucleus pulposus has been removed/ablated, the laser and dilators are removed. The incision can be closed with sutures or surgical adhesives. The patient is moved to recovery and sent home later in the day.

Indications for laser discectomy are presence of back and leg pain with a confirmed disc herniation. A ruptured annulus and lateral recess stenosis are not contraindications.[62,63] Newer transforaminal procedures can treat those patients with fragments in the epidural space.[63] In 2002, Tsou and Yeung[62] reported the 9-year retrospective results of their percutaneous transforaminal approach, with an 88.1% excellent to good result. Other studies report success rates from 78 to 85% in retrospective studies.[37,62,63] There is a scarcity of clinical trials regarding percutaneous lasers. Negative aspects of the laser include a steep learning curve for the physician. The use of lasers coupled with an endoscopic approach significantly increases the difficulty level for the surgeon.

ENDOSCOPIC DISCECTOMY

Burman in 1931 was the first reported author who introduced the concept of the direct visualization of the spinal cord. A few years later, Mixter and Barr[64] performed an open laminectomy with discectomy for the treatment of a disc herniation into the spinal canal. Later on, Pool[65] introduced the concept of intrathecal endoscopy and reported the outcomes of more than 400 myeloscopic procedures. Due to surgical complications of intraspinal surgery, endoscopy remained forgotten until the work carried out by Ooi et al.[66] during the 1970s.

Hijikata et al.[67] in 1975 demonstrated a percutaneous nucleotomy by means of an arthroscopy for disc removal for the treatment of posterior or posterolateral lumbar disc herniation under local anesthesia. Kambin described the safe triangular working zone (Kambin's triangle) and results of arthroscopic microdiscectomy, in which arthroscopic visualization of the herniation via the posterolateral approach was used for discectomy of contained herniations. In 1985, Onik et al.[68] reported the development of a 2-mm blunt-tipped suction cutting probe for automated percutaneous discectomy (PD) at L4-L5 or higher levels.

TRANSFORAMINAL ENDOSCOPIC MICRODISCECTOMY

The technique of foraminal epidural endoscopic discectomy (FEES) was developed from epidural endoscopy. FEES differs from other percutaneous discectomy procedures in that direct visualization of the epidural space, pathology, and neuroanatomic structures is possible.[69]

INDICATIONS

Indications for spinal endoscopy, approved by the FDA, include (1) documentation of pathological features and compression of structures, (2) direct nerve inspection, (3) inspection of internal fixation, and (4) delivery of therapeutic agents.

Recently, the use of spinal endoscopy has been expanded to include closed decompression of spinal roots, use with lasers, epidural biopsies, percutaneous interbody fusion, lysis of epidural adhesions, and decompression of thoracic disc herniation.

PATIENT SELECTION

As with other forms of minimally invasive surgical disc procedures, patient selection is critical. Patients should have leg pain more severe than back pain and 6 months of failed conservative therapy. The ideal pathology is a virgin paramedian, foraminal, or extraforaminal contained herniation, or one that is noncontained at less than 50% of the canal diameter.

GENERAL OVERVIEW OF TECHNIQUE

The patient is sedated and placed in the prone position (Figure 31-21). Fluoroscopic identification of the disc level is performed with the aid of a needle placed at the skin surface. The entry point is 9–13 cm from the midline. Discographic confirmation of the pathology is usually done prior to foraminoscopy. The goal is to access the "neural triangular working zone" defined by the exiting root, the proximal vertebral plate inferiorly, and the superior articular facet. This goal is facilitated by endoscopic entry under the pars and superior articular facet and through the foramen. The annulus is incised medial to the traversing root. A clinical trial is currently under way to investigate the foraminal approach by combining the posterolateral discographic with the laparoscopic retroperitoneal dissection.[70] This method utilizes a discographic needle with a bevel near the distal end, which deflects the nerve root anteriorly when inserted into the disc space. It also contains a hole oriented at 35 degrees to the long axis and through which a triangulated arm is placed. After the retroperitoneum is insufflated through a trocar placed in the flank, a second trocar is inserted through the arm and an endoscope introduced through the trocar. A variety of instruments can then be introduced to perform a foraminotomy, partial facetectomy, and decompression of the nerve root.

REGIONAL ENDOSCOPIC TECHNIQUES

Percutaneous Endoscopic Lumbar Discectomy

Percutaneous endoscopic lumbar discectomy is the ultimate form of minimal invasive spine surgery. In this technique, an endoscope is used. The whole procedure is done under local anesthesia and the patient is fully awake during surgery. The patient is made to lie prone on the operating table, and an exact entry point is mapped on the patient's body using an image intensifier x-ray system (Figure 31-22). A long spinal needle is passed from the posterolateral aspect of the lumbar spine. Through this needle, a guidewire is inserted. Then, a 5-mm incision is made on the skin. Following that, a dilator and a working cannula are inserted under local anesthesia, through which the endoscope is passed. A camera and monitor are attached to the endoscope, and the prolapsed part of the disc is removed under vision. The wound is closed with a single stitch. The patient usually gets immediate pain relief. After satisfactory vital signs and monitoring, the patient can go home in 24 hours.

ADVANTAGES

Surgery done under local anesthesia with conscious sedation.
5-mm skin incision.
Endoscope used for surgery.
No muscle, ligament, or normal tissue damage.
Targeted fragmentectomy (directly prolapsed disc tissue removed).
Minimal blood loss.
Patient can be discharged in 24 hours.
No prolonged bed rest required after surgery. Can resume work sooner.
Even prolapsed, migrated, extraforaminal, recurrent discs can be removed. Very good technique for old and medically compromised patients.

PATIENT SELECTION

Patient selection is based on clinical symptoms and radiological evidence. A rationale of strong hypothetical correlation between nucleus pulposus removal and decompression of the affected nerve root for pain alleviation is mandatory. Extruded fragments are assessed. Percutaneous discectomy (manual or automated) is only appropriate for patients with single-level disc disease.

PROGNOSIS

The size of the disc protrusion is an important factor in obtaining successful outcomes with PD. If the disc herniation is over 50% of the AP diameter of the thecal sac, the patient has a more than 90% probability of a poor outcome in pain relief using this technique.

Contraindications

- Previous history of chymopapain or surgical treatment for disc disease
- A solid bone fusion that does not allow access to the disc
- Progressive neurological deficits; bowel or bladder problems

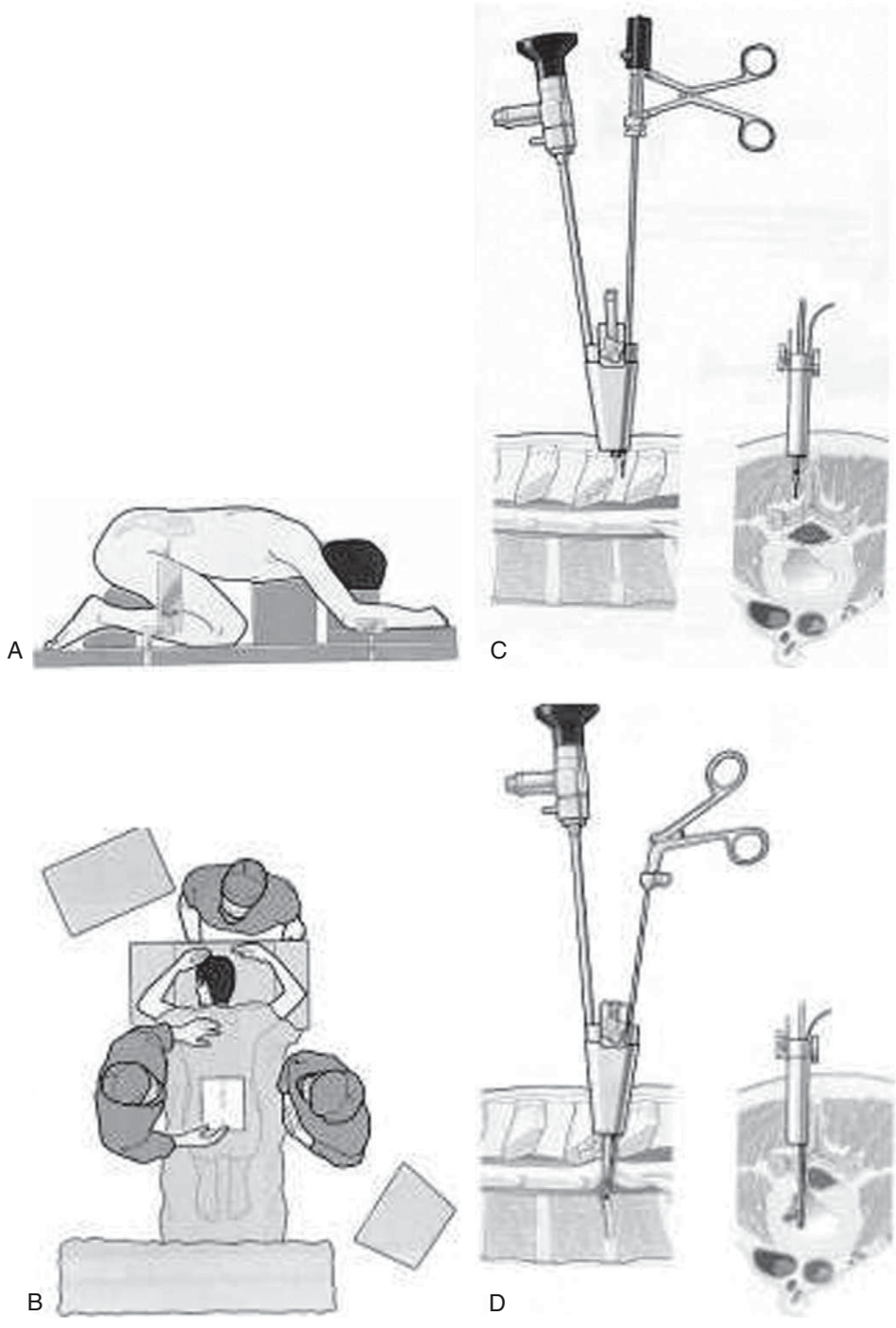


FIGURE 31–21

(A) Position of the patient during microdiscectomy. (B) Microdiscectomy procedure being performed. (C) Technique of passing the cannula and other instruments in the disc space. (D) The position of the cannula inside the disc.

- Disc disease with sequestered disc fragments
- Patients with evidence of structural vertebral disease (spinal stenosis, spondylolisthesis)
- Coagulopathy
- Pregnancy (teratogenic effects of radiation)
- Systemic infection or skin infection over the puncture site
- Discitis
- Multiple (more than one) level degenerative disc disease
- Severe allergy to any component of dye injection mixture (iohexol) or other medication

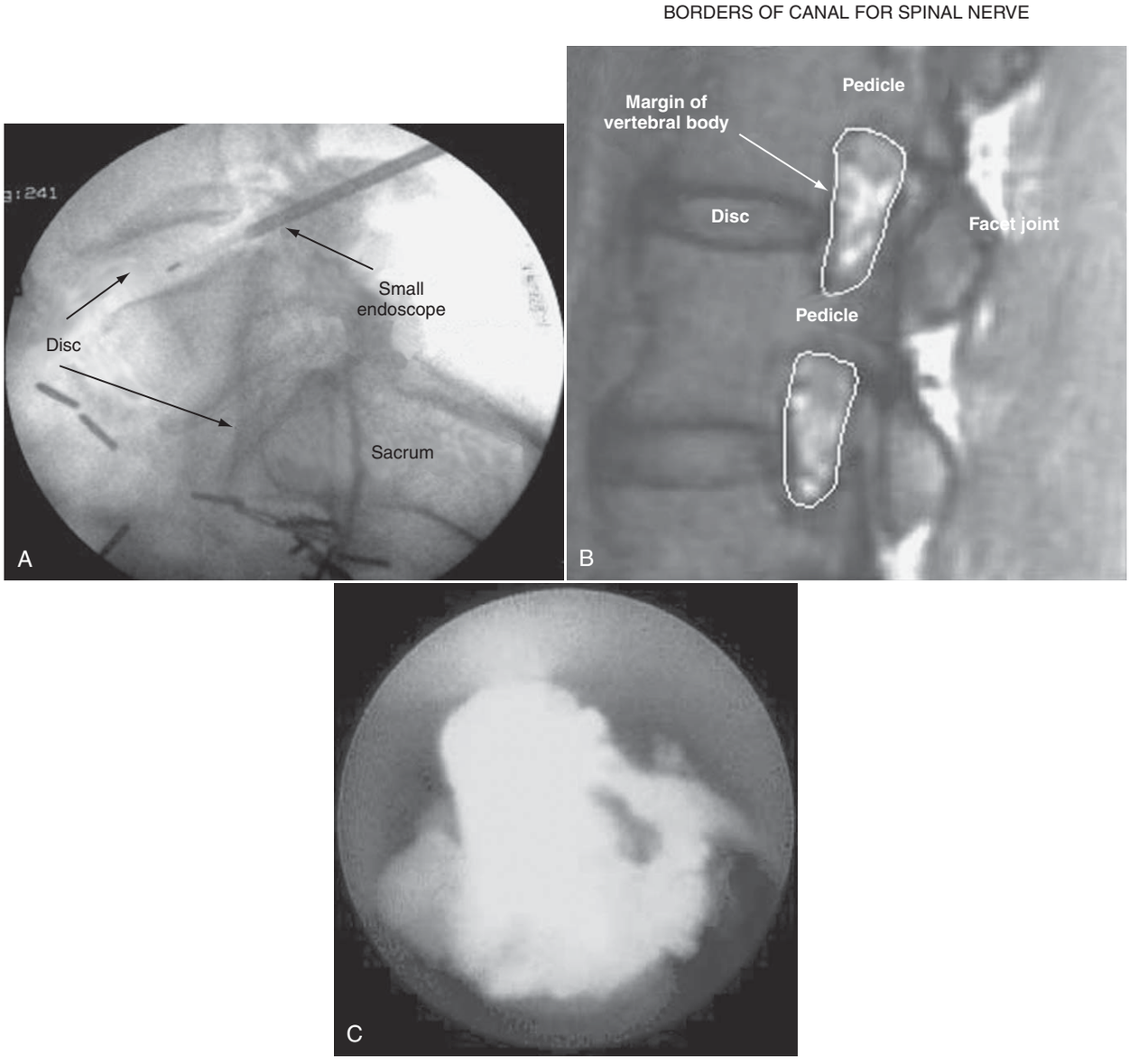


FIGURE 31-22

(A) This fluoroscopic view shows the placement of the endoscope into the L4-L5 disc. (B) The boundaries of the area (transforaminal) where the cannula and the endoscope have to be passed. (C) Piece of disc as seen through the endoscope.

Preoperative Assessment

- Obtain plain x-ray (AP, lateral, and obliques) prior to surgery.
- Evaluate CT and/or MRI scans of the entire abdomen through the involved disc space.
- Obtain informed consent.

Anesthesia

- IV sedation (conscious sedation).
- Mild analgesic medication can be administered during the procedure to make the patient more comfortable (midazolam 2–3 mg, and fentanyl, 100–150 micrograms).
- Local anesthesia of the area to be incised and also given along the expected path of the cannula tract.
- Prophylactic IV antibiotic therapy within 1 hour before the procedure.
- The patient must be able to fully understand the questions during the procedure and the quality of pain produced.

PROCEDURE

L1-L2 through L4-L5 Endoscopic Discectomy

The disc puncture should always be performed in the center of the field of view. The C-arm is rotated in an oblique angle until the superior articulating process (ear of the

"Scottie dog") is visible. The superior end plates of the same vertebral body should superimpose.

L5-S1 Endoscopic Discectomy

For insertion of cannulas into the L5-S1 level, significant caudal angulation is required for optimal visualization. The C-arm is rotated in a fashion similar to that to the upper lumbar discs. Generally, the window that the needle has to pass through is represented on the fluoroscopic images as a small triangle formed by the inferior end plate of L5, the superior articulating process of S1, and the iliac crest. However, the iliac crest may obstruct the approach irrespective of the angle of trajectory used. If one is not able to achieve a position that places the superior articulating process at the midpoint of the vertebral body, one obtains the best possible angle that allows visualization of the upside-down triangle. The puncture site for L5-S1 is usually higher than that for L4-L5.

Clinical Pearls

The cannula must be inserted parallel to the x-ray beam and advanced, using intermittent fluoroscopy, so that the needle tip stays just ventrolateral to the superior articulating process and midway between the vertebral end plates. Contact with the posterolateral margin of the disc can elicit a mild or moderate sharp pain as the needle passes through the disc's outer fibers (Sharpey's fibers).

CERVICAL PERCUTANEOUS ENDOSCOPY

The discs in the cervical region cannot be approached posteriorly because of the spinal cord, anteriorly because of the airway, or posterolaterally because of the vertebral artery and the uncinate process. Many authors believe that a right-sided approach should always be used for right-handed practitioners and vice versa.

The C-arm is placed in the AP position, and cranial or caudal angulation of the C-arm is used to optimally visualize the disc space. The cannula puncture site should be made between the carotid sheath and the airway. The carotid pulse at the disc level is then palpated with the index and middle fingers, and the carotid sheath structures are displaced laterally by manual palpation.

Complications

- Paraspinal or epidural hematoma
- Localized infection
- Epidural abscess
- Quadriplegia or paraplegia
- Myelopathy

Outcome Studies

One large-scale prospective nonrandomized investigation followed 402 patients who were treated for disc herniation–associated sciatica. Two groups were formed; the first consisted of 220 patients who had undergone surgery; the second consisted of 182 patients who had chosen to be conservatively (nonsurgically) treated. This study has demonstrated that surgical treatment for disc herniation–associated sciatica is faster and slightly more effective than conservative, nonsurgical conservative care. There have been reports of more than 7,000 automated or manual percutaneous lumbar discectomy procedures performed. Published results indicate an overall success rate of 75% with a complication rate of 1%[71] (Tables 31-3 and 31-4).

EVALUATION OF INTRADISCAL THERAPIES

INTRADISCAL ELECTROTHERMAL THERAPY

Appleby et al.[72] in a systematic review reviewed the literature from all the available studies and concluded that there was compelling evidence for the relative efficacy and safety of intradiscal electrothermal therapy. Freeman[73] performed a critical appraisal of the evidence of IDET and concluded that the evidence for its efficacy remains weak and has not passed the standard of scientific proof. The present evidence summarizes one positive randomized trial, one negative randomized trial, and seven positive prospective evaluations,[74–80] with two negative reports.[81,82] The evidence for intradiscal electrothermal therapy (IDET) is moderate in managing chronic discogenic low back pain.

TABLE 31–3 Success Rate of Surgical vs. Nonsurgical Treatment of Lumbar Disc Herniation: 5-Year Follow-up

Outcome at 5 Years	Surgical Group (%)	Nonsurgical Group (%)
Satisfied with outcome (delighted, pleased, very satisfied)	63	46
Patient is working	91	84
Patient's pain is completely gone	28	12
Patient's pain had worsened	13	14

Source: Atlas SJ, Deyo RA, Wu YA, et al: Long-term outcomes of surgical and nonsurgical management of sciatica secondary to a lumbar disc herniation: 10-year results from the Maine Lumbar Spine Study. *Spine* 30(8):927–935, 2005, with permission.

TABLE 31–4 Success Rate of Surgical vs. Nonsurgical Treatment of Lumbar Disc Herniation: 10-Year Follow-up

Outcome at 10 Years	Surgical Group (%)	Nonsurgical Group (%)
Reoperation/operation rate	25	25
Reported at least some improvement from their predominant symptom	69	61
Back or leg pain (main complaint) is completely gone or much better	56	40
Satisfied with current status	71	56

Source: Atlas SJ, Deyo RA, Wu YA, et al: Long-term outcomes of surgical and nonsurgical management of sciatica secondary to a lumbar disc herniation: 10-year results from the Maine Lumbar Spine Study. *Spine* 30(8):927–935, 2005, with permission.

DISCTRODE PROCEDURE

Finch et al.[83] studied 31 patients by heating of their annular tears with a flexible radiofrequency electrode placed across the posterior annulus and compared 15 patients with conservative management. The visual analogue scale decreased significantly. The evidence for radiofrequency posterior annuloplasty was limited for short-term improvement and indeterminate for long-term improvement in managing chronic discogenic low back pain.

PERCUTANEOUS DISC DECOMPRESSION

Waddell et al.,[84] in a systematic review based on Cochrane Collaboration Review and meta-analysis of surgical interventions in the lumbar spine,[85] identified three trials comparing automated percutaneous lumbar discectomy (APLD) with other surgical techniques and concluded there was limited and contradictory evidence. Randomized trials of APLD and microdiscectomy included Chatterjee et al.[86] and Haines et al.[87] Chatterjee and colleagues compared APLD to microdiscectomy in the treatment of contained lumbar disc herniation in a randomized study with blind assessment. The study included 71 patients with radicular pain as their dominant symptom after failure of conservative therapy for at least 6 weeks and with MRI demonstration of contained disc herniation at a single level with a disc bulge of less than 30% of the canal size. The study excluded patients with dominant symptoms of low back pain, disc extrusion, sequestration, subarticular or foraminal stenosis, or multiple levels of herniation. The results showed satisfactory outcomes in 29% of the patients in the APLD group and 80% of the microdiscectomy group. They concluded that APLD was ineffective as a method of treatment for small, contained lumbar disc herniations. The authors were criticized in that they failed to utilize CT discography. Haines et al.[87] conducted a randomized study comparing APLD with conventional discectomy as a first-line treatment for herniated lumbar discs. The study measured outcomes with physical signs related to the severity of low back pain and sciatica but used a modified Roland Scale for disability assessment and the SF-36 for general health status. The primary endpoint was the patients' outcome ratings 12 months after surgery. The study included patients with unilateral leg pain or paresthesia with no history of lumbar spinal surgery, whereas exclusions included moderate or advanced lumbar spondylosis, spondylolisthesis, lateral restenosis, herniated disc fragment occupying more than 30% of the AP diameter of the spinal canal, herniated disc fragment migrating more than 1 mm above or below the disc space, calcified disc herniation, lateral disc herniation, or posterior disc space height less than 3 mm. Success rate of the two procedures was identified as APLD at 41% compared with conventional discectomy at 40%. However, they concluded that the study did not have power to identify clinically important differences because of insufficient patient enrollment. Among the prospective evaluations and case series studies,[87,88] all of them reported positive results in greater than 50% of patients in a large population. The evidence is moderate for short-term and limited for long-term relief.[89]

PERCUTANEOUS LASER DISCECTOMY

Based on the systematic review by Waddell and colleagues,[84] there is no acceptable evidence for laser discectomy. Relevant studies evaluating the effectiveness of laser disc decompression included 14 studies meeting inclusion criteria. There were no randomized trials. The evidence is moderate for short-term relief and limited for long-term relief.[90–95]

NUCLEOPLASTY

There were no systematic reviews evaluating the effectiveness of nucleoplasty thus far in the literature. The effectiveness of nucleoplasty has been reported in six prospective studies.[96–101] Sharps and Isaac evaluated 49 patients.[97] The evidence of nucleoplasty is limited for short- and long-term relief.

The Dekompressor probe is a mechanical high-rotation-per-minute (RPM) device designed to extract the nuclear material through an introducer cannula using an auger-like device that rotates at high speeds. Dekompressor is one of the new methods that extract the nuclear material of the disc using a high RPM spiral tip instrument. There have been no systematic evaluations of percutaneous disc decompression utilizing Dekompressor. There also have not been any guidelines describing this technology. Amoretti et al.[98] published results of a clinical follow-up of 50 patients treated by percutaneous lumbar discectomy using the Dekompressor. Although the study is not a blinded and randomized study, the data collection was thought to be good. The evidence for percutaneous disc decompression utilizing ekompressor is limited for short- and long-term relief.

CONCLUSION

This chapter describes emerging techniques for treatment of disc pathology (contained or herniated). There have been advances in the type of needle to use, type of catheter to use, and the method of lysis of disc pathology. In addition, endoscopy and video photography have been added to these procedures. All comprise efforts to decrease the morbidity from classical open spinal procedures such as laminectomy.

Will these advances really decrease the morbidity and improve the safety? This cannot be answered yet. The evidence-based data are scant for these emerging techniques. Prospective head-to-head comparisons are few for these procedures. An effort should be made to

execute controlled comparative studies in the future. Multicenter studies with strict criteria need to be designed to answer the question of effectiveness and safety of these techniques.

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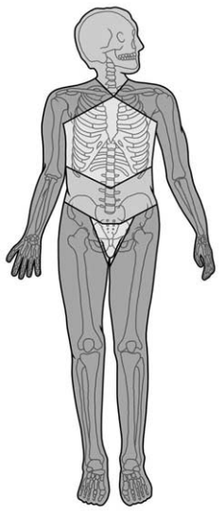

32 Vertebroplasty

RICARDO RUIZ-LOPEZ
AND CARMEN PICHOT

INTRODUCTION

Vertebral compression fracture due to osteoporosis is a common problem, with an estimated annual incidence of 500,000 new patients in the United States. Medical advances aimed at slowing or arresting bone loss from aging have only partially solved this problem, and the population affected is expected to grow steadily as life expectancy increases. Traditional conservative treatment for these fractures consists of nonsteroidal anti-inflammatory agents and brief use of narcotic analgesics; a short period of immobilization, followed by gradual mobilization; activity modification; and possibly a spinal orthosis and physical therapy. This regimen is often successful but has shortcomings: bed rest is fraught with complications in an elderly population, including pulmonary compromise and decubitus ulcer formation. Furthermore, pain relief is neither immediate nor a guaranteed outcome. Consequently, percutaneous vertebroplasty has been used to treat osteoporotic compression fractures, with a growing clinical experience suggesting considerable pain relief.

HISTORY

Vertebroplasty was originally developed in 1984 by Deramond and Galibert, a radiologist and a neurosurgeon, respectively, and in 1987, it was presented in the literature as a technique to percutaneously stabilize vertebral bodies affected by an hemangioma.[1] At the end of the same decade, another group reported that the technique could also be used to stabilize fractured vertebral bodies.[2] It has been performed in the United States since 1995. The vertebroplasty method was further developed by the introduction of the percutaneous balloon kyphoplasty technique.[3]

ANATOMY AND PHYSIOLOGY

The mechanism of analgesia during vertebroplasty is intriguing, as polymethylmethacrylate (PMMA) injection may offer its analgesic effect, not only by vertebral body solidification and mechanical stabilization of the fracture per se, but perhaps also by opposing osteoclastic activity, which causes neuropathic pain via vanilloid receptor (VR1) and acid-sensing ion channel (ASIC3) sensitization, due to changes in the acidotic extracellular microenvironment following PMMA injection, which may diminish pain signal transmission into the spinal cord and attenuate persistent neuropathic pain.[4]

Another interesting mechanism might be associated with the temperature elevation in the bone–cement interface, the epidural space, and the adjacent disk following PMMA injection: increased temperature may induce neuromodulatory effects on neighboring nervous structures such as the posterior annulus, the sinu-vertebral nerve, and the segmental dorsal root ganglion.[5]

INDICATIONS

- Vertebroplasty has three main clinical uses: for painful or collapsing vertebrae due to hemangioma, spinal metastases, or osteoporotic bone loss. It is a minimally invasive procedure that is effective in the treatment of pain resulting from pathologic compression fractures, osteolytic bone lesions, myelomas, hemangiomas, and osteoporosis.[6]
- The main indication is the symptomatic (painful) osteoporotic compression fracture of the thoracolumbar spine. It is essential to discriminate an acute event with sudden onset of pain and plain x-rays showing radiological signs of an acute

fracture from chronic painful disorders of the spine. Magnetic resonance imaging (MRI) is crucial to inform us of the age of a deformity of a vertebral body (acute fractures usually showing a significant bone edema in STIR sequences), and to exclude the protrusion of bone fragments into the spinal canal (Figure 32-1).

- Patients with chronic pain arising from vertebral fractures can be selected for those procedures on the basis of MRI or bone scintigraphy showing ongoing activity that correlates with painfulness.
- Malignant processes involving vertebrae are another indication. In this context, percutaneous vertebroplasty has been used for treatment of patients with painful, collapsing vertebrae due to metastatic cancer or myeloma. Specific indications include painful fracture refractory to the medical management and worsening collapse of a vertebral body.[7]
- Painful vertebral hemangioma constitutes an indication as well.

CONTRAINDICATIONS

- General contraindications include uncorrectable coagulation disorders, infectious processes of the spine, and allergies against PMMA or contrast medium.
- Poor pulmonary status and difficulty lying prone are relative contraindications. Multiple previous surgeries or obesity may impede proper identification of anatomical landmarks.

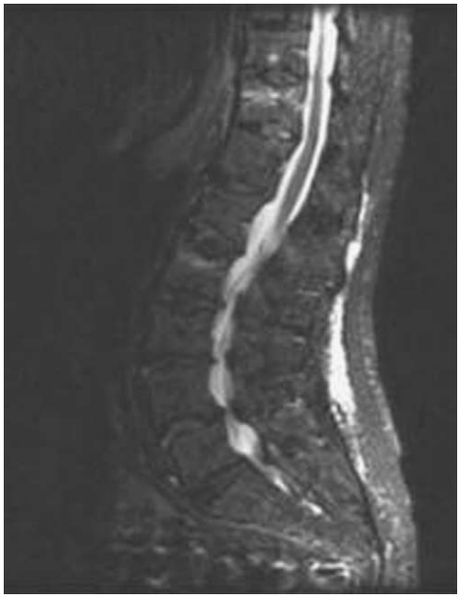

FIGURE 32–1

Magnetic resonance image showing bone marrow edema due to recent vertebral fractures at L2 and T11.

- Burst fractures with destruction of the posterior wall constitute a contraindication because of the risk of extravasation of the cement into the epidural space.
- Pre-existent neurological deficit is a contraindication, since any minor cement leakage may worsen the deficit. A previous spinal stenosis must be considered a relative contraindication. Damaged pedicles or articular facets can be considered as relative contraindications as well.
- Lack of coherence between the patient's pain and the radiological lesion.
- Loss of vertebral body height by more than 60–65%, soft tissue extension of metastasis, and cortical defects, especially of the posterior body wall, are also relative contraindications.[8]
- There is no absolute exclusion criteria based on the time of the fracture. However, fractures older than 3 months are less likely to benefit from vertebroplasty. The exceptions to this rule are the presence of instability or recurrent fractures.

PROCEDURE

EQUIPMENT

- High-quality fluoroscopic unit (biplanar)
- 11-gauge bone biopsy needle (trocar–cannula system)
- Cement based on polymethylmethacrylate (PMMA): a mixture of powder compound (a copolymer consisting of 68% methyl-methacrylate-styrene, 30% barium sulfate, and 2% benzoyl peroxide) and a liquid catalyst (methyl-methacrylate monomer in sterile fluid)

ANESTHESIA

Both procedures can be performed either under mild sedation with local anesthesia or general anesthesia, but there must be the possibility of converting the operation to an open emergency operation in cases of severe bone–cement leak affecting the spinal canal. The major disadvantages of general anesthesia are its risks in elderly patients and the inability to obtain verbal feedback from the patient regarding neurological symptoms during the procedure. Prophylactic antibiotics are given intravenously (1 g cefazolin), as this procedure involves injecting foreign material (PMMA).

PATIENT POSITIONING

Patients are placed in the prone position. Cylindrical cushions can be positioned across the table, one under the patient's chest and the other under the pelvis in order to

achieve maximum extension of the spine. Padding of pressure points is important to avoid rib fractures and to add comfort.[9]

TECHNIQUE

The skin is cleaned and draped in a sterile fashion. An anteroposterior (AP) projection is obtained (Figure 32-2). The vertebral body and the pedicles are identified, and the x-ray beam is obliqued enough to see the pedicle as a circle (Figures 32-3 and 32-4). The entry point for the transpedicular technique is in the lateral superior quadrant of the pedicle (Figure 32-5). In the higher thoracic region, an extrapedicular route lateral to the pedicle is recommended. This approach allows the needle tip to be angled more toward the center of the vertebra and thus allows easy filling of the vertebra with a single needle.

Local anesthesia is injected to the skin, subcutaneous layer, and the periosteum of the bone at the bony entry site. A 0.5-cm paramedian incision is made at each side of the spine for insertion of the trocar. An 11-gauge biopsy needle is inserted transpedicularly and advanced under AP fluoroscopic control, in an anteromediocaudal direction (Figure 32-6) with rotatory hand pressure or using a mallet. In the lateral view (Figure 32-7), the needle must be at the upper midpoint of the pedicle so that the needle advances in the midpoint of the pedicle (Figure 32-8). The needle should follow a path that is parallel to the superior and inferior edges of the pedicle. In fractured vertebral bodies with vertebral end plate depression, a more horizontal direction may be required to avoid traversing the end plate. A long needle holder can be used to avoid radiation to the surgeon's hand. In osteoporotic vertebrae, the insertion of the cannula is easy, but in osteoblastic tumors it can be harder.

With a lateral fluoroscopic projection, at the posterior vertebral body, the beveled needle is rotated 180 degrees to point medially and advanced into the anterior third portion of the vertebra (Figure 32-9), since this area is devoid of venous plexuses. If a bilateral technique is used, both cannulas should be inserted before the cement is injected

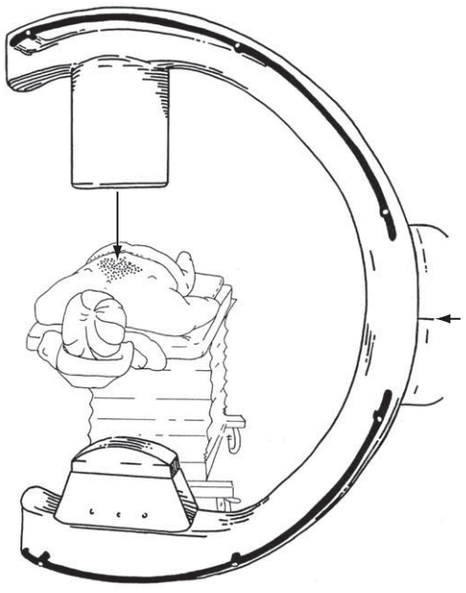

FIGURE 32–2
Drawing shows the fluoroscope positioned for a posteroanterior view of procedure side (vertebral level). The patient is prone.

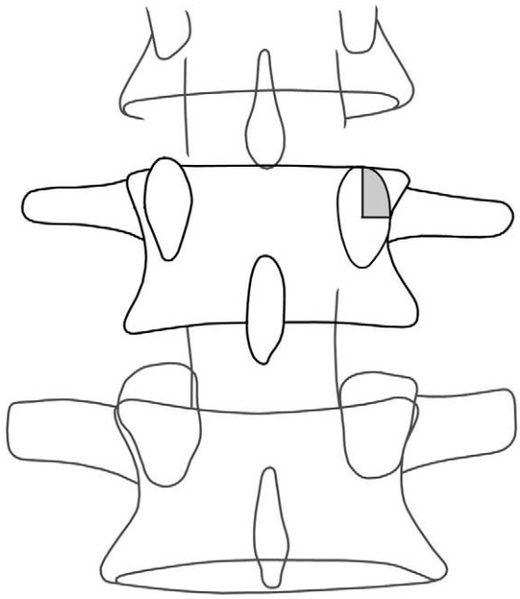

FIGURE 32–3
Anteroposterior projection showing the entry point at the pedicles.

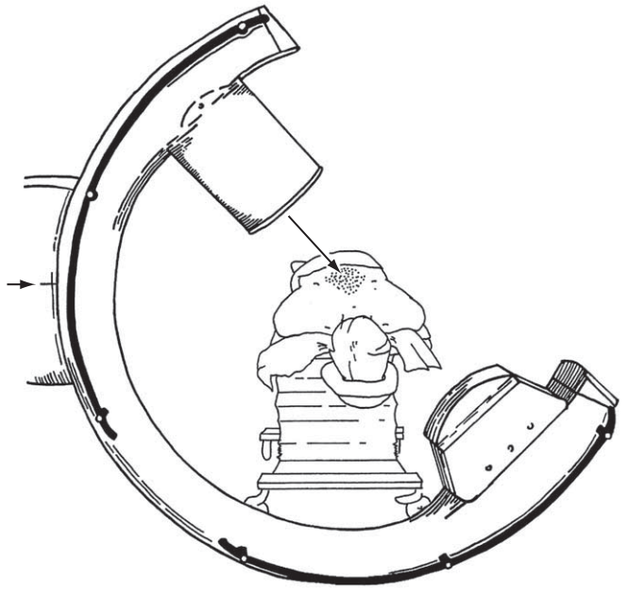

FIGURE 32–4
Drawing shows the rotation of the fluoroscope to obtain the oblique view of the vertebral level where the procedure is to be performed.

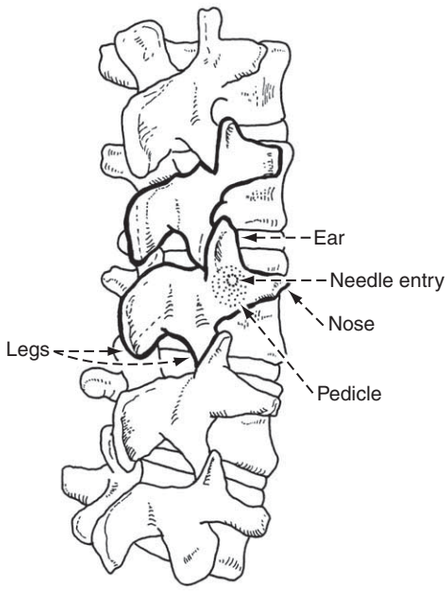

FIGURE 32–5
An oblique view of the lumbar vertebral bodies shows the Scottie dog image.

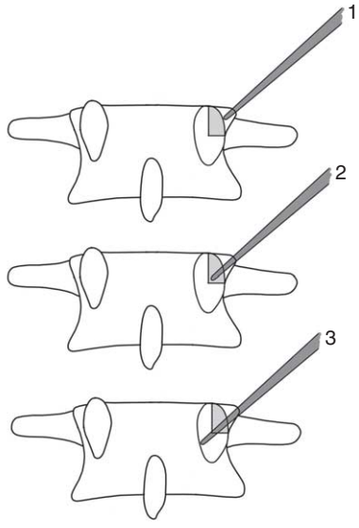

FIGURE 32–6
Anteroposterior view. Cannula entering through the pedicle into the vertebral body.

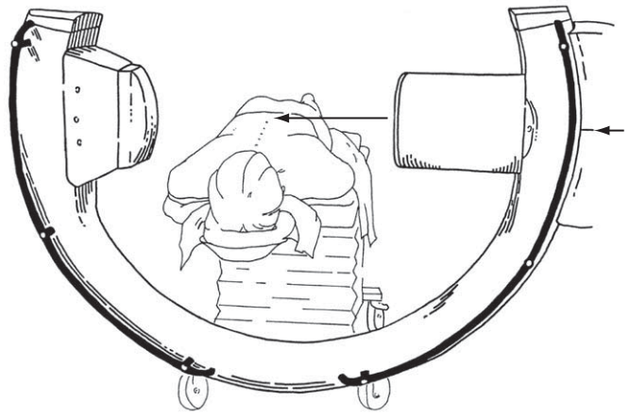

FIGURE 32–7
The position of the fluoroscope to examine the vertebral bodies in the lateral view.

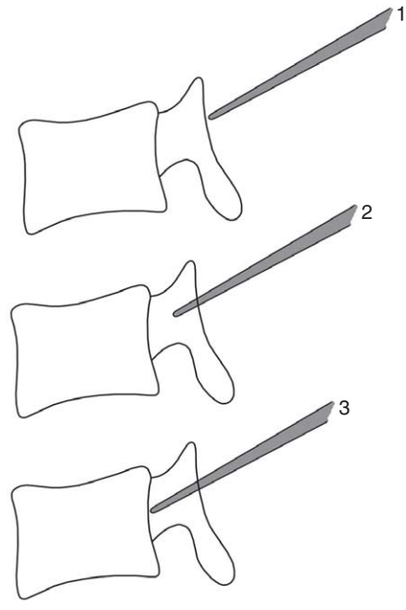

FIGURE 32–8
Lateral view. Cannula entering the pedicle as far as the vertebral body.

from both sides simultaneously. Then, a vertebral venography using iodated contrast material is injected to identify filling patterns and potential leakage sites. If present, a repositioning of the needle might be necessary.

Cement is prepared when the position of the needles is ideal and there is no significant extravasation on venography. Mixing of the cement should be done in a vacuum chamber. PMMA is mixed with barium sulfate (10–30%) and tobramycin.

Under lateral real-time fluoroscopic control to ensure early detection of cement leaking into the epidural space, vena cava, or disc space, injection of the opacified PMMA into the vertebral body is performed (Figure 32-10). With a biplanar fluoroscopy unit, the AP view can be monitored simultaneously to detect lateral leaking. The filling process needs monitoring for 6–8 minutes corresponding to the extended polymerization time. The cement injection is done with Luer-lock mechanism syringes and should start being injected when it is no longer in a liquid consistency.

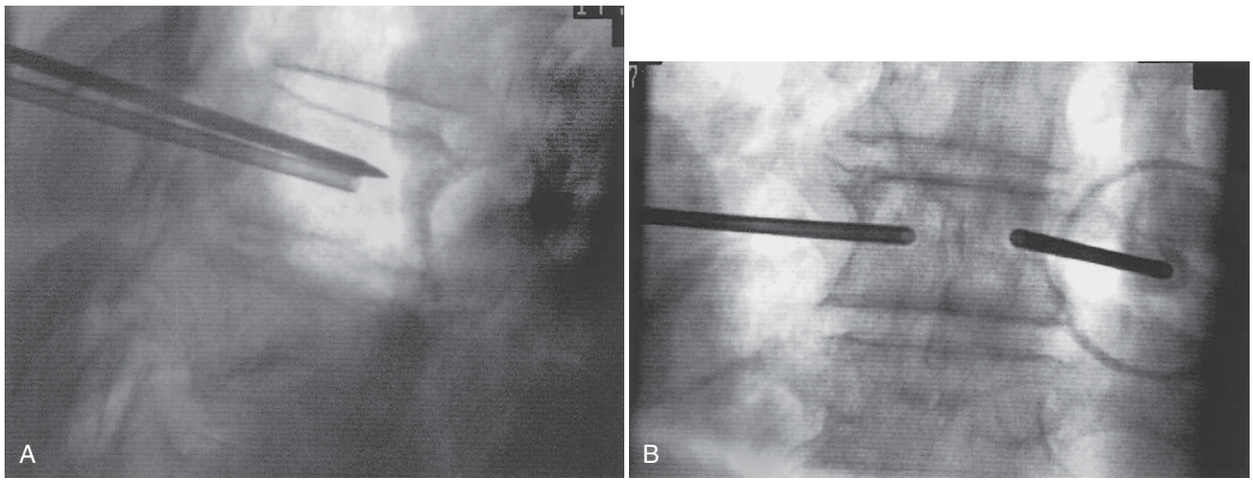

FIGURE 32–9
Bilateral technique. Both needles are placed in the anterior third of the vertebral body. (A) Lateral view. (B) Anteroposterior view.

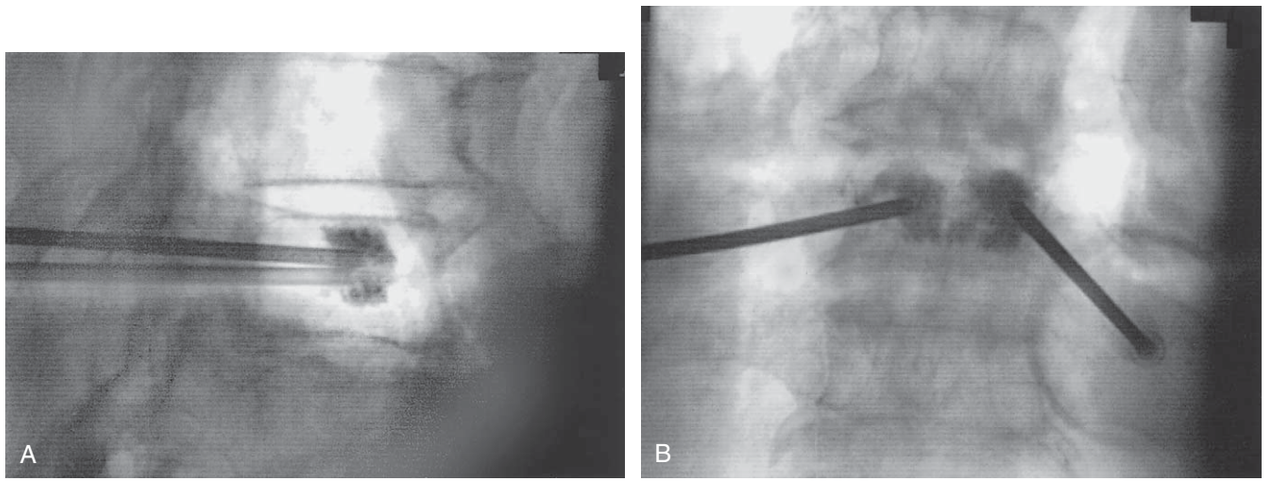

FIGURE 32–10
Injection of cement into the vertebral body. (A) Lateral view. (B) Anteroposterior view.

The amount of cement to be used to produce pain relief is still uncertain. The common recommendation is to use 2–3 ml of cement in the thoracic spine and 3–5 ml in the lumbar spine. When cement has been injected, the cannulas are removed immediately (Figure 32-11).

The maximum number of levels to be injected at one setting has not been determined, but there is a consensus that no more than three levels at a time should be treated. After injection of cement is completed, the patient is kept prone until the cement completely hardens. Postprocedure radiography or computed tomography is performed in all cases to assess the extent of filling and to look for cement leak. The procedure is considered satisfactory when cement fills the superior and inferior vertebral end plates.

EFFICACY

Patients are almost immediately relieved of back pain, whether after vertebroplasty or kyphoplasty. Most of the reports have shown a success rate of relieving pain of 75–90%.[10–18]

Good results have also been reported in metastatic bone fractures. The results in cases of vertebroplasty without accompanying radiation indicated that vertebroplasty is as effective as radiation, and in some ways more versatile.

Although percutaneous vertebroplasty harbors potentially life-threatening complications, these incidences seem prospectively extremely low[19] compared with the therapeutic benefit in refractory cases, including those already treated previously by vertebroplasty.[20]

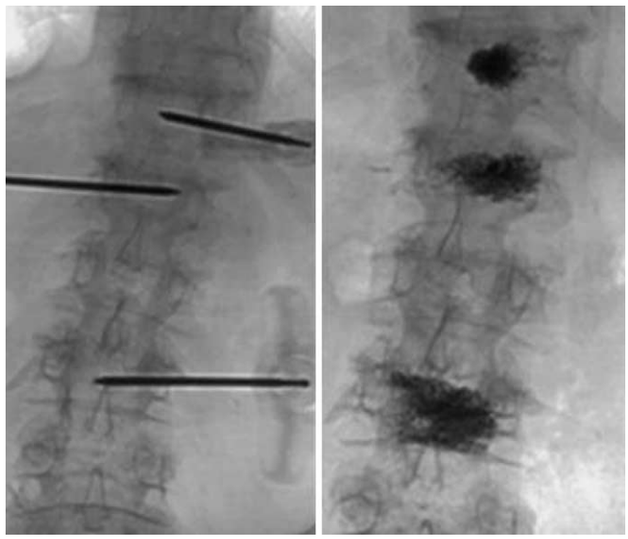

FIGURE 32–11
Vertebroplasty at multiple levels.

COMPLICATIONS

Cement leakage is a potential complication, which can occur via fracture clefts, improper instrument position, or vertebral venous plexus. The cement leakage is approximately 6% in osteoporotic compression fractures and 10% in metastasis. Accurate CT scans show leakage in almost all the procedures.[21] The risk of neurological sequelae ranges from 0 to 4% and includes radicular pain, bladder and bowel dysfunction, and paraplegia. If cement enters the spinal canal, it can produce nerve root and spinal cord compression with subsequent paraplegia.[22,23] Since the cement has to be injected at a high pressure with relatively low viscosity, the risk of extrusion is even more imminent in vertebral fractures, which affect the posterior wall (burst fractures) or osteolytic defects. Cement leakage can occur as well to the disc, epidural veins, vena cava, and vena azygos.

Other serious complications described are pulmonary embolism,[24,25] cardiac arrest and death,[26] and reactivation of infections.[27] Rib fractures have been reported.

It has been suggested that kyphoplasty is safer than vertebroplasty regarding the risk of cement leakage, even though a cement leakage is seen in 10% of cases. In comparison to vertebroplasty, the cement used in kyphoplasty has a higher viscosity and the pressure needed for application is considerably less.

During the procedures, fracture of the lamina and pedicles can occur. Other studies have reported an additional risk of the procedures in themselves producing altered mechanical forces that induce new fractures in the adjacent vertebrae.[28]

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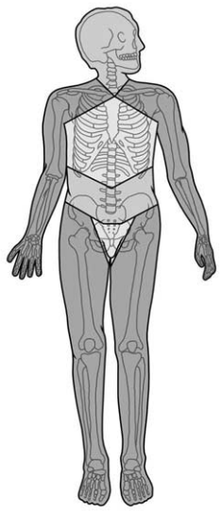

33 Balloon Kyphoplasty

RICARDO RUIZ-LOPEZ AND CARMEN PICHOT

INTRODUCTION AND HISTORY

Percutaneous vertebroplasty has been used to treat osteoporotic compression fractures, with a growing clinical experience suggesting considerable pain relief. The percutaneous kyphoplasty technique[1] is a further development of vertebroplasty.

During a vertebral compression fracture, the cortical bone buckles and cracks while the cancellous bone collapses and becomes compacted, thereby reducing the overall height and volume of the vertebra. Some fractures may collapse acutely while others collapse progressively over time.[2] If left untreated, spinal deformity can lead to subsequent fractures,[3] often resulting in kyphosis. Kyphosis compresses the chest and abdominal cavity with the potential consequences of chronic, debilitating pain, decreased lung function,[4,5] decreased activities of daily living, increased dependence on family members, and a 23% increase in mortality rate.[6]

Vertebroplasty can help to decrease a patient's pain, but the spine remains in its deformed state. Open surgical treatment can address the deformity but is typically reserved for cases of neurologic deficit. Balloon kyphoplasty addresses both the deformity and pain by stabilizing the fracture and helping to correct the vertebral body deformity (Figure 33-1).

ANATOMY AND PHYSIOLOGY

The mechanism of analgesia during vertebroplasty and kyphoplasty is intriguing as polymethylmethacrylate (PMMA) injection may offer its analgesic effect, not only by vertebral body solidification and mechanical stabilization of the fracture per se, but perhaps also by opposing osteoclastic activity, which causes neuropathic pain via vanilloid receptor (VR1) and acid-sensing ion channel (ASIC3) sensitization, due to changes in the acidotic extracellular microenvironment following PMMA injection, which may diminish pain signal transmission into the spinal cord and attenuate persistent neuropathic pain.[7]

Another interesting mechanism might be associated with the temperature elevation in the bone–cement interface, the epidural space, and the adjacent disk following PMMA injection: increased temperature may induce neuromodulatory effects on neighboring nervous structures such as the posterior annulus, the sinu-vertebral nerve, and the segmental dorsal root ganglion.[8]

INDICATIONS

The indications for balloon kyphoplasty are the same as in vertebroplasty. It is especially indicated in the presence of vertebral body deformity. The decision to perform a

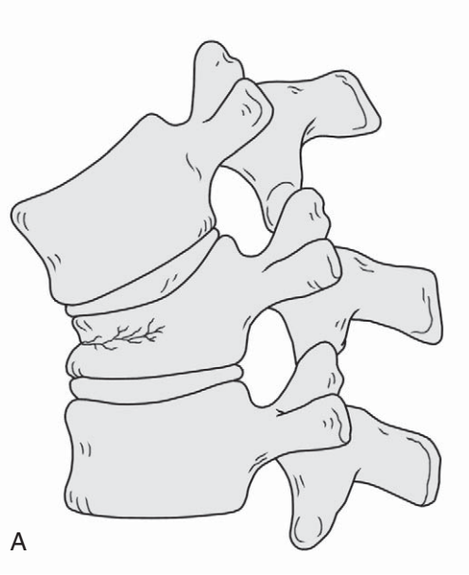

FIGURE 33–1

(A) Vertebral fracture with loss of height causing kyphosis.

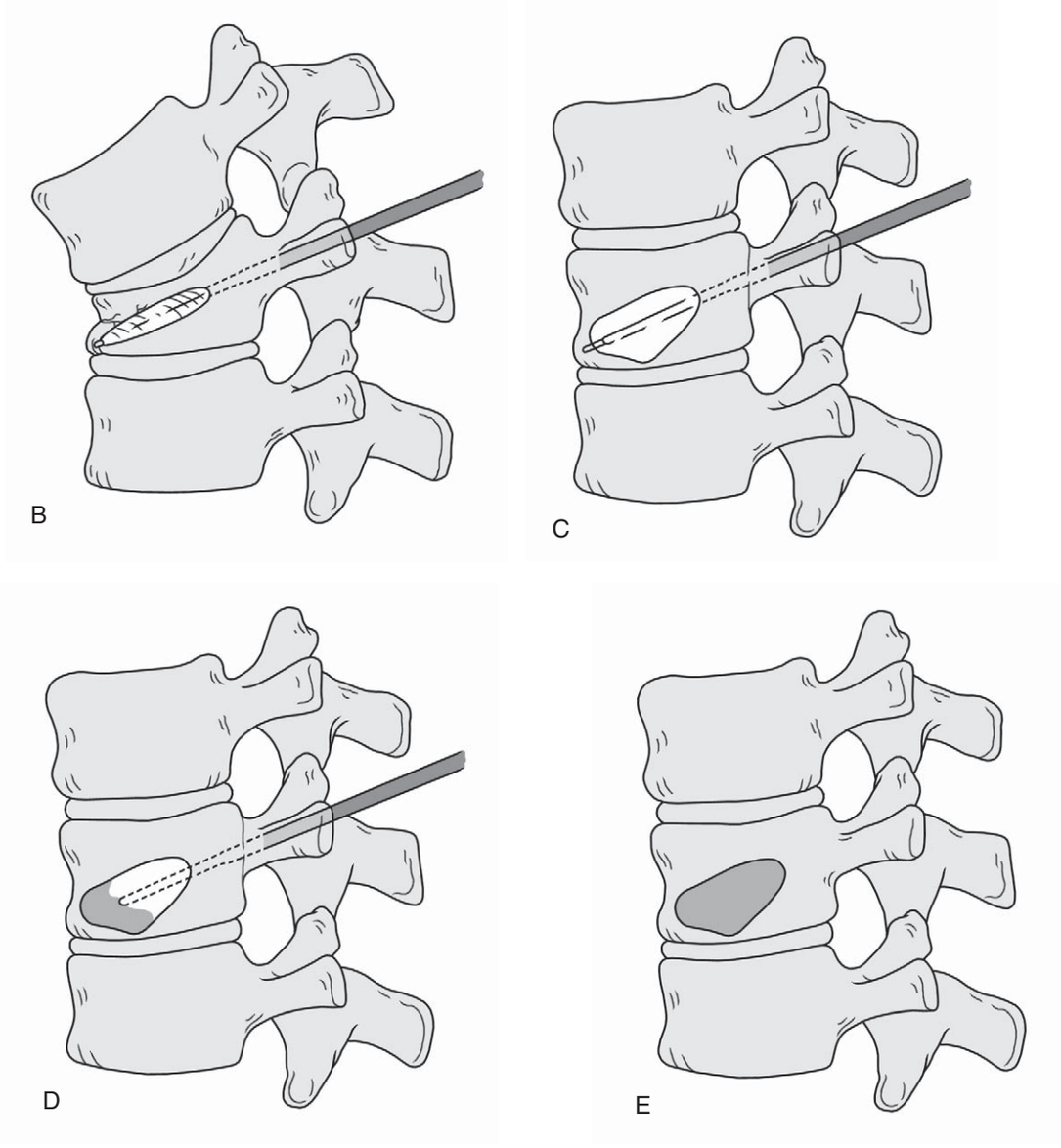

FIGURE 33–1

(B) Inserting the balloon through the working cannula. (C) Inflating the balloon. (D) After removal of the balloon, injection of cement. (E) Vertebral height restored.

kyphoplasty can be made upon deformity of the vertebral body and extent of disability.

CONTRAINDICATIONS

General contraindications include uncorrectable coagulation disorders, infectious processes of the spine, and allergies against PMMA or contrast medium. Poor pulmonary status and difficulty lying prone are relative contraindications. Multiple previous surgeries or obesity may impede proper identification of anatomical landmarks.

With kyphoplasty, burst fractures with destruction of the posterior wall constitute a relative contraindication because the risk of extravasation of the cement into the epidural space is theoretically reduced. Pre-existent neurological deficit is a contraindication, since any minor cement leakage may worsen the deficit. A previous spinal stenosis must be considered a relative contraindication. Damaged pedicles or articular facets and lack of coherence between the patient's pain and the radiological lesion also can be considered as relative contraindications.

Loss of vertebral body height of more than 60–65% is not a contraindication for kyphoplasty. There is no absolute exclusion criteria based on the time of the fracture. However, fractures older than 3 months are less likely to benefit from the technique. The exceptions to this rule are instability or recurrent fractures.

PROCEDURE

EQUIPMENT

- High-quality fluoroscopic unit (biplanar)
- 11-gauge bone biopsy cannula
- Catheter with inflatable balloon
- Bone cement (KyphX® HV-R™)

ANESTHESIA

The procedure can be performed either under mild sedation with local anesthesia or general anesthesia, but there must be the possibility of converting the operation to an open emergency operation in cases of severe bone–cement leak affecting the spinal canal. The major disadvantages of general anesthesia are its risks in elderly patients and the inability to obtain verbal feedback from the patient regarding neurological symptoms during the procedure. As in vertebroplasty, prophylactic antibiotics are given intravenously (1 g cefazolin).

PATIENT POSITIONING

Patients are placed in the prone position. Cylindrical cushions can be positioned across the table, one under the patient's chest and the other under the pelvis in order to achieve maximum extension of the spine. Padding of pressure points is important to avoid rib fractures and to add comfort.[9]

TECHNIQUE

The skin is cleaned and draped in a sterile fashion. An anteroposterior (AP) projection is obtained. The vertebral body and the pedicles are identified, and the x-ray beam is obliqued enough to see the pedicle as a circle. The entry point for the transpedicular technique is in the lateral superior quadrant of the pedicle. In the higher thoracic region an extrapedicular route lateral to the pedicle is recommended. This approach allows the needle tip to be angled more toward the center of the vertebra and thus allows easy filling of the vertebra with a single needle.

Local anesthesia is injected to the skin, subcutaneous layer, and the periosteum of the bone at the bony entry site. A 0.5-cm paramedian incision is made at each side of the spine for insertion of the trocar. An 11-gauge biopsy needle is inserted transpedicularly and advanced under AP fluoroscopic control, in an anteromediocaudal direction with rotatory hand pressure or using a mallet. In the lateral view, the needle must be at the upper midpoint of the pedicle so that the needle advances in the midpoint of the pedicle. The needle should follow a path that is parallel to the superior and inferior edges of the pedicle. In fractured vertebral bodies with vertebral end-plate depression, a more horizontal direction may be required to avoid traversing the end plate. A long needle holder can be used to avoid radiation to the surgeon's hand. In osteoporotic vertebrae, the insertion of the cannula is easy, but in osteoblastic tumors it can be harder.

With a lateral fluoroscopic projection, at the posterior vertebral body, the beveled needle is rotated 180 degrees to point medially and advanced into the anterior third portion of the vertebra, since this area is devoid of venous plexuses. If a bilateral technique is used, both cannulas should be inserted.

When the tips of the cannulas have passed the plane of the posterior wall of the vertebral body (Figure 33-2), the trocar is removed and a drill is introduced to make a channel in the vertebral body (diameter 3.3 mm) for insertion of the catheter with the deflated balloon (Figure 33-3). The exact position is checked with lateral fluoroscopy. The size of the balloon is selected on the basis of the vertebral body to be treated. The same technique is repeated on the contralateral side.

After checking the positioning by anterior and lateral fluoroscopy, the balloons are inflated with radiopaque contrast medium by a specifically designed syringe with continuous control of pressure and volume. This generates a radial force that compacts the cancellous bone in the periphery, increasing the strength of the cortical bone and creating a cavity inside the vertebral body. Once the voids in the vertebral body have been created, in many cases with a complete restoration of the vertebral body height (Figure 33-4), the balloons are retracted and bone cement is injected using a blunt cannula under low pressure (Figure 33-5), with visualization by fluoroscopy in two planes. The possibility of injecting the cement under low pressure after having created a cave with the balloon, together with the restoration of vertebral body height, is an obvious theoretical advantage of the kyphoplasty technique over the vertebroplasty technique.[10]

After injection of cement is completed, the patient is kept prone until the cement completely hardens.

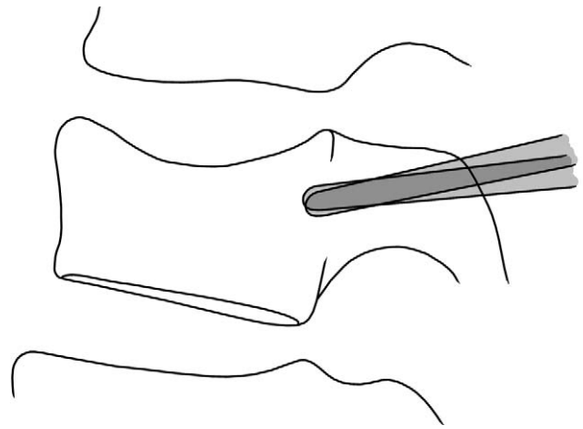

FIGURE 33-2
Lateral view. Positioning of working cannulas.

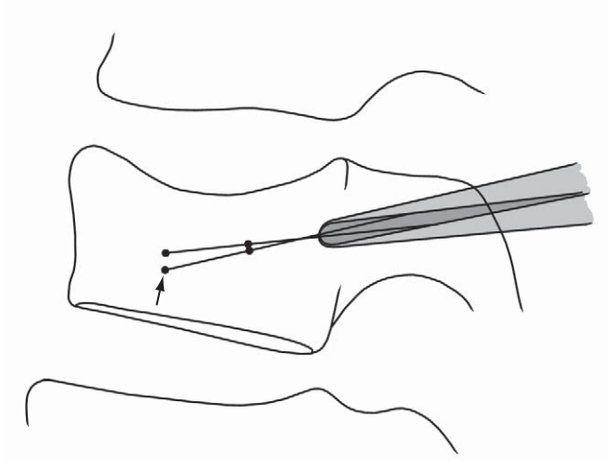

FIGURE 33–3
Insertion of catheters for balloons.

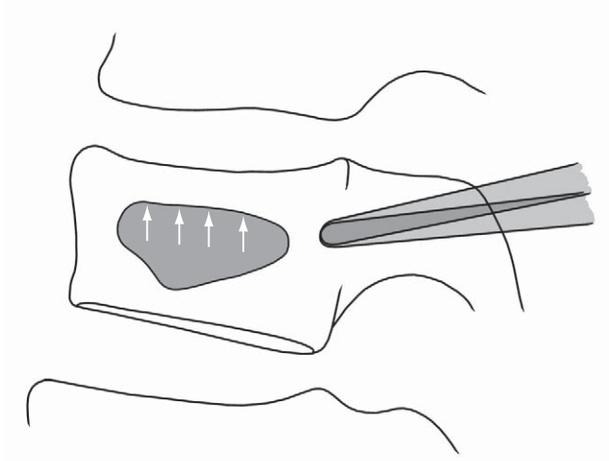

FIGURE 33–5
Lateral view. After inflating the balloon, the vertebral height is restored.

EFFICACY

Patients are almost immediately relieved of back pain, whether after vertebroplasty or kyphoplasty. Most of the reports have shown a success rate of relieving pain of 75–90%.[11–19] Kyphoplasty results in significant pain relief and improved physical function that can last for long periods.[20]

The kyphoplasty procedure results in some degree of height restoration in about two thirds of patients and a significant improvement in pain and physical function. It is not clear whether restoration of height is correlated with long-term clinical success. The location of the treated level seems to influence the extent of augmentation as well since kyphoplasties of the lumbar spine are more likely to achieve a significant height restoration than those of the thoracic spine. The majority of kyphosis correction by kyphoplasty is limited to the vertebral body treated.[21] Transpedicular balloon kyphoplasty for the direct restoration of burst fractures seems feasible in combination with posterior instrumentation.[22]

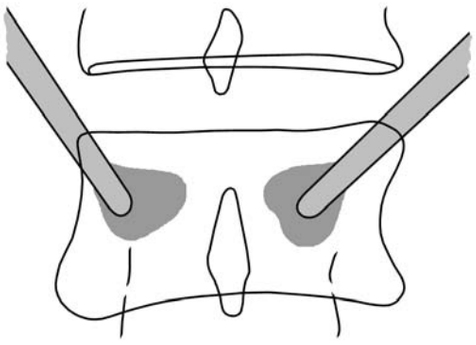

FIGURE 33–4
Anteroposterior view. Vertebral height restored.

COMPLICATIONS

Cement leakage is a potential complication that can occur via fracture clefts, improper instrument position, or vertebral venous plexus. It has been suggested that kyphoplasty is safer than vertebroplasty regarding the risk of cement leakage, even though a cement leakage is seen in 10% of cases. In comparison to vertebroplasty, the cement used in kyphoplasty has a higher viscosity and the pressure needed for application is considerably less.

In a U.S. study involving 214 fractures with 155 patients enrolled, there were no serious procedure-related adverse events.[23] In the literature review of 1342 vertebrae treated with balloon kyphoplasty, the pooled risk of an adverse event associated with bone cement was 0.2% per fracture (<0.3% per patient), while the pooled risk of any adverse event was 1% fracture (2% patient).[24]

During the procedures fracture of the lamina and pedicles can occur. Other studies have reported an additional risk of the procedures in themselves producing altered mechanical forces that induce new fractures in the adjacent vertebrae.[25]

CLINICAL PEARLS

A long needle holder can be used to avoid radiation of the surgeon's hand.

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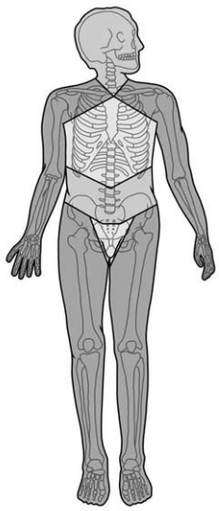
C H A P T E R



34 Cranial Stimulation

RICARDO RUIZ-LOPEZ

DEEP BRAIN STIMULATION

Deep brain stimulation (DBS) is a surgical technique that involves the placement of a fine electrode (wire) into specific parts of the brain. Most commonly, DBS is used to treat Parkinson's disease, but it can also be used in pain alleviation. DBS works by delivering a continuous electrical pulse to regions of the brain involved in the processing of pain signals. The exact mechanism by which this creates pain relief is yet to be fully understood. The advantages of this technique are that it is reversible and nondestructive, and can be modified by adjustment of the stimulator settings after implantation.

HISTORY

Deep brain stimulation was first introduced in the 1950s, when the study demonstrated the acceptance that surgical anesthesia could be produced in rats by electrical stimulation of the periaqueductal gray matter (PAG),[1] and the proposal of the gate control theory by Melzack and Wall,[2] made it acceptable clinical procedure to relieve chronic pain.[3] The first published report of periaqueductal and periventricular gray (PAG-PVG) region stimulation in humans appeared in 1973.[4] However, in subsequent years inconsistent results in small cohorts of patients, the debate regarding the best indications, the enhancement of oral opioid medications in the treatment of refractory pain syndromes, and the marked success of spinal neuromodulation (spinal cord stimulation or infusion pumps) led to abandonment of the technique by most neurosurgeons.[5] Combined stimulation of the PAG and sensory thalamus was first shown to be successful by Hosobuchi in 1983.[6]

In the late 1980s and 1990s, the use of DBS was prohibited by the U.S. Food and Drug Administration (FDA), and later it was designated as "off-label."

Results of two DBS multicenter studies with negative results further slowed down the use of DBS to treat chronic pain, and few reports have been published in the last decade.[7] The resurgence of functional surgery for movement disorders, and technological developments (stereotactic target localization with magnetic resonance imaging [MRI], safer electrodes, and more reliable pacemakers), led to a reappraisal of this indication.

ANATOMY AND PHYSIOLOGY

According to the gate control theory, stimulation of large diameter fibers is capable to inhibit nociceptive information. This idea led to the development of peripheral nerve and dorsal column stimulation. The thalamic nuclei (ventroposterolateral and ventroposteromedial) were considered an alternative substrate for activating the lemniscal system in certain pain syndromes where there is a lack of primary afferent fibers in the peripheral nerve or dorsal columns. Other structures in the brain have been explored as possible targets for stimulation, including motor cortex.

Different brain sites have been proposed as effective stimulation sites for pain relief, particularly the somatosensory area of the ventral thalamus,[8,9] the caudal medial thalamic areas around the third ventricle (periventricular gray, PVG),[1,10,11] the PAG near the Sylvian aqueduct,[4,12,13] and, more recently, the motor cortex.[14]

The use of sensory thalamic (ventral posterior lateral and medial [VPL/VPM]) stimulation for clinical pain relief was introduced by Mazars[15] for patients suffering from deafferentation pain, assuming that deafferentation pain is due to a lack of proprioceptive information reaching the thalamus from the deafferented region, and that somatosensory thalamic stimulation compensates for this deficiency. Nevertheless, the physiological explanation for pain relief by somatosensory thalamic stimulation is still unclear. Several neurophysiological mechanisms have been proposed, ranging from the activation of local inhibitory systems in the thalamus to activation of descending inhibitory systems.

Periaqueductal and Periventricular Gray Area

The initial 1969 report by Reynolds[1] that electrical stimulation of the rat midbrain can produce a powerful analgesia was soon confirmed by several other investigators. Subsequent laboratory investigators pointed out the close relationship between stimulation-produced analgesia (SPA) and the endogenous opioid system. The development of stimulation tolerance, the existence of cross-tolerance with exogenous morphine, and the reversal of SPA by naloxone were considered strong arguments for an endorphin mediation of stimulation-induced analgesia.

Electrophysiologic and anatomic studies have shown that the analgesic effect of PAG stimulation is at least partly mediated by descending control systems. Because direct projections from the PAG region to the dorsal horn are sparse, and lesions of the nucleus raphe magnus largely reduce the analgesia from PAG stimulation, it has been suggested that the effect of PAG stimulation is relayed via the nucleus raphe magnus. Descending pathways from the nucleus raphe magnus to the dorsal horn have been described.[16] When activated, by electrical stimulation or by the local administration of opioids, they exert a strong inhibitory effect on the responses of dorsal horn neurons to nociceptive stimulation. There is evidence for the existence of multiple descending antinociceptive systems in the midbrain, some involving endorphinergic and other monoaminergic mechanisms.[17] Besides the nucleus raphe magnus, several other structures also take part in these descending inhibitory control systems. Among these are the habenula, the locus coeruleus, the subcoeruleus-parabrachial complex, the magnocellular part of the nucleus reticularis gigantocellularis, and the Kölliker-Fuse nucleus region.

Numerous observations made in patients support the animal data that pain relief by PAG-PVG stimulation is mediated by endorphin-containing neuronal systems.[12] This hypothesis has been firmly challenged by Young and Chambi,[18] however. Using a double-blind, placebo-controlled study design, they found no evidence that PAG-PVG-induced SPA in humans is mediated by opioids.

Stimulation of the PAG-PVG and related targets as a therapeutic tool has been largely inspired by animal experiments, in which the effect of stimulation on acute pain responses (nociception) was investigated. Only a few studies have examined the effect of stimulation in animal models of chronic pain.[19] The fact that animal studies mostly focused on acute pain responses, whereas DBS is clinically used only for the treatment of chronic pain, may partially explain the discrepancy between the optimistic experimental findings and the often poor therapeutic results obtained in humans.

Ventroposterolateral and Ventroposteromedial Area

As noted earlier, behavioral studies in animals prompted neurosurgeons to try PAG-PVG stimulation for pain alleviation in humans. In contrast, no such experimental data were available for the somatosensory thalamus. The two that had been performed failed to show VPL-VPM stimulation-induced analgesia.[10,20] Experimental evidence for its presumed role in SPA was exclusively based on electrophysiologic data obtained from anesthetized animals. In this paradoxical stimulation, VPL-VPM stimulation was already successfully used in humans for more than two decades before the first behavioral data in the awake animal could show VPL-induced SPA.[21]

The mechanism by which VPL-VPM stimulation abolishes chronic pain is unclear. It is not likely to result from the activation of an endogenous opioid system because the analgesic effect of VPL-VPM stimulation is not reversed by naloxone. Although investigators found that after thalamic stimulation, B-endorphin levels were more than twice the resting level, no differences in B-endorphin levels could be demonstrated between patients reporting complete pain relief and those reporting only partial relief.[22] Moreover, a much higher increase in B-endorphin levels was found after PAG stimulation.

Some investigators have suggested that the neural substrate of VPL-VPM stimulation lies in its capacity to inhibit spinothalamic tract cells. No significant descending projections from the VPL-VPM region to the dorsal horn have been described. Anatomic studies have shown that spinothalamic tract neurons not only project to the thalamus but also send axon collaterals to the PAG and nucleus raphe magnus. Because stimulation of these structures may inhibit spinothalamic tract neurons, VPL-VPM stimulation may antidromically activate the descending inhibitory pathways in these structures. Tsubokawa and colleagues[22] have argued that the neural basis of this VPL-VPM–induced excitation of raphe-spinal neurons involves a dopaminergic mechanism. This hypothesis is supported by the clinical observation that administration of an antidopaminergic agent antagonized the analgesic effect of brain stimulation in patients with somatosensory thalamic, but not with PAG electrodes. Evidence also exists for the involvement of a serotoninergic mechanism in VPL-VPM–induced analgesia. For instance, microdialysis studies in anesthetized monkeys have shown that stimulation of the VPL releases serotonin in the lumbar spinal cord.

The relevance of these experimental findings to the explanation of the analgesic effect of the VPL-VPM stimulation in humans has been shown to be an effective treatment for chronic (neuropathic) pain, whereas most animal experiments studied the effect of acute noxious stimuli in intact animals. Second, although the inhibition of spinothalamic tract neurons is on the order of milliseconds, the observed clinical pain relief after VPL-VPM stimulation can last for hours and occasionally longer.

Duncan and colleagues[3] used positron emission tomography (PET) to study the mechanisms underlying VPL-VPM–induced analgesia. Five patients suffering from neuropathic pain for whom electrical stimulation of the somatosensory thalamus had produced satisfactory long-term pain relief were included in the study.

INDICATIONS

The main indication for this procedure is poststroke pain, especially if burning hyperesthesia in the affected area is present. Other deafferentation pain syndromes that can be considered an indication for DBS are failed back syndrome, peripheral neuropathy or radiculopathy, trigeminal neuropathy, and spinal cord lesions, among others.[23]

The procedure must be offered to patients with chronic pain that has proved resistant to all other treatment modalities including spinal infusion of opioids and other drugs.

A temporary trial stimulation is mandatory before a neurostimulation device is implanted. This test must be sufficiently long, and the results should preferentially be evaluated by an independent third party. The aim of the test is to ensure that the pain relief is sufficient to justify permanent implantation and that the patient is able to use the neurostimulator device properly.

Clinical data support the hypothesis that nociceptive pain is preferentially suppressed by stimulation of the PAG-PVG region, and neuropathic pain is preferentially suppressed by stimulation of the VPL-VPM region. Therefore, an analysis of the pathophysiology of the pain syndrome is of importance.

CONTRAINDICATIONS

- Clotting disorders
- Ongoing infection
- Local infection
- Patients unable to fully understand the objectives of the treatment
- Psychiatric comorbidity

EQUIPMENT

- MRI (T-1 weighted axial scan, with 2-mm thick slices parallel to the line connecting the anterior commissural with the posterior commissural or AC-PC line)
- Stereotactic CT-scan
- Radionics Image Fusion® and Stereoplan®
- Multipolar DBS electrode (Medtronic 3387 DBS®)
- Extension cables
- Pacemaker (Synergy, Medtronic)

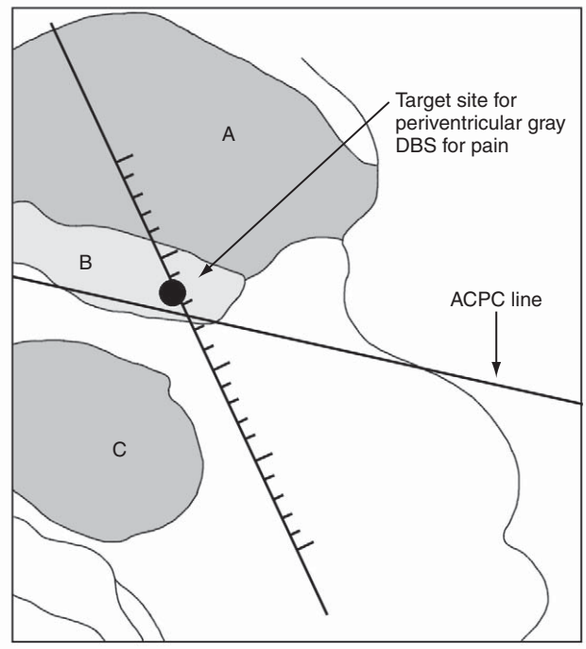
SURGICAL PROCEDURE

Deep brain stimulation is accomplished by means of a stereotactic neurosurgical procedure as the crucial point in the technique is to reach the correct target. Stereotactic calculations on the various targets were based on the results of contrast ventriculography and atlases of stereotactic anatomy and, more recently, stereotactic MRI. Since 1995, though, a stereotactic computed tomography is volumetrically fused to MRI (2-mm thick slices parallel to AC-PC line) by means of a special program (Radionics Image Fusion and Stereoplan) to eliminate the errors of MRI stereotaxy alone.

A CRW™ system for stereotactic surgery (Radionics) is used to fixate the patient's head under local anesthesia. Coordinates for PVG and VPL are calculated. A double oblique trajectory is defined to insert the electrodes (Figure 34-1). An entry point is marked just anterior to the coronal suture, which defines the laterality of approach based on the ventricular width. After washing and disinfecting the patient's scalp, a parasagittal posterior frontal scalp incision 3 cm from the midline is made contralateral to the side of the pain. A twist-drill skull perforation is made for each electrode.

The VPL is located from 5 mm above and up to 5 mm below the AC-PC line, and 5–8 mm posterior to the midcommissural point and approximately 12–14 mm lateral (Figure 34-2). As for the PVG/PAG, the proximal part of the electrode is located 2–3 mm lateral to the wall of the third ventricle and 2 mm anterior to the level of the posterior commissure, and distally, the deepest part of the electrode lay in the superior colliculus (Figure 34-3).

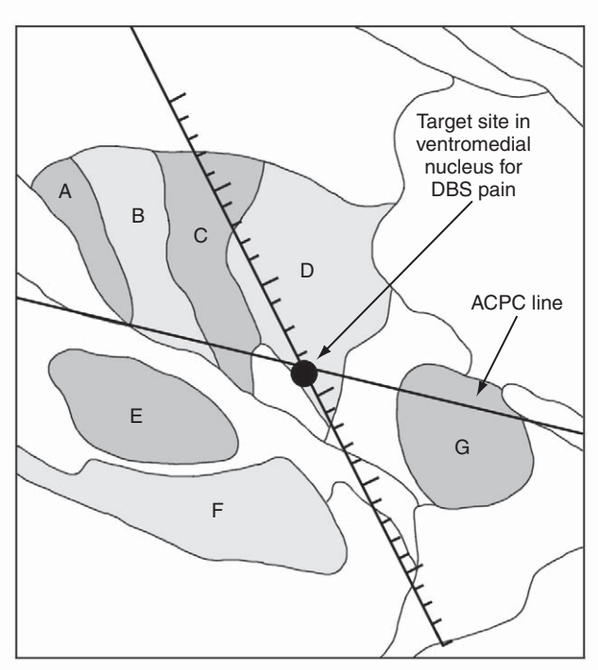
A Medtronic 3387 electrode is implanted in each site (PAG/PVG and VPL). In the VPL the electrode is implanted where stimulation induces paresthesias in the area of pain. In the PVG/PAG it is left where stimulation induces relief of pain or a sensation of warmth in the area of



A) Dorsomedian nucleus
B) Parafascicular nucleus
C) Red nucleus

FIGURE 34–1

This sagittal brain chart shows the electrode trajectories through the periventricular gray area and the target point for stimulation.



A) Anterior ventral oral nucleus
B) Posterior ventral oral nucleus
C) Ventral intermediate nucleus
D) Ventrocaudal nucleus
E) Subthalamic nucleus
F) Substantia nigra
G) Medial geniculate nucleus

FIGURE 34–2

Target site for deep brain stimulation (DBS) in ventromedial nucleus.

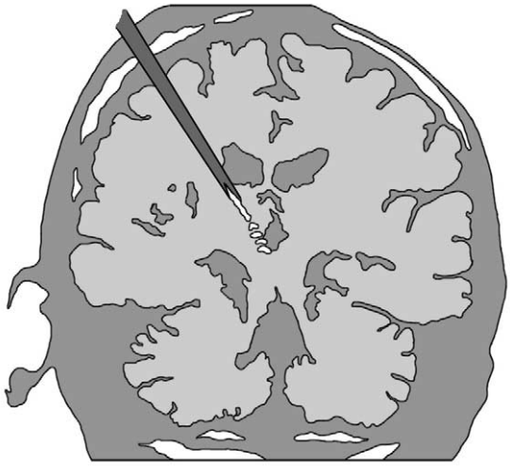


FIGURE 34–3

Magnetic resonance scan showing implanted periventricular gray electrode.

pain. In practice, capturing the whole area of pain can be challenging, particularly with hemi-body pain.

Each electrode is moved intraoperatively proximally or distally along its track until the widest possible involved area is covered with the middle two contacts. The electrodes are fixed to the skull prior to externalization for the trial stimulation phase.

The trial phase takes 1–2 weeks. Each electrode is tried individually (1–2 days of each) to cover the area of pain and achieve maximum pain relief.

If the patient is satisfied with the degree of pain relief from one electrode or a combination of both, full implantation of a Medtronic pulse generator (Synergy) is performed under general anesthesia (Figure 34-4).

EFFICACY

This technique was largely abandoned after the negative results published in two multicenter trials in the 1980s.[24] None of the reports available so far are in placebo-controlled studies, so caution is needed in evaluating and comparing the results. In a study by Nandi et al.,[25] six of the eight patients who had trial PVG stimulation had satisfactory pain relief and opted to have the pulse generator implanted.

A series by Owen et al.[26] in 15 patients with poststroke pain decreased average pain around 40–50%, with a great variability among patients. In this study, patients with subcortical strokes had a slightly better pain relief in the VAS than those with cortical strokes. If burning hyperesthesia was present, it was particularly reduced. The majority of patients preferred the analgesic effects of PVG stimulation. The effectiveness of the procedure is also shown in an N-of-one study[27] in which the patient with neuropathic pain records pain scores and the stimulator is randomly turned on or off.

A recent study suggests that this procedure can be helpful to reduce some patients' pain, but there is also an important placebo component in DBS.[14]

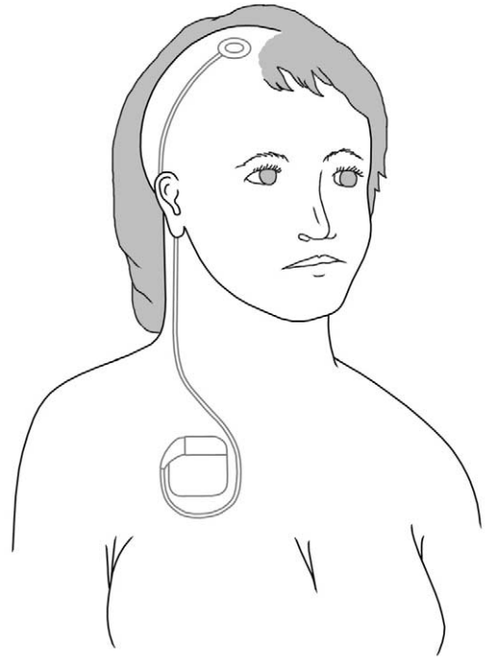


FIGURE 34–4

Implanted pulse generator.

A meta-analysis performed by Bittar et al.[28] showed that the rate of long-term pain alleviation was highest in those patients undergoing DBS of the periventricular gray region plus sensory thalamus (87%). A long-term success rate of more than 80% was attained in patients with intractable low back pain and failed back surgery syndrome. Trial stimulation was successful in approximately 50% of patients with poststroke pain, and 58% of patients with permanent implantation achieved ongoing pain relief. Moderately higher rates of success were seen in patients with phantom limb pain and radiculopathies.

COMPLICATIONS

Complications related to surgery include a mortality of 0.6–1.6%, intracranial hemorrhage with permanent sequelae in 1.4–5%, intracranial hemorrhage with transient sequelae in 1.9–8%, subdural hematoma in 1–1.6%, permanent neurological deficit in 0.6–3.4%, transient neurological deficit in 2–30%, diplopia (mostly transient) in 2.5–14.2%, seizures in 3–4.2%, and deep infections in 0.7–5.2% of the patients. Eye "bobbing" is a common problem when stimulating the lowest part of the PVG electrode at high voltages.

Complications related to the use of the hardware include infection in 3.3–15%, erosion of the skin in 1.6–9%, hardware failure in 4.9–30%, electrode displacement in 2–27.5%, and electrode fracture in 8.5% of the patients.

One potential complication of DBS is the onset of tolerance.

CLINICAL PEARLS

The use of prophylactic antibiotics minimizes the incidence of superficial infections. The type of electrode used (Medtronic 3387 DBS) locked into the burr hole with the Medtronic ring and dome apparatus has reduced the problems of electrode displacement and the necessity to revise the position of electrodes to optimize the paresthesias produced.

The use of a quadripolar electrode allows stimulating a variety of sites in the brain by simple parameter adjustment.

MOTOR CORTEX STIMULATION

HISTORY

The development of motor cortex stimulation (MCS) has been a consequence of the ongoing neurosurgical efforts since the early 1970s to treat neuropathic chronic pain conditions unresponsive to therapy, including pharmacological, minimally invasive therapies, and DBS.

Advances in the understanding of pain neurophysiology and pathways led to clinical application of electrical stimulation of specific areas in the nervous system, first in the peripheral nerves by Wall and Sweet in 1967,[29] the spinal cord by Shealy and colleagues in 1970,[30] and the experimental electrical stimulation of the brain stem by Mayer and collaborators in 1971.[31]

MCS or chronic epidural stimulation of the cortical motor area is being selectively used in cases of intractable chronic pain conditions, and until today only 350–400 patients have been treated with this neuromodulation modality.

ANATOMY AND PHYSIOLOGY

Animal experiments showed that electrical stimulation of the sensory motor cortex exerts a presynaptic inhibitory action on the spinal cord and that spinothalamic tract neurons are subject to corticofugal control. Of particular clinical importance are the findings by Tsubokawa and colleagues[32] that the neuronal hyperactivity in the subnucleus caudalis of the spinal trigeminal nucleus that appears after transection of the trigeminal nerve is better inhibited by motor cortex stimulation than by sensory cortex stimulation. Encouraged by these experimental findings and in view of the often unsatisfactory results of DBS in neuropathic pain and after central nervous system lesions, Tsubokawa and coworkers[32] published the first clinical results of motor cortex stimulation in 12 patients with central pain. Their hypothesis was that in deafferentation pain, sensory neurons below the level of the deafferentation cannot exert their normal influences on the deafferented nociceptive neurons. They further hypothesized that the pain inhibitory function of the somatosensory lemniscal system is still operative above the level of the deafferentation and that precentral gyrus stimulation selectively activates non-nociceptive neurons in the sensory cortex.

The corticospinal tract originates as the axons of pyramidal neurons in layer V of (mainly) primary motor cortex, travels through the pyramids of the brainstem, and finally ends on or near the α-motor neurons. It starts in the precentral gyrus, the fold of cortex just anterior to the central sulcus. The precentral gyrus provides the bulk of the corticospinal tract, but other cortical areas contribute as well. One such area is area 3a, part of the primary somatosensory cortex, which is hidden down inside the central sulcus. In motor cortex, the body is mapped out across the extent of the gyrus. Control of the feet lies near the midline at the top of the gyrus, whereas the lateral side of the gyrus controls the hands and face.

The internal capsule is a major two-way highway and very vulnerable to strokes. Sensory information travels up it on the way from the thalamus to the cortex, and motor information travels through on the way down to the spine.

Several mechanisms involved in pain relief have been suggested,[33] such as reduction in afferent responses in the spinal cord by presynaptic inhibitory mechanism, inhibition

of responses of wide-dynamic-range dorsal horn neurons to high-intensity stimuli by MCS, and attenuation of behavioral responses. Subthreshold electrical stimulation of the motor area leads to modulation of pain-related areas like the medial thalamus, anterior cingulated gyrus, and upper brainstem.

The mechanisms involved in pain relief with MCS are still poorly understood, and experimental data cannot explain why MCS appears to be exclusively effective for neuropathic forms of pain.

INDICATIONS

Specific guidelines for this surgery have not yet been implemented and universally accepted. Patients who could benefit from this therapy are those with poststroke pain, trigeminal neuropathic pain, and anesthesia dolorosa. Other indications include deafferentation pain as postherpetic neuralgia, brachial plexus or sciatic nerve avulsion injury, phantom limb and stump pain, and selected cases of pain after spinal cord damage.

As of this writing, a total of 44 publications exist with most of the data referring to central and facial pain than for other deafferentation syndromes.[34]

CONTRAINDICATIONS

- Clotting disorders
- Ongoing infection
- Local infection
- Patient unable to fully understand treatment objectives
- Psychiatric disorders, major depression, drug abuse

ANESTHESIA

Patients must be candidates for general anesthesia, albeit local anesthesia is currently used. In addition, patients must be able to communicate during the lengthy screening process before implantation.

Analgesic and sedative medication are recommended, as is prophylactic intravenous antibiotherapy.

SURGICAL TECHNIQUE

This invasive therapy must be performed with a rigid clinical protocol in a comprehensive inpatient hospital-based neurosurgical facility.

Preoperative Assessment

Exhaustive information about the procedure must be given, and written informed consent must be obtained from the patient. A neuropsychological evaluation must be performed by an independent neuropsychologist prior to surgery.

Preoperative imaging screening through a three-dimensional–volume MRI data set with adhesive fiducials and surface volume reconstruction must be achieved. Preoperative functional MRI data of motor function of the tongue, arm, or leg are achieved, and blood oxygenation level dependent signals are measured with standardized paradigms.[35,36] In most cases, these data are sufficient for matching with the neuronavigation data. In some cases of poststroke pain, the motor areas of the leg, arm, and tongue must be determined bilaterally, and identification of the precentral gyrus must be achieved by matching with the three-dimensional–volume MRI data set (Figure 34-5).

Operative Procedure

Sterile conditions are established in the neurosurgical operating room. The head is fixed in a three-point pin holder, after administering intravenous sedation and analgesia and local anesthesia of the areas to be incised. The neuronavigation system is initiated, and data are checked using anatomical landmarks like skin, nasion, and mastoid tip.

After drawing the anatomical landmarks of the central sulcus and its relation thereto with pre- and post-central gyrus, the placement of a single burr hole is made on the skin. The burr hole is performed, and a quadripolar paddle lead (Resume, Medtronic) is placed epidurally with all four contacts covering the precentral gyrus followed by connection to the electrophysiological monitoring system.

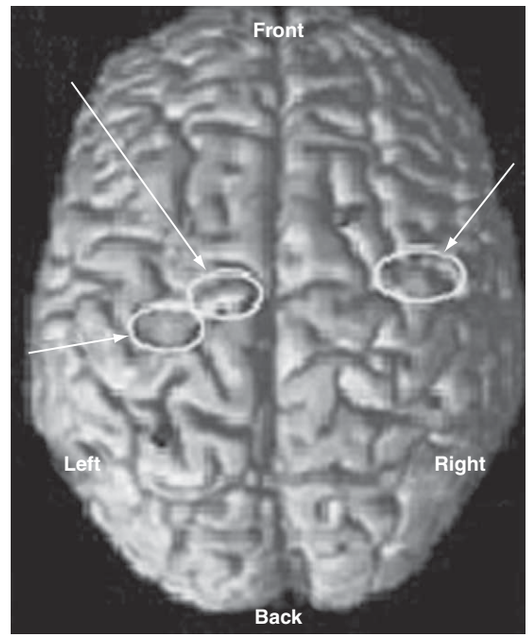


FIGURE 34–5

Magnetic resonance imaging data set showing areas that control finger movement after repetitive stimulations. (From Pray L: fMRI: the perfect imperfect instrument. *Scientist* 17(21):40, 2003, with permission.)

Intraoperative Monitoring

Somatosensory-evoked potentials of the media and/or tibial nerve are recorded in a bipolar mode using two electrodes of the quadripolar lead. After identifying the pre- and post-central gyrus with the help of the phase reversal of the N20 (median SEP) or P40 (tibial SEP), direct bipolar epidural cortical stimulation is performed using the quadripolar lead in the same position. Stimulus intensities for epidural stimulation usually vary from 3.5 to 10 V with an impulse duration at 21 milliseconds. The frequency of stimulus used ranges from 5 to 100 Hz.

The lead is placed and its extension cable is then tunneled subcutaneously and fixed in the frontotemporal region. The electrode is sutured to the outer, periosteal layer of the dura and the craniotomy bone flap is secured with plates.

Postoperative Test Trial

Testing of the electrode can begin immediately after the patient is fully recovered and should continue for 1 week or until the patient can confirm that stimulation reduces pain by at least 50%.

During this time patients must receive oral prophylactic antibiotherapy. A plain x-ray of the skull must be performed the day after the procedure to document the position of the lead (Figure 34-6).

Stimulation is generally performed with different electrode combinations and stimulation parameters (e.g., frequency 25–55 Hz, pulse width of 60–180 milliseconds and amplitude of 0.5–7.0 V, using an external stimulation device [DualScreen, Model 3628, Medtronic Inc., Minneapolis, MN]).

A VAS rating of pain must be assessed during stimulation, and pain relief is usually delayed by several minutes and may last beyond the period of stimulation.

A reduction of pain intensity of 50% or more as measured by the VAS, compared with the preoperative ratings, is considered to be effective.

In the case of a positive test trial, the neurostimulator (Itrel 2 or 3, Model 7425, Medtronic Inc., Minneapolis, MN) is implanted subcutaneously in the lower abdominal region or in the infraclavicular external area (Figure 34-4).

After implantation of the pulse generator, a regimen of intermittent stimulation is usually programmed in order to avoid habituation.

EFFICACY

The reports by Tsubokawa and coworkers[32] in patients with poststroke pain showed an initially good response in 75%, but after 2 years only 45% had pain relief from the procedure. Meyerson et al.[37] showed a higher efficacy of the procedure in patients with trigeminal neuropathic pain at 28 months follow-up. The overall success rate in other studies has similar figures.[35]

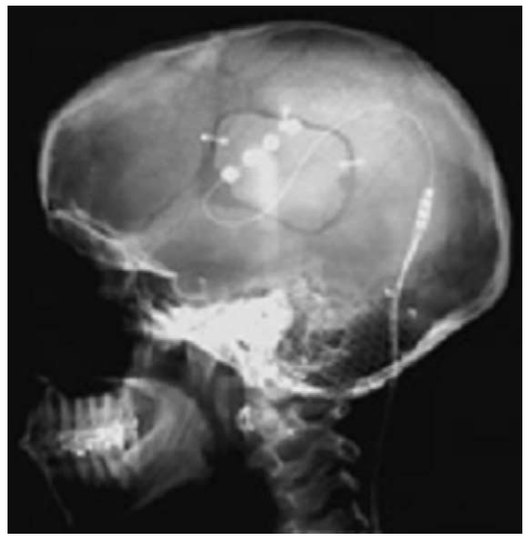


FIGURE 34–6
Plain radiograph of the skull showing the electrode placement at the motor cortex.

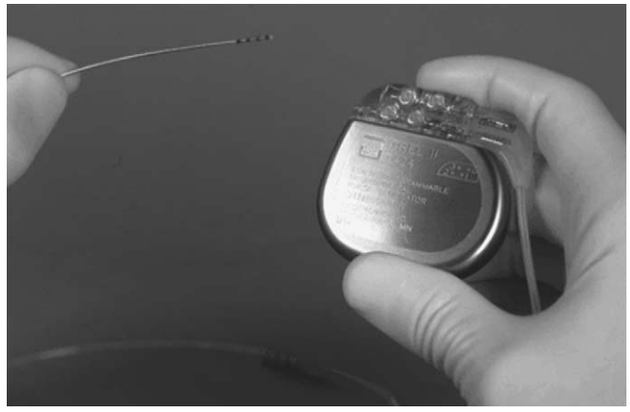


FIGURE 34–7
Itrel 3 Model 7425, Medtronic, Inc., which is implanted for motor cortex stimulation.

The technique is more effective in trigeminal neuropathic pain than in poststroke pain, with success rates from 50 to 80%.[36]

The most common successful application for neuropathic facial pain could be explained due to the large representation of the facial structures on the motor cortex, making easier the targeting of the electrode position.

Severe motor weakness or plebian in the area of pain is of negative predictive value for pain relief by MCS.[38]

COMPLICATIONS

There is no documentation of neurologic injuries resulting from this surgical procedure. Morbidity includes intraoperative seizures or even postoperative seizures lasting less than 3 days,[39] with no reports of long-lasting seizures.[40]

Complications related to surgery include stimulator-pocket infection or wound infection, epidural hematoma, subdural effusion, and dehiscence of the stimulator pocket.[32]

Complications related to hardware failure (lead fractures, migration, and insulation fractures) have been reported as occurs with any other implantable technology.[41]

CLINICAL PEARLS

Among the current invasive neuromodulation procedures, MCS allows a genuine placebo-controlled approach, since it is not dependent on the perception of paresthesias or other behavioral manifestations.

Computer-assisted neuronavigation will simplify this surgical method, making it a less invasive and ethically more acceptable option than DBS.

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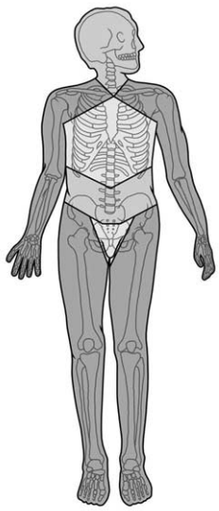
C H A P T E R



35

Retrograde Pelvic Stimulation

GABOR B. RACZ

HISTORY

Transsacral electrodes were introduced for bladder dysfunction procedures. During monitoring, incidentally, these patients also had pain relief from stimulation of the third sacral nerve root. The indication for pelvic stimulation has been for bladder dysfunction and detrusor sphincter dyssynergia as in spinal cord injury where the innervation pathway has not been interrupted.[1] Additionally, it was recognized that intractable rectal pain responds to stimulation of the S4 nerve root either singly or bilaterally, and some potential nerve-related problems could not be covered by the transsacral S3 and S2 electrode placements and required more stimulation. Additionally, transsacral stimulation has been plagued by electrode migration. The concept of retrograde electrode stimulation was introduced by Feler, Alo, and colleagues,[2,3] and even early experiences indicated certain hazards. The use of the retrograde Tuohy-type needle results in an unacceptably high incidence of dural puncture. The problem has been reduced by the development of the reversed R-X Coudé needle due to the cutting end of the needle being away from the dura as the needle is passed in a retrograde manner from the L3-L4 and L4-L5 areas. Initially, the evaluation and the temporary electrode placement occurred in a blind manner where the needle was placed and stimulated without fluoroscopy, and the electrode was placed in the retroperitoneal space surrounding the S3 nerve root.

ANATOMY

The sacral plexus usually is a collection of nerves and ganglia in pairs. However, occasionally one or the other nerve root may be missing, and this part of the anatomy and nervous system is considered to be one of the most variable parts of human anatomy. For this reason, it is imperative to carry out trial stimulation prior to placement of permanent electrodes. The anchoring system is tested every time the patient sits down, and sliding from one sitting position to the other makes the relatively thin covering layers of the sacrum to slide and drag the electrode from the sacrum into a displaced position. These concerns will be addressed by examples from our practice. The concept of retrograde stimulation was introduced primarily to allow the physician an option of covering more than one nerve root and to reduce the likelihood of electrode migration and displacement.

INDICATIONS

Among the accepted indications for sacral stimulation has been inability to void secondary to detrusor sphincter dyssynergia, urgency and frequency of micturition, and occasionally pain-related problems involving the pelvic structures. More recently, it has been found that rectal incontinence has responded to sacral stimulation primarily the S4 rather than bladder stimulation, which is by the S3 stimulation.[4–7]

CONTRAINDICATIONS

- Infection
- Arachnoiditis
- Spina bifida occulta
- Coagulopathy

TECHNIQUE

RETROGRADE APPROACH

The patient is placed in a prone position; fluoroscopic guidance is used to identify the L2-L3 or L3-L4 interspace. The C-arm is rotated from an anteroposterior position to slightly

cephalad and lateral. A paramedian approach close to the midline is used. The needle is advanced in such a manner that a small amount of the shaft of the needle is visible on fluoroscopy, indicating that the needle is angled relative to a tunnel view axis. Loss of resistance is used, and the needle is advanced into the epidural space near the midline position. The multicontact electrode is threaded in a caudad direction near the midline. The curved tip of the stylette allows steering, and the electrode advance is a repetitive, slightly twitchy manner in order to use the velocity of the electrode advance and rotation of the needle tip by repeated small movements (Figures 35-1, 35-2, and 35-3). The challenging part is getting past the lumbar sacral junction where there is some resistance, and at times scar formation. The electrode from the top of the sacrum is then steered in a lateral direction down toward the S3 neuroforamen. It is often necessary to place bilateral electrodes in a similar manner. Four to eight contact electrodes may be used, and the electromagnetic field is steered in such a manner that the underlining nerve roots can be included in the electromagnetic field. The electrodes are then stitched to the skin, and the patient is allowed to recover from the sedation that is used during the procedure. Test stimulation is used for 2–3 days, and the system is removed following the confirmation of successful stimulation. Unfortunately, a higher incidence of infection rate follows in cases where the same electrode system is used for trial and permanent electrode placement; therefore, our preferred practice is to separate the trial stimulation from the permanent implants by a 30-day waiting period if possible.

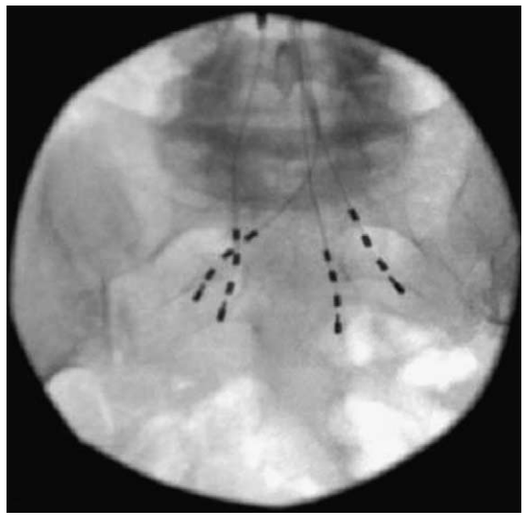


FIGURE 35–2

Retrograde placement of electrodes for pelvic pain stimulation (anteroposterior view). (Courtesy of Claudio Feler, MD.)

ALTERNATE TRIAL STIMULATION

The cost of trial stimulation can be reduced by the use of electrode placement from the sacral hiatus and going cephalad. Through a transsacral hiatus R-X Coudé needle and Stim Cath, which is a monopolar and injectable catheter, the catheter is advanced to the sacral neuroforamina, where the active tip of the catheter is just superior and medial to the neuroforamina. An alligator clip is attached to the needle for the positive electrode and the negative electrode is to the Stim Cath connection that comes out the end of the stimulating catheter (Epimed International). The patient receives the stimulation at 50-Hz low voltage, 0.5–1.5 volts amplitude, to the paresthesia threshold. Bilateral stimulation may be necessary for identifying the best target area for permanent stimulation. It is possible to move up to S2-S3 with two electrodes covering one side versus the other or bilaterally in any combination that one wishes to evaluate. The needles are removed

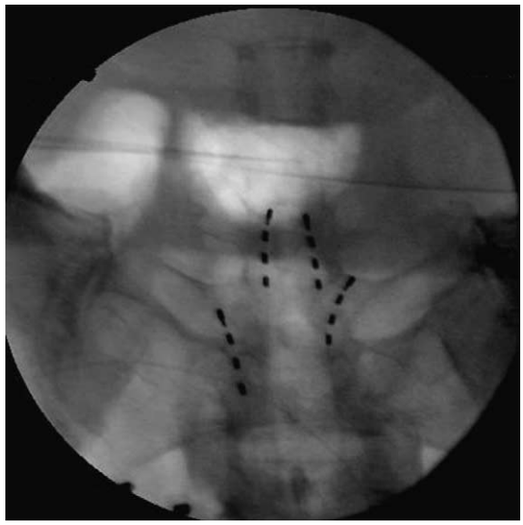


FIGURE 35–1

Retrograde placement of four ANS electrodes. (Courtesy of Claudio Feler, MD.)

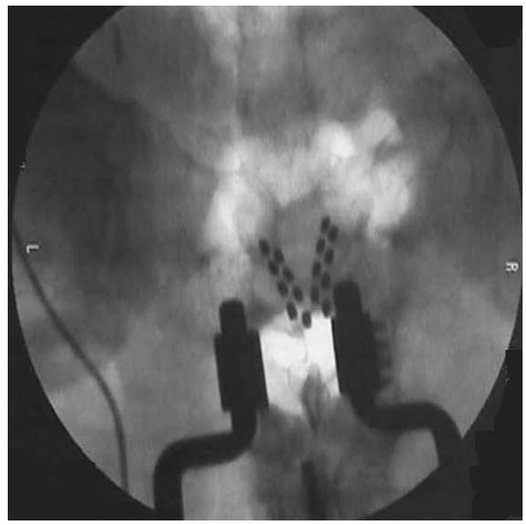


FIGURE 35–3

Placement of four electrodes at sacral nerve roots at L4-S1 via laminectomy. (Courtesy of Claudio Feler, MD.)

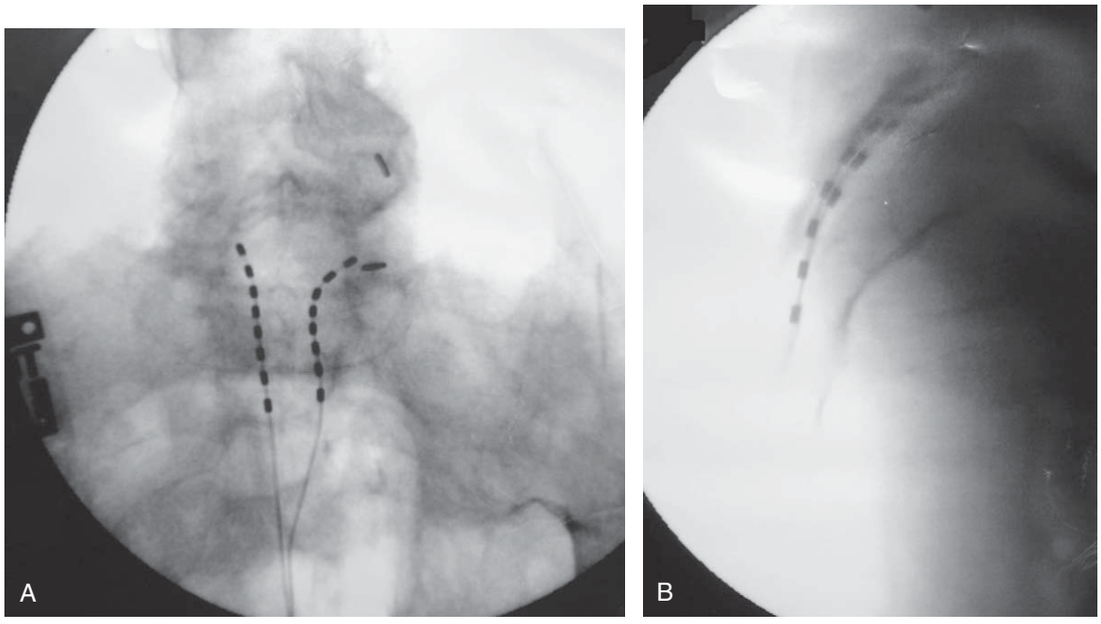


FIGURE 35–4
Transsacral hiatus antegrade electrode placement to L5 nerve root. (A) Anteroposterior view. (B) Lateral view. (Courtesy of Claudio Feler, MD.)

after appropriate satisfactory stimulation, and the stimulating catheters are sutured in place. An appropriate dressing is applied, and temporary stimulation is carried out for 2–3 days to evaluate whether the patient is a candidate for permenant stimulation. The decision regarding transsacral or retrograde stimulation then can be made, depending on the number of nerve roots that need to be covered.

ANCHORING THE SYSTEM

If a transsacral electrode placement is used, it is imperative that a few principles are observed (Figure 35-4). First, the electrode connecting cable should not cross posterior to the sacroiliac joint following placement and leading to the IPG battery pack. Second, an incision should be made and a loop

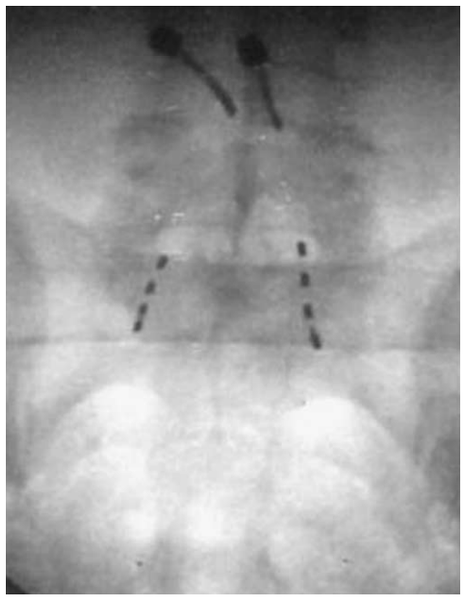


FIGURE 35–5
Retrograde double electrode placement through L4-L5 approach. (Courtesy of Claudio Feler, MD.)

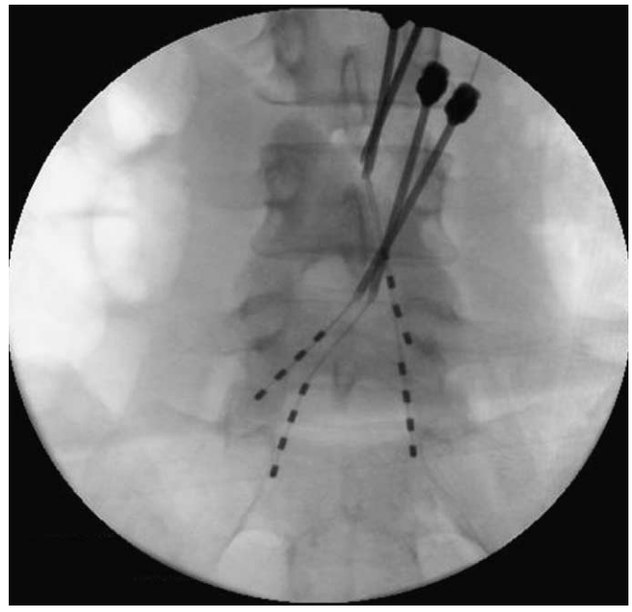


FIGURE 35–6
Retrograde quadruple electrode placement through L3-L4 and L4-L5. (Courtesy of Claudio Feler, MD.)

be placed under the skin (Figure 35-5 and 35-6). This loop should be used as a release mechanism for the event when the patient may slide from one sitting position to another. Because the surgical knot is variable from physician to physician and patient to patient, electrode migration has become an issue. Sacral stimulation has been accepted for functional problems of the bladder, but the significant benefit has also been in conditions such as pain secondary to interstitial cystitis, vulvar pain, post radiation neuritis, sacral plexopathy, rectal pain, and functional rectal disturbances in the form of rectal incontinence. Retrograde electrode approaches have been successful when the transsacral retrograde failed to cover pain related to multiple sacral nerve roots; however, the improved anchoring systems have made the transsacral approaches for electrode placement significantly better. Introduction of the twist lock (Medtronic) and the Titan anchor are likely to enhance the effectiveness of these techniques, particularly when the firmer and more predictably gripping anchor is sutured to the deeper structures such as the sacral periosteum.

EXPERIENCES WITH MULTIPLE CASE REPORTS

Case 1

IMPORTANCE OF ANCHORING

An example of benefit from the sacral stimulation to emphasizing the importance of anchoring is a patient suffering from extreme vulvar pain secondary to Lyme disease. The pain generators were identified to be bilateral S3 nerve roots by the use of monopolar Stim Cath (Epimed International) followed by double transsacral S3 electrodes with the use of the twist lock Medtronic anchor and 3487 electrodes (Figure 35-7). The patient received excellent pain relief and 6 years later turned off the battery (Medtronic Synergy System). A year later, the system was explanted with complete resolution of the vulvar pain. The lessons gained from this electrode placement were the use of the twist lock anchor, as well as the use of complete circle loop as a strained relief mechanism that clearly prevented migration and functioned throughout the 7 years.

Case 2

COMPLICATIONS OF DURA PUNCTURE

Another patient had retrograde electrode placement at L3-L4 for the treatment of bladder dysfunction and severe interstitial cystitis for arachnoiditis. During the procedure, the introducing modified Tuohy-type needle perforated the dura without obvious free flow of spinal fluid. The electrode placement resulted in the electrode perforating the arachnoid, and spinal fluid leak leading to repositioning of the needle and the electrode. Repeated attempts resulted in similar dural punctures three times, and the patient had severe postdural puncture headache. Two epidural blood patches were followed by elevation of temperature with antibiotic treatment and resolution of the spinal headache. At follow-up, the patient had persistent back pain that preceded the procedure. The pain and bladder dysfunction were most likely consequences of previous failed back surgery, and the pelvic pain complaints were best explained by the preexisting arachnoiditis. This is an example of the problem of dural puncture from the retrograde approach. Informed consent regarding dural puncture is important, and wet taps most likely occur at the L5-S1 approach.[2,3]

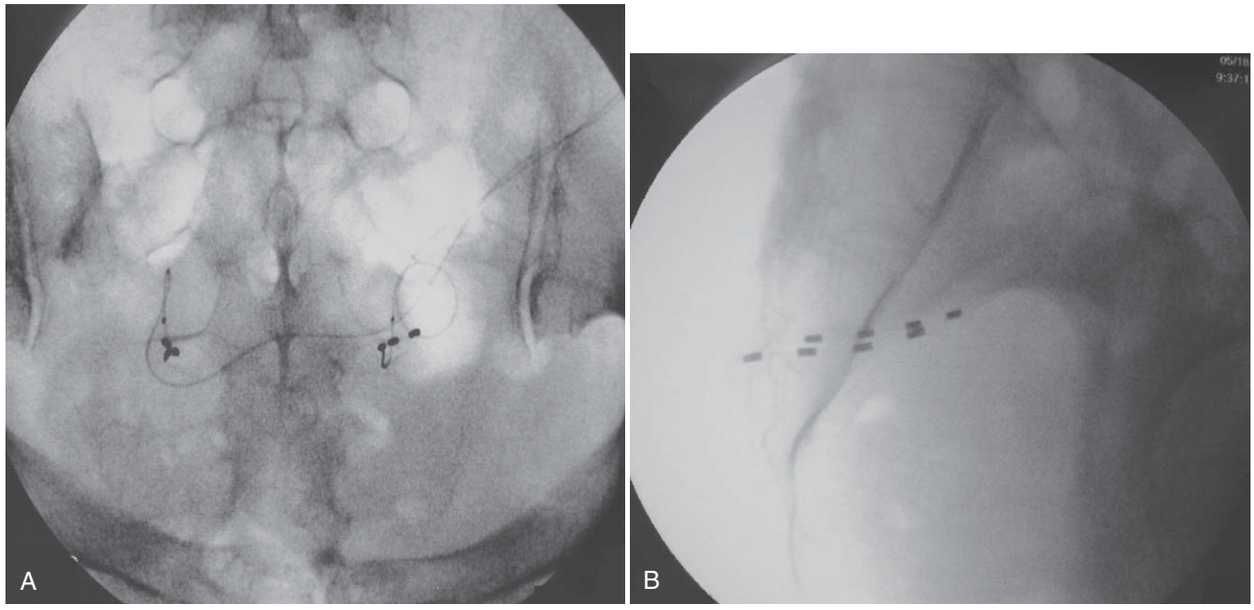


FIGURE 35–7

S3 transsacral electrode stimulation with lateral Medtronic 3487A electrodes. (A) Anteroposterior view. (B) Lateral view. (Courtesy of Claudio Feler, MD.)

CASE 3

NEED FOR ELECTRODE STIMULATION COVERAGE MORE THAN JUST IN THE INITIAL AREA OF PAIN

A patient with prostate cancer treated by radical prostatectomy and radiation therapy had severe rectal pain. Following mapping of the pain generator with the monopolar Stim Cath (Epimed International), the pain generator was found to be a left-sided S4 nerve root. Transsacral S4 electrode stimulation was followed by pain relief for over 2 years. The pain returned with a vengeance, spreading to an upper level, involving the posterior sacral structures as well as the scrotum and penis, in such severity that for sleeping, the patient assumed a knee–chest position. Retrograde electrodes were passed bilaterally with one of the electrodes on the right-hand side passing through the S1 ventral foramen. The other one passed down to the S3 neuroforamen on the left. The result of the bilateral retrograde electrodes and S4 electrode stimulation was significant pain relief, and the patient was able to sleep in the normal manner.

It is often necessary to use electrodes in excess of the usual two electrodes to have the option of manipulating the electromagnetic field in order to cover the neuropathic pain generators from the injured sacral nerve roots. Examples are given for the combined use of transsacral and retrograde stimulation to enhance the lives of our patients.

COMPLICATIONS

- Infection
- Postdural puncture headache
- Bleeding
- Delayed patient response or no pain relief (to determine efficacy, more large-scale case studies are needed)
- Infection, bleeding disorders, surgical fusion, spina bifida occulta

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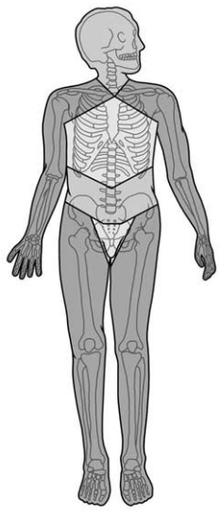
C H A P T E R



36 Ultrasound Imaging Techniques for Regional Nerve Blocks

JOSE DE ANDRÉS AND XAVIER SALA-BLANCH

The important medical diagnostic and therapeutic advances of the last two decades are largely attributable to improvements and innovations in imaging technology. The role to be played by such imaging diagnostic procedures in regional anesthesia and pain medicine will depend largely on the will of anesthesiologists to incorporate such technologies to their practice.

In the operating room, classically radiological procedures have been used for the performance of block techniques[1] and for assessing correct distribution of the administered volume of anesthetic solution,[2] or its maldistribution,[3] and/or the complications associated with anesthetic block.

The use of ultrasonography or ultrasound (US) has produced a conceptual change in the way the technique is performed. This change is based on the fact that the technique is performed under direct puncture visualization and, therefore, constitutes a much more anatomical approach.[4] During US guidance, the structures through which the needle is inserted are identified, and the plexus is directly localized; consequently, a reduction in complications and side effects is more likely attributable to optimized puncture than to an actual improvement in the clinical results.[5] In addition, the ultrasonograph machine can easily be moved to the operating room and is clearly advantageous from the economical perspective, if it proves able to replace neurostimulation in the finding of mixed nerves.[6] The cost of an ultrasound system for performing regional techniques (Sonosite™ 180 US) is in the range of US$17,000 (US$3.40 per block if 5000 blocks are performed). To these considerations we must also add the improved quality of puncture performed under direct visualization, and the possibility of using the equipment for other procedures and/or techniques.[6]

Nevertheless, clinicians interested in working with US must consider in the choice of machine for US-guided nerve blocks the cost, portability, and desired image quality.

PRINCIPLES OF ULTRASOUND

Ultrasound (or echography) is the result of technological developments in the application of ultrasound to imaging diagnosis. Sound is a vibratory phenomenon where *frequency* defines the number of vibrations, oscillations or cycles per second (measured in hertz, where 1 Hz equals one oscillation/second). Ultrasound is defined as sound at a frequency above the human auditory threshold (over 20,000 Hz). The *piezoelectric* principle allows the generation of ultrasound with applications to imaging techniques. This effect is based on the capacity of certain crystals (piezoelectric crystals) to generate mechanical energy in the form of ultrasound waves in response to the application of electric energy, and vice versa.

The physical characteristics of ultrasound are defined by the wavelength, period, amplitude, frequency, and velocity of the waves. The *wavelength* is the distance traveled by sound in the course of a single cycle and is measured in millimeters. The *period* is the time required to complete a full cycle and is measured in seconds, while the *amplitude* corresponds to the square root of the energy of the wave, and *frequency* is the number of periods per second. US frequency in turn depends on the generating piezoelectric material used. The frequencies employed in clinical practice range from 1 to 20 MHz, while in application to brachial plexus block the range is typically 3.5–10 MHz. Wave *velocity* is the displacement of sound per unit time (measured in millimeters per second), and depends on the medium through which the sound travels—approximately 1540 mm/sec in the case of biological tissues.

Echogenicity is the capacity of structures standing in the way of the ultrasound beam to reflect the waves back to their source. This capacity depends not only on the characteristics of the ultrasound waves but also on the properties of the medium through which the sound travels. The interface is

the limit or contact zone between two distinct media that transmit sound at different velocities. The *acoustic impedance* is in turn defined as the resistance of the medium to the passage of sound. When an ultrasound beam penetrates a given structure, the beam intensity decreases as a result of attenuation on one hand, and wave reflection on the other. *Attenuation* represents the loss of wave amplitude (energy) on traveling through a medium and depends on the wavelength, density of the medium or tissue, and heterogeneity (number and type) of the interfaces present (attenuation being 1 dB/MHz on average). Wave *reflection* in turn conditions the formation of ultrasound images; it is proportional to the difference in acoustic impedance between two media that form an interface standing in the way of the US beam. In terms of reflectivity, the resulting images can be regarded as hyperechogenic, normoechogenic, or hypoechogenic. In turn, hypoechogenic structures may appear anechogenic (anechoic) when US is completely attenuated or trans-sonorous when the waves are neither attenuated nor reflected back toward the emitting source.

A characteristic of ultrasound echoes applied in the clinical setting is the so-called Doppler effect, which occurs when the ultrasound beam encounters a moving structure in its path. As a result of such contact, the frequency of the reflected echo is modified and an analysis of the corresponding frequency difference can inform us of the velocity of the moving structure (e.g., blood within the vascular lumen).

The images seen on the echograph screen can depend upon the tissue through which the sound travels (tissue images) or on the separation zones between tissues (contour images). In turn, contour images can be (1) *anatomical* (or wall) images, when two tissues are separated by an anatomically identifiable structure with a distinct acoustic impedance; or (2) *interface* (or separation) images, in the presence of various acoustic impedances without any actual anatomical separation between them. On the other hand, tissue images can exhibit (1) *fluid patterns*, characterized by the absence of echoes with posterior enhancement and lateral shadowing (e.g., blood vessels); (2) *solid patterns*, characterized by disperse internal echoes that can be either homogeneous or heterogeneous; (3) *mixed patterns*; and (4) *acoustic shadows*, beyond which echoes are no longer generated. Acoustic shadowing occurs when ultrasound crosses interfaces with great differences in acoustic impedance (e.g., air/bone interfaces).

The ultrasound characteristics of the different body tissues are shown in Table 36-1.[7,8] Water is the body element that best transmits US waves, generating a black (anechoic) image. Thus, highly cellular tissues containing abundant water can be expected to be hypoechoic, while more fibrous tissues containing less water and a larger number of interfaces are characteristically hyperechoic.

Beam penetration and image resolution have an inverse ratio: the higher the resolution, the lower the working depth (tissue penetration). For deep location we need to use a low-frequency probe (<7 MHz) that will produce poor resolution. On the contrary, for superficial structures (1–2 cm from skin), we will use a high-frequency probe (>7 MHz), producing at the same time high-resolution images.

TABLE 36–1 Ultrasound Images of Tissues Identifiable during Sonographic Study of Brachial Plexus Territories

Tissues	Ultrasound image	Artifacts
Venous vessels	Compressible, anechoic	
Arterial vessels	Pulsatile, anechoic	Anisotropy (hypoechoic)
Fat	Hypoechoic	Anisotropy (hypoechoic)
Muscle:	Hyperechoic	
Perimysium	Hypoechoic	
Muscle tissue	Intensely hyperechoic	
Tendons	Fine band, anechoic	
Cartilage	Hyperechoic	
Nerves	Intensely hyperechoic line, with acoustic shadow	
Bone	Anechoic	
Air (lung)		

The learning curve of the managing probe and needle toward target is relatively rapid according to a resident study performed by Sites et al.[9] The practitioner must not forget that needle visibility during advancement decreases linearly with reduced needle size, insertion angle, (>60 degrees) and depth.[10]

ULTRASOUND-GUIDED BRACHIAL PLEXUS BLOCKS

An essential requirement for applying US in locating the brachial plexus is a detailed classical topography (i.e., structures by planes) and sectional anatomic knowledge of the region in which the plexus is found. The topographic anatomy of the zone allows us to identify the successive structural layers down to the actual plexus and facilitates identification of near-lying related elements. Sectional anatomy in turn facilitates identification of the structures seen on-screen during US guidance.

The various brachial plexus anesthetic approaches involve puncture in quite distinct anatomical zones that must be familiar to the anesthetist in order to ensure a safe and successful technique. From its origin in the neck, the brachial plexus ends in the form of terminal or endnerves in the arm, running through three anatomically differentiated zones that in turn correspond to the various anesthetic approaches used: supraclavicular region, infraclavicular (or anterior shoulder) region, and axillary (or arm root) region. The supraclavicular region is used to perform interscalene and supraclavicular punctures, while the infraclavicular region is used to perform infraclavicular techniques, and the axillary or armpit and root of the arm region is used for axillary access to the brachial plexus (Table 36-2).

TABLE 36–2 Anatomical Characteristics of Interest for Accessing Brachial Plexus under Ultrasound Guidance

	Interscalene	Supraclavicular	Infraclavicular	Axillary
Anatomical zone	Supraclavicular fossa	Supraclavicular fossa	Pectoral	Arm
Plexus zone	Roots–trunks	Trunks–divisions	Divisions–cords	Terminal nerves
Bony landmarks	Cervical transverse processes	Collarbone	Collarbone, coracoid process	Humerus
Vascular landmarks	Carotid and vertebral artery, internal jugular vein	Subclavian artery and vein	Axillary artery and vein	Humeral artery and vein
Muscular landmarks	Sternocleidomastoid, anterior, middle and posterior scalene		Greater and smaller pectoral	Greater pectoral, coracobrachial, biceps, triceps
Depth	1–3 cm	1–3 cm	3–6 cm	1–3 cm

SUPRACLAVICULAR REGION

Topographic Anatomy

The supraclavicular region has well-defined limits with the collarbone at the base, the posterior margin of the sternocleidomastoid muscle anteriorly, and the trapezius muscle posteriorly. This triangle is identified by the skin depression found within its limits: the supraclavicular fossa.

The supraclavicular fossa is covered by the skin and subcutaneous lax tissue, and the cutaneous supra-acromial and supraclavicular branches of the superficial cervical plexus. The second layer comprises the superficial cervical aponeurosis or fascia that envelops the muscles defining the limits of the supraclavicular region, that is, the sternocleidomastoid and trapezius muscles. A third anatomical layer in turn contains the middle cervical aponeurosis, enveloping the omohyoid muscle that crosses the supraclavicular region; the muscle can be easily identified by palpation, and the external jugular vein runs along its surface.

The deep zone of the middle cervical aponeurosis contains structures that the anesthetist should know and be able to associate to the corresponding sectional anatomical characteristics. The anterior scalene muscle originates in the anterior tubercles of the fourth, fifth, and sixth transverse processes of the cervical vertebrae, and inserts in Lisfranc's tubercle on the anterior aspect of the first rib. At its insertion, the anterior scalene muscle divides two important vascular structures—the subclavian artery (located posterior to the muscle) and the subclavian vein (lying anterior to the muscle). Posterior to the subclavian artery we have the brachial plexus, which lies on the anterior belly of the middle and posterior scalene muscles. The middle scalene muscle originates in the posterior tubercles of the transverse processes of the first four or five cervical vertebrae and inserts in the external margin of the first and second ribs. The posterior scalene muscle in turn originates in the posterior tubercles of the fourth, fifth, and sixth cervical vertebras and inserts in the external aspect of the second and third ribs.

This zone possesses an important vascular component. The subclavian artery gives rise to the cervical artery, which penetrates through the intertransverse foramina (from the sixth cervical vertebra) located anterior to the outlet of the spinal nerves through the conjugate foramina. The thyrocervicoscapular trunk gives rise to the inferior thyroid artery, the ascending and superficial cervical artery, and the suprascapular artery. Finally, the superior scapular (or coracoid) artery crosses the supraclavicular region to become the axillary artery.

Sectional Anatomy

The sectional anatomy depends on the level at which the US image is acquired, which is in turn dependent on the specific anesthetic technique employed. When performing the interscalene technique, the region of interest is the neck, while in the case of the supraclavicular anesthetic approach US imaging focuses on the supraclavicular region.

The cross-sectional anatomy at the level of the sixth cervical vertebra allows us to identify the sternocleidomastoid muscle, while medial to the latter lies the common carotid artery and internal jugular vein. Posterior to these vessels we have the scalene muscles: the anterior scalene muscle posterior to the vessels, followed posteriorly by the middle and posterior scalene muscles, which conform a single muscle mass. Between the scalene muscles, we can identify small round or oval nodules corresponding to the nerve roots and/or trunks of the brachial plexus. More medial to the interscalene region lies the transverse process of the sixth cervical vertebra, with the vertebral artery and vein located anterior to the latter.

A sagittal study at the level of the supraclavicular fossa allows us to identify the collarbone, with the subclavian muscle lying caudad and the subclavian vein on the first rib. In a posterior plane we find the omohyoid muscle and subclavian artery. In the posterosuperior portion of the artery lies the brachial plexus. These structures are easily identified by US, with the exception of those elements located posterior to the collarbone (due to the acoustic shadowing effect of the bone).

Ultrasound Anatomy Applied to Plexus Anesthesia

The interscalene techniques are performed over the cervical region, in the interscalene sulcus or groove. Ultrasound assessment of the neck region requires the use of high-resolution devices, due to the anatomical complexity of the zone. The availability of Doppler imaging in turn

allows us to identify the vascular structures, thereby greatly facilitating localization of the different anatomical spaces. The US transducer should operate at 7.5–10 MHz in order to identify and evaluate the important muscle and vascular references found superficially. The global anatomy can be identified by cross-sectional imaging at the level of the sixth cervical vertebra (Figure 36-1). Superficially we have the clavicular belly of the sternocleidomastoid muscle, while internal to the latter lie the internal jugular vein and carotid artery, along with the anterior and middle-posterior interscalene muscles. Between the anterior and middle scalene muscles, we observe a separation with US characteristics corresponding to fatty tissue, where hypodense nodules (trunks of the brachial plexus) can be identified.[4] More in depth we have the acoustic shadowing effect (i.e., bone pattern) of the cervical transverse process, and Doppler imaging can help us to identify the vertebral vessels.

In order to perform the anesthetic technique, the needle should be inserted deep into the interscalene space under direct visual guidance. The injected anesthetic solution floods the interscalene space but also spreads around the anterior scalene muscle to reach the carotid artery,[11] thereby inducing the most constant undesirable effect associated with interscalene block of the brachial plexus: phrenic nerve paralysis.[12]

Perlas et al.,[13] by introducing the block needle from the end of the US probe and advancing in line with the plane of the beam, showed that US imaging can direct the needle to reach the brachial plexus under real-time guidance.

The supraclavicular techniques are performed over the supraclavicular space, where the superior, middle, and inferior primary trunks divide into their respective anterior and posterior branches. The plexus runs very superficially at this level; high-frequency (10 MHz) transducers should therefore be used to identify the structures. In addition, technical difficulties are found when studying the supraclavicular zone, due to the presence of the supraclavicular depression, which complicates both manipulation of the transducer and puncture. Finally, although the important primary trunks of the brachial plexus are easily identified in the interscalene space, identification is more difficult in the supraclavicular zone. Although color Doppler imaging is not necessary to identify the subclavian vein, it greatly facilitates identification of the brachial plexus by differentiating between the nerves (hypoechogenic structures with no Doppler effect) and arterial and venous branches located in the zone (likewise hypoechogenic, although producing a Doppler effect). The US anatomy of the supraclavicular region can be seen in Figure 36-2.

In order to perform the anesthetic technique, the identification of the nerve branches is an important consideration. In effect, when the nerves have been identified, the needle is inserted into the depth of the plexus along its vertical axis, under visual guidance to avoid medial displacement and the risk of pleural puncture. However, if the nerves have not been identified, half of the anesthetic solution should be injected into the zone posterior to the subclavian artery, reserving the other half of the solution for the posterosuperior zone of the artery below the omohyoid muscle.[14]

In a recent clinical study, Chan et al.[15] achieved success in 95% of the cases. They showed that needle movement could be tracked under US guidance and different local anesthetic spread patterns observed. In the performance of supraclavicular blocks, Williams et al.[16] found that US guidance provided higher-quality results than the nerve stimulator (NS) technique.

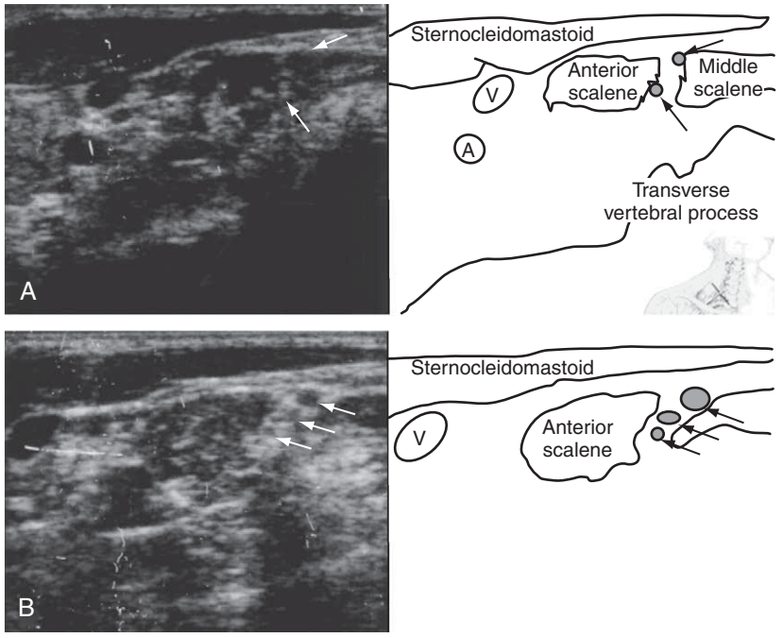


FIGURE 36–1

Two-dimensional ultrasound image across the transverse interscalene at C6 level, identifying the different muscles and vascular structures in the neck region (A). We can observe three hypodense nodular structures located between the scalene muscles (arrows) (B) and corresponding to the trunks of the brachial plexus. A, carotid artery; V, internal jugular vein. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, figure 17-21, p. 271, with permission.)

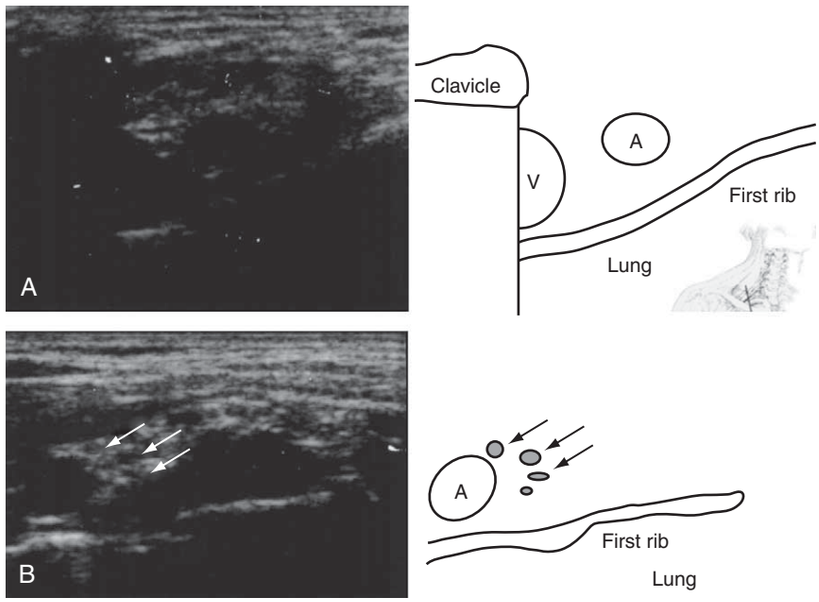


FIGURE 36–2

Sagittal supraclavicular cross-section. Sectional image at the level of the first rib, showing the different vascular structures of the supraclavicular region (A), and identifying hypodense nodular structures in the supero-posterior portion of the subclavian artery, corresponding to the divisions of the brachial plexus (arrows) (B). A, subclavian artery; V, subclavian vein. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, figure 17-22, p. 272, with permission.)

INFRACLAVICULAR REGION

Topographic Anatomy

The limits of the infraclavicular region are well defined, with the collarbone above, the lower margin of the greater pectoral muscle below, the deltopectoral sulcus externally, and the vertical traced from the center of the collarbone to the lower margin of the greater pectoral muscle internally.

The infraclavicular region is covered by skin and subcutaneous lax tissue. The second layer in turn contains the superficial aponeurosis or fascia that covers the greater pectoral muscle and extends toward the deltoid muscle through the deltopectoral sulcus. The third layer or plane contains the greater pectoral muscle, while a fourth anatomical layer presents the middle axillary aponeurosis or clavipectoral fascia, containing the subclavian muscle and extending past the smaller pectoral muscle to the axillary fascia. The deep zone of the clavipectoral fascia contains the axillary fossa (of which it forms the anterior wall). The axillary fossa in turn contains the neurovascular bundle. The most medial structure of the latter is the axillary vein, followed by the axillary artery and the three cords or fascicles of the brachial plexus laterally. On reaching the region of the smaller pectoral muscle, the neurovascular bundle runs deeper, and the nerve fascicles are found around the artery. The axillary vein is formed as a result of the joining of the brachial veins and receives the cephalic vein along its axillary trajectory, at the level of the deltopectoral triangle. The axillary artery in turn gives rise to the thoraco-acromial, long thoracic, subscapular, and humeral circumflex arteries.

Sectional Anatomy

The sagittal plane sectional anatomy of the infraclavicular region corresponds to the anterior wall of the axillary space or fossa. Consequently, here we find the collarbone and greater pectoral muscle anteriorly, followed medially by the subclavian muscle, the clavipectoral fascia, and the smaller pectoral muscle. Within this muscle-aponeurotic wall, we identify the neurovascular bundle—the vein lying caudad, the artery medial and the secondary trunks of the brachial plexus cranially. If the imaging section is acquired at the distal infraclavicular level (i.e., in the subacromial zone), we can identify the acromion with the insertion of the smaller pectoral muscle and the neurovascular bundle lying immediately posterior to this muscle. At this level the secondary trunks and end-branches of the brachial plexus are distributed around the axillary artery.

Ultrasound Anatomy Applied to Plexus Anesthesia

The infraclavicular techniques are performed over the pectoral region, which is easily evaluated by US in view of the simplicity of its anatomical structures. Doppler imaging in turn allows us to identify the vascular components, particularly the divisions of the axillary vessels, thereby avoiding accidental vascular puncture (this being the most common complication of the infraclavicular techniques).[17] Because of the greater depth at which the brachial plexus is found in this territory compared with other anesthetic approaches, a lower-frequency (5–7.5 MHz) transducer may be indicated to identify the structures.

Several locations between the mid-clavicle (infraclavicular vertical approach)[17,18] and the coracoid process

(coracoid approach) are suitable for scanning the brachial plexus in the infraclavicular region. At mid-clavicle, one can visualize the subclavian artery and vein with cords of the brachial plexus most commonly cephaloposterior to the subclavian artery. In the parasagittal plane, at 2 cm medial to the coracoid process, the cords of the brachial plexus (N) appear hyperechoic and are deep to the pectoralis major and minor muscles, in close proximity to the axillary artery and vein.

The proximal infraclavicular techniques are performed over the mid-clavicular point. At this level, the plexus is relatively superficial (3–5 cm) and is located beneath the subclavian muscle and clavipectoral fascia. The plexus lies cranial and external to its most important reference—the axillary artery. At this level the anterior and posterior divisions of the plexus group merge to form the secondary trunks. Doppler US can identify the thoracoacromial branch of the axillary artery, which arises at this level. In order to perform puncture, most of the local anesthetic volume should be injected into the posteroexternal part of the axillary artery; however, Ootaki et al.[19] obtained excellent clinical results (95% success rate) by administering equivalent 30-ml volumes at both sides of the axillary artery.

The distal (or coracoid) infraclavicular techniques are performed in the distal zone of the infraclavicular plexus, at the level where the terminal nerves are formed. The anatomical zone corresponds to the axillary neurovascular bundle, which runs beneath the smaller pectoral muscle. At this level the neurovascular bundle depth is 4–5 cm; that is, lower-frequency (3.5–7.5 MHz) transducers are required for US imaging. At this level (Figure 36-3), we can identify the thick muscle space of the greater and smaller pectoral muscles, separated by a hyperechogenic band corresponding to the perimysium. The end-branches of the brachial plexus are located at this level, around the axillary artery. The branches of the brachial plexus are difficult to visualize at this level, however; consequently, the local anesthetic solution should be administered around the axillary artery. The technique proposed by Ootaki et al.[19] is, therefore, not applicable here.

PROXIMAL REGION OF ARM AND AXILLARY FOSSA

Topographic Anatomy

In the region of the arm, the neurovascular bundle is located in the internal bicipital sulcus or groove separating the flexor muscle mass (biceps) from the extensor muscle (triceps). Here again, the first layer comprises the skin and subcutaneous lax tissue, while a second layer presents the superficial aponeurosis or fascia that covers the medial bicipital sulcus and continues with the aponeuroses of the flexor and extensor muscles of the arm. Following the superficial aponeurosis and within the bicipital sulcus lies the neurovascular bundle, with vein and nerves superficial and the brachial artery in depth. Beyond the superficial aponeurosis lies the flexor muscle layer, composed ventrally and superficially of the inferior insertions of the deltoid and biceps muscles, and with the coracobrachial and anterior brachial muscles located more in depth. In the posterior or extensor region of the arm we find a single muscle layer corresponding to the three portions of the triceps muscle. The humerus is in turn located in the deepest region.

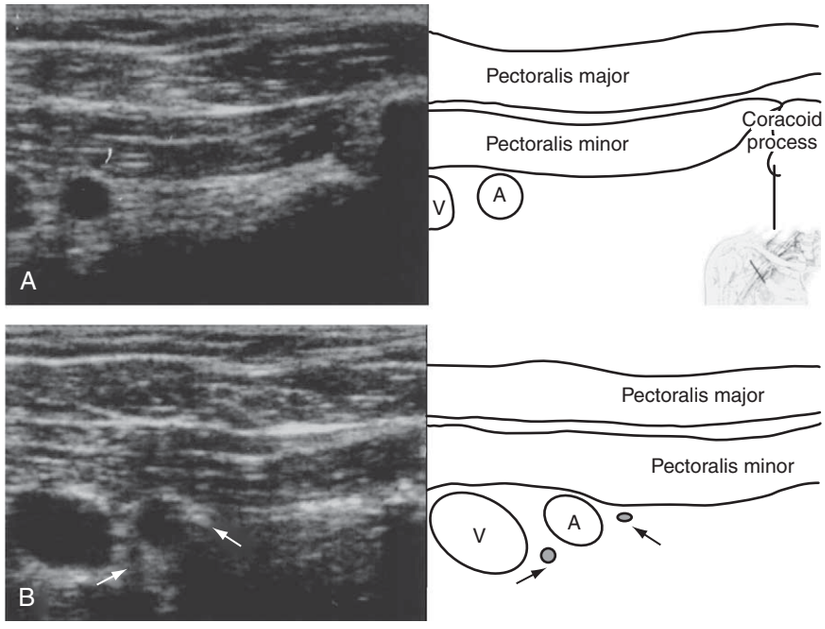


FIGURE 36–3

Sagittal infraclavicular cross-section. Sectional image corresponding to the pectoral zone (anterior wall of the axilla), showing the different muscles and vascular structures in the region (A); hypodense nodular structures are seen around the axillary artery (B) (arrows), corresponding to the cords of the brachial plexus. A, axillary artery; B, axillary vein. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, figure 17-23, p. 273, with permission.)

Sectional Anatomy

The sectional anatomy of the zone medial to the insertion of the deltoid muscle presents the internal bicipital sulcus (seen as a hyperdense structure on US), with two humeral vessels—the vein superficially and the artery lying internal to the latter. At this level, the end-branches of the brachial plexus separate to terminate in their corresponding innervation territories. The radial nerve is located in the depth of the sulcus, while the median and ulnar nerves accompany the artery over its more superficial portion (Figure 36-4).

ULTRASOUND ANATOMY APPLIED TO PLEXUS ANESTHESIA

The axillary techniques are performed in the region of the internal bicipital sulcus. US assessment requires the use of high-resolution and high-frequency (10 MHz) transducers, due to the superficiality of the neurovascular structure (1–2 cm). Terminal branches of the brachial plexus can be easily identified in close relationship to the axillary artery and vein. Color Doppler imaging provides little additional information, since the vascular elements are easily identifiable and palpable at this level, but veins can be differentiated from arteries by their ease of compressibility. At this level, US imaging often shows two or three distinct hypoechoic nodules representing the median, ulnar, and radial nerves within 1 cm from skin surface.

Using ultrasound, Retzl et al.[20] examined anatomic variations of the terminal branches of brachial plexus in the axilla. These authors found that at the usual level of axillary block, the radial nerve is most often located in the posterior and anterior lateral positions in reference to the axillary artery. The median nerve is most commonly in the anterior and posterior medial position, and the ulnar nerve in the posterior medial position. However, these nerves can be found in many other locations.

Puncture under US guidance consists of distribution of the local anesthetic solution both behind and in front of the humeral artery. However, Kapral et al.[14] obtained their best results by placing the anesthetic volume between the humeral artery and vein, an observation that can be explained by their choice of puncture site, located very close to the root of the arm.

Ultrasound guidance for axillary catheter placement has been described.[21,22]

PERIPHERAL NERVES

In the area distal to the axilla, peripheral nerves of the brachial plexus can be visualized with a 10–15 MHz probe. They appear hyperechoic in most cases. The musculocutaneous nerve is best seen in the coracobrachialis muscle. The median nerve is best seen medial to the brachial artery at the elbow and can be traced in the forearm all the way to the carpal tunnel. The ulnar nerve can be seen in the olecranon fossa and in the forearm.[23] The radial nerve can be found lateral to the shaft of the humerus just above the elbow.

The peripheral nerve that is of potential interest to the pain physician is the superficial radial nerve. This nerve may be seen a few centimeters distal to the elbow just after the division of the radial nerve into the deep and superficial branch. At this place, the superficial radial nerve lies directly between the brachioradial and the supinator muscle and meets the radial artery a few centimeters more distally.

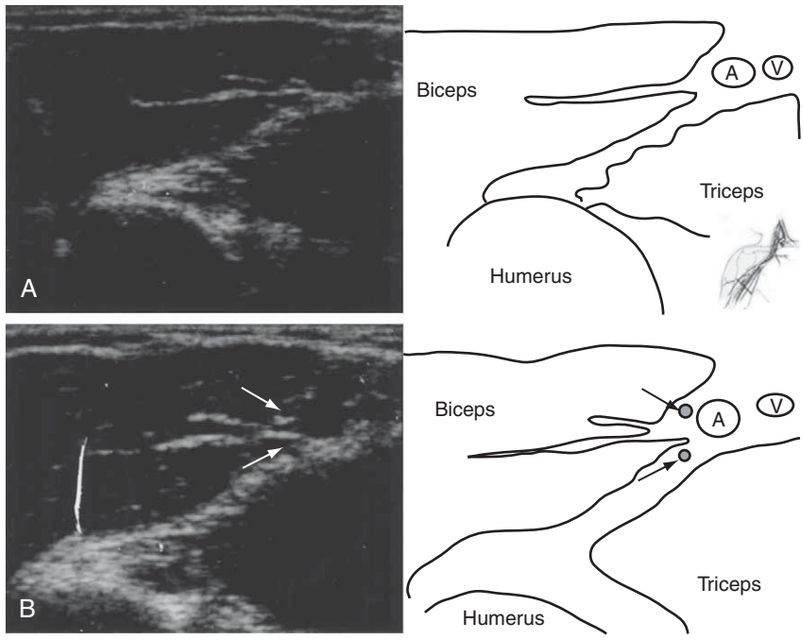


FIGURE 36–4

Axillary transverse cross-section. Sectional image corresponding to the proximal zone of the arm, showing the different muscles and vascular structures (A), and identifying hypodense nodular structures around the axillary artery (B) (arrows), corresponding to the terminal nerves of the brachial plexus. A, humeral artery; B, humeral vein. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, figure 17-24, p. 274, with permission.)

CLINICAL TRIALS WITH ULTRASOUND-GUIDED PLEXUS ANESTHESIA

Friedl and Fritz[24] in 1992 presented a technique for accessing the brachial plexus at axillary level, using a linear 7.5-MHz transducer, recommending its application when the brachial artery cannot be well identified by clinical examination, especially in obese patients. Wu et al.[25] in 1993 reported the US location of the subclavian artery for performing infraclavicular plexus block in nine patients, with a success rate of 89% and an average of three (plus/minus one) needle penetrations. In 1994, Kapral et al.[14] compared two groups of 20 patients subjected to upper limb surgery. The patients were randomly assigned to either US-guided axillary puncture ($n=20$), or the supraclavicular technique ($n=20$). The efficacy was 95% in both groups, and no complications were recorded. Based on these results, the authors concluded that an US-guided approach for supraclavicular block combines the safety of axillary block with the larger extent of block of the supraclavicular approach. Guzeldemir and Ustunsoz[22] in turn introduced an axillary catheter under US guidance, and in 1998, Yang et al.,[11] using high-resolution sonographic guidance with a broadband L10 5-MHz probe (HDI 3000, ATL Bothell), inserted a catheter into the interscalenic brachial plexus sheath and evaluated the location using radiography and computed axial tomography (CAT) after injection of contrast medium. The authors described a complex anatomy at the interscalenic level, where the brachial plexus appeared as three discrete, rounded hypoechoic nodules between the scalenus anterior and medius muscles on transverse US in the lower cervical (C6) region, representing the trunks in the sagittal oblique section. A cluster of hypoechoic nodules corresponding to the divisions was seen cephalad to the subclavian artery on sagittal scans of the supraclavicular region. Successful neural block at 20 minutes and postoperative analgesia were achieved in all patients. In 2000, Ootaki et al.[19] performed infraclavicular block of the brachial plexus under US guidance (7 MHz US probe) in 60 patients, with a 95% success rate. The distinguishing characteristic of this approach was the distribution of the volume of anesthetic solution on either side of the axillary artery, with description of the so-called "donut" sign. Sandhu and Capan,[26] following a controlled study in 126 patients, suggested puncture at three levels in the infraclavicular approach below the minor pectoral muscle (medial, lateral, and posterior to the axillary artery, respectively). This afforded a rate of conversion to general anesthesia of 2.4% and a time to block of 10 (±4.4) minutes, and a complete anesthesia was detected in 6.7 (±3.2) minutes (Figure 36-5).

Chan presented a clinical case in which US-guided puncture was successfully performed after two interscalenic puncture attempts with the NS technique.[27] Gray et al.[28] in turn presented two cases of cubital nerve block in the forearm under US guidance. Perlas et al.[13] conducted a US study of

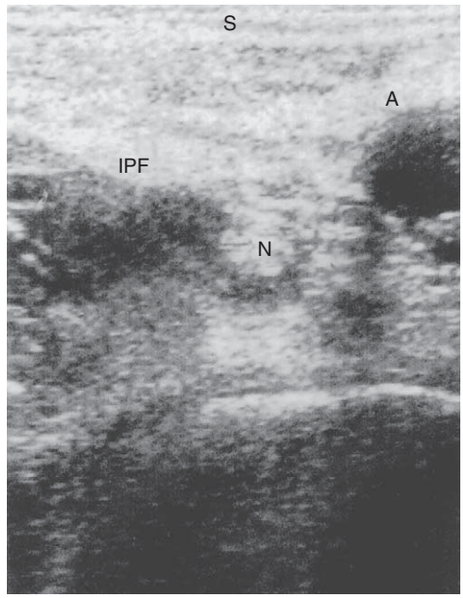


FIGURE 36–5

Ultrasonographic picture of the major anatomical structures of the 3-in-1 block (right side). N, femoral nerve; A, femoral artery; IPF, iliopectineal fascia; S, skin surface. (From Raj PP, editor: *Textbook of Regional Anesthesia*. Philadelphia, Churchill Livingstone, 2002, figure 17-25, p. 275, with permission.)

the brachial plexus in healthy volunteers to evaluate the capacity of the imaging technique to identify the different nerve components of the plexus. The procedure was carried out at the interscalene, supraclavicular, infraclavicular, axillary, and mediohumeral levels, and the different plexus components were visualized in 100% of the patients at the various levels–with the exception of the infraclavicular region, where the components were identified in 27% of the cases (4 out of 15), using a high-frequency transducer (5–12 MHz). However, the structures could be identified with lower-frequency transducers affording increased tissue penetrability (4–7 MHz).[29] Having established the possibility of identifying the nerve plexus components with the technique, the authors performed US-guided puncture in five patients at each level: interscalene, supraclavicular, and axillary. Following US visualization of contact with the plexus nerve, the intensity of stimulus capable of inducing a motor response was evaluated (0.36 ±0.11 mA). In 14% of cases, no movement was observed at intensities of up to 1.5 mA; this may have been due to a lack of NS response to nerve contact. However, it is not possible to rule out other possible causes such as tissue interpositioning (i.e., no direct nerve–needle contact) or incomplete visualization of the needle.[30] In any case, these observations point to the need for further studies in this field.

Chan et al.[15] used a US-guided supraclavicular technique in 40 patients, with the intervention of five anesthetists (three with prior US-guided puncture experience of five or fewer than five cases). Puncture was performed in 9 (±4.4) minutes. Following puncture, NS showed motor response of the brachial plexus at a stimulation intensity of 0.46 mA (range, 0.2–0.7 mA). After administering 40 ml of

local anesthetic (20 ml of 2% lidocaine and 20 ml of 0.5% bupivacaine), complete block was achieved in 95% of the patients within 16.7 (±5.5) minutes on average. An important aspect of this article was the observation of nerve mobility on coming into contact with the needle, and a diffusion effect of the anesthetic solution in two phases. In a first phase, the solution enveloped the nerve (circumferential spread), exhibiting a containing membrane suggestive of the plexus sheath, while the second phase was characterized by asymmetrical distribution in partial contact with the nerve. These observations by US could correspond to initial intraepineural injection of the solution—aphenomenon observed by Sala-Blanch et al.[31] in two cases of epineural puncture following sciatic nerve block via an anterior approach under CAT guidance and controlled by NS.

Williams et al.[32] carried out a randomized comparative study to evaluate the differences observed in supraclavicular block guided by US and NS versus the classical technique based on anatomical evaluation and NS control. The US-guided approach was seen to be faster both as regards evaluation of the references (21 ±17 vs. 57 ±14 seconds) and performance time (5 ±2.4 vs. 9.8 ±7.5 minutes). Moreover, in the US group this latter time shortened significantly in the last 20 patients versus the first (5.8 ±3.4 vs. 4.2 ±2.2 minutes). These data support the economical considerations of Sandhu et al.[6] in relation to the time saving afforded by the US-guided technique.

In conclusion, US guidance for accessing the brachial plexus will undoubtedly find a place in plexus anesthesia for the teaching of anesthetic techniques, application to concrete clinical situations (involving patients in which the classical anatomical landmarks for blind puncture are difficult to identify), or for systematic application in clinical practice.

ULTRASOUND-GUIDED LUMBOSACRAL PLEXUS BLOCKS

As in brachial plexus, an essential requirement for applying US in locating the lumbosacral plexus is a detailed classical topographic (i.e., structures by planes) and sectional anatomic knowledge of the region in which the plexus is found. The topographic anatomy of the zone allows us to identify the successive structural layers down to the actual plexus and facilitates identification of near-lying related elements. Sectional anatomy in turn facilitates identification of the structures seen on-screen during US guidance. Nevertheless, and in comparison with brachial plexus, the information and number of published papers in this area are relatively scant.

The superficial femoral nerve often appears triangular and hyperechoic at the inguinal crease. Ultrasound localization of the femoral nerve and its surrounding structures has been found to reduce the onset time, improve the quality of sensory block, and minimize the risk of vascular puncture (Figure 36-6).

Marhoffer and colleagues[33,34] studied the benefits of US guidance versus NS in performing the 3-in-1 block, administering 20 ml of 0.5% bupivacaine to both groups. After US- or NS-based identification of the femoral nerve, the local anesthetic solution was administered. The onset of sensory block was significantly shorter in the US group compared with the NS group (16±14 vs. 27±16 minutes, respectively, $p<0.05$). The quality of sensory block after injection of the local anesthetic was also significantly better in the US group (US 15±10% of initial value, NS 27±14% of initial value, $p<0.05$). A good analgesic effect was achieved in 95% of the patients in the US group and in 85% of the patients in the NS group. In the former group, visualization of the cannula tip, the femoral nerve, the major vessels, and local anesthetic spread was possible in 85% of patients. Associated morbidity was recorded only in the NS group, in the form of accidental arterial puncture ($n=3$). In another related study, it was found that the amount of local anesthetic for 3-in-1 blocks can be reduced by using US guidance compared with the conventional NS-guided technique.[34] The authors concluded that a US-guided approach for the 3-in-1 block reduces the onset time, improves the quality of the sensory block and minimizes the risks associated with this regional anesthetic technique.

Kirchmair et al.[35,36] in turn evaluated the possibility of accessing the lumbar plexus adopting the posterior approach (psoas muscle) under US guidance in 20 patients. They were able to identify the different anatomical structures, but not the actual lumbar plexus. The authors concluded that the

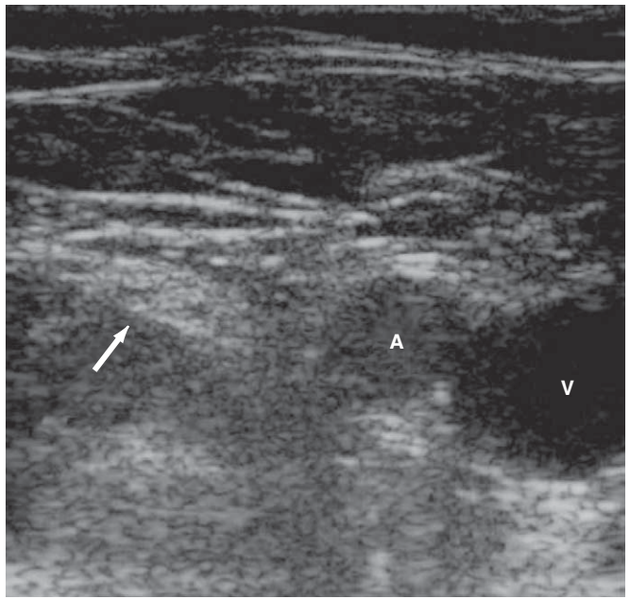


FIGURE 36–6

Ultrasonographic picture of the femoral block. White arrow, femoral nerve; A, femoral artery; V, femoral vein.

depth of the plexus can be predicted with this approach, with visualization of the trajectory and thus reduction of the possible complications. In children,[37] the lumbosacral plexus is more superficial and it is technically easier to image the nerve in this location.

The US-guided approach to the saphenous nerve has also recently been reported.[38] Ultrasound identification of the sciatic nerve (hyperechoic) in the gluteal region can be difficult due to the depth of beam penetration required and lack of defined interface between the nerve and surrounding fat and muscles. Nevertheless, Gray et al.,[39] in a 7-year-old boy, have recently described the US-guided approach to the sciatic nerve via a subgluteal approach (Figure 36-7).

Further distal in the popliteal fossa, Heinemeyer and Reimers[40] failed to identify the sciatic nerve in 26% of the subjects. Successful use of US to guide popliteal nerve block has been reported in a number of recent articles.[41–43] Sites et al.[41] described via a posterior popliteal approach in two diabetic patients. Sinha and Chang[42] in turn published the first series of 10 cases of posterior popliteal sciatic block

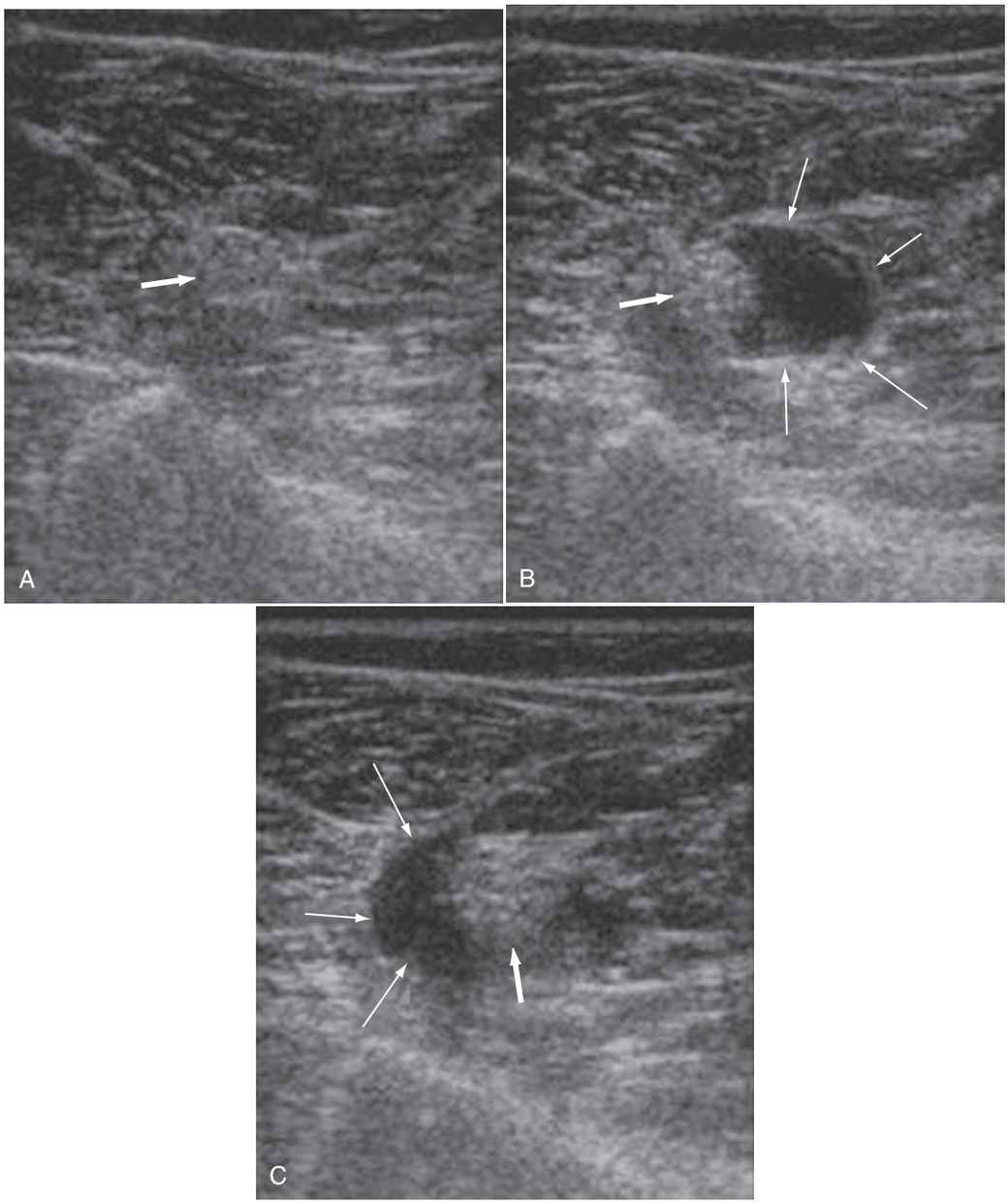


FIGURE 36–7

Ultrasonographic picture of the sciatic block at the popliteal level. (A) White arrow, sciatic nerve. (B) Large White arrow, sciatic nerve, small arrows are showing the injection of 10 ml of local anesthetic. (C) Large White arrow, sciatic nerve, small arrows are showing the distribution after 5 minutes of the injection of 10 ml of local anesthetic.

using a stimulating catheter under US guidance. Their preliminary conclusions point to the simplicity and easy performance characteristics of the technique, although the need for further studies in this field is also pointed out.

ULTRASONOGRAPHY IN CENTRAL BLOCK

The identification of needle depth to the epidural space for the conduction of anesthetic puncture in this space has been well documented and is indicated in patients with difficulties for identifying the interspinous space, as in pregnant or obese individuals.[44–49] The procedure has also been used for combined intradural–peridural anesthetic techniques.[50,51] The control of epidural space depth and of puncture site location is interesting in terms of the quality of epidural anesthesia, particularly in patients presenting problems for locating the anatomical references (e.g., pregnant women).[52] An additional possibility of the US technique when color Doppler is available is the identification of the epidural vessels—with avoidance of the latter during puncture.[53] The technique is also advantageous for identification of the thoracic epidural space.[54] Application of the technique in pregnant patients is able to minimize the undesirable effects and the number of punctures required, while improving performance of the analgesic technique and patient satisfaction.[55,56] The applicability of US for teaching the epidural anesthetic technique in delivery has been well established by Gray et al.[30] in a controlled study involving residents in training. A successful epidural technique was recorded in 60 ±16% of the first 10 cases of the control group and in 86 ±16% in the US guidance group. After 60 cases, the residents achieved success rates of 86 ±15% in the group control and 94 ±9% in the US group. These data are truly indicative of the interest of this imaging modality in application to the teaching of puncture techniques.

PAIN PROCEDURES UNDER ULTRASOUND GUIDANCE

The published data available in regional anesthesia application suggest that US might have potential usefulness in interventional pain management. Possible applications are nerve blocks of the cervical and lumbar zygapophysial joints, stellate ganglion block, intercostal nerve blocks, peripheral nerve blocks of the extremities, blocks of painful stump neuromas, caudal epidural injections, and injections of tender points. US can be used not only for local anesthetic blocks, but has a potential application for destructive procedures, such as cryoanalgesia, radiofrequency lesions, or chemical neurolysis.[57]

Nevertheless, and in comparison with the increasing experience in the use of US in regional anesthesia, the use of US for pain management is in development and there are very few published studies in this field. Efficacy and safety data are not yet available.

Recently, a US-guided methodology for nerve blocks of the lumbar zygapophysial joints has been developed.[58] Authors used a curved array US transducer with a frequency of 2–6 MHz to reliably guide the needle to the L2 to L4 medial branches.[58] Accuracy of US-guided T12 to L4 medial branch blocks was recently confirmed in a cadaver study by CT control, with a rate of over 90% of successful needle placements.[59] According to the presented results, the procedure has limitations, and it is difficult to identify the bony landmarks to block the dorsal ramus of L5 at the junction between the ala and the superior articular process of the sacrum. In the obese patient, poor US image quality is obtained, and according to the level of the treatment, considering a significant increase in skin-to-target distances from the third to the fifth lumbar vertebra.

In the treatment of patients suffering from vascular diseases or sympathetically maintained pain of the head or the upper extremity, US allows the visualization of all relevant anatomical structures of the stellate ganglion region.[60] In the study by Kapral et al.,[60] compared with the blind-puncture group, a reduction in the amount of local anesthetic to 5 ml was achieved, and no hematoma in the US group was recorded, but 3 of 12 in the group with the blind approach had a hematoma.

There are no published data on US-guided intercostal nerve blocks, basically because the nerves are rarely seen as they are lying close to or recovered by the caudal edge of the rib. Eichenberger et al.[57] stated in their paper that 2 ml of local anesthetic is sufficient to fill the intercostal space, and the spread of the injected solution can mostly be seen clearly by US during the injection. These authors add the concept of dosing based on observed clinical anatomy considering the width of the intercostal space may vary and sometimes more volume has to be applied to fill the intercostal space.

In the practice of anesthesiology, the ilioinguinal and the iliohypogastric nerve are often blocked without the use of an imaging technique as technique for postoperative pain relief, but most important for chronic pain relief after inguinal hernia repair. US offers a perspective of precise percutaneous approach to these nerves using high-resolution probe.[57] With important anatomical variations in location, they lie between the external and internal oblique or the internal oblique and transverse abdominal muscles, just medial and cranial to the superior iliac crest. The genitofemoral nerve, probably because of its deep course, is unlikely to be displayed by US.

In peripheral nerves, the treatment of stump neuroma after amputation is made using initially diagnostic blocks. Using US helps to identify the anatomical source of pain in these patients[61] and also helps in later use of either radiofrequency neurotomy, cryoanalgesia, or chemical neurolysis of phenol.[62]

For the performance of the caudal injection of steroids, the point of entrance of sacral hiatus may be difficult to identify and US may be a useful tool for appropriate caudal needle placement.[63]

In patients with a diagnosis of chronic pain of myofascial origin, a trigger point may be defined as a focal, hyperirritable spot located in a taut band of skeletal muscle. According to Eichenberger et al.,[57] in the performance of trigger/tender point infiltrations under US visualization, it is possible to see a muscle twitch when the needle is entering the trigger point in the target muscle. Nevertheless, the authors pointed out that US is not a means to justify the use of a treatment modality that is still controversial, but rather an additional tool for practitioners who believe in the efficacy of trigger/tender-point injections.

Montero et al.[64] in 1989 described the neurolytic block of the coeliac plexus through the anterior abdominal wall using ultrasonic guidance. The anterior approach is simple and useful in patients with chronic pancreatic pain undergoing biopsy of the pancreas, and in terminally ill or heavily sedated patients who have difficulty in tolerating the prone flexed position. After this first publication, the usefulness of sonographically guided percutaneous neurolysis of the celiac plexus in patients with abdominal tumors or chronic pancreatitis has been evaluated in several papers.[65,66] The ultrasonic-guided anterior approach to US was used for needle placement and examination of the spread of injection. The aorta and discharge of the truncus coeliacus or the arteria lienalis are ultrasonographically presented, and the celiac plexus as echogenic foci were observed around the origin of the coeliac trunk and superior mesenteric artery in all cases.

CONCLUSION

Imaging techniques as a complementary approach appear to have a bright future in the clinical, teaching, and research settings of regional anesthesia and pain medicine practice. However, US has limitations that preclude its use for several currently performed pain procedures for which fluoroscopy still is the main indication.

The principal clinical utility of US seems to be in the operating room, affording a very likely improvement in the quality of techniques performed. Future studies will undoubtedly serve to more clearly define the possibilities of imaging techniques in clinical anesthesiology, but also their effectiveness and safety.

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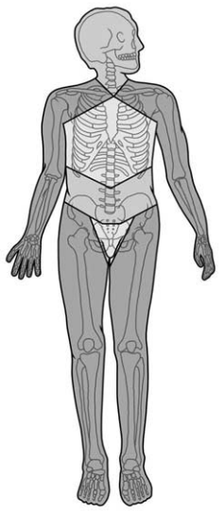
C H A P T E R



37

Interventional Pain Practice Equipment and Devices

JOSE RODRÍGUEZ, LELAND LOU, AND STEVEN LORETZ

For the safe practice of interventional techniques, it is mandatory to know the equipment and devices available to the pain physician for safe and precise practice. In this chapter, we will show and describe equipment and devices currently available to the physician, which will allow the interventional pain physician to set up his or her pain practice. Much thought has been put into cautioning the physician to strive for safety and efficiency at all times.

To that end, Figures 37-1 and 37-2 describe a picture of the operating room for performing a typical interventional procedure.

PROCEDURE ROOM SETUP

PROCEDURE ROOM TABLE

Because of the extensive use of fluoroscopy, a radio-lucent table is a preferred choice (Figure 37-3). The two most common types are with a distal base with a distal counter weighted base in a "dive board" design and a "four-legged" table. Movement of the table is also important. To reduce costs, many tables are supplied with one, two, or three movements. Typical movements are up and down, rotation left and right, and Trendelenburg and reverse Trendelenburg. For cervical and head and neck procedures, add-on frames or a "cut out" hole in the table are optional.

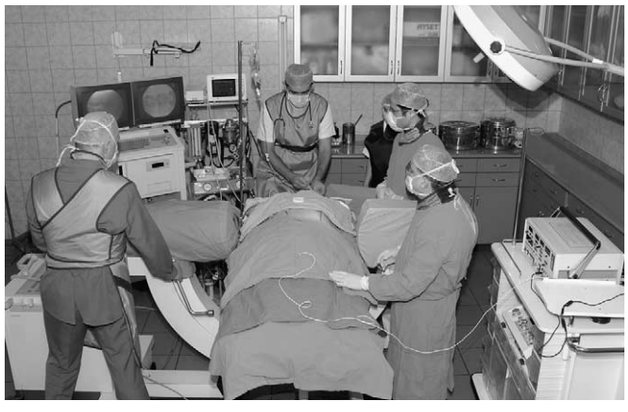


FIGURE 37–1
Procedure room setup. (Courtesy of Serdar Erdine, M.D.)

RADIATION GARMENTS

RADIATION GOWN

A variety of gowns are available (Figures 37-4 and 37-5). Regulated standards are set for the amount of radiation protection provided. For example, in the United States,

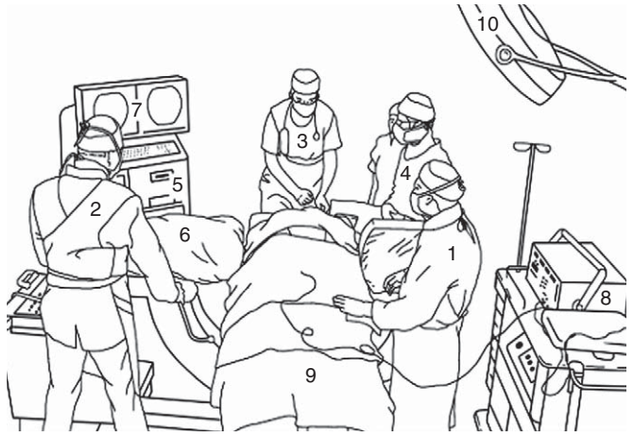


FIGURE 37–2
Procedure room. 1, Physician (interventionalist) appropriately attired with sterile gown, gloves, lead jacket, thyroid collar, and glasses. 2, Radiology technician. 3, Physician or nurse personnel monitoring the patient's vital signs during the procedure. 4, Assistant to the interventionalist appropriately attired. 5, Monitoring cart and equipment. 6, C-arm appropriately covered with sterile drapes. 7, Monitor for C-arm images. 8, Special equipment (radiofrequency) for the procedure. 9, Operating table. 10, Operating light.

FIGURE 37–3

Pain table and accessories. Companies that provide pain tables and accessories include Morgan Medesigns Inc., Oakworks, and Surgical Tables Inc.

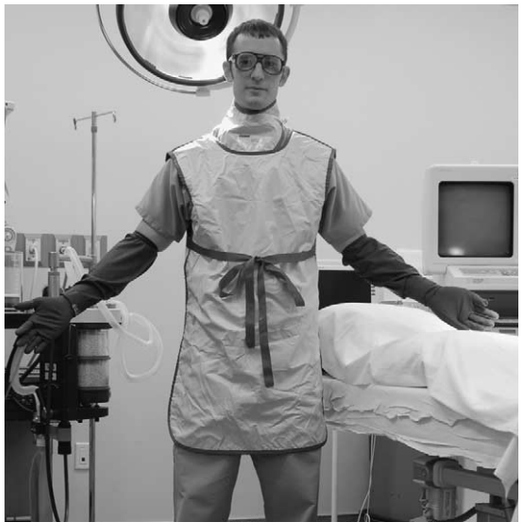


FIGURE 37–4

Proper setup for radiation protection: radiation protective eyewear, thyroid shield, radiation protective apron, and radiation protective sleeves and gloves.

standard is 0.5 mm of lead or equivalent, but in the United Kingdom the minimum protection set is at 0.35 mm lead. Because of the weight of these gowns, a two-piece gown is available for all users, not just women. For comfort, shoulder straps and waist support can be added. To decrease gown weight, a radioprotective material other than lead has been integrated into them for public purchase and use.

RADIATION GLOVES

The gloves used in the procedure room are commonly lead impregnated with latex construct (Figure 37-6). Like all surgical gloves, left and right gloves are packaged. As a reminder, these gloves are radiation resistant, but not protective. For this reason, hands should not be left in the radiation field even in lead gloves when the fluoroscope is in use.

THYROID SHIELD

One of the more important glands to protect due to its radiation sensitivity is the thyroid. Poor coverage or lack of coverage of the thyroid may result in damage to the gland and may lead to hypothyroidism (Figure 37-7).

GAUNTLETS

Where radiation exposure is high or exposure to the physician is detrimental, forearm lead covers can be acquired. Typically, these gauntlets are wrapped around the arms and secured with straps or Velcro (Figure 37-8).

RADIATION EYEGLASSES OR SHIELDS

Recent data show that there is increased risk to the lens of the eyes from radiation. Physicians who regularly used leaded eye-protective glasses (Figure 37-9) had delayed cataract changes, when compared with those who did not use eye protection. The eyeglasses can be ground into a prescription lens for those who need visual correction.

FLUOROSCOPY EQUIPMENT

The fluoroscopy unit used in the interventional pain procedure room has evolved to become an essential tool in itself (Figure 37-10). By facilitating visualization for more accurate needle placement, the safety and efficacy of the many pain procedures has been enhanced. More aggressive procedures can now be performed in the minimally invasive therapeutic pain treatments. The physician should be familiar with the function, purpose, and potential adverse effects associated with that particular fluoroscopy equipment.

The fluoroscope has become the mainstay tool for the interventional pain physician. By being able to see the bony structures and by accommodating the identification of vessels and spaces with the assistance of contrast agents, safety and accuracy of the procedures are enhanced. Most fluoroscopes come in two parts: the monitor with the processing electronics and various additions such as a printer. Radiation is produced in the second part, which is commonly called the “C-arm.” With these two pieces, live images are created and viewed.

A variety of software packages are available for the fluoroscopes. Other options, such as an enlarged imaging head or laser pointers, can be added. For user safety, the newer machines are now limited to a maximum of 120 Kvp and have a spacer at the radiation source to prevent too-close contact of the radiation source to the patient.

FIGURE 37–5
Radiation-protective aprons. Radiation-protective products are distributed by several companies, including Barrier Technologies, Epimed International, Protech, and Shielding International.

FIGURE 37–6
Radiation-protective gloves.

FIGURE 37–7
Thyroid shield.

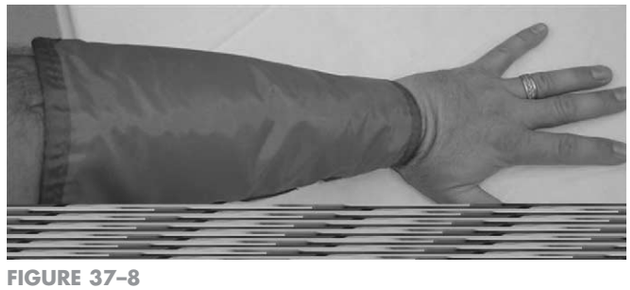


FIGURE 37–8
Forearm shields.

FIGURE 37–9
Radiation-protective eyewear.

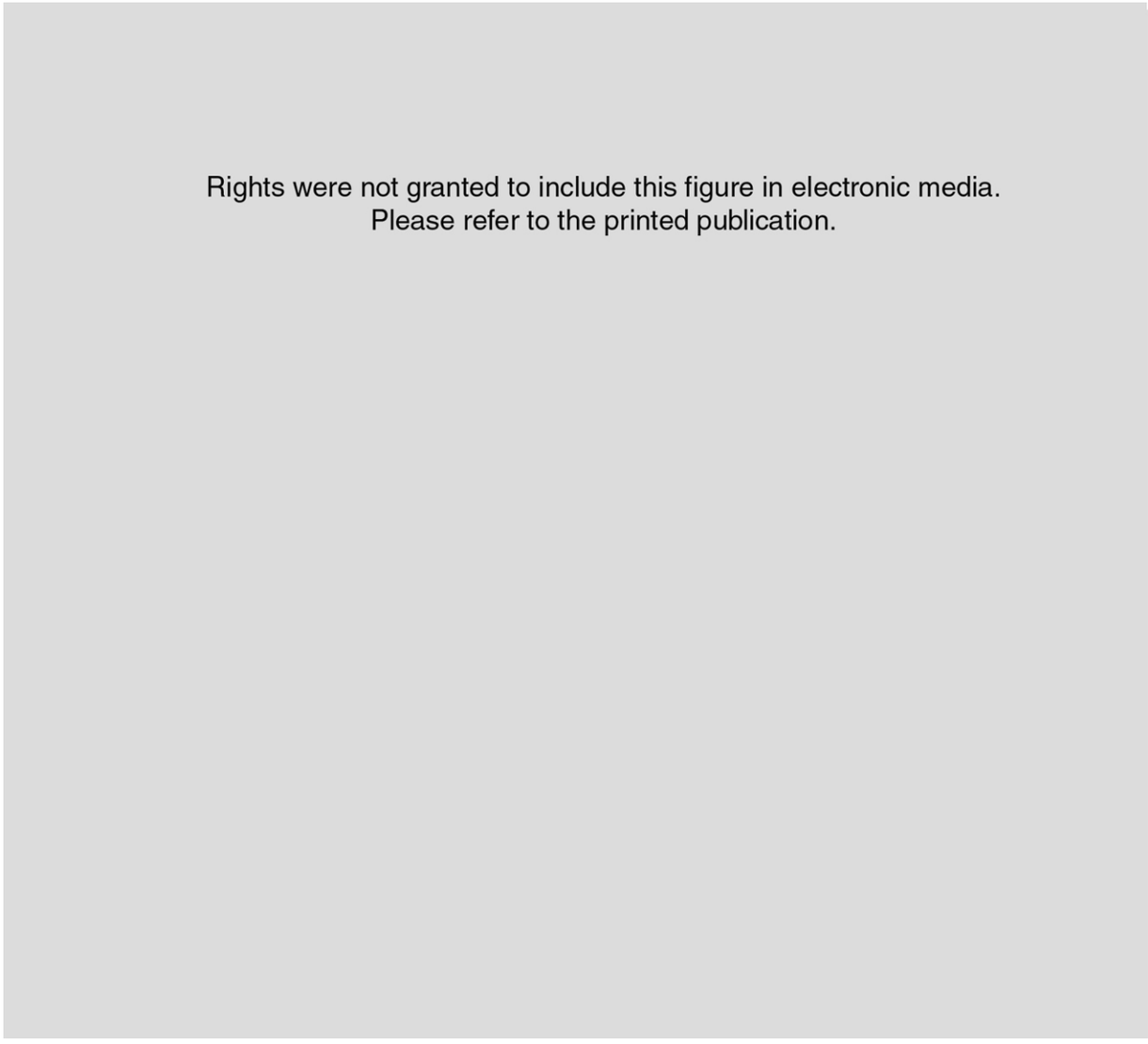


FIGURE 37–10
(A) C-arm fluoroscope by GE Healthcare. (B) C-arm fluoroscope by Ziehm Imaging. (C) C-arm fluoroscope by Siemens.

NEEDLES

Needles are a common denominator in all of the interventional pain procedures. The most common needle used for many procedures is a Quincke spinal needle. For this reason, the need for describing the needles seems to be a moot one. As the science for each procedure catches up to the technical aspects, potential risks and dangers of each procedure are better understood. This development is driving innovation in the creation of safer approaches and the need for specialized needles to facilitate those changes. Also driving this variety of choices is the availability of a wealth of therapeutic devices (Figure 37-11).

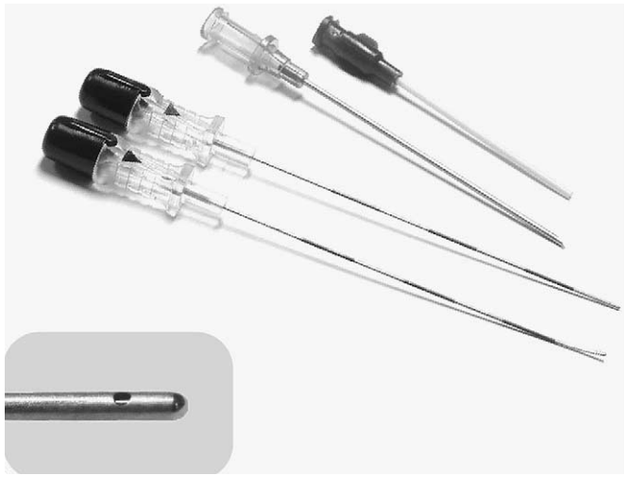


FIGURE 37–11

Blunt needles are available in 20-, 22-, and 25-gauge sizes from 3.0 inch (7.6 cm) to 6.0 inch. Needles pictured are courtesy of Epimed International.

Tradition has supported the use of sharp needles for many invasive procedures. A sharp needle can easily penetrate the skin and inserts through the tissue smoothly.

There are also blunt-tipped needles available for the procedure, which are commonly used. Proponents of blunt needles maintain that neural and vascular structures are less likely to be damaged while traveling to the target area. Proponents of sharp needles advocate the ease of which the sharp needle penetrates the different layers of the body to the target area.

EPIDURAL NEEDLES

Epidural needles come in various lengths and bevel designs. The most common epidural needle used is the Tuohy needle. Other epidural needles available are the Crawford and Hustead needles. A curved directed orifice is a hallmark of the Tuohy and Hustead, with the sharper tip on the Hustead needle. With a Crawford epidural needle, the tip is sawed off, straight, and short beveled (Figures 37-12 and 37-13).

Nonshearing epidural needles are made specifically for catheter insertion and manipulation. The newer needles, the RX Coudé® Epidural Needle and the RK™ Epidural Needle are examples of smooth, blunted bevel needles that are specifically created to direct the intraspinal catheter out and into the epidural space. Minimal shearing is accomplished by rounded inner edges.

SPINAL NEEDLES

The spinal needle comes in many shapes and sizes (Figures 37-14, 37-15, and 37-16). Spinal needles are also used for peripheral nerve blocks because of its convenient length (3.5 inch/8.75 cm) and being styletted. Quincke needles are sharp-tipped spinal needles. The Quincke needles have a longer beveled tip. The Sprotte® spinal needle is a bullet-tipped needle. A pencil-point–tipped spinal needle is called the Whitacre. Both the Sprotte® and Whitacre needles have a lateral orifice near the tip. The Chiba needle is a frequent choice and is especially popular for use in discograms. The marking on the Chiba needle is helpful in monitoring the depth of penetration.

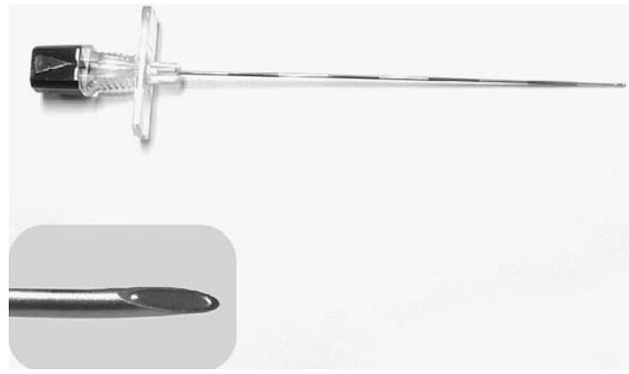


FIGURE 37–12

Tuohy needles. They are available in several gauge sizes and lengths. Most commonly used are 18–22 gauge in 3.5 inch (8.75 cm) to 6.0 inch (15.2 cm).

FIGURE 37–13

The RX Coudé®, RX Straight, and RK Straight epidural needles are designed for use with a catheter to reduce shearing and allow for catheter insertion and manipulation. They are available in 14–18 gauge sizes in several lengths and are manufactured by Epimed International.

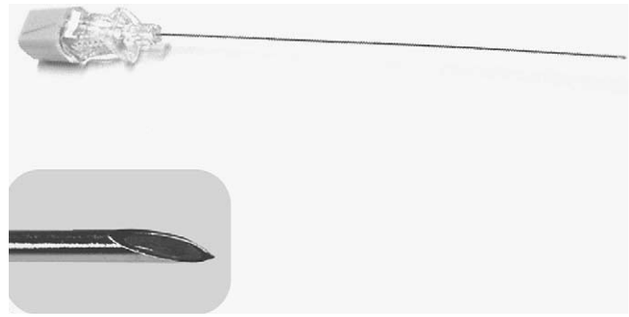


FIGURE 37–14

Quincke needle. These needles are available in several lengths (1–7 inch) and gauge sizes (18–27 gauge) from several manufacturers.

RADIOFREQUENCY NEEDLES

The main difference in the needles used for radiofrequency procedures is that they are insulated, except for the "active tip." For interventional pain management, there are two

FIGURE 37–15
(A) Sprotte® needle and tip. (B) Whitacre needle tip.

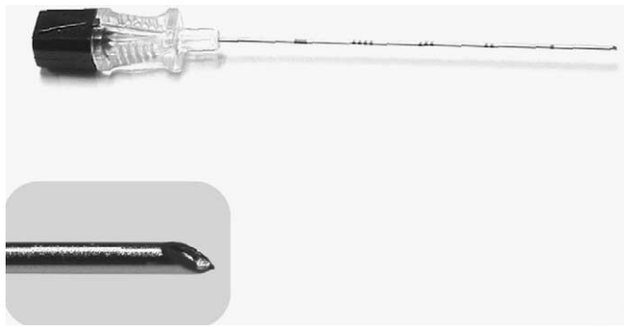


FIGURE 37–16
Chiba needle. This needle is commonly used for discograms and available in several lengths and gauge sizes.

predominant types of needles (Figures 37-17 and 37-18). The Sluijter-Mehta (SMK)–type needle is popular.

Another popular needle is the Racz-Finch (RFK), typically blunt tipped with a 10-degree curve at the distal end.

CATHETERS

Catheters can be used where a continuous infusion is desired for pain relief. The most common technique is the placement of a catheter in the intrathecal space with an implantable pump. Another common technique is the epidural infusion for prolonged chronic pain relief. Recently, it is also being used for infusion of major plexuses, such as brachial and lumbar plexus. There are also reports in the literature of catheters being placed on the peripheral autonomic nervous system such as on splanchnic, celiac plexus, or lumbar sympathetic chain. Catheters are made from inert

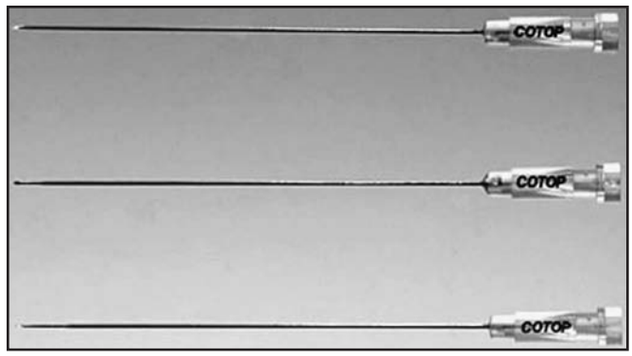


FIGURE 37–17
Sluijter-Mehta (SMK)–type needles.

FIGURE 37–18
Radiofrequency needles: curved blunt, straight sharp, and curved sharp.

noninflammatory plastic polymer (Figures 37-19 to 37-22). They are resistant to heat changes, nonkinkable, and able to maintain the patency for long time periods. Advances have been made to prevent shearing of the catheter due to stretching. Examples of these are Racz®, DuPen®, intrathecal, epidural, and peripheral catheter.

EQUIPMENT

In this section, radiofrequency pulse generators, cryotherapy machines, implants, and other pain devices will be presented and discussed.

RADIOFREQUENCY GENERATORS

Radiofrequency is becoming a commonly performed procedure. For principles, mechanisms, indications, and contraindications of pulsed thermal and radiofrequency,

FIGURE 37–19
(A) Racz® epidural catheter. (B) Stingray™. (Courtesy of Connector.)

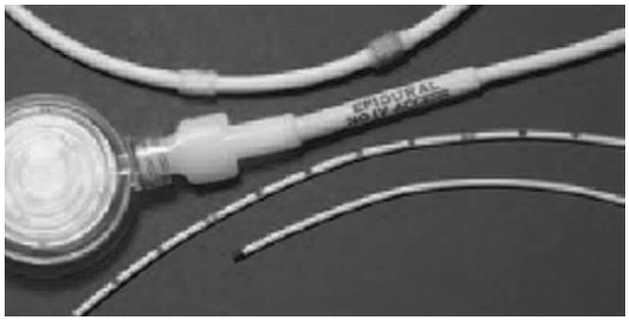


FIGURE 37–20
DuPen® long-term epidural catheter. (Courtesy of C.R. Bard.)

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FIGURE 37–21
InDura® 1P intrathecal catheter by Medtronic.

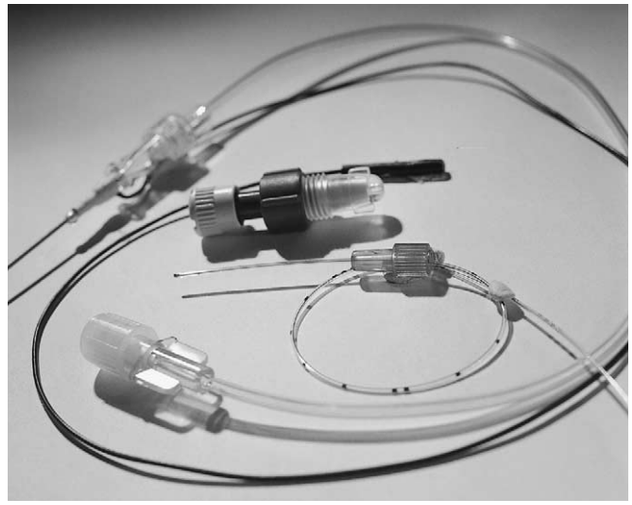

FIGURE 37–22
Peripheral catheter.

see Chapter 3. Various generators are available today (Figures 37-23 and 37-24), most of which are manufactured to generate heat. The features of these machines are constructed to allow sensory and motor testing. Temperature monitoring is an essential feature in the safe use of a radiofrequency generator. A timer is now considered a basic feature in modern machines. With the discovery and presentation of other treatment modalities using radiofrequency, some machines are built to perform those functions as well. To match the technological advances and user needs, some manufacturers have automated their machines with preset modes desired by physicians. Other features are constantly being added to match new technological advances and physicians' needs.

CRYOTHERAPY MACHINES

Cryotherapy requires specialized probes and delivery systems. Because liquid nitrogen is used to produce the freeze "burn" or lesion, the probes are typically bigger than can be used by the radiofrequency lesioning machines. However, they are a preferred choice in areas where the risk of neuritis is a concern.

The machine features are otherwise similar to the radiofrequency generators (Figure 37-25). They have a testing mode for sensory and motor checks. A timer is included for monitoring the time of the freeze and warming of the probe.

This procedure is not commonly performed. See Chapter 4 on cryolysis and the details of its mechanism.

SPECIAL EQUIPMENT

The following special procedures are commonly used. Descriptions of the equipment for these techniques are provided in the following illustrations and legends.

- Epiduroscopy (Figures 37-26 and 37-27)
- Percutaneous endoscopic discectomy (Figure 37-28)
- Other disc therapeutic procedures (Figures 37-29 to 37-32)
- Vertebroplasty/kyphoplasty (Figures 37-33 and 37-34)
- Intrathecal pumps (Figure 37-35)
- Stimulating devices (Figure 37-36)
- Peripheral nerve stimulation needles (Figure 37-37)
- Ultrasound (Figure 37-38)

FIGURE 37–23

Pictured are the products needed to perform a radiofrequency procedure: generator, electrode (probe), grounding pad, and (insulated) needle and injectates.

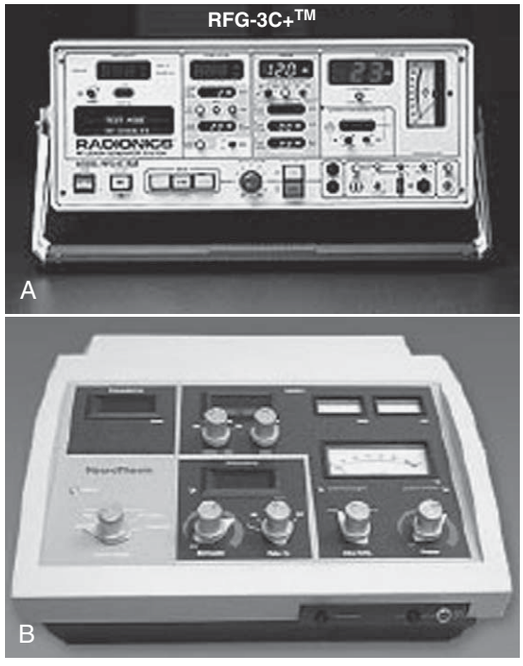

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FIGURE 37–24

(A) Radiofrequency unit. (B) Neuro-therm radiofrequency generator. (C) Radiofrequency Lesion Generator-Cosman®.

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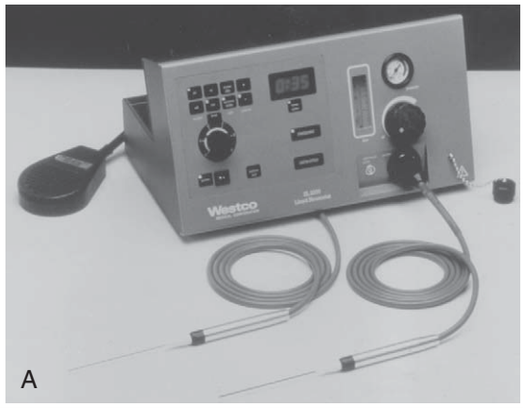
FIGURE 37–24

(D) Baylis® Pain Management Generator (PMG)/RF Generator.



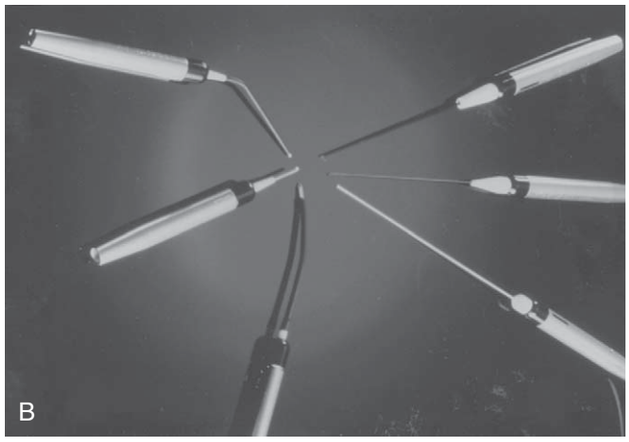


FIGURE 37–25

(A) Cryoneurolysis machine and cable. (B) Cryo probe set, including 12-, 14-, 16-, and 18-gauge probes.

FIGURE 37–26

Epiduroscope with catheters and introducers. (A) Various components of the epiduroscopy system. (B) Dilator (top) and introducer cannula (bottom).

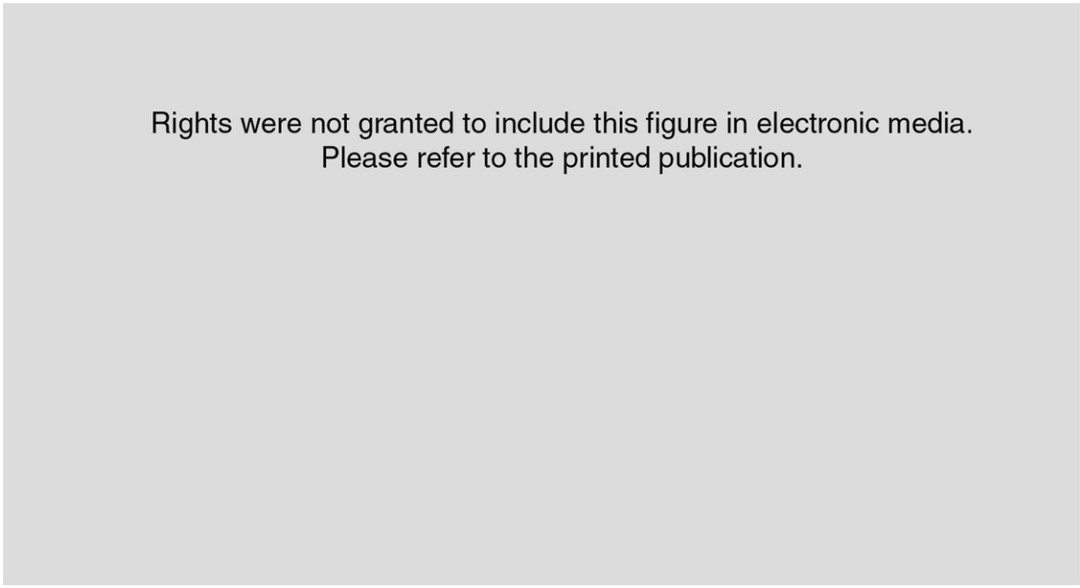
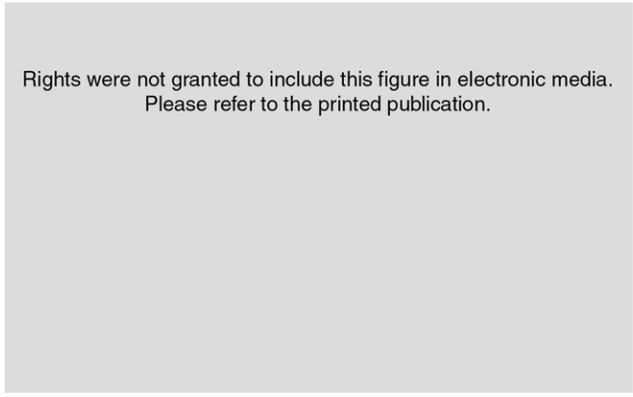


FIGURE 37–27
(A) Product brochure for the Myelotec NaviCath®. (B) Myelotec NaviCath®.



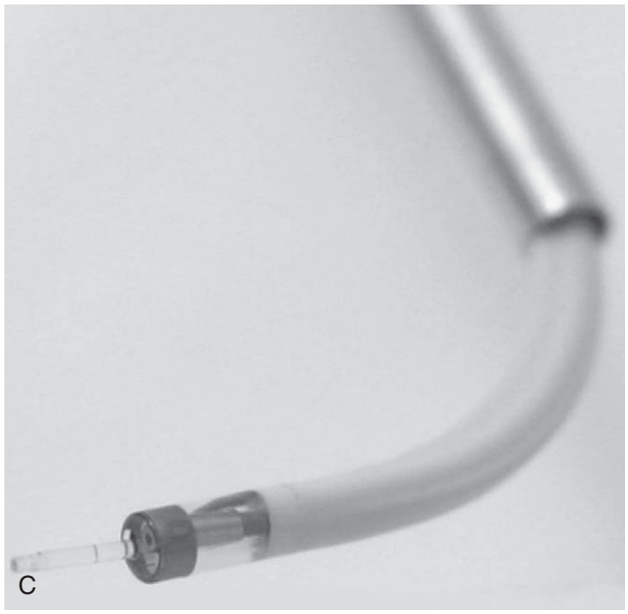


FIGURE 37–28
(A) Devices used for the LASE® procedure from Clarus Medical. (B) This is a picture of forceps used for percutaneous endoscopic discectomy. (C) Products used for the percutaneous endoscopic discectomy.

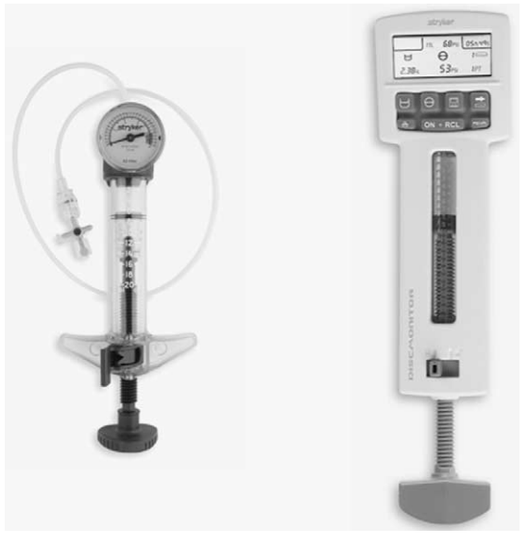


FIGURE 37–29

Disc pressure monitors. (Courtesy of Stryker, ArthroCare, Spinal Specialties, and Smith & Nephew.)

FIGURE 37–30

SpineCath Intradiscal Catheter for Intradiscal Electrothermal Therapy (IDET). (Courtesy of Smith & Nephew.)

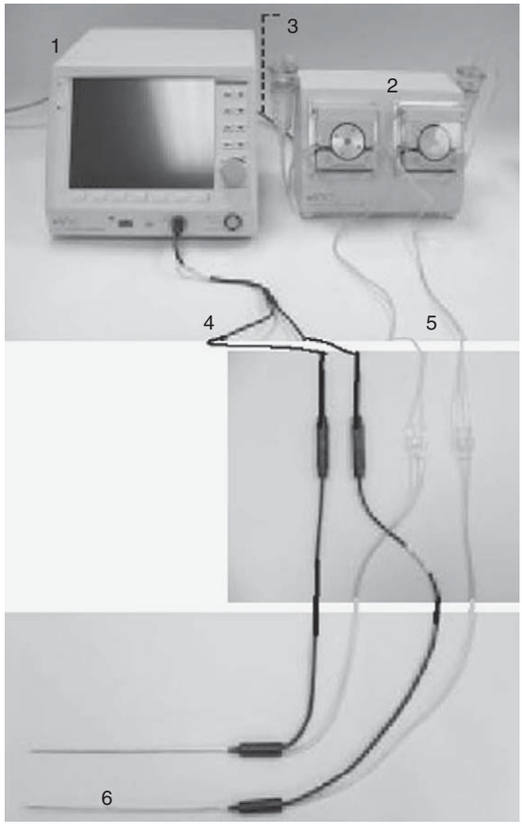


FIGURE 37–31

TransDiscal™ system for disc biacuplasty. (Courtesy of Baylis Medical Company.)

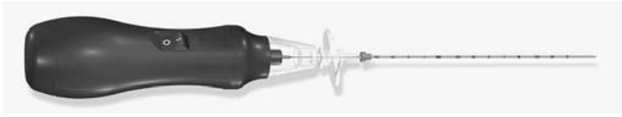


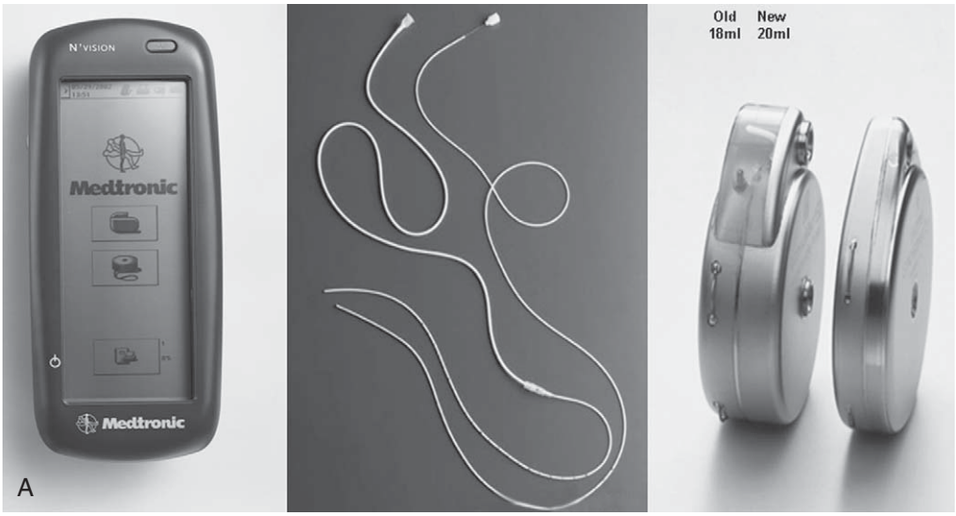
FIGURE 37–32

Pictured is the Dekompressor®, which is a percutaneous discectomy probe. (Courtesy of Stryker.)

FIGURE 37–33

Products used for vertebroplasty. (Courtesy of Stryker.)

FIGURE 37–34
Products used for kyphoplasty. (Courtesy of Kyphon.)



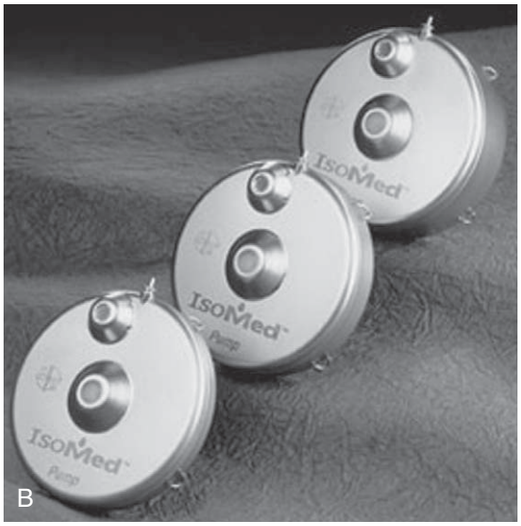


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FIGURE 37–35
(A) Infusion devices. (B) Medtronic IsoMed®. (C) Tricumed/Codman "Archimedes."

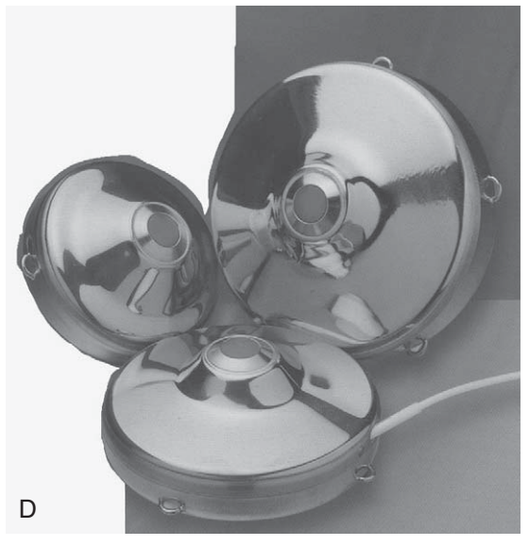

FIGURE 37–35 (Cont'd)
(D) Arrow Model 3000. (Courtesy of Marshall Bedder, MD.)

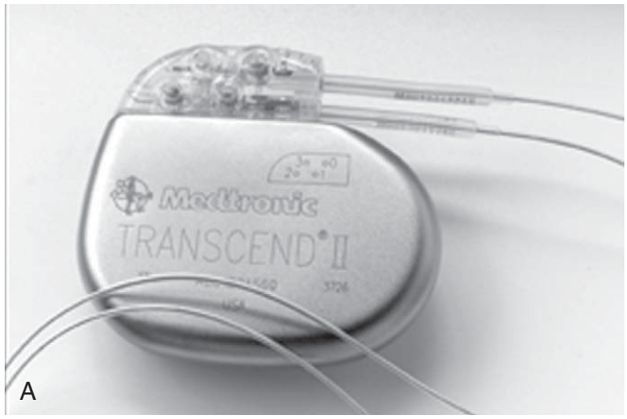

FIGURE 37–36
Implantable stimulating devices, (A), Medtronic. (B), Advanced Bionks. (C), ANS.

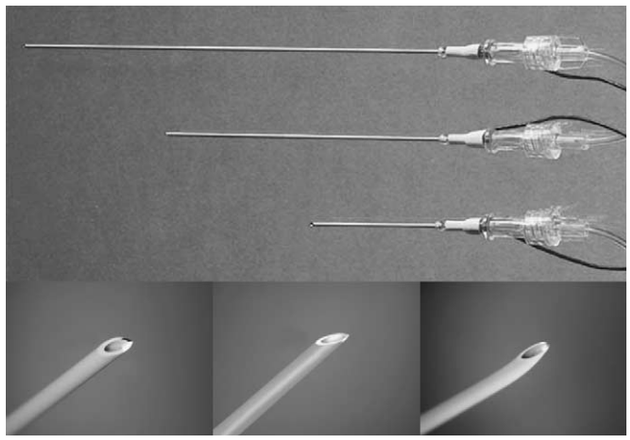

FIGURE 37–37
Needles for peripheral nerve block procedures with the use of a nerve stimulator. They are mainly insulated needles and are available from several companies. (Courtesy of Life-Tech, Inc.)

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FIGURE 37–38
Ultrasound systems by GE Healthcare and SonoSite.

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